

GUIDELINES FOR SURGICAL EMERGENCIES

Recommended by



THE ASSOCIATION OF SURGEONS OF INDIA

FOREWORD

The Association of Surgeons of India founded in 1938 is the second largest professional body of Surgeons in the world next to the American College of Surgeons. The role of ASI in India is unique compared to the position of similarly placed organizations in the rest of the world where such bodies have specific statutory role in academics, training, examination and credentialing. This has a negative impact in policy making as the data is grossly inadequate when the professional is kept away. The Association of Surgeons of India is making an earnest attempt to set in practice Guidelines for its members to aid them in their daily practice.

This book comprises of Guidelines to address common emergencies confronted by Surgeons practicing in India.

Each of the topics is written by one or more Consultants and had been peer reviewed by a Panel of experts to increase the credibility of its contents.

Hope this will ensure a better and safe practice in India.

We hope to present this to the various statutory authorities in India so that this becomes the benchmark for reference in policy making, legal disputes and even part of the curriculum of training specialists.



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List of Emergencies covered

1. Abdominal colic
2. Abscess
3. Acute abdomen
4. Acute Aortic Dissection
5. Acute Appendicitis
6. Acute Cholecystitis
7. Acute Limb Ischaemia
8. Acute Mesenteric Ischemia
9. Acute Pancreatitis
10. Acute Retention of Urine
11. Acute Scrotum
12. Acute Traumatic Haematuria
13. Blunt Abdominal Trauma
14. Burns
15. Chest Trauma
16. Haematuria- Non Traumatic
17. Head injury
18. Hollow viscous injuries
19. Intestinal Obstruction
20. Liver trauma
21. Painful Hernia- Irreducibility/Obstruction/Strangulation
22. Peptic Ulcer Perforation
23. Perianal Pain
24. Pneumothorax
25. Primary Care of Trauma: ABCDE
26. Severe Soft tissue infections- Cellulitis
27. Small Bowel Perforation
28. Splenic trauma
29. Stridor
30. Upper GI Bleeding
31. Vomiting

Topics & Authors

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5	Acute Appendicitis	Dr Manish Joshi
6	Acute Cholecystitis	Dr Ramesh Agarwalla
7	Acute Limb Ischaemia	Dr Sreekumar R C Dr Sunil Rajendran Dr Binni John
8	Acute Mesenteric Ischaemia	Dr Prasad S.S
9	Acute Pancreatitis	Dr Gajanan Wagholikar Dr Prakash Valse
10	Acute Retention of Urine	Dr Sham Singla
11	Acute Scrotum	Dr Ashok Godhi Dr Siddalingeshwar Neeli Dr Santosh Kurbet Dr Kishor Bandagar
12	Acute Traumatic Haematuria	Dr Shamita Chatterjee
13	Blunt Abdominal Trauma-General approach	Dr Girish Bakhshi Dr Sanjay Shah Dr Vineet Kumar
14	Burns	Dr Chacko Cyriac
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16	Haematuria Non Traumatic	Dr Kalyan Kumar Sarkar
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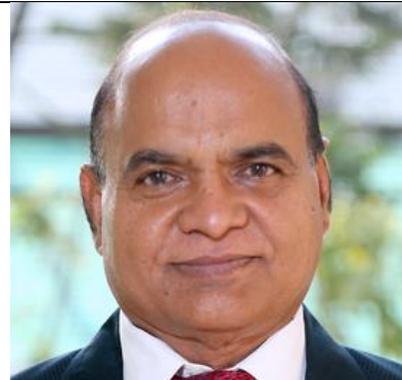
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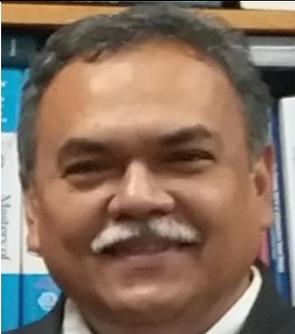
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12. Dr Achal Gupta, MS; DNB; D.Uro; FIAGES; FICLS

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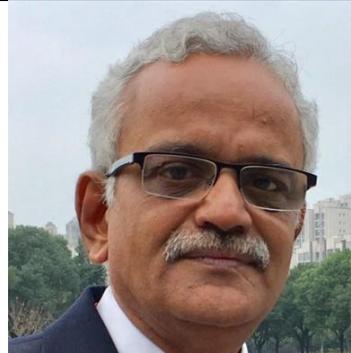
18. Dr G Siddesh, MBBS; MS

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21. Dr Manoj Kumar, MBBS; MS; FRCS(Edin); DNB; MNAMS; FIAGES

<p>Dr Manoj Kumar Professor & Head, Department of Surgery AIIMS Patna, Bihar</p> <p>Email:-docmk_2002@yahoo.co.in</p>	
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22. Dr Nirmal Narain, MS; D.Ortho; PhD (Surg); DNB; FRCS(Eng); FRCS(Edin); FRCS(Glas); FICS

<p>Dr Nirmal Narain</p> <p>Professor & Head, Department of Surgery Patna Medical College Patna, Bihar</p> <p>Email:- narainnp53@gmail.com</p>	 A portrait of Dr Nirmal Narain, a middle-aged man with dark hair and glasses, wearing a dark suit, white shirt, and a red tie.
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23. Dr A M Quraishi, MS; FRCS; DNB; FMAS; FACRSI; FIAGES

<p>Dr A M Quraishi</p> <p>Professor of Surgery Government Medical College Nagpur, Maharashtra</p> <p>Email:-am_quraishi@hotmail.com</p>	 A portrait of Dr A M Quraishi, a middle-aged man with grey hair and glasses, wearing a dark suit and a yellow shirt.
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24. Dr Pankaj R Modi, MS

<p>Dr Pankaj R Modi</p> <p>Professor of Surgery B.J Medical College Civil Hospital Ahmedabad</p> <p>Email:-prmodi13@hotmail.com</p>	 A portrait of Dr Pankaj R Modi, a middle-aged man with grey hair and a mustache, wearing a blue and white checkered shirt.
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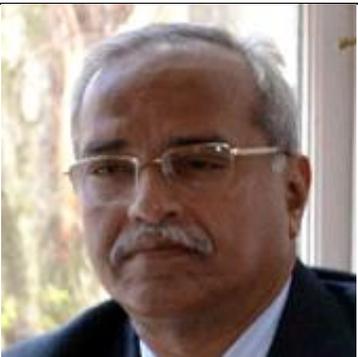
25. Dr Probal Neogi, MBBS; MS; FRCS (Edin)

<p>Dr Probal Neogi Professor of Surgery, M L N Medical College Allahabad (Prayagraj) Uttar Pradesh</p> <p>Email:- neogiprobald@yahoo.co.in</p>	 A portrait of Dr. Probal Neogi, a middle-aged man with short dark hair and glasses, wearing a dark suit, white shirt, and red tie. He is looking directly at the camera with a neutral expression.
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26. Dr Siddharth Dubhashi, MS; PhD; FACS; FAIS; FICS; FACRSI; FIMSA; FAIMER Fellow

<p>Dr Siddharth Dubhashi Professor and Head of Surgery M G M Medical College Navi Mumbai, Maharashtra</p> <p>Email:- spdubhashi@gmail.com</p>	 A portrait of Dr. Siddharth Dubhashi, a man with dark hair, smiling. He is wearing a blue and white striped shirt.
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27. Dr Sanjay De Bakshi, MS; FRCS(Eng; Edin {ad eundem})

<p>Dr Sanjay De Bakshi Head of Department Division of Surgical Gastroenterology Calcutta Medical Research Institute Kolkata, West Bengal</p> <p>Email:- scmdbakshi@hotmail.com</p>	 A portrait of Dr. Sanjay De Bakshi, an older man with grey hair and a mustache, wearing glasses and a dark suit. He is looking slightly to the left of the camera.
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31. Dr Anthony Rozario DNB; FRCS; FMAS; FACRSI

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Abdominal Colic

The present guidelines are an annotation of scientific material and evidence as available on the present topic regarding Abdominal Pain-specially as Abdominal colic.

1. Definition.
2. Introduction to guideline
3. Section on Various common types of Colic in Surgical practice.

Each section will have

- a. Clinical Practice points
- b. Red Flag signs
- c. Documentation (Medicolegal) points
- d. Investigation (evidence based)
- e. Treatment overview.

1. Definition

A **colic** is to be described as an Abdominal Pain originating in the confines of the Abdomen & progress in. a crescendo- decrescendo manner with period of painless episodes in between.

A **colic** typically occurs arising from a Hollow viscera with a muscular wall capable of peristaltic movements.

A **Pseudo colic** typically mimic's a Colic arising from hollow muscular viscera prompting peristaltic movements and largely follows a gradual crescendo- decrescendo but the painless interval is absent.

Acute Abdomen is defined as a constellation of signs of symptoms occurring in a span of less than five days.(1)

Abdominal Pain is an unpleasant sensation with physiological and psychological effects arising from the Abdominal contents (viscera) or Panicles or Retro peritoneum.

Abdominal pain arising from hollow viscera with muscular walls is commonly called colic.

2. Introduction to Guideline

The present guidelines are based on the Oxford centre for Evidence Based Medicine (2011) levels of Evidence guideline.

For this implementation a set of Question has been raised in each topic with. Mechanism based reasoning as weakest evidence and systemic review of cross sectional studies as strongest.(2)

Q1. What is the colic (eg: ureter)?

Q2. What are the clinical guidelines to diagnose this colic?

Q3. What are the red flag signs?

Q4. What is the minimum documentation required for medico legal purpose?

Q5. What will happen if not treated?

Q6. What are the common treatments available?

Q7. What are the treatment complications (side effects).

Q8. What is the sequence of investigations?

SECTION A

BILIARY COLIC

- A Biliary colic is defined arising from the Gallbladder in relation to a stone disease.
- This is a pseudo colic as the pain does not reach a decrescendo.
- The pain is usually described for a Gallstone obstructing the cystic duct or infundibulum of Gallbladder.

Clinical Guidelines

- a. Vague pain starting at epigastrium and radiating to right upper abdomen or poorly localised to upper abdomen. (Essential criteria).
- b. Occasionally aggravated to fat containing food (Non essential criteria).
- c. Rarely radiation to inter-scapular region or right shoulder [Non essential criteria).
- d. Associated nausea & rarely vomiting. (Essential criteria)
- e. Intensity of pain is variable (Non Essential criteria)
- f. Duration usually in hours. Maximum 8- 10hr (Essential-criteria)
- g. Acute cholecystitis is diagnosed as per Tokyo Guidelines 2018. (Essential criteria)(3)

Investigations

- a. Ultrasound whole abdomen (Essential criteria)

Must document (i) Stone

(ii) Wall-thickness

(iii) Peri-cholecystic Fluid.

- 1 CBD diameter (not mandatory)
- 2 IHBR dilatation (mandatory)

- b. Investigations to support Acute cholecystitis (Tokyo 2018)

Practice Guidelines (4,5)

- a. Diagnosis of Gallstone disease by USS should be followed by elective Cholecystectomy at short interval.
- b. Acute Cholecystitis should be graded and Emergency surgical intervention should be planned as per as Tokyo criteria.
- c. Analgesics as Paracetamol, Drotavanine one usually used as first line analgesic.
- d. Opioids should be avoided in emergency.
- e. Diclofenac and ketorolac should be used with caution in emergency setting. History of gastric disorders, asthma and renal disease are contraindications.
- f. Intravenous PPI/H2receptor blockers have been found useful in adjunct setting.

Referral Criteria

- a. Peritonitis as a part of complicated gallstone disease.
- b. Acute pancreatitis suspected to be severe and needing specialty critical care support.
- c. Immunosuppression with sepsis in need for isolation and specialist opinion.
- d. Pregnancy in association with Gallstones for perioperative maternal and child monitoring.

Applicable only when expertise and experience in treating such situation is lacking from the part of the Concerned Surgeon or when equipment to manage the condition is limited in the centre.

Medicolegal Points

- a. All clinically diagnosed Acute cholecystitis patients are to be admitted for emergency management.

- b. Description as Biliary colic or clinical diagnosis of biliary pathology needs to be followed by investigations as ultrasound as first time.
- c. Vital signs to be recorded at the time of discharge.
- d. Cholecystectomy as definite treatment modality to be mentioned as advice in the discharge summary.

The surgical management of Acute cholecystitis is the standard of care at present. Depending upon the local infrastructure, an expectant management with surgery at the earliest opportunity should be mentioned in the discharge.

Resource utility

1. Emergency Surgeon with adequate experience with performing emergency cholecystectomy and Laparotomy for peritonitis.
 2. Emergency physician with experience in diagnosis of acute abdomen and differentiating common non surgical disorders with basic knowledge in interpretation of USG abdomen and ECG and common Blood parameters.
 3. Access to Interventional Gastroenterology referral is preferable.
 4. Emergency instrumentation Bundle (annexure 1)
 5. Emergency Drug trolley (annexure 2)
 6. Emergency Investigation support (annexure 3)
 7. Emergency Non medical Manpower team(annexure 4)
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URETERIC COLIC

- a. This is a true colic in relation to an expulsion of a stone along the ureter.
- b. The classical nature of the colic is rarely missed in a stone in upper one third of ureter.
- c. Mid third ureter stone mimic Appendicitis (Dysuria) or Diverticulitis / Amoebic colitis
- d. Distal third stones mimic Bladder calculi (strangury), uterine colic and commonly cystitis.

Clinical Guidelines (6)

- a. Acute onset Crescendo with a rapid decrescendo and a pain free interval. (Essential)
- b. The pain free interval is marked by dysuria or occasional hematuria.(Essential)
- c. The onset of pain is typical of anatomic location of the stone in ureter.
- d. The upper third ureter calculi will start with pain at Renal angles and occasional radiation upto Testis. (Essential)

- e. The mid-third calculi cause flank discomfort (Essential)
- f. The lower third calculi are most poorly localized and present as suprapubic pain with radiation to thigh. (Essential)
- g. Passage of a calculus is pathognomonic. (Nonessential).

Investigations

- a. X Ray KUBP and/or USG of whole abdomen must be done in all cases.
- b. Urine examination should be done with culture sent before starting antibiotic.
- c. Uro-sepsis must be ruled out by clinical and lab criteria
- d. Renal insufficiency to be investigated to calculate dose adjustments.
- e. Non contrast CT is a highly specific tool to rule out sub centimeter stones and is to be done only in cases where clinical suspicion is high and when KUB or USG are non contributory to establish the diagnosis.

Practice Guidelines

- a. Parenteral Diclofenac and Ketorolac are first line drugs used.
- b. Non relief of pain suggests impaction of stone or infected urinary retention.
- c. Antibiotics should be started at clinical signs of Uro sepsis.
- d. Emergency Urology consultations should be requested whenever available.
- e. Endo urological- treatment should be preferred to conventional procedures if indicated.
- g. Monitoring the urine output in post pain episode acts as a rough guide to therapy.
- h. Medical expulsion therapy with alpha blockers have a high success rate in stones less than 10mm in size.

Referral criteria

- a. Acute renal failure in need for renal replacement therapy.
- b. Onset of pain in setting of chronic renal failure or in patient undergoing regular Haemodialysis.
- c. Immunosuppression with sepsis in need for isolation and specialist opinion.
- d. Pregnancy in association with urolithiasis for perioperative maternal and child monitoring.
- e. Lack of expertise and facility for Image guided nephrostomy in the hospital in case of Urosepsis refractory to antibiotics.
- f. Endourologic procedures for ureteric and renal stones.

Medicolegal Points

- a. Suspected stone not seen on X Ray or ultrasound(at emergency) to be followed by a NCCT and urology opinion.

- b. Demonstration of adequate renal function and judicious use of Antibiotics.
- c. Endo urological interventions have associated specific complications which need a special mention.
- d. Residual stones (in bladder) may need future surgical interventions. This should be mentioned in consent for urologic procedures

Resource utility

1. Emergency Surgeon with adequate experience with Urology and Surgical access for Open or percutaneous Nephrostomy and Suprapubic cystostomy.
 2. Emergency physician with experience in diagnosis of acute abdomen and differentiating common non surgical disorders with basic knowledge in interpretation of USG abdomen and ECG and common blood parameters.
 3. Access to Urology referral for Endourology procedure
 4. Emergency instrumentation Bundle (annexure 1)
 5. Emergency Drug trolley (annexure 2)
 6. Emergency Investigation support (annexure 3)
 7. Emergency Non medical Manpower team (annexure 4)
-

INTESTINAL COLIC

- a. The most common cause of colic, this represents the prototype of a colicky abdominal pain.
- b. The spectrum of colic in children and adults is complex with “Gas” colic in children being common to cancers presenting with Colicky pain in adults.

Clinical Guidelines (7)

- a. The colic represents a classical slow crescendo & decrescendo and does not have a painless period. (Essential).
- b. Intake of food aggravates the pain (Non essential)
- c. Passage of liquid faeces / blood after an colic strongly suggest a mechanical Cause eg: Intussusception (Non essential)
- d. The origin of the pain provides an insight to anatomic origin of the pain.
 - Foregut causes of pain are felt as epigastric pain
 - Midgut causes of pain are felt as periumbilical pain.
 - Hindgut causes of pain start as hypogastric pain. (Essential)
- e. A visible peristalsis with pain suggests a sub-acute and mechanical bowel obstruction and needs a surgical treatment. (Essential)

- f. Common causes of mechanical causes of pain include extrinsic compression (eg: Adhesions, hernia) and intramural causes (strictures) in adults and need surgical intervention (Essential)
- g. Colics in children need to be evaluated to rule out mechanical causes needing surgery (eg: Intussusception) with common cause like roundworm bolus which may not require surgical intervention primarily. (Essential)

Investigations

- a. Persistent pain in children & adults should be evaluated by Xray and or Ultrasound.
- b. Ultrasound is the investigation of choice in children particularly if intussusception is the suspected etiology.
- c. CT scans are to be used judiciously.
- d. Contrast enema in children should be used with caution and peritonitis must be ruled out before its administration.
- e. Fluid and electrolyte deficiency in adults and children needs serial investigations before operation on a patient put on expectant management

Practice Guidelines

- a. Drotavarine, Meperidine Dicyclomine are common antispasmodics in use. (8)
- b. The use of antispasmodics is limited and is only till etiology is identified.
- c. Mechanical causes of pain should have a surgical intervention as definitive treatment.
- d. Bentimidazoles (eg: Albendazole) are contraindicated in treatment of active "worm" colic. Pyrantel Palmoate is an alternative choice.
- e. Acute intestinal obstruction (mechanical) needs a Surgical intervention.

Referral criteria

- a. Peritonitis as a part of clinical presentation, if hospital is unable to provide emergency surgical intervention.
- b. Acute pancreatitis suspected to be severe and needing specialty critical care support.
- c. Immunosuppression with sepsis in need for isolation and specialist opinion.
- d. Pregnancy in association with peritonitis for peri-operative Maternal and child monitoring

Medicolegal Points

- a. Surgical causes of pain should be followed by surgical interventions.
- b. Suspected subacute intestinal obstruction needs to be evaluated for the etiology before discharge.

- c. If symptoms are persistent, evaluation by clinical and radiological methods are to be done as when necessary.
- d. Resuscitation is important before surgery. Documentation of the same is an integral part.

Resource utility

- a. Emergency Surgeon with adequate experience for doing laparotomy and bowel resection and anastomosis and ostomy formation.
 - b. Emergency physician with experience in diagnosis of acute abdomen and differentiating common non surgical disorders with basic knowledge in interpretation of USG abdomen and ECG and common blood parameters.
 - c. Access to Gynaecology and gastroenterology referral for specialty opinion and care. (eg: Gynaecologic endoscopic intervention, gastrointestinal endoscopy).
 - d. Emergency instrumentation Bundle(annexure 1)
 - e. Emergency Drug trolley (annexure 2)
 - f. Emergency Investigation support (annexure 3)
 - g. Emergency Non medical Manpower team(annexure 4)
-

APPENDICULAR COLIC (9)

Appendicitis initially, starts as a colicky pain located in the lower quadrant of abdomen due to distension of the lumen by fecolith, foreign body etc.

Nausea and vomiting are more frequently associated with appendicitis than with ureteric colic

Alvarado system scoring is a reliable tool for emergency decision making.

Diagnosis of appendicitis is still by clinical examination only. Radiological adjuncts are helpful in doubtful cases and for complications of appendicitis.

Diagnostic laparoscopy is a particularly helpful investigation in women and children with suspected appendicitis.

Laparoscopic or open appendectomy serves as a standard treatment for Appendicitis.

Common complications such as wound infection need a mention in consent for the procedure.

Type of Colic	RED FLAG SIGNS
Intestinal Colic	<ol style="list-style-type: none"> 1. Change in the nature of pain (Constant dull aching pain) not relieving with regular doses of analgesic 2. High fever 3. Signs of generalised peritonitis Suggest “STRANGULATION”
Ureteric Colic	<ol style="list-style-type: none"> 1. High swinging fever 2. Renal angle tenderness 3. Palpable lump in loin 4. Ballottable kidney 5. Anemia Suggest “PYONEPHROSIS” <ol style="list-style-type: none"> 1. Anuria 2. Elevation of Serum creatinine Suggest “END STAGE KIDNEY DISEASE”
Appendicular Colic	<ol style="list-style-type: none"> 1. Extremes of age 2. Signs of generalised peritonitis 3. High swinging fever 4. Fluctuant tender mass at RIF Suggest “PERFORATED APPENDICITIS”
Biliary Colic	<ol style="list-style-type: none"> 1. Presence of SIRS 2. Deteriorating general condition 3. Evidence of sepsis 4. Generalised peritonitis 5. High swinging fever. Suggest: “PERFORATED/GANGRENOUS/SEVERE CHOLECYSTITIS”

Annexure 1

Emergency Instrumentation Bundle

1. Open Surgical Trolley (Laparotomy set)
 - Scalpel handled blade
 - Hemostats
 - Tissue holding forceps
 - Radio opaque tagged gauze & pads [mops] (Preferable)
 - Needle holder (at least one vascular)
 - Needles and sutures
 - Suction apparatus & Suction nozzle.
 - Diathermy pencil
2. Standard Anaesthesia Trolley
3. Operation theatre arrangement as for emergency setup.
4. Emergency drug & resuscitation trolley

Annexure 2

Emergency Drug Trolley

Injections - Adrenaline, Hydrocortisone, Deriphylline, Atropine, Calcium gluconate /
Defibrillator,
IV Cannula.
Normal Saline infusion bottle

Annexure 3

Emergency Investigation Support

Investigations

1. Haemogram
2. Blood Sugar
3. Renal function test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG
8. X- Ray – Chest

Annexure 4

Resources required for one patient / procedure (Patient weight 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anesthetist – 1---- Services from outside can be availed
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

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Abscess

Introduction

The incidence reported ranges from 2.5% in general population to 21.5% to I.V. drug abusers (1,2). The most frequent causative organism is Staph aureus or Streptococcus pyogens. In the community acquired abscesses Methicillin Resistant Staph. aureus is most common offending organism. The infection varies by location, race, & age. There are several risk factors like immunocompromised status (steroid therapy, chemotherapy, malignancy, AIDS, alcoholic and I.V. drug abuses), diabetes, sickle cell disease, Crohn's disease. A breach in epithelial continuity is another common factor for abscess. The common sites are skin surface particularly axillae, groin, perianal region, around vagina and hair follicles.

Definition

An abscess is a localized collection of pus within the tissue which could be superficial & deep. The collection of pus is nothing but liquefied tissue debris & inflammatory cells (3).

Required history – Trauma, Scratch, Diabetes, infections, FB and drugs.

Clinical Features

Clinical presentation is acute onset fever, painful swelling which is red with raised local temperature, tenderness and fluctuation. This could be localized or may have surrounding edema and induration. Patients may have tachycardia, features of shock in case of bacteremia particularly in immunocompromised patients.



Figure 1: clinical signs

Deep seated abscess may lack local signs.

Diagnosis:

Diagnosis is usually by clinical examination. Investigations are required for deep seated abscess, recurrent abscess and following failure of antibiotics.

Bed side ultrasound – A hyperechoic (4) or anechoic area of fluid collection (5-8) with clinical features is suggestive of an abscess.

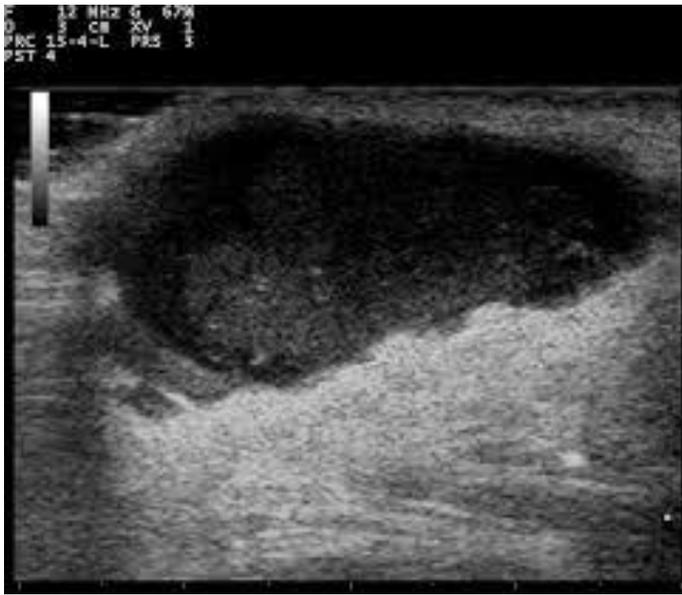


Figure 2 : Anechoic area on ultrasound

CT is done only for suspected deep abscess.

Pus culture is routinely not recommended since it does not alter the management and outcome. Patients improve even when pathogen is resistant to empirical antibiotics.

CBC and Blood culture are done in patients with Septicemia.

Treatment: (Latin phrase is - Ubipus, ibievacua – where there is pus evacuate it)

Components of treatment

- a. Incision and drainage (8-10)
- b. Anesthetics (Local or General)
- c. Antibiotics in specific situations
- d. Analgesics
- e. Post op dressings – irrigation and packing
- f. Follow up

What not to do

- Do not squeeze
- Do not stick a needle or sharp instruments
- Avoid aspiration

When to consult

- > 1cm swelling or enlarging painful swelling
- Sore in groin or genitals
- Fever
- Spreading swelling
- Associated medical conditions causing immune suppression

Commonly asked questions

- Pain control LA vs GA
- Empirical antibiotics / No antibiotics
- When to use it ?
- What antibiotics ?
- Open vs closed wound
- Swab culture / no culture

Incision and drainage is the standard treatment.

Types of anesthesia

Age and patient's preference and site of abscess are the criteria for the decision. The options are sedation, local and general anesthesia. When all the options are available patient preference could be taken unless contraindicated.

Majority of superficial abscesses can be done under local anesthesia (level III evidence).

IV opiates, benzodiazepines, Ketamine, nitrous oxide and regional blocks can also be used.(11-13)

Larger abscesses and abscesses in certain locations like perineum and perianal region should be drained under regional anaesthesia.

Children and deep seated abscess are drained under deep sedation if general anesthesia is not available.

Antibiotics:

- No empirical antibiotics are to be used. There is increasing evidence of CA-MRSA infection following use of empirical antibiotics.(14-15)
- There is some recommendations for empirical antibiotics if the abscess is larger than > 2cm particularly in immunocompromised individuals.

- Routinely it is not recommended since there is no effect on outcome irrespective of Sensitive / resistant organism level I-II evidence) (12,13)
- Antibiotics are to be used in following situations
 - Immuno-compromised patients
 - Large area of associated cellulites & lymphangitis
 - Patients with evidence of systemic toxicity (temp > 38°C, tachycardia, hypotension, Leukocytosis, abscess in perianal and vulval regions.

What antibiotics:

- Cephalosporin
- Trimethoprim
- Sulfamethoxazole
- Clindamycin
- Tetracycline
- According culture report, if done

Open vs closed wounds

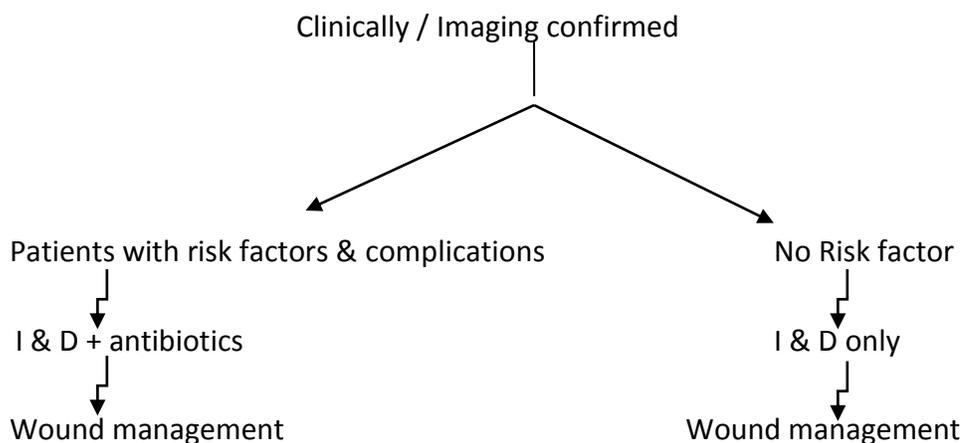
Open wound is better since it allows drainage and there is less chance of recurrences. It is packed with iodoform dressing for 24-48 hours and is changed if needed. Closed wounds have more chances of recurrence hence it is not preferred (Level I evidence).

Culture:

Routine culture is debated and not recommended. It does not alter the management & outcomes Level II evidence) (14- 15).Patients improve even when pathogens are resistant to empirical antibiotics. (16-19).It is recommended under following conditions:

- Immunocompromised patients with risk factors
- Recurrent abscess.

Algorithm in abscess management:



Precautions for performing procedure:

Informed consent is always taken before making the decision and performing procedure.

Important points in management

- Clinical diagnosis by symptoms and signs of acute inflammation,
- imaging only in deep abscesses
- Incision and drainage is the treatment under sedation/ Local anesthesia
- Regional block and general anesthesia in specific situations
- Packing of the wound and iodoform dressing
- Routine culture and antibiotic is not recommended
- Antibiotics only in specific situations
- Oral analgesics for analgesia
- Refer the patient tertiary care center with toxemia

Follow-up: Till wound heals

Resources

Surgeon: one

Assistant: one

Janitor: one

Lab attendant: One

Equipment, Drugs and consumables

OT Table and light

IV cannula and fluids

Analgesics, sedatives

Instrument trolley

Scalpel, artery forceps, Sinus forceps

Packing materials, iododorm solution

Monitor/ Pulse oximeter

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Acute Abdomen

Introduction

The acute abdominal pain is defined as a pain that arises suddenly and is of less than a week's and in most cases less than 48 hours' duration [1, 2]. The term acute abdomen defines a graver presentation of abdominal pain, accompanied by guarding and muscular rigidity, which essentially describes the clinical picture of peritonitis and usually calls for an emergency operation [3]. This led to the common misconception that the acute abdomen is synonymous with the surgical abdomen. However, not all cases of acute abdomen are best treated with surgery. In literature as well as in clinical practice the borders between the acute abdominal pain and acute abdomen overlap and are used interchangeably [4].

'Acute abdomen' encompasses a spectrum of surgical, medical and gynecological conditions ranging from the trivial to life threatening conditions, which require hospital admission, investigations and treatment. Acute abdominal conditions occupy one of the few areas of medical practice where the surgeon often reaches a diagnosis without resorting to numerous investigations. The accurate diagnosis and management of patients with acute abdominal pain remains one of the most challenging tasks for the surgeons. The wide range of causes and the varied spectrum of patient presentation pose a formidable diagnostic and therapeutic challenge.

Indian Statistics

Acute abdomen is the most common surgical emergency in India. The spectrum of etiology of diseases causing acute abdomen ranges from appendicitis, appendicular abscess, hollow viscus perforation, Intestinal obstruction and others.

In a study by Jain et al attempts [5] to identify the epidemiological pattern and to determine the spectrum of disease causing non-traumatic acute abdomen in central India. Amongst the study of 98 patients, males have higher incidence of acute abdomen with the young age group (21-30 years) most commonly affected. In a study done by Memon et al, highest incidence was found in patients between 21-30 years i.e. (27.81%) with more male predominance [6]. This clearly points that younger age group is more commonly affected.

Jain et al [6] found perforation peritonitis constituted the most common cause of acute abdomen (39.7%), followed by acute appendicitis (37.7%), followed by intestinal obstruction (14.2%). In a study done by Chanana et al, the commonest symptom was abdominal pain (76.9%) followed by vomiting (57.2%) and in study done by Berhane et al in 2016, the commonest symptom was abdominal pain (100%) followed by vomiting (80%) [7,8]. In a study done by Singh et al in 2014, it was concluded that abdominal tenderness and abdominal

distension as the commonest sign. Jain et al (2017) found the commonest sign is abdominal tenderness (99.9%) followed by abdominal guarding/rigidity (61.2%) [9].

Recently Thakur et al [10] studied 8688 patients were admitted through emergency department. 1236 (14.2%) of them were admitted with complains of pain abdomen. Frequency of surgical and non-surgical pain abdomen was 6.0% and 8.2% respectively. Most consistent symptom and sign were pain abdomen and abdominal tenderness respectively. Most common cause of acute abdomen was acute appendicitis. It was present in 38.9% of cases followed by gall bladder pathology, renal/ ureteric colic, perforation peritonitis, intestinal obstruction, bowel ischemia in 21.0%, 14.2%, 13.7%, 7.8%, and 4.4% of cases respectively.

Clinical Presentation:

The causative pathologies of the acute abdomen range from intra-abdominal to extra-abdominal and metabolic diseases.

Causes of acute abdominal pain (modified according to Soybel et al. 2006 [1])

Abdominal Causes

Inflammatory:

- Peritoneal:
 - Bacterial peritonitis: perforated hollow viscus, spontaneous bacterial peritonitis
 - Chemical & nonbacterial peritonitis: perforated peptic ulcer, pancreatitis,
 - Others: Mettelschmerz
- Hollow visceral: gastroenteritis, appendicitis, cholecystitis, diverticulitis, acid peptic disease
- Solid visceral: pancreatitis, hepatitis, splenic abscess
- Mesenteric: Lymphadenitis
- Pelvic: Pelvic inflammatory disease, endometriosis

Mechanical (obstruction, acute distention):

- Hollow visceral: acute intestinal obstruction, choledocholithiasis, ureterolithiasis
- Solid visceral: acute splenomegaly, acute hepatomegaly
- Ogilvie-syndrome

Hemoperitoneum:

Ruptured viscera: AAA, spleen, liver; ruptured graffian follicle, ruptured ectopic pregnancy

Vascular:

- Thrombosis or embolism
- Dissecting aortic aneurysm
- Sickle cell anaemia
- Rectus sheath hematoma

Abdominal trauma**Extra-abdominal Causes:**

- Cardiothoracic: Bronchopneumonia, pulmonary embolism, myocardial infarction
- Genitourinary: testicular torsion, cystitis, pyelonephritis, nephrolithiasis, UTI, dysmenorrhea
- Neurogenic: herpes zoster

Metabolic Disorders:

Exogenic Causes: lead poisoning, mushroom poisoning

Endogenic Causes:

- Uremia
- Diabetic keto acidosis
- Acute intermittent porphyria
- Addisonian crisis
- Hemolytic crises
- Familial Mediterranean fever
- Hyperparathyroidism (hypercalcemia)

The most frequent diagnosis made is with 34% that of a non-specific or undifferentiated abdominal pain [3, 11-16]. Of the remaining 66% three-quarters are diagnosed as either acute appendicitis, small bowel obstruction or are ascribed to gynaecological causes [11,15]. Age differences play a crucial role and are important to be considered when assessing the acute abdomen, seeing as with age not only the incidence of certain pathologies changes but also the clinical presentation varies [16]. The World Organization of Gastroenterology's (OMGE) epidemiological survey [15], with a sample of more than 10000 patients the most representative epidemiological study on abdominal pain so far places 50 years of age as a threshold to differentiate younger from older patients with acute abdomen [11,14].

The ten common aetiologies of acute abdominal pain (Modified according to de Dombal 1988)[15]

OMGE-study (n=10 320)	Prevalence (%)
Nonspecific abdominal pain	34.0
Acute appendicitis	28.1
Acute cholecystitis	9.7
Bowel obstruction	4.1
Gynaecological pathologies	4.0
Acute pancreatitis	2.9
Renal colic	2.9
Peptic ulcer (perforated)	2.5
Cancer	1.5
Diverticulitis	1.5

Age-dependent prevalence of acute abdominal pain aetiologies (modified according to de Dombal 1988) [15]

Cause of acute abdomen	Prevalence (%)	
	< 50 years	> 50 years
Nonspecific abdominal pain	40	16
Acute appendicitis	32	15
Acute cholecystitis	6	21
Bowel obstruction	3	12
Acute pancreatitis	2	7
Diverticulitis	< 0.1	6
Cancer	< 0.1	4
Hernia	< 0.1	3
Vascular disease	< 0.1	2

According to those results there is a significant increase in organic causes of acute abdominal pain in the older population [14]. Comparing the < 50 years and > 50 years age groups one finds that especially vascular and oncologic abdominal pain aetiologies increase in frequency and gain in significance. The older the patient, the more important it is to “think vascular” and to “think cancer” [14,16]. Biliary tract disorders, like cholecystitis with 21% become the most frequent cause of acute abdomen in the older age group [14-16]. In the > 75 year olds the “leaderboard” is again headed by non-specific abdominal pain [3, 14-16]. Partly responsible for that might be that elderly patients present with nonspecific symptoms more often and the clinical assessment oftentimes poses a challenge for the physician due to altered mentation

from fever, electrolyte abnormalities, dementia and other co-morbidities [16]. With that in mind, the job of the emergency physician, namely to rapidly identify whether the underlying cause requires an urgent or even immediate surgical or medical intervention [11] does not get any easier. Especially so, as the lethality of the acute abdomen increases with the age of the patient, lying at 10% in the > 80 year olds, meaning that there is less tolerance for initial diagnostic falsities [14].

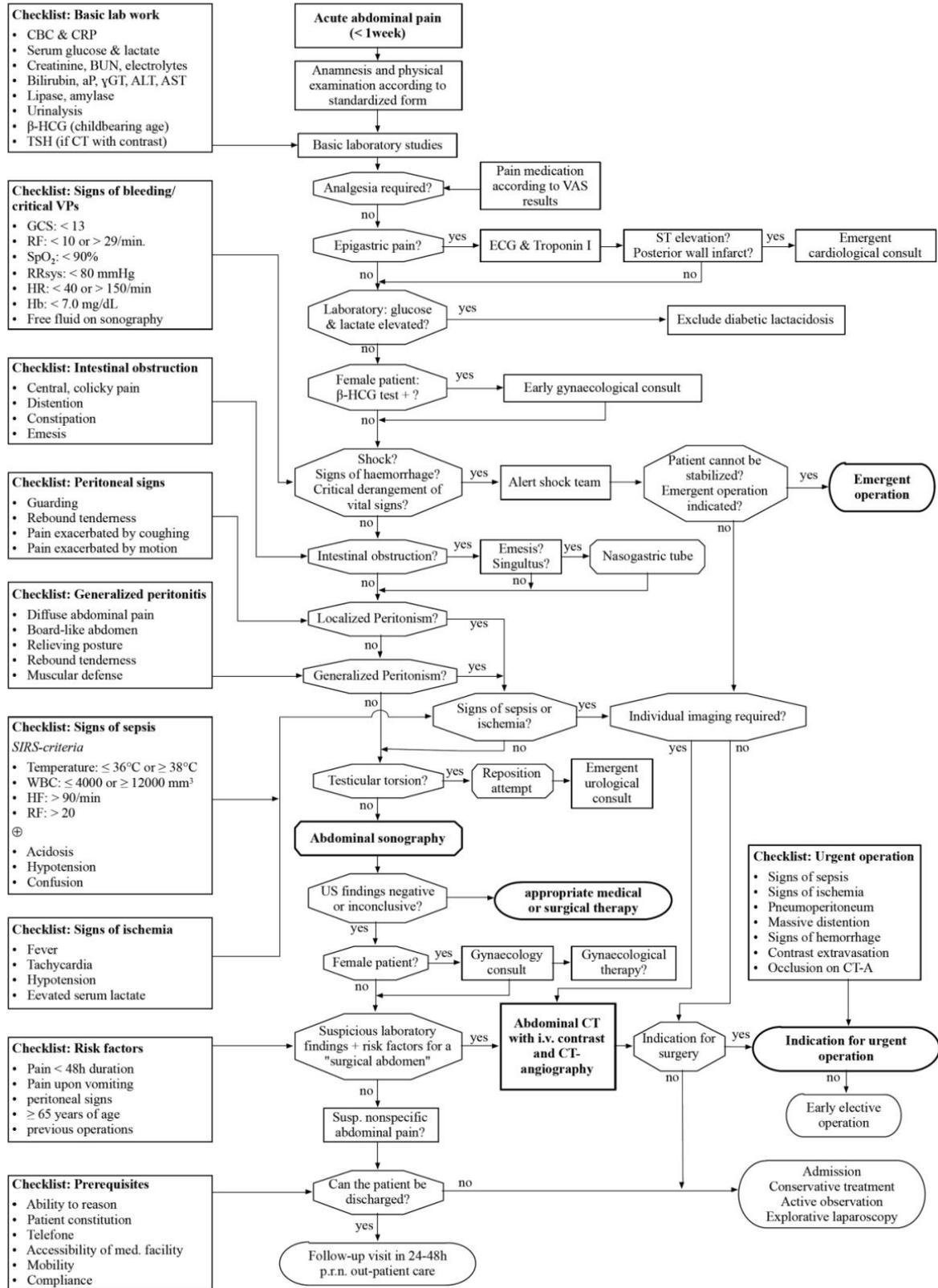
Workflow

Acute abdominal pain is one of the most diagnostically challenging presenting symptoms encountered by the ED physician. A long list of possible differential diagnoses which range from intra-abdominal to extra-abdominal as well as metabolic derangements must be taken into consideration and treated according to severity and urgency [14]. In order to improve the diagnostic accuracy and the triage of the patient into emergent, urgent and non-urgent, the use of standardized forms for the clinical evaluation and the use of work-up algorithms have proven worthwhile [1, 11]. The utilisation of these helping tools is aimed at improving patient care and decreasing diagnostic errors. One often employed, repeatedly cited and modified standardized form is the abdominal pain chart developed by the OMGE [1, 11]. Following work-up protocols enables an evidence-based problem and priority oriented approach to acute abdominal pain and allows for young yet inexperienced physicians to make sound and reproducible decisions regarding the diagnostic evaluation and treatment plan [11].

The patient work-up should follow the renowned SOAP (subjective - objective - assessment - plan) scheme and guide the physician through the patient evaluation and management.

An interdisciplinary approach is of great importance in the assessment of acute abdominal pain. Although the acute abdomen is oftentimes viewed as a surgical problem, a surgical intervention is needed only in 1/3 of cases [1, 11]. Nevertheless the need for a surgical consult or intervention should be evaluated early in the course of the patient assessment in order to prevent unnecessary delays.

Most clinical workflow algorithms available [1,3, 13] concentrate on the logical sequence of individual decision-making processes but do not take into consideration the patient's general state when suggesting a management model [11]. Trentzsch et al. [11] created an algorithm with the goal in mind to provide a time-critical and multidisciplinary approach to the acute abdomen, not only distinguishing between medical and surgical treatment but also giving advice about the necessity to ask for urological or gynaecological consults.



Clinical algorithm for the workflow in acute abdominal pain in adults (Modified according to Trentzsch et al. 2011) [5]

Clinical Evaluation

The most important tools, aiding in evaluating the acute abdomen are the detailed history of the patient's ailment and an accurate physical examination [1, 11, 17]. In some circumstances, as in the case of a typical presentation of acute appendicitis or an incarcerated hernia, the diagnosis can be made even without the help of imaging modalities [1, 11,17]. Solely by means of the clinical evaluation the correct diagnosis can be ascertained with a sensitivity of 88% and a specificity of 41% [11]. Although in most cases this is not enough to make a safe diagnosis, which makes further laboratory and imaging studies indispensable. The importance of this step in the assessment of the acute abdomen should not be underestimated. Patient history and physical examination findings should build on sufficient clinical experience and a detailed knowledge of the anatomy and physiology of the abdominal cavity and a clear conception of the pathological processes at work [5, 17]. Unfortunately though, when the ability of clinicians to take an organized and accurate history has been studied, the results have been rather disappointing, which is why the use of standardized history and physical examination forms has been recommended and yielded an improvement of the diagnostic accuracy by 20% [1, 11].

Patient history

The leading symptom of the acute abdomen is pain. It is imperative to inquire about the dimensions of pain, which are summarized under the mnemonic acronym SOCRATES, helping to ensure a thorough history. Much as its name giver's methods of questioning should the patient history be conducted, with systematic and a critical mind.

- Site: Location of pain
 - Onset: Exact time and mode of onset-sudden or gradual? Setting - what was the patient doing when the pain first started? Is it progressive or regressive?
 - Character: Localized or diffuse? Dull or sharp?
 - Radiation or referral of pain?
 - Associated Symptoms: Jaundice, Anorexia, nausea, vomiting, diarrhoea Constipation Pyrosis and/or singultus?
- Genitourinary symptoms - dysuria, pollakisuria, hematuria?
- Time course: Is the pain continuous or intermittent? Does it follow a pattern?
 - Exacerbating/Relieving factors: Are there palliating or provoking factors – breathing, coughing, food intake, defecation, vomiting?
 - Severity: from 1-10 on the visual analogue scale (VAS)

Site: Pain location

Based on the type of afferent nerve innervation three basic patterns of pain perception can be differentiated: Visceral pain, somatic pain and referred pain [11].

Visceral pain:

Visceral pain results from stretching or distention of the intestine or excessive contraction against resistance [1, 11, 17]. Touch or inflammation as long as they do not irritate the parietal peritoneum do not lead to a pain perception [17]. Visceral nociceptive stimuli are conducted to the brain via C-fibres, which means that the pain elicited will generally be perceived as dull in character [11,12,14]. Due to the fact that the innervation of abdominal organs is arranged bilaterally, the pain is oftentimes referred to the midline, but is mostly of poor localization [11, 12, 14, 17].

Somatic (parietal) pain:

Somatic pain originates from irritation or inflammation of the peritoneum parietale, mesentery, mesocolon or retroperitoneum and is of a sharp, stabbing character [14]. Patients can discern its localization much better than visceral pain, because in somatic pain the painful stimulus is conducted via A-delta-fibres, which are unilaterally arranged and can therefore be better lateralized by the patient [11].

Visceral pain, as demonstrated by the case of acute appendicitis can change over to somatic pain [14]. In acute appendicitis, typically the pain starts as a poorly localized periumbilical pain, which as the inflammation progresses, irritating the peritoneum, shifts to the right lower quadrant becoming increasingly better localized.

Referred / Radiating pain:

Referred pain arises from a convergence of somatic and visceral afferent fibres. This type of pain is not perceived at the point of origin, but may be projected to a corresponding possibly far away lying dermatome. Examples of referred pain include the pain of a renal colic which radiates to the groin or the right subscapular pain felt in a biliary colic [1]. Generally the chance of perceiving referred pain is higher, the more severe the painful stimulus is [1].

All three types of pain perception may appear together, which has to be taken into account when assessing the patient [11]. On the basis of the pain localization a preliminary differential diagnosis can be formulated [18].

Mode of onset and duration:

The mode of onset and the acuteness of abdominal pain give important clues about the severity of the underlying pathology [1, 14,17]. Abdominal pain that sets in suddenly, particularly if severe, is suspicious of an intra-abdominal catastrophe of the likes of a ruptured abdominal

aortic aneurysm (AAA) or perforated viscus [1, 14, 17]. In the case of such a sudden-onset pain it is useful to ask whether there was a concomitant loss or near loss of consciousness [1, 17]. According to Cope's Early Diagnosis of the Acute Abdomen [17], the perforation of a gastric or duodenal ulcer, acute pancreatitis, ruptured aortic aneurysm or a ruptured ectopic pregnancy are the only abdominal conditions likely to cause a person to faint in the setting of acute abdominal pain. Abdominal pain that awakens the patient from sleep should also be considered serious until proven otherwise [1, 12, 14, 17]. The duration of pain can be an early clue, whether surgical treatment is necessary or not. Acute abdominal pain that lasts 6 hours or longer is likely to be caused by pathologies requiring surgical intervention [1, 11].

Character:

The assessment of the character of pain helps giving further insight into the nature of the underlying condition [17]. Abdominal pain can be characterised as dull, burning, tearing, sharp stabbing, as well as localized or diffuse. Particular types of pain seem specific and characteristic for certain pathologies [1, 17]. The pain of a perforated gastric ulcer for example is of a burning character, the pain of a dissecting aneurysm tearing and that of a bowel obstruction gripping [1, 17]. In most cases the pain described by the patient does not fit in these profiles [1], which makes a sound comprehensive approach even more important.

Associated symptoms:

The assessment of associated symptoms and their relation to the abdominal pain is of great significance. Besides gastrointestinal symptoms it is important to inquire about urinary symptoms and in female patients a full gynaecological and obstetric history should be taken [1, 11, 12].

Vomiting: Vomiting commonly accompanies all acute abdominal pain and can be encountered in almost every abdominal disease [1, 12]. Apart from acute gastritis, vomiting in association with abdominal lesions is either due to a severe irritation of peritoneal or mesenteric nerve endings as in the case of a perforated ulcer or is due to the obstruction of an involuntary muscular tube, as seen in the case of colics [17]. It is important to enquire about the timely relationship between vomiting and pain onset [1, 12, 17]. In surgical conditions, pain generally precedes vomiting, whereas in medical conditions, like for example acute gastroenteritis, the pain usually comes after the vomiting [1, 12, 17]. The character of the vomitus should be noted. Here it is important to ask whether the vomitus was bilious or contained blood [12, 17]. The presence of blood or coffee ground emesis suggests gastric disease or might be caused by a complication of hepatic disease [12]. Bilious vomiting is commonly seen in colics [17]. A way of differentiating benign causes of vomiting like viral gastroenteritis or food poisoning from rather sinister ones is that they are usually self-limited [12].

Nausea and anorexia: Cope [17] states that nausea, anorexia and vomiting represent different grades of the same kind of stimulus, hence it is important to question about their presence or absence [17]. Anorexia is closely linked to the diagnosis of acute appendicitis and is often used as a discriminatory symptom for that matter [12]. Yet it is important to keep in mind that only 68% of patients with acute appendicitis also report it as a symptom [12].

Bowel symptoms:

- **Diarrhea:** The passage of diarrheal stools typically characterizes gastroenteritis but should not be used as a marker to rule out more serious pathologies [1, 12]. Mesenteric ischemia, whose early symptoms are often missed, also presents with frequent loose bowel movements [14,17]. Other differential diagnoses that have to be considered are acute appendicitis and partial small bowel obstruction or early complete small bowel obstruction, where in both cases in an attempt to clear itself bowel peristalsis becomes hyperactive [12, 17].
- **Constipation:** Constipation by itself is no reliable symptom unless other symptoms are associated, most importantly the presence or absence of flatus [12, 17]. The clearing of gas happens more quickly than fluid, which is why the absence of flatus is a better sign of intestinal obstruction than constipation [12].
- **Hematochezia/melena:** The passage of bloody stools in patients presenting with abdominal pain should alert one's mind to the possibility of mesenteric ischemia [12]. Melena is usually indicative of upper gastrointestinal bleeding, for example from a peptic ulcer [12].

Genitourinary symptoms: Due to the fact that the genitourinary and gastrointestinal organ systems lie in such close proximity to each other inflammatory processes in either of them may lead to a symptomatology in the other [12]. Such as for example testicular torsion may present with acute abdominal pain, nausea and vomiting, so can acute appendicitis lead to dysuria and pyuria [12]. Therefore it is important to take an exact history asking about dysuria, frequency, urgency and hematuria. In women, it is imperative to ask detailed questions about the menstrual cycle, including cycle length, date of last menses, changes in menstrual blood loss, dysmenorrhea and the use of contraceptives [1]. Furthermore, inquiries should be made about previous pregnancies or miscarriages and risk factors for ectopic pregnancy [1].

Time course and progression pattern:

Pain can be either of a constant, continuous nature or as in the case of colics intermittent [1]. Colics are defined as paroxysmal attacks of cramping pain that typically last a few minutes and are followed by periods of pain relief [1, 17]. Causative for this type of pain is the violent peristalsis of the muscular wall of hollow viscera like the intestines, bile ducts, ureters or fallopian tubes, in an attempt to overcome an obstruction in the tube [1, 17]. Continuous pain

is more common than intermittent pain and usually points to a pathological process that will in its course cause ischemia or inflammation of the peritoneum [1].

Exacerbating / Relieving factors:

During the patient history it is very important to ask about the settings, in which the pain worsened or was palliated. The pain of peritoneal irritation is typically exacerbated by jarring motions as those of a cough or while walking and should be enquired about [1, 11, 12]. Abdominal pain in the upper quadrants might originate from pleural irritation caused by pathological processes in the thorax, for example pneumonia [1, 12, 17]. Hence, it should be established, whether the pain is influenced by respiration. Eating might also be a provoke or palliate pain. In the case of peptic ulcer disease, eating exacerbates the pain if gastric and alleviates the pain if duodenal in origin [6]. Pain following especially fatty meals is oftentimes reported by patients suffering from gallstones. In abdominal angina, in the case of mesenteric ischemia, as the blood supply cannot meet the oxygen demand of the intestine created by food digestion, pain ensues [12, 17].

Severity:

Pain that is severe should heighten the concern for a serious underlying cause [11]. Descriptions cannot always be relied upon to exclude serious illness, especially in older patients who may underreport symptoms [12,14]. As an attempt to objectivise and categorise the pain perceived by the patient, the visual analogue scale (VAS) may be used. The score obtained dictates, whether analgesics are of need and what type of pain medication should be used. Herein the use of opiate analgesia comes into play at a VAS of >3 [11].

The question whether the patient has felt a pain like this before should be asked at any rate [12,17]. Except for mesenteric ischemia, cholecystolithiasis or partial intestinal obstruction, recurrent episodes generally suggest that the pain is of a medical causative origin [12].

After a thorough history of the presenting symptoms and abdomen focused patient history, the physician should obtain a history of past illnesses, medical and surgical [1, 11]. Inquiries should also be made about allergies and medications, since both of which might be causative aetiologies or have an impact on the diagnostic measures taken and treatment plan.

Physical examination

The abdomen focused physical examination of a patient with acute abdominal pain follows an IAPP (Inspection - Auscultation - Palpation - Percussion) scheme.

Inspection: Prior to focusing purely on the abdominal wall of the patient, the treating physician should actively look for signs indicating a systemic pathological process [1]. Inspecting the patient's skin for signs of jaundice or spider naevi is as much part of the physical exam as the palpation of the abdomen. The patient's general appearance, vigilance and vital signs, which

mostly are ascertained during the course of the triage give important information about the acuteness of the problem as well as clues which might point towards a certain differential diagnosis [11, 17, 19]. The patient's position and attitude in bed is worthy of attention. Note for example the contrasting nature of those enduring severe colics squirming restlessly about in bed and those suffering from peritonitis, who is trying to avoid any movement [1, 17, 19]. For the acute abdomen, every anomaly of vital signs or vigilance should lead suspicion to an intra-abdominal catastrophe and prompt urgent intervention [11]. An old surgeons' lore is that an abdominal examination is from nipples to knees. That means that the patient must be undressed and inspected accordingly, in order not to miss possible skin changes or masses, for example the signs of herpes zoster or the ecchymoses typical of intra-abdominal haemorrhage (Grey-Turner's sign, Cullen's sign) [12]. Furthermore, the treating physician must take notice of any abdominal distention, which might signify intestinal obstruction or ascites [1, 12].

Auscultation: It is imperative that the auscultation is performed before the abdominal palpation and percussion, since these could encourage bowel peristalsis and thereby falsify the findings [14]. The clinical significance of abdominal auscultation is quite a controversial topic though [20-23]. Cope (Silen 2010) states: "Of all the modalities of physical diagnosis of the abdomen, auscultation is one of the least valuable and most misleading." Despite its inherent role in the examination process, we still have an insufficient understanding of the acoustic properties of bowel sounds and a lack of proper systematic training of abdominal auscultation [20, 24]. According to a traditional saying silence at auscultation indicates peritonitis and loud rumbling or gurgling sounds point to intestinal obstruction [17]. However, among the different sounds of peristalsis, whether present or not, one cannot infer a pathological process, except for the high-pitched borborygmi of small bowel obstruction [11, 25]. Furthermore, it was shown that there is a lack of inter-observer agreement, which reflects the need for improvements of skill in differentiating bowel sounds [20, 24]. Although abdominal auscultation by itself is not useful in differentiating normal from pathological sounds of peristalsis, with patient history and clinics its diagnostic value may be increased [20]. Under these circumstances, namely the aforementioned improvements in education and training, the abdominal auscultation should be continued [20, 24].

Palpation: The palpation of the abdominal wall is the most important part of the abdominal examination [11]. Its main purpose is to look for signs of peritonitis, to localise tenderness and to detect organ enlargement, as for example that of the liver, spleen or possibly the aorta [12]. The palpation of the abdomen should be started at the point farthest away from the point of maximum tenderness in order to avoid unnecessary discomfort for the patient during the process [1, 17]. A too rough examination may by itself elicit pain and be therefore misleading for the examiner, which is why it should be done carefully and gently [11,12,17]. The classical signs of peritonitis are rebound tenderness and muscular rigidity or "defense musculaire" [2].

The test for rebound tenderness is performed by deeply pressing down on the abdominal wall and then suddenly releasing the pressure. Pain felt upon the pressure release means that the test is positive. Studies have shown that the test for rebound tenderness has a sensitivity and specificity of 80 % and 40-50 % respectively [12]. A way to increase the specificity is to use the “cough test”, which is an indirect test for rebound pain has a similar sensitivity but an even higher specificity of 79 % [12].

Abdominal wall rigidity or “defense” is an involuntary reflex, whereby the abdominal wall muscles contract in order to protect and prevent movement of the underlying viscera [12,17]. The term “guarding” is often used instead of and synonymously with “rigidity”. The underlying pathophysiological process is in both cases the same. In some texts a differentiation is made by saying that rigidity represents the extreme form of guarding [12]. In practice though, this is a purely nomenclatural problem. The differentiation of guarding and rigidity is of less importance. The crucial point is to differentiate between voluntary and involuntary guarding. This can be achieved by assessing the muscular rigidity throughout the respiratory cycle. In case of true involuntary guarding, the abdominal musculature will remain stiff throughout inspiration and expiration. In voluntary guarding the muscle tone decreases during the inspiratory phase.

It is important to bear in mind that the manifestation of peritoneal signs might be diminished or simply overseen even though a serious peritonitis is looming beneath the examiner’s fingers. This might especially be the case in elderly patients with weakened and lax abdominal musculature, fat abdominal wall or in instances of severe toxemia [1, 11, 12, 17].

Percussion: Gentle abdominal percussion can be viewed as a miniature version of the rebound tenderness, which does not only yield more precise results with respect to the localization of the peritoneal irritation but also spares the patient from unnecessary pain that results from the “rebound” during palpation [14, 17]. In suspected peritonitis the careful percussion of the abdominal wall is the examination technique of choice [14]. Percussion of the abdomen is also of aid in case of abdominal distention. Large bowel obstruction which leads to a drum-like tympanic note on percussion can be discerned from the shifting dullness indicative of advanced ascites [12].

Digital rectal examination

In conformance with the accepted standards of care, every patient who presents to the emergency department with acute undifferentiated abdominal pain should in order to complete the physical evaluation be examined by digital rectal examination [26,27]. Without it, the physical exam is considered to be incomplete [27]. As Cope (Silen 2010) phrases it: “It is as important to insert a finger into the lower end as it is to order a plain film of the abdomen.” Although both, the digital rectal examination as well as the plain abdominal film (vide infra) were and still are used as diagnostic modalities, studies have shown that their diagnostic utility

is of less value than previously believed [11, 26,27]. Thus the digital rectal exam's meaningfulness in the assessment of acute abdomen in the emergency department must be questioned [11, 12, 27]. There is no evidence in the literature supporting the routine performance of a digital rectal examination in patients with acute, undifferentiated abdominal pain and acute appendicitis [12, 26, 27]. However, it should not be entirely discarded. In cases of specific differential diagnoses, as that of suspected gastrointestinal bleeding, intestinal ischemia or colorectal cancer the digital rectal examination remains to have a clear indication and justification [12, 27].

Special abdominal examination manoeuvres:

Tailored to the preliminary differential diagnoses formulated further examination manoeuvres can be useful in establishing a diagnosis [19]. Before the advent of imaging modalities the diagnosis was based solely on the clinical evaluation of the patient. In order to increase accuracy special signs and manoeuvres were utilized and still are in use. Studies which assess their sensitivity and specificity are scarce and thus diminish their value [14], which is why they have to be interpreted with care. Some examples are listed here:

The iliopsoas sign: The iliopsoas sign is performed by having the patient roll onto his/her left side and hyperextending the right hip joint. If pain is elicited the sign is positive and suggests an irritation of the iliopsoas muscle by a retrocecal appendicitis [1, 17, 25]. Other pathologies that might lead to a positive iliopsoas sign are pyelonephritis, pancreatitis and psoas abscess [12].

The obturator sign: With the patient supine, the thigh of the patient has to be flexed passively and fully rotated inward. The test is positive, if pain is elicited by that manoeuvre and means that the obturator muscle is inflamed because of pathology of a neighbouring viscus [1, 17, 25]. Causative pathologies might be a pelvic appendicitis, diverticulitis, pelvic inflammatory disease or ectopic pregnancy [12].

The Rovsing sign: The Rovsing sign is being tested by applying pressure the left lower quadrant. If pain is being referred to the Mc Burney point the sign is positive and increases the likelihood of appendicitis [1, 12, 25]. However, the test is not perfect, since it has both false positives and negatives [25].

Murphy sign: The Murphy sign is performed by asking the patient to take a deep breath while palpating the right upper quadrant of the abdomen. If the patient abruptly stops the inspiration, the sign is positive and is suggestive of acute cholecystitis [1, 12, 17, 25]. Numerous studies were conducted, which evaluated the sensitivity and specificity of the Murphy sign, leading to varying results ranging from 44%-97% and 48%-96% respectively [25]. Although it is the most reliable clinical indicator of acute cholecystitis, with a positive LR, of 2.8 and a 95% CI of 0.8-8.6, since the 95% CI includes 1, further diagnostic testing is necessary to secure the diagnosis [12, 28].

Cullen and Turner signs: Both represent ecchymoses on the skin of the abdomen, which result from intra-peritoneal or retroperitoneal haemorrhage which dissects through the skin. The Cullen sign was first described in case of a ruptured ectopic pregnancy and represents a periumbilical ecchymoses and the Turner sign is a discolouration of the flanks and is suggestive of haemorrhagic pancreatitis [1, 25]. Strictly speaking, the only difference between those two signs is the eponym, especially as the location of the bruise does not give clues about the origin of the bleed [25]. The sensitivity of these tests is very limited, with less than 1% for a ruptured ectopic pregnancy and 3% for acute pancreatitis. Taking into account that a whole array of different pathologies may cause these signs, their specificity and thus their relevance for the diagnosis of ruptured ectopic pregnancy and acute pancreatitis is limited [25].

Analgesia

Regarding the use of analgesics in patients with acute abdomen a change of paradigm has taken place [11]. For generations the regrettable doctrine of not administering analgesics until the diagnosis was secured, ruled [17, 29]. The fear was, that analgesics might mask symptoms, obscure findings of the physical exam and delay the diagnosis [19]. A Cochrane analysis [19] refuted this and showed that the administration of analgesics in fact does not obscure relevant findings of the physical exam, nor does it increase the risk of diagnosis errors or errors in decision making with respect to the treatment. Furthermore this not just fulfills the physician's duty of relieving pain and suffering but also by increasing patient comfort adds to the quality of the emergency care [11,12]. What should be stressed though is, that prior to the administration of pain relieving medication the clinical findings are evaluated with ample security [11].

Investigative Studies

In light of the fact that at least one third of patients with acute abdomen present with atypical features [1], the diagnosis should and cannot be based solely on the clinical evaluation but makes investigative studies (laboratory tests and imaging) ultimately indispensable [11]. All diagnostic tests, whether laboratory tests or radiological studies have a false-negative rate and therefore, if the clinical evaluation of the patient leads to a high pre-test probability of a disease, the initially suspected diagnosis should not be discarded [12, 30]. For example, in the case of suspected appendicitis, the use of laboratory markers (WBC, granulocyte count and proportion of polymorphonuclear cells, CRP) is insufficient to ascertain the diagnosis and the use of computed tomography (CT), even though it is the most accurate diagnostic modality bears the burden of radiation [21]. This emphasises the importance of clinical experience and an understanding of the "inner workings" of the abdominal cavity. Moreover, the over-reliance on laboratory tests and radiological evaluation might be misleading, especially if the clinical evaluation of the patient was not conducted properly [17]. It is important to stress that one's clinical judgement should not be led astray or blinded by an alleged infallibility of investigative

studies. The ordering of laboratory or radiological tests should not be done blindly but build on a sound differential diagnosis.

Laboratory Tests

The extent of laboratory testing varies and is dictated by the suspected diagnosis and differential diagnoses. The minimal panel of laboratory tests in a patient with acute abdomen entails [3, 11,12]:

Complete blood count (CBC): The CBC is useful in detecting changes in plasma volume due to dehydration (hematocrit), diagnosing anaemia and by virtue of the WBC detecting an inflammatory process [1]. The finding of an elevated WBC does not necessarily exclude, nor prove an intra-abdominal inflammatory process though. Besides this, it is important to remember that in the early stages of a hemorrhagic process the RBC count might be normal and should not delay appropriate treatment unnecessarily [17].

C-reactive Protein (CRP): The CRP is an acute phase protein and of the β -globulin fraction. Its levels start to rise 6-12h after the beginning of an inflammatory process. In adjunct with the WBC it increases the confidence in the diagnosis of an acute inflammatory condition [1, 21, 30].

Procalcitonin (ProCT): Procalcitonin, the precursor hormone of calcitonin, was found to be a useful marker in the assessment of sepsis and systemic inflammatory response and has also proven its worth in the differentiation of infectious from non-infectious inflammation [31,32]. Although it is mainly produced by the C-cells in the thyroid gland and neuroendocrine cells in the lungs many other tissues have shown the capacity to synthesise it [32]. In the event of a systemic inflammatory process, hypersecretion of ProCT follows [32]. In the assessment of acute abdominal pain, ProCT can be of particular help, when acute mesenteric ischemia, necrosis in acute bowel obstruction or abdominal sepsis are suspected [31]. A systemic review by Mofidi et al. [33] concluded that ProCT may also be valuable in predicting the severity of acute pancreatitis and the risk of infected pancreatic necrosis.

Liver function tests: Liver function tests including a total bilirubin count, alkaline phosphatase, γ -glutamyltransferase (γ -GT), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are mandatory in abdominal pain suspected to be of hepatobiliary origin [1].

Lipase/amylase: The evaluation of amylase and lipase is obligatory in suspected pancreatitis [1]. Caution is in order though, as normal levels of amylase cannot exclude pancreatitis, but elevated levels do not necessarily prove it either, since the levels might be also elevated in conditions like intestinal obstruction, mesenteric thrombosis or a perforated peptic ulcer [1, 14]. A study conducted by Gomez et al. [34] showed that lipase has a higher sensitivity and specificity in the diagnosis of acute pancreatitis, with 96.6% and 99.4% respectively, and is therefore more suitable in the diagnosis of acute pancreatitis than amylase. Gomez et al. [34] concluded not only that determining lipase levels alone is sufficient to diagnose acute

pancreatitis but also showed that by this approach, a substantial amount of money may be saved. It is important to mention that although higher serum enzyme levels make the diagnosis of acute pancreatitis more likely they do not reflect the severity of the pancreatic involvement [14].

Serum electrolytes & kidney function tests: Assessing creatinine and blood urea nitrogen (BUN) levels may give important information about the hydration status of the patient and the nature of fluid loss [1]. BUN/creatinine, electrolytes together with CBC are the most commonly ordered laboratory tests in the ED [30]. Although these parameters might not be as useful in the detection of the underlying pathology as for example lipase levels in pancreatitis, they are of clinical importance as they serve as indicators of systemic involvement [30].

Serum Glucose: Derangements of the glucose metabolism, be it hypo-, or hyperglycemia can present in different colours and forms. It is not by chance that disturbances of glucose metabolism are seen as the “chameleon of emergency medicine”. To prove the point: Pseudoperitonitisdiabetica is a condition that may be encountered in patients with decompensated diabetes mellitus in the course of keto acidotic coma [2]. These patients present with the clinical picture of peritonitis, with severe abdominal pain and possibly vomiting, mimicking an inflammatory intra-abdominal condition.

Lactate: An increase in lactate levels indicates anaerobic glycolysis and therefore is a parameter for inadequate perfusion, oxygenation and an estimate of tissue oxygen deficiency [31]. Although not being specific to abdominal conditions increased lactate levels were found to be a possible marker of mesenteric ischemia [30]. Besides this elevated lactate levels may be found in conditions like general bacterial peritonitis, strangulated intestinal obstruction, diabetic keto acidosis and acute pancreatitis [31].

Urinalysis: Urinalysis is a frequently ordered laboratory test and next to the abdominal CT the most useful test in the assessment of acute abdominal pain in the ED [30]. Erythrocytes in the urine suggest nephrolithiasis or urolithiasis, leucocytes and nitrites are suggestive of a urinary tract infection, and an elevation of glucose and ketones might point to diabetes [1].

In women of childbearing age it is imperative to order a β -human chorionic gonadotropin (β -hCG) test [1, 4, 11]. This is not only important since a complication of pregnancy might be the cause of the abdominal pain but also because a positive result has an impact on the evaluation of the patient with respect to the choice of imaging modality and treatment [4].

In patients with upper quadrant and epigastric pain troponin levels should be assessed in order to exclude myocardial ischemia [11]. Furthermore, it is mandatory to do an electrocardiography in elderly patients, patients who present with epigastric pain and patients with risk factors for cardiovascular disease (CVD) or a history of the same [1, 11,14].

Imaging

Imaging diagnostics have an undisputed central role in the evaluation of the acute abdomen in the ED [11]. With plain X-rays, computed tomography (CT), ultrasonography (US) and magnetic resonance imaging (MRI) different imaging modalities are at disposal. In order to prevent the patient from being exposed to ionising radiation unnecessarily or undergo time-consuming and costly procedures which might delay treatment it is important that the treating physician chooses the imaging modality accordingly.

X-ray: Plain abdominal radiography (AXR) has until recently been in use as the initial imaging modality in the investigation acute abdominal pain but has since been surpassed by ultrasound imaging and CT [1, 3, 11,22,30]. Although current literature advises against the routine use of abdominal X-ray imaging in the work-up of patients with acute abdominal pain in the ED, it is often being utilised [4, 22]. Nevertheless there remain some conditions where plain films may be useful: air-fluid levels and the presence of dilated loops of bowel are suggestive of intestinal obstruction, suspected intestinal perforation might be proven by demonstrating the presence of free air (pneumoperitoneum), plain films also have a role in the detection of foreign bodies and in the follow-up of renal tract calculi [1, 22, 30].

Ultrasound (US): The ultrasound examination can and should be viewed as the continuation of the clinical evaluation of the acute abdomen and should rank first among the investigative studies [11,14]. Thus, all patients with suspected acute abdomen, after a comprehensive patient history and thorough physical examination should undergo an abdominal sonography [11]. Plain film or computed tomography can only make a snapshot of a pathological process. A true advantage of the abdominal sonography is that the examination is dynamic and takes place in real time [30]. In this way not only the intestinal peristaltic but also the perfusion of abdominal vessels can be evaluated [11]. Ultrasound imaging is non-invasive and repeatable as often as desired, which can be very useful in assessing the dynamic of the pathological process [1, 30]. The main drawback regarding its precision is its high user-dependency, which narrows its diagnostic conclusiveness [11]. Another disadvantage of the US is patient factors including the patient's body habitus and bowel gas, which may limit the examination [30]. Nevertheless, abdominal sonography is an easily accessible imaging modality that may be performed by the ED physician at the patient's bedside, which makes it especially useful in the evaluation of unstable patients [24].

Computed tomography (CT): The diagnostic value of CT remains undisputable [11]. It is as a method more objective, reproducible and with less inter-observer variability than the US [11, 30]. A study by Stromberg et al. [23] showed that by the use of CT the correct diagnosis could be ascertained in 96.8%. Moreover it was found that the early use of CT leads to a decrease in duration of hospital stay and a decrease in hospital admission overall [3, 11]. However, a clear and major drawback to the regular use of CT imaging is the considerable radiation exposure and

the thereby increased lifetime risk of a radiation-induced cancer [3, 11, 30]. This risk rises indirect proportionally with the age of the patient. The radiation dose of an abdominal CT lies between 10-30 mSv [11,30, 33].

CT- angiography: CT-angiography is the diagnostic modality of choice in the evaluation of suspected mesenteric ischemia or disturbances of renal blood flow [3, 14]. Since mesenteric blood flow disturbances are detected relatively late in its course should the indication for a CT-angiography be posed quite liberally [14]. Furthermore the CT-angiography can also be of use if the source of gastrointestinal bleeding cannot be found or if renal damage is assumed [14].

Endoscopy: Endoscopy represents the link between diagnostics and intervention but has a rather minor role in the evaluation of the acute abdomen in the ED [3, 11, 14, 17]. Nevertheless an upper GI endoscopy may be helpful in detecting peptic ulcers as the causative pathology in cases of unspecific acute abdominal pain [14].

Magnetic resonance imaging (MRI): Currently MRI plays a rather minor role in the evaluation of acute abdominal pain in the emergency department [11, 35]. The reason for that is its low availability compared to CT, the time consumption and the cost of the procedure [11, 35]. The advantages of magnetic resonance imaging on the other hand, namely the lack of ionising radiation and a proven diagnostic track record make in light of the increased awareness about the detrimental effects of ionising radiation an increasingly attractive alternative to CT [35]. MRI has an established role as the imaging modality of choice in acute abdominal pain in the pregnant population, where the US was bland [14, 33].

Choice of imaging modality

The choice of the imaging modality should be based on two things: Firstly whether thereby a diagnosis can be ascertained or excluded and secondly whether this justifies the radiation exposure [3, 11, 13]. The OPTIMA study [13] was conducted with that goal in mind. On this basis a strategy was formulated which implies that all patients, who present with acute abdominal pain, should undergo an ultrasonographic examination. CT with i.v. contrast should be employed when the US exam is bland or yields non-specific results. Thereby a sensitivity of 94% and specificity of 68% could be achieved, which under the precondition of radiation protection produced the best results and should therefore be the diagnostic protocol to follow [3, 11, 13].

When the physical examination, laboratory tests and abdominal sonography do not show any abnormalities and therefore the aetiology remains unclear and the patient does not present with a symptomatology suggesting a surgical condition, the diagnosis undifferentiated abdominal pain can be assumed. In such an instance CT imaging might not be necessary [1, 11].

If the patient has risk factors for a surgical problem, namely abdominal pain that lasts less than 48h, pain after vomiting, involuntary guarding and rebound tenderness, is ≥ 65 years old or has

a history of previous abdominal surgery, the indication for CT imaging should be posed generously [11]. This is especially true in older patients, as they are more likely to present with atypical and unspectacular symptoms, yet harbouring a significant underlying illness [11].

Management

On the basis of clinical evaluation and investigative studies a working diagnosis should be established and according to the SOAP scheme a treatment plan formulated [1]. In doing so the ED physician must first decide whether surgical treatment is required or not and if so, whether the patient must undergo emergent, urgent or early elective surgery [1, 3, 11]. If there is no clear indication for a surgical intervention or medical treatment, one of two routes can be chosen: Hospitalisation and conservative therapy under active observation or evaluation of the patient in an outpatient setting preferably by a Surgeon [1, 11].

Fortunately intra-abdominal calamities that call for emergent operation are a few [1]. They include the rupture of abdominal viscera – abdominal aortic aneurysm, liver or spleen, or the rupture of an ectopic pregnancy [1, 11]. The pointers of such crises are severe abdominal pain and haemodynamic instability represented by a critical derangement of vital signs and symptoms of shock [1, 11]. Obviously in such circumstances time is of essence and does not allow for an exhaustive evaluation but still if the patient's condition makes it possible, the basic diagnostic steps should be completed and a probable diagnosis found [1, 11].

Urgent surgical interventions are generally necessary in conditions like perforated hollow viscus, acute appendicitis, acute diverticulitis, acute cholecystitis, mesenteric ischemia, intestinal obstruction or incarcerated hernia [1, 11, 13]. The clinical picture that indicates such an approach is that of generalized or localized peritonitis or massive abdominal distention, accompanied by signs of sepsis and/or ischemia [1, 5]. Investigative studies that show findings of pneumoperitoneum, extravasation of contrast material or occlusion of blood flow are as well indications for urgent operation [1, 11]. An important exception to this "rule" is the acute pancreatitis, which might present with a similar symptomatology, is treated mostly conservatively [11].

A conservative approach with active observation may be chosen, when despite the exhaustion of noninvasive diagnostic efforts the aetiology of abdominal pain remains unclear [1, 3, 11]. Patients having severe abdominal pain, who are dehydrated and have an electrolyte imbalance or elevated inflammatory parameters should be admitted and treated accordingly [1, 11]. If the patient's constitution and compliance allow it, the patient might be followed-up 24-48h later in an outpatient setting alternatively [1, 11]. The fear that such an approach opens the door for complications could not be proven true [1, 11]. Studies have shown that although the diagnosis and therapy did change at the follow-up visit in 30% and 17% respectively, this fact did not influence the rate of morbidity [37].

Next to surgical causative pathologies, the ED physician must think of extra-abdominal causes too. Hereto it is important to stress once again that two thirds of patient who present to the ED with acute abdominal pain do not require surgical treatment [1].

In female patients special care has to be taken to exclude gynaecological pathologies and in male patients the possibility of an acute scrotum should be taken into consideration. In such circumstances it is necessary to call for gynaecological or urological consults [11].

Invasive diagnostics

An alternative to the “wait and see”-approach is the diagnostic laparoscopy. According to the guidelines laid out by the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) the explorative laparoscopy is indicated, when all non-invasive diagnostic measures were exhausted and yet the aetiology of the acute abdominal pain is unresolved [3, 4, 11, 38]. By the use of diagnostic laparoscopy a diagnosis can be made in 90-98% of cases [4]. However, there is not enough evidence to favour the explorative laparoscopy over active observation in suspected undifferentiated acute abdominal pain [3, 11].

Team responsibilities

Acute abdominal pain frequently poses a diagnostic dilemma. These patients may exhibit non-specific signs and symptoms such as vomiting, nausea, and leukocytosis. The cause of acute abdominal pain may be due to a myriad of diagnosis including gynecological, obstetrical, gastrointestinal, urological, metabolic and vascular etiologies. While the physical exam may reveal that the patient has a surgical abdomen, the cause is difficult to know without proper imaging studies.

While the general surgeon is almost always involved in the care of patients with an acute abdomen, it is important to consult with an inter professional team of specialists that include an obstetrician, gynecologist, and a vascular surgeon. The nurses are also a vital member of the inter professional group as they will monitor the patient's vital signs. In the postoperative period for pain, wound infection and ileus; the pharmacist will ensure that the patient is on the right analgesics, antiemetics, and appropriate antibiotics. The radiologist also plays a vital role in determining the cause. Without providing a proper history, the radiologist may not be sure what to look for or what additional radiologic exams may be needed. This problem gets even more complex when women of childbearing age present with an acute abdomen. The American College of Radiology Appropriateness Criteria® are evidence-based guidelines for specific clinical disorders that are reviewed by an inter-professional expert committee every three years. The current guidelines have been developed after an exhaustive review of current medical literature from peer-reviewed journals to determine the appropriateness of radiological imaging and treatment procedures by the committee. In cases where evidence is not definitive or minimal, expert opinion from the specialist may be utilized to recommend the

type of imaging or treatment. The outcomes of an acute abdomen depend on the cause. However, to improve outcomes, prompt consultation with an inter-professional group of specialists is recommended.

Resources required for one patient/ related

Since most patients with an acute abdomen are seniors, they are best managed in an ICU setting. Intravenous hydration, Nasogastric decompression and pain control are often required. The role of antibiotics depends on the cause. Close monitoring is required as patients may develop complications like atelectasis, ileus, wound infections, DVT and pneumonia.

Acute abdomen is a condition that demands urgent attention and treatment. The acute abdomen may be caused by an infection, inflammation, vascular occlusion, or obstruction. The patient will usually present with sudden onset of abdominal pain with associated nausea or vomiting. Most patients with an acute abdomen appear ill.

The history and physical exam serve to eliminate some diagnoses and suggest others. Acute care physicians are well aware of the modes of presentation of these disease entities.

An acute abdomen may present in an obvious or subtle manner, but must always be recognized. Rapid, appropriate testing and concomitant resuscitative therapy are mandatory. If the condition is even possibly surgical, early consultation with a surgeon is mandatory.

Conclusion

The acute abdominal pain represents a diagnostic challenge for the ED physician. Apart from intra-abdominal pathologies, extra-abdominal and metabolic causes must be considered. Therefore a multi-disciplinary approach is of great importance. In order to improve patient care and forestall diagnostic errors, diagnostic algorithms and patient evaluation forms should be used. They serve as a means for viewing highly complex clinical pictures, such as the acute abdomen, from a lucid, logically coordinated and systematic overall perspective, in order to maintain a problem- and priority oriented approach. In the assessment of acute abdominal pain many pitfalls have to be avoided and tiptoed around. While these diagnostic algorithms are conceptualised to provide the best possible care for the majority of cases they do not substitute for the clinical experience and judgement of the physician.

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Acute Aortic Dissection

Introduction

Acute aortic dissection is the most catastrophic event affecting the aorta, with an incidence exceeding that of ruptured abdominal aortic aneurysm. In a population-based epidemiologic study by Clouse et al, 38% of dissections were diagnosed at autopsy¹. This substantial mortality underscores the importance of early diagnosis and initiation of appropriate therapy. 'Dissection' implies that a false lumen is created within the layers of aortic wall leading to compromise of the lumen by dissection flap; this may ultimately lead to aneurysm formation.

Epidemiology

Recent population-based studies have estimated the incidence of acute aortic dissection to range from 2.9 to 3.5 per 100,000 person years². Men are more frequently affected with a male/female ratio of 4:1 reported by IRAD³. The incidence of Type A dissection occurs more frequently between 50 and 60 years while Type B dissection occurs more frequently between 60 and 70 years of age.⁴

Classification

Aortic dissections need to be viewed in terms of time since onset of symptoms, and the anatomical location of origin of dissection flap.

1. Duration of Symptoms

Acute- From onset of symptoms & within 2 weeks.

Subacute- Onset of symptoms from the period of 2 weeks to 90 days.

Chronic - Onset of symptoms for more than 90 days.

2. Anatomic

DeBakey Classification

Type I- Dissection originates in the ascending aorta, extends through aortic arch, and continues into descending aorta and /or abdominal aorta.

Type II- Dissection originates in and is confined to the ascending aorta.

Type III a- Dissection originates in the descending aorta and limited to the same.

Type III b- Dissection originates involves descending aorta and variable extents involve abdominal aorta.

3. Stanford classification

Stanford type A - this dissection originates in the ascending aorta and therefore includes DeBakey type I and type II dissections.

Stanford type B – this dissection originates in the descending aorta distal to the origin of left subclavian artery.

The Stanford Classification is most widely followed. It has also clear implications for management: Type A Dissection is treated by Surgery, whereas Type B Dissection is treated medically. There are specific indications for early surgical treatment of Type B dissection.

Risk Factors

1. Connective tissue disorders: Marfan's Syndrome, Turner's Syndrome, Noonan's Syndrome and hereditary conditions are risk factors.
2. The aortic wall structural abnormalities and the presence of bicuspid aortic valve with or without aortic root dilation are known risk factors for ascending aortic dissection⁵.
3. Pregnancy: It is associated with a fourfold increased risk of aortic dissection and rupture. Preclampsia and resultant hypertension is the most common etiology⁶.
4. Cocaine Abuse: Cocaine ingestion is a rare cause of aortic dissection.

Pathogenesis of Mal-perfusion syndromes

It occurs when there is end organ ischemia secondary to aortic branch compromise from the dissecting process.

Two mechanisms for aortic branch compromise have been identified

- a. Dynamic Obstruction: The compressed true lumen is unable to provide adequate volume flow or the dissection flap may prolapse into the vessel ostium.
- b. Static Obstruction: The mal-perfusion is caused by dissecting process extending into the branch vessel proper, narrowing it to a variable degree.

Clinical Presentation

- a. Pain: Is the most common presentation, abrupt onset pain (located in the back, abdomen or chest), reported in more than 93% of patients.
- b. Hypertension: It is present in 70-90% of type B dissection, but only in 25% to 35% of type A dissections.
- c. Syncope/Neurologic Symptoms: It may complicate the presentation of acute aortic dissection in 5% to 10% of patients and often indicates development of cardiac tamponade.
- d. Peripheral Vascular Complications: Are common and occur in 30% to 50% patients in whom aortic arch and thoraco-abdominal aorta is involved.

Complications

1. Syncope
2. Cardiac tamponade
3. Stroke
4. Death
5. Mesenteric ischemia
6. Spinal cord ischemia
7. Paraparesis: due to interruption of intercostal vessels
8. Acute limb ischemia

Diagnosis

1. Chest X-RAY: there is widening of the mediastinum, which is the most common finding on X-ray and aortic calcification is usually seen with type A dissections.
2. . CT Angiography: It has a sensitivity of 83% to 95% and specificity of 87% to 100%, for acute aortic dissection. It is the Imaging modality of choice.
3. ECHOCARDIOGRAPHY: The sensitivity and specificity of transthoracic echocardiography (TEE) range from 35% to 80% and 40% to 95% respectively. The chief limitation of TEE imaging is the anatomic blind spot in the distal ascending aorta and arch.
4. MRI: the overall sensitivity and specificity for the diagnosis range from 95% to 100%.

Treatment

Initial Management

The principles of immediate treatment are:

1. Rapid Control of BP.
2. Pain relief. If agents like diclofenac or tramadol do not work
3. IV morphine 3-5 mg push may be required.
4. Maintaining hemodynamic stability
5. Early Diagnosis
6. Supplemental Oxygen
7. Steroids. IV Methylprednisolone may be given if paraparesis is setting in. Precise role in outcome is unknown.
8. Rapid evacuation to a CTVS Center. Definitive treatment of aortic dissections is beyond the scope of a general surgeon. These patients should be transferred to a CTVS center by fastest available means.

Definitive Treatment

TYPE A Dissection: Surgical

Type A dissection is a surgical emergency. Immediate surgery in a CTVS unit gives the only chance of Survival; un-operated the mortality approaches 100%. Replacement of ascending aorta / arch may be required; sometimes this may be combined with aortic valve replacement (AVR) if significant aortic regurgitation is present.

Treatment of acute type B dissection: Medical; Surgery (or stent grafting)

The primary treatment of Type B Aortic Dissection is Medical (control of BP, pain relief, and close observation in ICU). Surgery is indicated in specific situations.

Medical therapy: Primary treatment involves reducing systolic blood pressure and dP/dT, which in turn reduces intimal flap mobility and relieve forces predisposing to dissected aorta to rupture or compromise blood vessels. In standard practice, a combination of beta blocker and vasodilator is standard medical therapy. Pain relief, and close observation in an ICU are essential components of Medical Therapy.

Surgical Treatment: The standard surgical treatment used to be thoracotomy and replacement of thoracic aorta with a tube-graft, often with re-implantation of renal, SMA, and celiac axis (and sometimes intercostals arteries as well). The procedure carries a mortality of 10-15%, and morbidity (including paraplegia) of about 25%. This has largely been replaced by – TEVAR (Thoracic Endovascular Aneurysm Repair). In this procedure, a stent graft is introduced via a small cut-down in the groin. Stent graft coverage of the aortic entry tear provides means to accomplish logical short- and long-term goals of sealing proximal entry tear and induce false lumen thrombosis.

Indications for Surgical Treatment in Acute Type B Dissection

1. Visceral Malperfusion: renal failure, acute mesenteric ischemia
2. Acute Limb Ischemia
3. Paraparesis / Paraplegia
4. Rapidly enlarging false lumen (more than 4.5 cm diameter)

Long Term Survival

The INSTEAD TRIAL prospective trial conducted in Europe that compared medical treatment of Type B dissections with stent grafting (TEVAR) for sub-acute and chronic uncomplicated type B aortic dissection. There was no difference in survival at 2 years, but 5-year survival was better in TEVAR Group.

Conclusion

Acute aortic dissection is a surgical emergency which demands urgent attention. Diagnosis is often not straightforward. Type A dissections need immediate surgical treatment, whereas the majority of Type B dissections can be managed conservatively. The mortality in the condition remains high. The surgeon in periphery should stabilize the patient, control the blood pressure, relieve the pain with adequate analgesics, and arrange for rapid transfer of the patient to the nearest CTVS Center.

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Acute Appendicitis

Introduction

Appendicitis remains one of the most common diseases faced by the surgeon in practice. Despite improving diagnostic technology, there is still no single test or clinical finding that is 100% reliable. The cause of acute appendicitis is unknown but is probably multifactorial; luminal obstruction with an appendicolith is quite common. Lymphoid hyperplasia, parasitic infections, and neoplasm are less common causes

In 1886, Reginald Fitz's seminal publication, 'Perforating Inflammation of the Vermiform Appendix' advocated early appendectomy for appendicitis. Appendectomy is traditionally the treatment of choice and is increasingly done as a laparoscopic procedure.

Classification

Classified as uncomplicated or complicated based on clinical, radiologic, intra-operative, and/or histologic findings

Uncomplicated appendicitis (simple or non-perforated appendicitis)

Inflamed appendix **without any** evidence of gangrene, perforation, purulent intraperitoneal fluid, appendicular phlegmon, or intra-abdominal abscess.

Complicated appendicitis (includes perforated appendicitis)

Gangrenous inflamed appendix, formation of appendicular phlegmon, perforation, purulent- intraperitoneal fluid, , or intra-abdominal abscess.

The appendiceal wall has been compromised due to pressure and inflammation and the intraluminal contents have leaked out into the peritoneal cavity.

Incidence

Western data estimates that as much as 6% to 7% of the general population will develop appendicitis during their lifetime, with the incidence peaking in the second decade of life. Indian data is lacking.

Symptoms

1. Patients typically complain of anorexia followed by epigastric or periumbilical abdominal pain. Usually the pain localizes to its classic location in the right lower quadrant, so referred to as 'migrating pain'.
2. Pain gets worse after oral ingestion or movements

3. Feeling of nausea and vomiting (usually after onset of pain)
4. Low grade fever (absence of fever does not rule out appendicitis)
5. Weakness

Signs

1. Typical appendicitis patient appears ill and prefer to lie still because of the presence of localized peritonitis, which makes any movement painful.
2. Mild to moderate tachycardia.
3. Normal to low grade elevation of temperature.
4. Abdominal examination typically reveals tenderness and guarding on palpation of the right lower quadrant. The location of the tenderness is classically over Mc Burney's point (located one-third the distance between the anterior superior iliac spine and the umbilicus)
5. Rebound tenderness is also commonly elicited.
6. Occasionally if perforation occurs, diffuse peritonitis is seen.

A number of clinical tests and signs have been described to help in the diagnosis of acute appendicitis, however they are all indicators of peritoneal irritation and may differ depending on the location of the appendix.

Investigations

The diagnosis of acute appendicitis is predominantly a clinical one; many patients present with a typical history and examination findings. However, in atypical presentation diagnosis may be clinched with the help of imaging modalities like ultrasound scan and / or CT scan. The diagnosis of acute appendicitis can be challenging. Scoring systems devised are not definitive. A high index of suspicion—clinical, biochemical and radiological findings put together, may help a better decision making in the management of the patient with suspected appendicitis.

Diagnostic aides:

Laboratory tests are non specific and need to be correlated clinically.

- White cell count - usually elevated
- C-Reactive Protein - usually elevated
- Urine examination including pregnancy test (for female patient in child bearing age) – to be done to rule out ectopic pregnancy in atypical presentation.

Imaging aides

1. USG abdomen/pelvis –
 - preferred initial test in most patients
 - risk of radiation – Nil
 - usually seen is a distended, non-compressible appendix with an at least 6 mm diameter

2. CT scan abdomen /pelvis :
 - For selected patients especially with diagnostic dilemma, perforation and obese patients
 - Risk of radiation exposure – present
 - Plain or with IV contrast is recommended (Oral and rectal administration of contrast material is not routinely required)
 - Appearance of a thickened, inflamed appendix with surrounding fat “stranding” indicative of inflammation, is usually suggestive
 - High sensitivity and high negative predictive value –may help reduce the rate of negative appendicectomy
 - Not preferred as first line modality during pregnancy

3. MRI abdomen/pelvis
 - Reserved for suspected appendicitis during pregnancy
 - Recommended without use of contrast agents
 - Risk of radiation exposure– Nil
 - Appendiceal enlargement (>7 mm), thickening (>2 mm), and the presence of inflammation are usually suggestive of appendicitis
 - Low availability and expertise during non-working hours

For fitness for Surgery & Anaesthesia

- Complete Haemogram
- Blood sugar – if diabetic
- Serum Electrolytes – in selected patients with profound vomiting
- Blood Urea, Serum creatinine – in selected patients
- Bleeding time, clotting time and /or prothrombin time
- X’ray chest – in elderly and with history of pulmonary diseases
- ECG & 2D echo – in selected patients when cardiac compromise is suspected or evident
- PFT/ ABG –optional in high risk cases
- Other patient profile specific tests – as per pre-anaesthetic check up

Management of Acute appendicitis:

The following comorbidities are associated with poorer outcomes following treatment of appendicitis:

- Diabetes
- Immunocompromised state
- Obesity
- Crohn's disease
- HIV infection
- End-stage renal disease

I. Management of Acute Uncomplicated Appendicitis

A. Operative management of Uncomplicated appendicitis

The appropriate treatment of acute uncomplicated appendicitis is prompt appendectomy.

1. Timing of Surgery: (Urgent, not emergent)

The timing of surgery has been categorized as urgent intervention and not as emergent and can be performed within 6-24 hours of diagnosis without any statistically significant difference in length of hospital stay, operative time or rates of complications.

2. Resuscitation, optimization & Antibiotics:

Patient should be fluid resuscitated as many present with mild dehydration.

Appendicitis is considered polymicrobial infections and IV broad spectrum antibiotics need to be administered to cover Gram-ve bacteria and anaerobes (as per institutional policy).

3. Surgical approach

Open Appendectomy: Traditional approach

Position: supine

Anaesthesia: General / Regional / Even local

Incision: The choice of incision is surgeon's preference,

Commonly used are:

- Oblique muscle-splitting (Grid iron) incision (Mc Burney),
- Transverse incision (Rockey-Davis),
- Conservative midline incision, as indicated
- Lane's crease incision

Common Steps:

- The cecum is grasped by the taeniae and delivered into the wound
- Allows delivery of the appendiceal tip & visualization of the base of the appendix.
- The mesoappendix is divided sequentially.
- Appendix is crushed just above the base, ligated with an absorbable ligature at the site of crush, and divided.
- The stump is then either cauterized or inverted by a purse-string or Z suture technique.
- Abdomen is thoroughly irrigated if indicated, haemostasis noted and the wound closed in layers

Laparoscopic Appendicectomy

Laparoscopic appendicectomy is to be offered only when facilities and expertise is available and only when the charges are acceptable to the patient and not a procedure to be recommended as the only option to the patient.

Position: supine (The bladder is emptied by a straight catheter or by having the patient void immediately before the procedure). Both the surgeon and assistant stand to the left side of the patient with the left arm tucked. Patient strapped to table to prevent sliding off during table tilts.

Anaesthesia: General

Access & Port placements:

- Open or Veress needle access
- Ports: Supra/infra/trans umbilical camera port and 2 working ports (5mm) typically in the left / right lower quadrant and one in either suprapubic, supra umbilical or right upper quadrant as per surgeons preference to allow optimum triangulation.

Common Steps:

- 30 degree telescope is used routinely used, a four-quadrant exploration is performed quickly and diagnosis confirmed.
- The patient is placed in reverse Trendelenburg position with right side up to gain exposure to the appendix and caecum.
- In a retrocecal appendix, sometimes the lateral peritoneal attachments, the white line of Toldt, must be divided to mobilize the cecum and expose the appendix.

- Atraumatic bowel graspers are used to elevate the appendix and inspect the base.
- Mesoappendix is carefully divided using the cautery (preferably bipolar) or harmonic scalpel and scissors after clipping or ligation.
- The base is then secured with endoloops and the appendix divided.
- Alternatively, the appendix may be divided with an endoscopic stapler (prefer to use a blue loadstapler in cases in which the entire appendix is friable to get a healthy base stapled close to the caecum)
- Appendix is usually retrieved in an endobag.
- The pelvis is irrigated, haemostasis noted, trocars are removed under vision, and the wounds are closed.
- Laparoscopic appendectomy may also be performed with single-site laparoscopic surgical techniques as well in expert hands.

Although the laparoscopic approach is increasingly becoming the procedure of choice in most patients, open appendectomy still remains the choice of treatment on a global basis.

Post Operative Care: No further need for antibiotics. Patient is usually discharged after passage of flatus and tolerating oral diet.

Common Complications of Appendicectomy

- Bleeding
- Ileus
- Bowel obstruction
- Stump leakage
- Stump appendicitis
- Intra-abdominal abscess
- Wound infection

B. Nonoperative Management of Uncomplicated Appendicitis

- Current best practice at this time for uncomplicated appendicitis is prompt appendectomy either by open or by laparoscopic approach. This concept has been challenged with non-operative management of acute appendicitis and is evolving.
- If Non-operative management is chosen for some reason, the surgeon must remain extremely vigilant.
- Serial examinations and imaging are necessary to monitor for treatment failure especially if there's an appendicolith.

- The rate of recurrence of appendicitis treated with antibiotics alone is shown to be 7% to 14% at 1 year from the indexed episode especially in children.
- Treatment is by IV antibiotics, analgesics, fluids and serially initiating enteral feeds depending on abdominal findings.
- In future, the algorithm may move towards less invasive approaches, however, at this time appendicitis is still a surgical disease

II. Management of Acute Complicated appendicitis:

- Delayed Presentation with septicemia
- Appendicular perforation
- Appendicular phlegmon or Mass
- Appendicular abscess

Patients with diffuse peritonitis are challenging to manage with a high morbidity and sometimes mortality as well. These patients can be managed with multidisciplinary approach eg:

- interventional radiologists inserting a percutaneous drain for an abscess or collection
- may require ICU admission
- In these cases, the treatment should be individualized on the basis of the nature of the presentation.
- In general, treatment for these patients is initially accomplished non-operatively. Fluid resuscitation is initiated, and broad-spectrum antibiotic therapy is initiated. A CT scan is obtained, and perforated appendicitis with a localized abscess or phlegmon is confirmed.
- Due to the extreme induration and friability of the involved tissues, Immediate exploration and attempted appendectomy in these patients may result in substantial morbidity, including
 - failure to identify the appendix,
 - postoperative abscess or fistula, and
 - unnecessary extension of the operation to include ileocectomy.
- If a localized abscess is identified,
 - Antibiotic therapy for 5-7 days
 - Drainage of abscess
 - CT-guided percutaneous drainage is performed for source control. The drainage catheter is typically left in place for 4 to 7 days
 - Laparoscopic drainage is another option that can be exceptionally useful.

This technique is performed by visualizing the inflammatory mass with the laparoscope and then entering the abscess with a laparoscopic suction tip, evacuating the purulent material, and placing a drain within the residual abscess cavity.

- Postoperative management is identical to that of patients who are successfully drained percutaneously.

- If an appendiceal phlegmon is present or if the amount of fluid present is not sufficient to drain, the patient may be treated with antibiotics alone, typically for 4 to 7 days also, as recommended by institutional guidelines for treatment of intra-abdominal infection.
- Interval appendicectomy can be performed based on risk assessment and for patients harbouring appendicolith. The current data is inclined towards an interval appendicectomy in children, however in adults the evidence in favour of interval appendicectomy is declining.

Referral Criteria:

- All patients with suspected appendicitis need to be referred for a Surgical Consultant and be managed by him.

ICU admission criteria:

- usually for complicated appendicitis
- ICU care may be needed in patients who present late with shock, septicemia or with perforation peritonitis and / or have other systemic illnesses.
- Patients with appendicitis who are not improving in spite of intra-venous antibiotics and/or require tertiary care including ventilator support.

MDT approach:

- Patients with complicated appendicitis requiring interventional radiologists support for abscess drainage or fecal fistula, intensivists for nutrition and sepsis management or experienced surgical team for further management.
- Pregnancy related management of acute appendicitis including need for MRI abdomen/pelvis.

Who does what?

Doctor:

Surgeon:- (Surgical team –PG's , Registrar's, Consultants)

- Diagnosis & Work up
- Pre operative planning
- Operative procedure
- Peri operative care in conjunction with Anaesthetist / Intensivist
- Post operative follow up

Anesthetist:- Pre Anaesthesia Check up
 Part of resuscitation and stabilization
 Performing anesthesia
 Post op ICU management in conjunction with Surgeon

Nurse:-(OT , ICU, ward & OPD) Pre/Intra/Postop comprehensive care
 Dressing of the wound

OT Technician:- Pre op equipment and drugs to be checked and kept ready
 Assist anesthetist in the OT
 Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (Patient weight -approx 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG-if justifiable clinically
8. X- Ray – Chest- if justifiable clinically
9. Histopathology

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments (open and/or Laparoscopic)
6. Suction (open and/or laparoscopic)
7. Sutures / endoloops / stapler
8. Drains
9. Catheters
10. Cautery – a basic set (monopolar and/or bipolar) or harmonic shears
11. Antibiotics
12. Analgesics
13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically or open surgery as deemed appropriate by the Surgeon.

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Acute Cholecystitis

Acute Cholecystitis is the acute inflammation of the gall bladder and is associated in majority of cases with gall stones (90%) and without gall stone in 10% of the cases

Pathogenesis

Calculous - Stone causing obstruction of the Hartmann's pouch or cystic duct.

Acalculous – Common in patients who have undergone major trauma, burn, sepsis.

Causative bacteria- E. Coli (most common), Klebsiella, Pseudomonas, Proteus, Streptococcus Faecalis, Salmonella, Clostridium Welchii.

Symptoms

- Right upper quadrant or epigastric pain which may radiate to the back
- Pain is often dull and constant, may be colicky
- There may be dyspepsia, flatulence, intolerance to fatty food
- Biliary colic presents as severe right upper quadrant pain that is associated with nausea and vomiting and may radiate to the chest, may last for a few minutes to several hours. Pain starts at night, awakening the patient.

Signs

- Fever (may be present)
- Tenderness – Right upper quadrant. Positive Murphy's sign
- Palpable tender mass in right upper quadrant of abdomen (may be present)
- Tachycardia
- Jaundice (may be present)

Investigations

- Haemogram– Leucocytosis, Raised CRP
- Liver function tests - deranged
- Blood sugar
- Urea / Creatinine- if needed
- Prothrombin Time
- Chest X-ray
- ECG
- Echocardiogram – In selected cases

- USG examination of abdomen – Gall bladder distended, wall thickened, oedematous, with or without pericholecystic fluid. Gall stones present in 90% of the cases. Pooled sensitivity and specificity of USG in the diagnosis of gallstones were 84 % and 99 %
- MRCP – In case of concomitant jaundice
- CECT abdomen – In case of complications
- Hepatobiliaryiminodiacetic acid scan (HIDA scan) has the highest sensitivity and specificity for acute Cholecystitis, although its scarce availability, long time required to perform the test, and exposure to ionizing radiation limit its use (LoE 2 GoRB)

Diagnosis

Combining clinical, laboratory and imaging investigations is recommended, although the best combination is not yet known (LoE 4 GoRC)

Differential Diagnosis

Common

- Acute appendicitis
- Perforated peptic ulcer
- Acute pancreatitis

Uncommon

- Acute pyelonephritis
- Acute myocardial infarction
- Pneumonia – Right lower lobe

Complications

- Mucocele
- Empyema of the gall bladder
- Perforation
- Obstructive Jaundice
- Acute Pancreatitis
- Acute Cholangitis
- Intestinal Obstruction due to Gall Stone Ileus

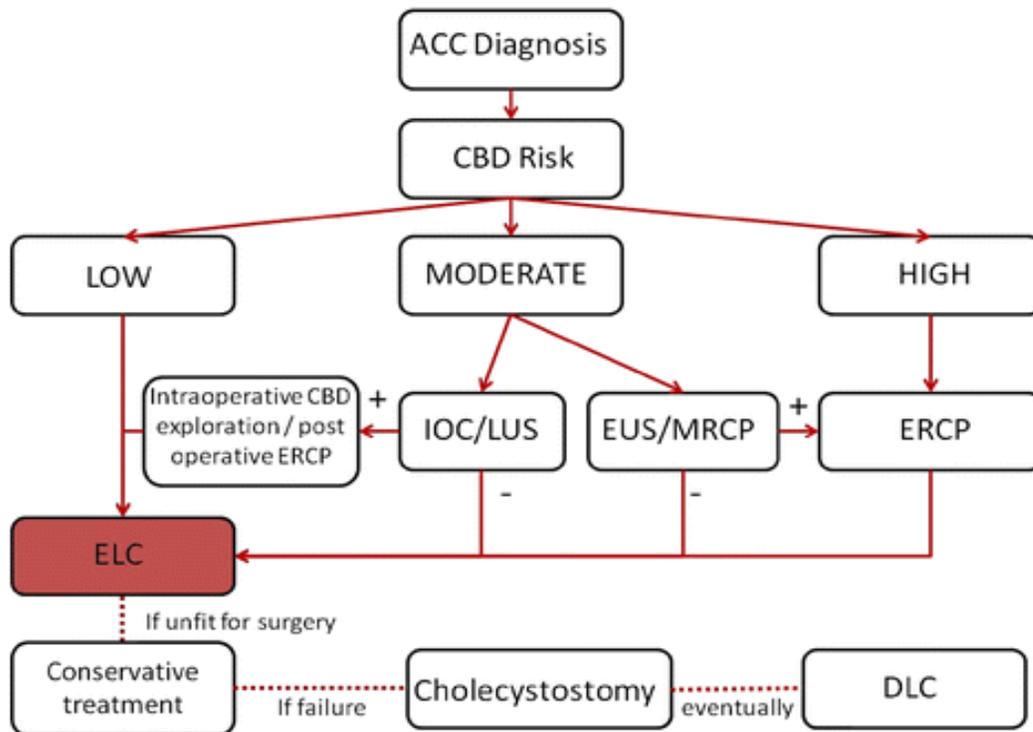
Risk Stratification

Patient's age above 80 in ACC is a risk factor for worse clinical behaviour, morbidity and mortality (LoE 3 GoR B)

The co-existence of diabetes mellitus does not contraindicate urgent surgery but must be re-considered as a part of the overall patient comorbidity (LoE 3 GoR C)

Currently, there is no evidence of any scores in identifying patient's risk in surgery for ACC

Treatment



Comprehensive algorithm for the treatment of Acute Calculous Cholecystitis

ACC: Acute Calculous Cholecystitis

CBD: Common Bile Duct

DLC: Delayed Laparoscopic Cholecystectomy

ELC: Early Laparoscopic Cholecystectomy

ERCP : Endoscopic Retrograde Cholangio Pancreatography

EUS : Endoscopic Ultrasound

IOC : Intraoperative Cholangiography

LUS: laparoscopic ultrasound

MRCP: Magnetic Resonance Cholangiopancreatography

The treatment for acute Cholecystitis is according to aforementioned algorithm.

Conservative treatment

Nil per mouth, IV fluid, analgesics, broad spectrum antibiotics – effective against gram negative aerobes (E.g.- Cephazolin, Cefuroxime, Gentamicin), regular monitoring of the temperature, pulse and other physical signs of the patient to assess the response to the conservative treatment. Once the pain has subsided, and the temperature and pulse have become normal, the patient can be fed orally and be sent home and kept on regular follow up, and taken up for cholecystectomy after 3 to 6 weeks (Delayed laparoscopic cholecystectomy)

Types of Surgical Approach-

-In ACC, a laparoscopic approach should initially be attempted except in case of absolute anaesthesiology contraindications or septic shock (LoE 2 GoR B)

- Laparoscopic cholecystectomy for ACC is safe, feasible, with a low complication rate and associated with shortened hospital stay (LoE 1 GoR A)

- Among high-risk patients, in those with Child A & B cirrhosis, advanced age >80, or pregnant women, laparoscopic cholecystectomy for ACC is feasible and safe (LoE 3 GoR C)
- Laparoscopic or open subtotal cholecystectomy is a valid option for advanced inflammation, gangrenous gallbladder, or any setting of the “difficult gallbladder” where anatomy is difficult to recognize and main bile duct injuries are more likely (LoE 2 GoR A)
- In case of local severe inflammation, adhesions, bleeding in Calot’s triangle or suspected bile duct injury, conversion to open surgery should be strongly considered. (LoE 3 GoR B)

Timing of Surgery

- Early laparoscopic cholecystectomy is preferable to delayed laparoscopic cholecystectomy in patients with Acute Cholecystitis as long as it is completed within 3 days of onset of symptoms (Level 1 Evidence; Grade A recommendation)
- Laparoscopic cholecystectomy should not be offered for patients beyond 10 days from the onset of symptoms unless symptoms suggestive of worsening peritonitis or sepsis warrant an emergency surgical intervention. In people with more than 10 days of symptoms, delaying cholecystectomy for 45 days is better than immediate surgery (LoE 2 GoR B)

Associated common bile duct stone: suspicion and diagnosis at the presentation

- Elevation of liver biochemical enzymes and/or bilirubin levels is not sufficient to identify ACC patients with choledocholithiasis and further diagnostic tests are needed. (LoE 2 GoR B)
- At AUS, the visualization of CBDS is a very strong predictor of choledocholithiasis. (LoE 5 GoR D). Indirect signs of stone presence such as increased diameter of common bile duct are not sufficient to identify ACC patients with choledocholithiasis and further diagnostic tests are needed. (LoE 1 GoR A)
- Liver biochemical tests, including ALT, AST bilirubin, ALP, gamma glutamyltransferase (GGT), AUS should be performed in all patients with ACC to assess the risk for CBS. (LoE 2 GoR B)
- Common bile duct stone risk should be stratified according to the proposed classification, modified from the American Society of Gastrointestinal Endoscopy and the Society of American Gastrointestinal Endoscopic Surgeon Guidelines (LoE 5 GoR D)
- Patients with moderate risk for choledocholithiasis should undergo preoperative MRCP, EUS, intraoperative cholangiography, or Laparoscopic ultrasound depending on the local expertise and availability. (LoE 1 GoR A)
- Patients with high risk for choledocholithiasis should undergo preoperative ERCP, intraoperative cholangiography, Laparoscopic ultrasound, depending on the local expertise and the availability of the technique. (LoE 1 GoR A)
- CBDS could be removed preoperatively, intraoperatively, or postoperatively according to the local expertise and the availability of the technique. (LoE 1 GoR A)

Alternative treatments for high risk patients

- Gallbladder drainage, together with antibiotics, converts a septic Cholecystitis into a non-septic condition; however, the level of evidence is poor (LoE 4, GoR C)
- Among standardized gallbladder drainage techniques percutaneous trans hepatic gallbladder drainage (PTGBD) is generally recognized as the preferred technique due to the ease and the reduced costs. (LoE 4, GoR C)

- PC could be considered as a possible alternative to surgery after the failure of conservative treatment in a small subset of patients unfit for emergency surgery due to their severe co-morbidities (LoE 2 GoR B)
- Delayed laparoscopic cholecystectomy could be offered to patients after reduction of operative and anaesthesiology related risks to reduce further hospitalization (LoE 5 GoR D)

Post-Operative Care

Antibiotic Therapy-

- Patients with uncomplicated Cholecystitis can be treated without post-operative antibiotics when the focus of infection is controlled by cholecystectomy.(LoE 1 GoR B)
- In complicated acute Cholecystitis, the antimicrobial regimens depend on presumed pathogens involved and risk factors for major resistance patterns. (LoE 3 GoR B)
- The results of microbiological analysis are helpful in designing targeted therapeutic strategies for individual patients to customize antibiotic treatment and ensure adequate antimicrobial coverage in patients with complicated Cholecystitis and at high risk for antimicrobial resistance. (LoE 3 GoR C)

Referral Criteria

ICU care may be needed in patients who present late with severe sepsis and have other systemic illnesses.

Medico legal Issues

- Failure to diagnose and institute immediate proper treatment

Who does what?

Surgeon- Establishing the diagnosis and working up the patient

- Follow the abovementioned treatment algorithm
- Post-operative care and follow up

Anaesthetist-Pre Anaesthetic work up

- Anesthetizing patient during surgery and post op management in critical patients

Nurse - Pre, Intra and Post op care

Technician-Pre op equipment and drugs to be checked and kept ready

- Assist the anaesthetist in the OT
- Assist the surgeon, positioning of patient

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anaesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test - in selected cases
4. LFT
5. S. Electrolytes in selected cases
6. USG
7. ECG
8. Echocardiogram – in selected cases
9. X- Ray – Chest
10. MRCP – in selected cases
11. CECT – in selected cases
12. HIDA scan – selected cases
13. Histopathology- following surgery

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anaesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains

9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as decided by the Surgeon.

Abbreviations

CBDS - common bile duct stones
GoR - Grade of Recommendation
IOC - Intraoperative cholangiography
LC- Laparoscopic cholecystectomy
LoE - Level of Evidence
LUS - Laparoscopic ultrasound;
PC - Percutaneous Cholecystostomy

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Acute Limb Ischemia

What is acute limb Ischemia

1. Symptoms less than 2weeks
2. Due to the lack of blood perfusion of extremities
3. Always risk of limb loss and life loss
4. Needs active intervention

How to diagnose?

Classical Symptoms are six Ps -pain, pallor, paralysis, pulse deficit, paresthesia, and poikilothermia.

Pain- constant or elicited by passive movement.

History – duration, site, intensity and rapidity

- Claudication, previous vascular procedures like bypass, cardiac symptoms like arrhythmias, angina, rheumatic fever, and aortic aneurysms
- smoking, hypertension, diabetes, hyperlipidemia,
- enquire for any history suggestive of any vasospastic diseases and vascular interventions

Clinical examination

Pulse – Irregular in atrial fibrillation, level of pulse deficit indicate site of occlusion. Absence of pulse is mandatory.

Pallor and poikilothermia - indicate level of occlusion and progression indicates progression of thrombus

Absence of sensation is diagnostic and progression of disease

Complete paralysis with no sensation – irreversible damage and need of amputation

Look for any indwelling catheters including venous or arterial catheters or cannulas.

Auscultate for any murmur

Investigations

Blood investigations

1. Blood Hb, TC, DC and platelet
2. ESR

3. Blood sugar
4. Renal profile
5. ECG

Haemoglobin and Packed cell volume (PCV), will be raised in patients with secondary polycythemia secondary to lung diseases.

Renal functions tests (S. Creatinine and Urea): To assess a baseline renal function and also to pick up any undiagnosed chronic kidney disease. Altered renal parameters would guide surgeon to decide on the type of investigation and to limit use of contrast in the subsequent imaging.

ECG: A baseline ECG to look for abnormalities in cardiac rhythm like atrial fibrillation and to rule out an acute myocardial infarction. ALI may be the presenting feature of an acute coronary event, and this would also guide surgeon to decide the type of treatment in a particular patient, based on his cardiac co-morbidities.

Work up for thrombosis

1. Bleeding time and clotting time
2. Platelet count
3. Total count and Hb
4. Serum Homocysteine

Diagnostic Work up

1. Doppler evaluation
2. CT angiogram (if diagnostic dilemma or planning for revascularisation)
3. Echocardiogram
4. USG abdomen – to rule out abdominal aortic aneurysm

Doppler: A bedside hand held Doppler assessment to look for arterial and venous signals at the ankle should be done to categorize patient in to Rutherford class of ALI.

Ultrasound scan: A rapid bedside ultrasound can pick up the level of occlusion and state of distal arteries. Presence of calcium and plaques in the arterial wall would point to an underlying chronic atherosclerotic disease. An expert sonologist can also differentiate between and acute and chronic intra-luminal thrombus. Abnormalities like an underlying thrombosed popliteal aneurysm and arterial dissection flaps causing acute ischemia can also be picked up by an US scan.

CT angiography (CTA): CTA is the mainstay of investigations, provides a good road map in a case of ALI and should be done prior to surgical management if available. CTA being a non-invasive has largely replaced conventional angiography in the initial evaluation of patients with ALI. CTA might pick up ulcerated aortic plaque, a proximal aortic thrombus or an aortic dissection as an aetiology of acute ischemia. Patients should be adequately hydrated prior to the CT imaging to prevent contrast induced nephropathy.

Conventional angiography: This was the gold standard investigation, but has largely replaced by other non-invasive modalities for the initial evaluation ALI. Being an invasive modality, this is preferred investigation in patients in whom a catheter based treatment like thrombolysis is being planned in a setting of a hybrid operating suite. Disadvantages are invasive nature and difficult to visualise distal run-off in the setting of acute thrombosis due to lack of collaterals and presence of spasm unlike in a chronic occlusion. Non-visualisation of distal run-off in a catheter angiogram should not preclude operative management in ALI and in a salvageable limb it is worthwhile exploring distal arteries in such clinical scenarios.

Magnetic resonance imaging is very time consuming and is prone to movement artifacts and does not seem to have much role evaluation of a patient with ALI.

Echocardiography should be done as a part of evaluation, but is not mandatory prior to definitive management. Non-visualisation of thrombus in the heart does not necessarily rule out a cardiogenic source of embolus. Also irrespective of presence or absence of thrombus in the heart most patients are anti coagulated for life after an event of ALI.

Consultations

1. Anaesthesia
2. Cardiologist regarding any possible source
3. Radiologist

Severity based on

1. Degree of obstruction
2. Site of occlusion
3. Collaterals
4. Tissue involved- Muscle -4 hours
 - Nerve 8 hours
 - Fat 13 hours
 - Skin 24 hours
 - Bone 4 days

Classification

category	Description/prognosis	Sensory loss	Muscle weakness	Arterial	venous
1	No immediate threat	None	none	present	audible
11A	Salvageable if urgently treated	Minimal	none	absent	audible
11B	Salvageable if urgently revascularised	Moderate with rest pain	moderate	absent	audible
111	Non salvageable	Profound anesthetic	profound	absent	absent

Medical Management

Heparin

Systemic anticoagulation with heparin should be administered unless contraindicated. Unfractionated heparin is the agent of choice -100 units per kg bolus on diagnosing the condition – to arrest the process and to prevent the propagation of thrombus, followed by 18mg/kg/hour. Maintenance infusion is to be started only if no immediate surgery is planned. Then continue infusion at the dose of 1000unit per hour.

Monitoring of Heparin- Serum fibrinogen, Platelet count, APTT

Choice of interventions

Accepted treatments for prompt revascularization consist of catheter-directed thrombolysis (CDT), percutaneous mechanical thrombus extraction with or without thrombolytic therapy, and surgical thrombectomy or bypass.

Patients with limb-threatening ischemia are not candidates for local fibrinolysis, which usually takes between 6 and 72 hours to achieve clot lysis. These patients require emergency embolectomy. CDT is reserved for patients with non-life-threatening limb ischemia due to in-situ thrombosis of less than 14 days' duration. Consider that patients with thrombosis of more than 30 days' duration are not likely to respond to local fibrinolysis.

Thrombolysis

Agents

Streptokinase is produced by beta-hemolytic streptococci. By itself, it is not a plasminogen activator, but it binds with free circulating plasminogen (or with plasmin) to form a complex that can convert additional plasminogen to plasmin. Because streptokinase is produced from streptococcal bacteria, it often causes febrile reactions and other allergic problems. It can also cause hypotension that appears to be dose-related. Streptokinase usually cannot be administered safely a second time within 6 months, because it is highly antigenic and results in high levels of antistreptococcal antibodies. Hence it is not a preferred agent now except its cost.

Urokinase is the fibrinolytic agent most often used for peripheral intravascular thrombus and occluded catheters. Urokinase is a thrombolytic agent that is produced in renal parenchymal cells. Unlike streptokinase, urokinase directly acts on plasminogen to produce plasmin. Urokinase is also produced by tissue culture from *E coli* cultures. In plasma, urokinase has a half-life of approximately 20 minutes. Allergic reactions are rare, and the agent can be administered repeatedly without antigenic problems.

Tissue plasminogen activator (tPA) is a naturally occurring fibrinolytic agent found in vascular endothelial cells. It exhibits significant fibrin specificity and affinity, which include alteplase (tPA), reteplase (recombinant plasminogen activator [r-PA]), and tenecteplase. Fibrinolytic agents can be administered systematically or can be delivered directly into the area of the thrombus. Peripheral arterial thrombi are most often treated via a catheter-directed approach.

The choice of a lytic agent must be based both on the results of ongoing clinical trials and on the clinician's experience. The most appropriate agent and regimen for each clinical situation will change over time and may differ from patient to patient.

Indication: Catheter-based thrombolysis is effective for patients with ALI and a salvageable limb. In patients with ALI with a salvageable limb, percutaneous mechanical thrombectomy can be useful as adjunctive therapy to thrombolysis.

Dosage of thrombolysis -The standard regimen for reteplase in PAD consists of 0.5 U/hr by intra-arterial infusion.

The standard regimen for alteplase consists of 0.05-0.1 mg/kg/hr intra-arterially. The high-dose regimen consists of three doses of 5 mg over 30 minutes followed by 3.5 mg/hr for up to 4 hours.

The regimen for urokinase consists of 4000 U/min intra-arterially until initial recanalization, then 1000-2000 U/min intra-arterially until complete lyses.

Indications of conversion

1. Progression of thrombus
2. Worsening of the limb
3. Evidences of bleed from other sites
4. Intolerant to thrombolysis
5. Myoglobinuria

Thrombectomy

Surgical thrombectomy is still the mode of treatment of acute limb in many indications. Fogarty catheter is the main device required for the same.

Instruments required for the thrombectomy

1. Fogarty's catheter of different sizes. It ranges from number 1 to 5. Number 1 is the smallest.
2. Curved femoral clamp and bull dog vascular clamps
3. 2 self retaining mastoid retractors
4. No 11 knife for arteriotomy
5. Castroviejo vascular needle holder
6. Fine tipped vascular forceps
7. Metzenbaum fine scissors
8. Potts scissors
9. Regular instruments for surgical dissection

Procedure

Patient is put in supine position. This can be done under GA, SAB or local anesthesia. Drape the groin with both femoral areas to be exposed. Longitudinal incision along the mid inguinal point. (Curvilinear incision can also be done). Femoral sheath is opened longitudinally. Artery is dissected out and takes distal and proximal control. All the branches around are looped and facilitate clamping of all all branches with clamps, vascular tapes or thick silk. This is to avoid bleeding during the procedure and migration of thrombus during embolectomy. Identify the

instruments and sutured available on the table. Feel the opposite femoral pulse to make sure that it is normal. Inform the anaesthetist regarding clamping. Heparinise the patient with 1mg/Kg body weight. If the patient is already heparinised, reload according to the ACT value if facilities available or half dose heparin if facilities not available. Clamp all the vessels from proximal to distal sequence. Do a horizontal arteriotomy preferably half the diameter of the vessel. Arteriotomy is initiated with no11 knife and then complete with Potts scissors.

Check the balloons of the Fogarty catheter and identify the maximum volume to inflate the balloon. Remove the stylet of the catheter and introduce it proximally. Once you are sure that it has passed beyond the embolus or thrombus inflate the balloon and slowly pull back with the balloon inflated. Remove the thrombus completely. If you are not happy attempt it once again. Multiple attempts may damage the endothelium and make it more thrombogenic.

Repeat the same procedure distally also. Distally make sure that the catheter has gone to all three branches – posterior tibial, anterior tibial and peroneal. If the back flow from the profunda is poor attempt the same in profunda also. Remove the clamps from each vessel also to make sure that the back flow is good. If the flow is poor distal we can infuse steroids, mannitol and heparin through a catheter. Arteriotomy can be closed with interrupted or continuous sutures. If facilities available- cath lab, hybrid theatre or a C arm – we can do a check angiogram at this stage to identify and recording of the procedure.

Opposite femoral pulse is palpated and make sure that it is normal. If it is absent it may be due to the migrated embolus and the same step has to be repeated in that side also.

Shift the patient to ICU, monitor the cardiac function and perfusion status.

Clot has to be sent for microbial culture and histopathological analysis.

Fasciotomy

Fasciotomy is the opening of the deep fascia to reduce the muscular compartment pressure.

Indications

1. Delayed revascularisation
2. Doubtful about the vascularity
3. Poor texture of muscles
4. Worsening of limb after the revascularisation though it is early
5. Increased compartmental pressure more than 40mms of Hg
6. Oedema of the distal limb

If there is any doubt of vascularity better go for fasciotomy

Procedure

Longitudinal skin incisions are made over the anterior, lateral and posterior compartments up to deep fascia. . Deep fascia is opened with a Metzenbaum scissors, preferably under direction up to the proximal and distal joints. Make sure that fascia is well opened and muscle is relaxed and bulged out. Observe the colour of the muscle. Wound can be kept open or skin can be closed if it doesn't cause any tension.

Revascularisation

Indications

1. Acute on chronic occlusion
2. Failure of embolectomy
3. Lack of improvement or worsening of limb after embolectomy

Depending on the length, site and type of occlusion and general condition of the patient we can opt for endovascular or surgical options. This includes angioplasty, stenting of the lesion and various bypass procedures.

Amputation

Amputation may be the option in a considerable group of patients. They are indicated in class3 Limbs in the initial stage itself, worsened limb after the procedures, worsening of the general condition, and in life threatening conditions like sepsis, acute renal failure etc. Amputation should be planned well depending upon the proximal perfusion pressure and future rehabilitation.

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Acute Mesenteric Ischaemia

Introduction

Acute mesenteric ischemia is an abdominal emergency associated with high rates of morbidity and mortality. A review of literature reveals very few studies from India (1, 2). Therefore, true magnitude of AMI in Indian population is unclear. With increasing awareness and increase in older population, AMI is being more often diagnosed thereby contributing to the perception that the incidence of AMI is on the rise.

Classification

Based on the type of occlusion AMI is classified as follows –

- Acute mesenteric arterial occlusion
- Mesenteric venous thrombosis
- Non-occlusive mesenteric ischemia

Acute mesenteric arterial occlusion

This is the commonest type of AMI. Acute mesenteric arterial occlusion could be due to embolus or thrombosis. Embolism to mesenteric arteries is more often encountered than thrombosis. Such emboli can originate from heart or aorta. Thrombosis causing mesenteric ischemia is usually an acute on chronic thrombosis. Both thrombosis and embolism involve superior mesenteric artery. This subtype of AMI is encountered among patients with significant cardiac comorbidities.

Mesenteric venous thrombosis

The basic pathophysiology in mesenteric venous thrombosis is occlusion of mesenteric veins resulting in congestion and subsequent ischemia of corresponding segment of bowel. Mesenteric venous thrombosis is encountered in two subsets of patient populations. The first category comprises of patients in procoagulant states. Eg. Deficiency of protein C, protein S and antithrombin III. The second category includes patients with low flow splanchnic circulation (eg. Portal hypertension) or regional inflammatory conditions (eg. Pancreatitis). Often mesenteric venous thrombosis extends as portal vein thrombosis.

Non-Occlusive mesenteric ischemia (NOMI)

In NOMI the basic pathology is spasm of arterioles. Such spasm could be either a result of sympathetic response to cardiogenic/hypovolemic/septic shock or induced by drugs like

inotropes. Most of the affected patients will be critically ill being treated in ICU. Their poor general condition may hamper the elicitation and interpretation of symptoms and signs.

Clinical Features

Clinical presentation of AMI is very non-specific. Most patients present with acute onset of severe abdominal pain. Such acute onset of abdominal pain may be associated with nausea, vomiting and urge to defecate (3). Few studies (4,5) corroborate the symptom of **“acute abdominal pain associated with sudden passage of stools in AMI”** as described by Klass (6). The classical teaching of **“symptoms being out of proportion to signs in AMI”** has stood the test of time. The findings on physical examination of patients with AMI are deceptively minimal. Clinical features like fever, shock, progression of abdominal pain to a more severe/diffuse type and bloody diarrhea are suggestive of progression of bowel ischemia to infarction (3). Although the clinical features of AMI are non-specific, such clinical features in a patient with co-morbidities like ischemic heart disease, atrial fibrillation, hypertension, diabetes mellitus or renal insufficiency should alert the treating doctor towards the possibility of AMI

Investigations

There are no laboratory tests which can be employed for the early detection of AMI (3). Commonly employed laboratory parameters which can be deranged in AMI include, hematocrit, white cell counts, amylase levels, serum lactate levels, aspartate aminotransferase, lactate dehydrogenase and creatinine phosphokinase. However none of these tests are either sensitive or specific to AMI. When subjected to serial monitoring, these tests can point out the progression of AMI.

The single most sensitive and specific imaging modality for AMI is biphasic multidetector computed tomography (MDCT) with intravenous contrast (3, 7). The findings in MDCT suggestive of AMI include –

- Occlusion of mesenteric vessels.
- Diminished enhancement of bowel wall, pneumatosis intestinalis and gas in portal vein radicals – all of which are suggestive of bowel gangrene.
- Bowel wall edema with ascites is likely to be a feature of mesenteric vein thrombosis

MDCT might not be helpful in diagnosis of non-occlusive mesenteric ischemia (NOMI). The only investigation which can pick up NOMI is selective mesenteric angiography which shows –

- Narrowing of multiple branches of superior mesenteric arteries
- Impaired filling of intramural vessels
- String of lakes appearance of run off vessels

Treatment

Effective management of AMI can be aptly summarized as 3 “R” s namely- rapid diagnosis, resuscitation and early revascularization (3). Following are the general principles for managing AMI –

- Restore the circulating volume which prevents further hypo perfusion of bowel (3).
- Address and optimize co-morbidities. This would be a team effort requiring inputs from other specialties like cardiology and nephrology.
- Avoid vasopressors whenever possible (3).
- AMI compromises mucosal barrier thereby facilitating bacterial translocation. Hence it is recommended to start broad spectrum antibiotics
- Prompt laparotomy should be considered if there are any signs of peritonitis / bowel infarction.
- When such laparotomies are performed, principles of “Damage control surgery” should be adopted (3, 8, 9, 10, 11), which comprises of resection of evidently gangrenous bowel, deferring anastomosis and scheduling a second look laparotomy in 48 hours (12). In the time interval between two surgeries, aggressive resuscitative measures should be continued.

Specific treatment measures for each type of AMI are as follows –

AMI due to arterial embolization

The key factor in decision making is the presence or absence of bowel gangrene. Any clinical /radiological suspicion of bowel infarction mandates damage control laparotomy. In stable patients with established diagnosis and no evidence of bowel infarction the choice between open and endovascular embolectomy can be made depending upon the experience and expertise of the treating team. Such endovascular interventions include percutaneous aspiration (14), thrombolysis (14,15) and percutaneous transluminal angioplasty with or without stenting (16,17).

AMI due to arterial thrombosis

Where expertise is available endovascular treatment is the approach of choice for arterial thrombosis accounting for AMI. The endovascular techniques that are employed include percutaneous transluminal angioplasty (PTA) and stenting, percutaneous aspiration thrombectomy, local fibrinolysis or intra arterial drug perfusion (e.g. Heparin or papaverine).

Where surgery is required to resect gangrenous bowel, following options may be considered –

- Retrograde open mesenteric stenting (ROMS) (18)
- Conventional bypass surgery (19) which may be antegrade or retrograde with vein or synthetic grafts

When expertise for vascular intervention / surgery is not available, it may be reasonable to perform damage control surgery and refer the patient to a center where expertise for vascular intervention is available.

Non-Occlusive mesenteric ischemia (NOMI)

The objective of treating NOMI should be to relieve mesenteric vasospasm/vasoconstriction and restore circulating fluid volume. Relieving vasospasm in NOMI by selective intra-arterial infusion of vasodilators (3, 19) is the key treatment. The commonly used vasodilators are –

- Prostaglandin E1 - administered as 20 mcg bolus followed by 60-8- mcg / 24 h infusion.
- Alternatively papaverine (30-6- mg/h) can be used.

Patients with NOMI are generally poor risk patients for surgery (when indicated for bowel gangrene). Such patients considered unsalvageable should be considered for palliative care (3).

AMI due to mesenteric vein thrombosis

Venous thrombosis causing AMI can be isolated superior mesenteric vein (SMV) thrombosis or superior mesenteric vein with portal vein thrombosis. Patients with isolated SMV thrombosis fare well. Systemic anticoagulation with unfractionated / low-molecular weight heparin is the cornerstone of treatment of such patients. Patients who deteriorate while on systemic anticoagulation may be considered for endovascular interventions like –

- Trans-jugular intrahepatic porto-systemic shunting
- Percutaneous transhepatic thrombolysis
- Catheter directed thrombolysis

Few studies (20) have suggested favorable short term and long-term outcomes with thrombolytic therapy. Absolute contraindications for thrombolysis include central nervous system tumors, recent hemorrhagic stroke, gastro intestinal bleeding and uncontrolled hypertension. Pregnancy, remote history of GI bleeding and recent major surgery are relative contraindications for thrombolytic therapy.

Summary

- Diagnosis of AMI requires high degree of suspicion. Severe abdominal pain which is out of proportion to the clinical signs in a high-risk patient should alert the treating doctor regarding possibility of AMI
- There are no reliable laboratory investigations for early diagnosis of AMI
- Multi detector Computed tomography with intra venous contrast is the imaging modality of choice in suspected cases of AMI
- Resuscitation and restoration of circulating fluid volume are of paramount importance.
- Damage control laparotomy is recommended when bowel gangrene is strongly suspected.
- Open / endovascular embolectomy is required in AMI due to embolus
- Endovascular approach is the preferred line of treatment in arterial thrombosis
- Intra arterial prostaglandin E1 / Papaverin infusion is the treatment of choice for NOMI without bowel gangrene
- Systemic anticoagulation forms the cornerstone of treatment of mesenteric vein thrombosis

Referral Criteria

ICU care may be needed in patients who present late with severe sepsis and have other systemic illnesses.

Medico legal Issues

- Failure to diagnose and institute immediate proper treatment

Who does what?

Surgeon	- Establishing the diagnosis and working up the patient -Follow the above mentioned treatment algorithm -Post-operative care and follow up
Radiologist	-USS, CT, & Intervention if needed
Anaesthetist	-Pre Anaesthetic work up -Anesthetizing patient during surgery and post op management in critical patients
Nurse	-Pre, Intra and Post op care
Technician	-Pre op equipment and drugs to be checked and kept ready -Assist the anaesthetist in the OT -Assist the surgeon, positioning of patient

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1 - If trained can take up all interventions
2. Medical Officer / Assistant Surgeon – 1
3. Anaesthetist – 1
4. Radiologist – 1 with interventional experience.
5. Pathologist – 1---- Services from outside can be availed
6. Staff Nurse – 1
7. Technician – 1
8. Nursing Orderly – 1
9. Cleaning staff-1

Drugs & Consumables

1. OT Table & lights
2. Cath lab with guidewires, catheters, stents and other hard wares for endovascular manipulations.
3. Instrument trolley
4. Anaesthetic Machine, instruments including endotracheal tubes & drugs
5. Monitor
6. Set of surgical Instruments
7. Suction
8. Sutures
9. Drains
10. Catheters
11. Cautery – a basic set
12. Antibiotics
13. Analgesic
14. I.V. Fluids
15. Dressings
16. Diagnostic Laparoscopy may be attempted in select few cases when expertise and equipments are available.

Abbreviations used

AMI- Acute Mesenteric Ischemia

NOMI- Non-occlusive mesenteric ischemia

PTA- Percutaneous transluminal angioplasty

MDCT- multidetector computed tomography

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Acute pancreatitis

Introduction

Acute pancreatitis (AP) is an acute inflammatory condition of the pancreas leading to injury or destruction of acinar components and clinically characterized by abdominal pain and elevated blood levels of pancreatic enzymes. The clinical spectrum is as diverse as its causes and pathogenesis; AP can range from relatively mild to severe with potentially life-threatening complications.

Gallstones and alcohol account for approximately two thirds of the causes of AP with gallstones being the most common cause. Other causes are hyperlipidemia, hypercalcemia, post procedural (ERCP), viral, drug related and genetic. In a subset of patients, AP is idiopathic and the exact cause is not discerned even after comprehensive aetiological work-up.

While fortunately the majority of patients (80-85%) with AP have mild disease which usually recovers uneventfully, in a small subset of patients (15-20%) it is a severe, potentially life-threatening illness that can result in prolonged hospital admission and significant mortality. The management of pancreatitis requires early recognition of this high-risk subgroup and multidisciplinary care and management of complications under the supervision of specialists. Management is challenging and centers on diagnosing the etiology, assessing the severity, and treating the disease and its associated complications.

Indian Statistics

While exact incidence of AP in our country is unknown due to lack of national registry, the experience from leading academic departments (personal communication) seems to suggest that the incidence appears to be rising.

AP is the most common diagnosis for hospitalization amongst the gastrointestinal conditions in the United States, accounting for as many as 275,000 hospitalizations per year. The incidence is on an increasing trend world-wide during the past few decades and has ranged from approximately 15 to 42 per 100,000 population per year.

Clinical Presentation

The majority of patients with acute pancreatitis have acute onset of persistent, severe epigastric pain. In some cases the pain may be in left upper abdominal pain and at times it can be a diffuse abdominal pain. In patients with gallstone pancreatitis, the pain is usually well localized and the onset of pain is rapid, reaching maximum intensity in 10 to 20 minutes. In

contrast, in patients with pancreatitis due to other causes - alcohol or metabolic causes, the onset of pain is usually less abrupt and the pain may be poorly localized. In approximately 50 percent of patients, the pain radiates to the back. The pain persists for several hours to days and may be partially relieved by sitting up or bending forward. The majority of patients have associated nausea and vomiting which may persist for several hours.

Patients with severe acute pancreatitis may have dyspnea due to diaphragmatic inflammation secondary to pancreatitis, pleural effusions, or adult respiratory distress syndrome. Approximately 5 percent of patients with acute severe pancreatitis may have painless disease and have unexplained hypotension (eg, postoperative and critically ill patients, patients on dialysis, organophosphate poisoning).

Physical findings vary depending upon the severity of acute pancreatitis. In patients with mild acute pancreatitis, the epigastrium may be minimally tender to palpation. In contrast, in patients with severe pancreatitis, there may be significant tenderness to palpation in the epigastrium or more diffusely over the abdomen. Patients may have abdominal distention and hypoactive bowel sounds due to an ileus secondary to inflammation. Patients may have scleral icterus due to obstructive jaundice due to choledocholithiasis in case of gallstone pancreatitis or edema of the head of the pancreas. Patients with severe pancreatitis may have fever, tachypnea, hypoxemia, and hypotension. Presence of ecchymotic discoloration in the periumbilical region (Cullen's sign) or along the flank (Grey Turner sign) has been described in fair skinned individuals in severe AP and suggests presence of retroperitoneal bleeding. They are not seen in the context of the Indian population. AP can at times mimic an acute surgical abdomen with rigid abdomen and peritoneal signs. It may also be uncovered in critically ill patients as a cause of acute respiratory distress syndrome.

Atlanta Classification of AP

According to the revised Atlanta classification system of 2012 which is presently followed, AP is divided into two broad categories:

1. Interstitial edematous acute pancreatitis, which is characterized by acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis.
2. Necrotizing acute pancreatitis, which is characterized by inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis.

According to the severity, AP is divided into the following grades of severity:

1. Mild AP which is characterized by the absence of organ failure and local or systemic complications.

2. Moderately severe AP which is characterized by transient organ failure (resolves within 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours)
3. Severe AP which is characterized by persistent organ failure that may involve one or multiple organs.

Local complications of AP include acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection, and walled-off necrosis.

Investigations

Investigations in AP are mainly done for following reasons:

1. To confirm the diagnosis (exclude other possibilities)
 - a. Biochemical – serum Amylase/Lipase
 - b. Imaging – X-ray/ USG / CT scan / MRI abdomen
2. To predict the severity
3. To establish the etiology

Diagnosis

Diagnosis of AP is based on presence of two of the following three criteria.

1. Clinical presentation in accordance to AP
2. Biochemical – elevated levels of pancreatic enzymes; serum Amylase/ Lipase more than 3 times normal levels.
3. Imaging (US/CT/MRI) suggestive of AP

Routine use of CT scan of the abdomen is not necessary for diagnosis of AP in all cases. CT scan is needed in cases of suspicion of hollow viscus perforation/ mesenteric ischemia, in case of delay between onset of symptoms and presentation (where enzyme levels will have normalized) and sedated patients where clinical assessment may be fallacious.

Serum amylase in AP patients generally rises within a few hours after the onset of symptoms and returns to normal values within 3 – 5 days. It is hence important to infer the serum amylase levels in context with the time duration since the onset of symptoms and conduct of the test. Serum amylase concentrations may not be significantly elevated in alcohol-induced AP especially after previous attacks and hypertriglyceridemia (true values are apparent after diluting the serum). There is no relation between severity of AP and amylase levels. The normalization of serum levels has no prognostic relevance.

Serum amylase concentrations might be high in the absence of AP in macroamylasaemia (a syndrome characterized by the formation of large molecular complexes between amylase and abnormal immunoglobulins), in patients with decreased glomerular filtration rate, in diseases of the salivary glands, and in extrapancreatic abdominal diseases associated with inflammation, including acute appendicitis, cholecystitis, intestinal obstruction or ischemia, peptic ulcer, and gynecological diseases although levels may not be as high as in AP. Serum lipase appears to be more specific and because of a longer half life levels remains elevated longer than amylase after disease presentation. Hence serum lipase should be preferred over amylase.

Role of imaging

Abdominal X-ray (AXR) and chest X-ray (CXR) — The radiographic findings in AP range from unremarkable in mild disease to localized ileus of a segment of small intestine (sentinel loop) or the colon cut off sign in more severe disease . The colon cut off sign reflects a paucity of air in the colon distal to the splenic flexure due to functional spasm of the descending colon secondary to pancreatic inflammation. Approximately one-third of patients with acute pancreatitis have abnormalities visible on CXR such as elevation of a hemidiaphragm, pleural effusions, basal atelectasis, pulmonary infiltrates, or acute respiratory distress syndrome. Presence of pleural effusion is one of the parameter of BISAP criteria and may indicate severe disease. AXR and CXR are quite unreliable and contribute little to the diagnosis of AP. The utility of AXR is mainly to rule out other common surgical causes of acute abdominal pain – perforation (AXR showing gas under diaphragm) or intestinal obstruction (AXR showing air-fluid levels).

Ultrasonography (USG) of the abdomen - On Abdominal USG in patients with AP, the pancreas appears diffusely enlarged and hypoechoic. Gallstones may be visualized in the gallbladder or the bile duct. Peripancreatic fluid appears as an anechoic collection. These collections may demonstrate internal echoes in the setting of pancreatic necrosis. However, in approximately 25 to 50 percent of patients with acute pancreatitis, bowel gas due to an ileus precludes evaluation of the pancreas. In addition, USG cannot clearly delineate extrapancreatic spread of pancreatic inflammation or identify necrosis within the pancreas. The main role of USG abdomen in AP is to diagnose gallstones and dilated intrahepatic bile ducts which can indicate bile duct obstruction. It is also helpful as a fairly inexpensive and non-invasive modality for follow-up evaluation of pancreatic fluid collections.

CT scan - CT scan is not necessary for the diagnosis of AP in the majority of cases. The indications for a CT scan in AP are selective in the following situations:

- 1) Diagnostic uncertainty – sedated patients, clinical suspicion of perforation/ mesenteric ischemia. In these cases CT scan is usually done at admission

- 2) To confirm severity of AP based on clinical predictors suggestive of severe AP
- 3) Patients who fail to respond to conservative treatment
- 4) To select the appropriate modality and approach in patients who need intervention

In these cases (Points 2 and 3) CT scan is usually done after at least 4- 5 days of onset of symptoms since the complete extent of necrosis is manifest after 96 hours and an early CT may underestimate the severity of AP. Furthermore it is unlikely that a CT can change the management of the disease in this period. CT scan carries with it the concerns of renal impairment in patients with deranged or borderline renal function due to contrast nephrotoxicity. The concerns of the contrast aggravating pancreatic necrosis are probably unfounded.

Follow up CT or MRI in acute pancreatitis is indicated when there is a lack of clinical improvement, clinical deterioration, or especially when invasive intervention is considered. While necrosis on CT predicts a severe attack, clinical studies have failed to show any uniform correlation with the extent of necrosis and organ failure and/or mortality.

It is recommended to perform a multi-detector CT with thin collimation and slice thickness (i.e. 5mm or less), 100-150 ml of non-ionic intra-venous contrast material at a rate of 3mL/s, during the pancreatic and/or portal venous phase (i.e. 50-70 seconds delay). During follow up only a portal venous phase (monophasic) is generally sufficient. A CT severity score (the Balthazar score) has been developed based upon the degree of necrosis, inflammation, and the presence of fluid collections. It although is not found to be superior to the other severity predictors and can be applicable only after 4-5 days (when the CT signs become manifest).

MRI- MRI has a higher sensitivity for the diagnosis of early AP as compared with contrast-enhanced CT scan and can better characterize the pancreatic and bile ducts and complications of acute pancreatitis. Magnetic resonance cholangiopancreatogram (MRCP) is comparable to endoscopic retrograde cholangiopancreatogram (ERCP) for the detection of choledocholithiasis. MRI has the advantage of not requiring radiation, and gadolinium has a lower risk of nephrotoxicity as compared with iodinated contrast. In addition, in patients with renal failure, a non enhanced MRI with T2 weighted images can identify pancreatic necrosis. MRI is capable of demonstrating the presence or absence of solid necrosis in a fluid collection which is often missed on CT scan.

Management

Initial management of AP consists of supportive care with fluid resuscitation, oxygen supplementation, pain control and nutritional support.

Although majority of patients with AP will have mild disease which is self limiting, it is difficult to identify patients with severe attack at admission. It is hence prudent to manage every patient of AP aggressively in the initial period till disease severity is established.

Fluid resuscitation:

The adequacy of fluid replacement is the single most important aspect of the medical management. Fluid therapy to prevent hypovolemia and organ hypoperfusion is a long-established cornerstone of the initial management of AP. Initially fluids are given as goal directed fluid therapy at a rate of 5-10ml/kg/hr in patients with AP, except those with cardiac, renal or other co-morbidities which preclude aggressive fluid replacement, till resuscitation goals are reached. Goal-directed therapy is defined as the titration of intravenous fluids to specific clinical and biochemical targets of perfusion (eg. heart rate, mean arterial pressure, central venous pressure, urine output, blood urea nitrogen concentration, and hematocrit) and is putatively superior to non-targeted fluid therapy. In patients with severe hypotension and tachycardia, fluids can be given as rapid infusion bolus of around 15-20 ml/kg over the first hour. In goal directed therapy, fluid requirements are assessed at frequent intervals in the first six hours and over the next 24-48 hours. The fluid of choice is Ringer's lactate and is preferred over Normal saline (NS) which can cause acidosis. Colloids have no demonstrable benefit over crystalloids.

Adequate fluid replacement can be assessed by an improvement in vital signs (goal heart rate <120 beats/minute, mean arterial pressure between 65 to 85 mmHg), urine output (>0.5 to 1 cc/kg/hour) and reduction in hematocrit (goal 35 to 44 percent) and Blood urea Nitrogen (BUN) over 24 hours, particularly if they were high at the onset. Monitoring the BUN may be particularly important, as both the BUN at the time of admission and the change in BUN during the first 24 hours of hospitalization predict mortality.

In the initial stages (within the first 12 to 24 hours) of AP, optimal fluid replacement has been associated with a reduction in morbidity and mortality. It is important to limit fluid resuscitation mainly to the first 24 to 48 hours after onset of the disease. Continued aggressive fluid resuscitation after 48 hours may not be advisable as overly-vigorous fluid resuscitation is associated with an increased need for intubation and increased risk of abdominal compartment syndrome.

Oxygen supplementation:

Another fairly simple and efficacious strategy is the administration of supplemental oxygen to maintain saturation above 95% which is known to limit the extent of necrosis. Patients with acute pancreatitis are at high risk of hypoxia because of one or more of abdominal splinting,

atelectasia, pulmonary oedema, or acute respiratory distress syndrome. Hence it is important to monitor arterial oxygenation. During the initial management, consider arterial blood gases every 12 hours for the first 3 days to assess both oxygenation and acid-base status in patients who have features of SIRS.

Nutrition:

The traditional philosophy of giving rest to pancreas by keeping patients NBM (Nil by mouth) so as to avoid stimulation of pancreas is totally obsolete and current evidence is to the contrary. Oral feeding should resume as soon as possible in any patient of AP. Traditionally, patients have been advanced from a clear liquid diet to solid food as tolerated. In patients who are unable to resume oral feeds (eg. patients on ventilatory support, nausea or vomiting related to gastroduodenal inflammation and/or extrinsic compression from fluid collections) enteral nutrition is preferred to parenteral nutrition. The routes for enteral feeding are either nasogastric or naso-jejunal. Naso-jejunal feeding has advantages in terms of avoiding risk of aspiration which can happen with nasogastric route. When the local complications start improving, oral feeds can be initiated and advanced as tolerated. Enteral nutrition maintains gut barrier and prevents bacterial translocation from gut. It is associated with low incidence of infective complications. It also has advantages of better glucose control and less incidence of central line associated infective complications which are known in patients on TPN. This translates into better outcomes and lesser mortality in those receiving enteral feeding. There is no specific advantage of elemental/ semi-elemental /immunonutrition formulas and inexpensive polymeric feeds are equally effective. The recommended nutrient requirements in severe acute pancreatitis are as follows: energy 25 to 35 kcal/ kg/day, protein 1.2 to 1.5 g/kg/day, carbohydrates 3 to 6 g/kg/day, and lipids 2 g/kg/day

Parenteral nutrition is preferred in patients who do not tolerate oral/ enteral nutrition and in whom the nutritional goals cannot be reached in 3-5 days of attempted enteral feeding. It is always second line option in view of the above enumerated disadvantages in comparison to enteral feeding.

Pain relief:

Pain in AP is a severe pain and usually will merit opioids. The choice of analgesic is largely dependent on local protocols and physician preference, as no one approach has been shown to be more beneficial. Opioids are safe and effective at providing pain control in patients with acute pancreatitis. Adequate pain control requires the use of intravenous opiates. If available, a patient-controlled analgesia pump is ideal. Fentanyl is being increasingly used due to its better safety profile, especially in renal impairment. As with other opiates, fentanyl can depress respiratory function and needs to be given only with strict monitoring in ICU/ HDU setting. In

mild cases, the standard World Health Organization pain ladder can be used. Overall, opioids were associated with a reduction in the need for supplementary analgesia and therefore, for the majority of patients, opioid analgesia remains the treatment of choice.

It is important at this period, as the initial management of AP is being carried out, to assess and predict the severity of AP.

Predicting severity of AP:

Around 15-20% of patients with AP develop severe AP. Although there have been developments in the management approach towards AP, the morbidity and mortality of severe AP even today is quite high. The ability to predict severity can help in identifying this high risk group of patients thereby assisting in appropriate counseling of family, early triage to tertiary care referral centres. Although number of predictive models, scoring systems based on clinical / laboratory/ radiological parameters and serum markers have been proposed and studied, they all have low specificity (high false positive) and thereby result in low positive predictive value. Two relatively easy to follow, inexpensive and practical tools are SIRS score and BISAP score.

SIRS (Systemic Inflammatory response syndrome) score

SIRS is defined by the presence of two or more of the following four criteria:

- Temperature <36 or 38°C
- Respiratory rate $>20/\text{min}$ ($\text{PaCO}_2 < 32 \text{ mm Hg}$)
- Pulse $> 90/\text{minute}$
- WBC <4000 or $>12000 /\text{cu.mm}$ or $> 10\%$ immature bands

Persistent ($>48\text{hrs}$) SIRS is associated with multi-organ failure and mortality in AP. In one study persistent SIRS was associated with a mortality of 25% compared with 8% for transient SIRS. The advantages of using persistent SIRS as a marker is the widespread familiarity and simplicity. None of the different predictive scoring systems – APACHE II, Ranson's, Glasgow score, etc. described for AP have been found to be superior or inferior to persistent SIRS as a severity predictor.

The BISAP – Bedside Index for Severity of Acute Pancreatitis score is a simple, clinically oriented severity scoring system that can predict mortality of Acute Pancreatitis. It has been devised and validated on a cohort of around 18000 patients with AP. Individual components of the BISAP scoring system are:

- BUN $> 25 \text{ mg/dl}$
- Impaired mental status (Glasgow coma scale score < 15)

- SIRS as defined by two or more:
 - Temperature <36 or 38°C
 - Respiratory rate > 20/min (PaCO₂ < 32 mm Hg)
 - Pulse > 90/ minute
 - WBC <4000 or >12000 /cmm or > 10% immature bands
- Age > 60 years
- Pleural effusion detected on X ray or ultrasound

One point is assigned for each variable within 24 hours of presentation and then added for getting the BISAP score.

Patients with a score of zero had a mortality of less than one percent, whereas patients with a score of five had a mortality rate of 22 percent. Usually a BISAP score of ≤ 2 is associated with a mortality of < 2% which increases significantly with a score of 3 or more.

Role of antibiotics

Prophylactic antibiotics are not recommended in patients with acute pancreatitis, regardless of the type (interstitial or necrotizing) or disease severity (mild, moderately severe, or severe). According to a recent meta-analysis of 14 RCTs, there is no evidence to support routine use of prophylactic antibiotics.

Antibiotics are although indicated in presence of extra-pancreatic infections and in patients with suspected infected necrosis. Up to 20 percent of patients with acute pancreatitis develop an extra pancreatic infection (eg. bloodstream infections, pneumonia, and urinary tract infections) [45]. Extra pancreatic infections are associated with an increase in mortality [46]. When an infection is suspected, antibiotics should be started while the source of the infection is being determined. However, if cultures are negative and no source of infection is identified, antibiotics should be discontinued.

Infected necrosis should be suspected in patients with pancreatic or extra pancreatic necrosis who deteriorate (clinical instability, increasing white blood cell count, fevers) or fail to improve after 7 to 10 days of hospitalization. Clinical signs of infection and abdominal imaging demonstrating the presence of gas within the necrosis are reasonably suggestive of infection and antibiotic therapy can be initiated without aspiration and culture. If empiric antibiotics are initiated, antibiotics known to penetrate pancreatic necrosis (eg, carbapenem, or quinolone, or fourth generation cephalosporin -[ceftazidime](#), or [cefepime](#) combined with an anaerobic agent such as [metronidazole](#)) should be used. Imipenem is the antibiotic which has been evaluated in many clinical studies and is usually preferred in these situations.

Specific drug therapy

There is no proven benefit in treating AP with antiproteases (eg., gabexatemesilate, aprotinin), platelet-activating factor inhibitors (eg. lexipafant), or pancreatic secretion inhibitors (eg. Octreotide)

ERCP

There is no role of routine use of urgent ERCP and endoscopic sphincterotomy (ES) in patients with acute gallstone pancreatitis. ERCP and ES are indicated only in patients with bile duct stone and biliary obstruction and/or cholangitis.

Role and timing of Cholecystectomy

In patients with mild gallstone pancreatitis it is advisable to perform an early cholecystectomy to prevent recurrent pancreatitis. It can safely be performed in the same admission after the attack settles. In a large review it was found that there was an 18% risk of recurrent pancreatitis at a median of 6 weeks. It is essential to have a preoperative EUS or MRCP in these patients to rule out a CBD stone before doing the cholecystectomy.

Cholecystectomy should be delayed in patients with severe biliary pancreatitis with peri-pancreatic collections until the collections either resolve or stabilize and the inflammation resolves which may take 6 weeks or beyond.

In very elderly patients and those who are unfit for cholecystectomy, an ERCP and endoscopic sphincterotomy will prevent them from risk of recurrent biliary pancreatitis and should suffice. It although does not avoid the risk of developing biliary colic.

Indications for intervention in AP

- Clinical suspicion or documented infected necrosis in patients with clinical deterioration especially when necrosis has become walled off
- In absence of infected necrosis in cases of Walled off pancreatic necrosis with ongoing organ failure not improving with medical management
- Less common indications are Gastric outlet obstruction, biliary obstruction or intestinal obstruction due to mass effect from large WOPN (arbitrarily > 4-8 weeks after onset of AP)
- Abdominal Compartment syndrome, Bowel ischemia, GI bleeding. In these situations necrosectomy is not attempted as it can increase risk of developing infected necrosis
- In patients with sterile necrosis persistently unwell with failure to thrive (arbitrarily more than 8 weeks after the onset)

Routine percutaneous fine needle aspiration (FNA) of pancreatic necrosis/ fluid collections is not indicated. Clinical signs of sepsis with deterioration in patients with pancreatic necrosis or presence of gas in necrosis are reliable signs of infection. Percutaneous FNA can be false negative in 12-25% of patients.

Timing of intervention: It is advisable if possible to delay the intervention (percutaneous catheter drainage, endoscopic drainage/necrosectomy, minimally invasive necrosectomy, open necrosectomy) upto the fourth week of illness or beyond. It allows for clinical stabilization of patient, resolution of early organ failure and decrease in the inflammatory reaction in the retroperitoneum. In a subset of patients it will not be feasible to delay the intervention until 4 weeks. In such cases preferably percutaneous drainage can be undertaken early. Necrosectomy should ideally still be delayed until the collection has got walled off. Surgery in early phase of illness is associated with higher morbidity and mortality. There is difficulty to discriminate necrotic tissue from viable pancreas making surgery imprecise, bloody with increased risk of iatrogenic damage and incomplete removal of necrotic tissue.

In a randomized trial it was observed that in comparison to the standard open surgical approach of necrosectomy, a step up approach of percutaneous (retroperitoneal) catheter drainage followed by a minimally invasive necrosectomy resulted in significantly lesser major short term complications and new onset multiorgan failure as well as endocrine insufficiency.

The overall philosophy based on prospective comparative studies is towards lesser and later interventions. Percutaneous catheter or endoscopic transmural drainage should usually be the first intervention. Percutaneous drainage is usually technically feasible in the great majority of patients with necrosis. It can suffice in 30-40% of cases and can avoid the need for further necrosectomy. The choice of approach -percutaneous or endoscopic should be selected by the morphology of collections and depending on the available expertise. The endoscopic approach is associated with lower incidence of pancreatic fistula. There are no strict guidelines for the timing of repeat intervention. The decision making is governed by the clinical condition and findings on imaging.

Aetiological evaluation in AP

History

- Previous gallstones, Biliary colic
- Alcohol intake
- Drug intake
- Family history
- Exposure to known viral causes or prodromal symptoms

Initial investigations (acute phase)

LFTs, USG

Follow up investigations (recovery phase)

Fasting lipid profile

Serum Calcium, serum Parathyroid hormone

Viral antibody titres

Repeat USG (gallstones)

MRCP, CT (pancreatico-biliary anomalies, neoplastic)

Further investigations (especially in recurrent Idiopathic AP)

EUS

Autoimmune markers – IgG4

Pancreatic function tests (early chronic pancreatitis)

Genetic analysis (Family history, Ty 1 DM)

Referral criteria

The majority of patients of AP has only mild attack and can be managed at secondary care centres (private nursing homes). The following group of patients need specialized care and should be referred to tertiary care centres specialized in management of AP

- Patients with predicted severe AP – persistent SIRS
- BISAP score ≥ 3
- AP with associated major co-morbidities, altered mental status, obesity (BMI > 30)

Team responsibilities

Management of severe AP calls for a team approach. The Team and prerequisites are:

- Clinical: multi-disciplinary team of specialists in Gastroenterology proficient in therapeutic pancreatco-biliary endoscopy, GI surgery, Intensivists, Interventional radiology, dietitian and nursing staff. The team leader has to be a surgeon if possible with GI interest or training / Gastroenterologist with special interest in pancreatic disorders.
- ICU care with facilities for renal and respiratory support
- Facilities for CT, MRI and interventional radiology procedures
- Facilities for ERCP and therapeutic endoscopy

In case if some of the investigational / endoscopic backup is not available it needs to be coordinated with centres where these facilities are available and where patient can be shifted.

Acute Retention of Urine

Introduction

Acute retention of urine implies that inspite of accumulation of urine in the urinary bladder and a strong desire to urinate, patient is not able to pass urine. It is different from anuria where there is no or little urine in the urinary bladder and hence patient does not pass any urine and has no intense desire to micturate. Acute Retention of urine gradually becomes painful. In chronic retention there is more than 200ml of urine at any given time and is painless. Retention with overflow is when the bladder is full and patient passes small amount of urine or in drops. Acute on chronic retention is there when acute retention superimposes on chronic retention.

Males are more commonly affected. Common causes of acute retention of urine are:

In Men:

- Diseases of Prostrate: Benign hyperplasia of prostrate, carcinoma of prostrate, prostatitis, prostatic abscess.
- Diseases of urethra: Urethritis, stricture, traumatic rupture of urethra, impacted stone.
- Disease of urinary bladder : calculus, carcinoma
- Phimosis

In Women:

- Prolapse of uterus, retroverted gravid uterus
- Bladder neck obstruction
- Uterine, ovarian surgeries
- Urethral stricture, meatal stenosis
- Non-congenial place to pass urine leading to over-distension and inertia eg. Long drive/travelling

In both men and women, other causes are:

- Stone in bladder/urethra
- Urethritis, Cystitis
- Excessive fluid intake (alcohol)
- Blood clots in urinary bladder
- Injury or diseases of spinal cord
- Impacted faecal matter

- After surgeries of the Anal and perianal region: haemorrhoidectomy, fissurectomy, fistulectomy. Painful anorectal stimuli after these operations may lead to anovesical inhibition of the detrusor. Pelvic surgeries
- Spinal Anesthesia
- Drugs like narcotics, anticholinergics and antipsychotics and heavy metals.

Narcotics decrease the sensation of bladder fullness. Anticholinergics inhibit detrusor contractility and anti-psychotics have anti-cholinergic effects.

In infants and children:

- Posterior urethral valves
- Meatal stenosis
- Phimosis
- Impacted urethral stone.

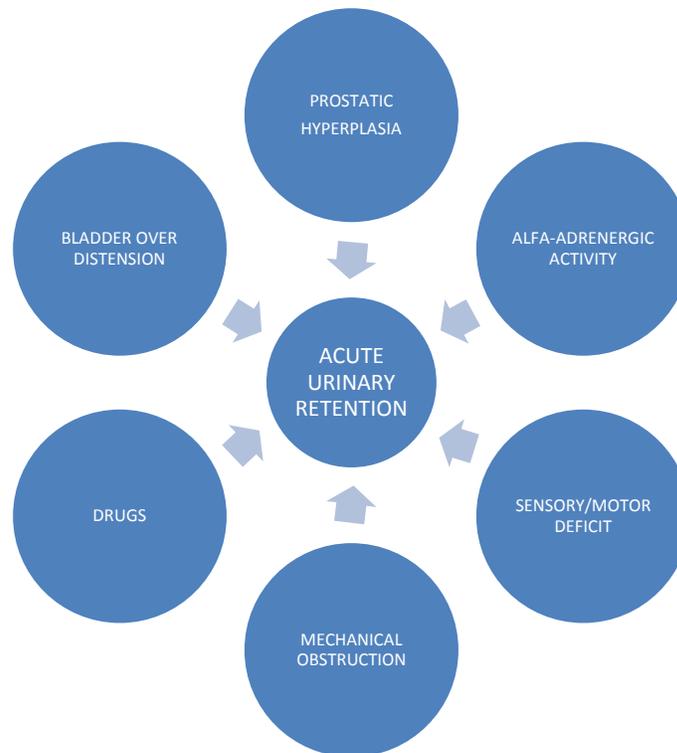


Figure 2 showing aetiology and pathogenesis of acute urinary retention

Incidence

The annual incidence of acute urinary retention varies from 2.2 to 6.8 /per 1000 men.

Symptoms

Patient has not passed urine for many hours. Patient experiences pain and is in distress. There is excessive desire to pass urine but to not possible.

Examination

- Examine the genitalia for: phimosis, meatal stenosis, impacted stone or a stricture.
- Urinary bladder is palpable as smooth, soft and tender swelling in hypogastrium. It is dull on percussion.
- Per rectal examination shows that prostate or uterus has been pushed backward and downwards by the bladder. Bladder is felt as a cystic mass.

The assessment of sensory, motor and reflex functions of nerves of perineum and lower limbs may reveal neurological problem.

Investigations

At presentation, complete blood count and blood urea should be done. Subsequently get X-ray KUB to see for calculi, urine examination, serum electrolytes, ultrasonography of kidneys, urinary bladder and prostate.

Treatment

Conservative Treatment

In a post-operative case before a catheter is passed following measures should be tried:

- Relieve pain
- Maintain privacy.
- Change posture from recumbency to sitting or standing
- Sound of running water may help.
- Hot fomentation should be done over hypogastrium.

In case foregoing methods fail then do a single catheterization and a voiding trial may be allowed. Later on, if catheterization is required again, better leave an indwelling Foley's catheter for two to three days and a trial with α -blockers may be started.

- Impacted stone in urethra:
 - Push back the stone into urinary bladder and catheterize.
 - Meatotomy/urethrotomy for stone extraction may be done.

- Pin-hole meatus: Dilation/Meatotomy.

Urethral Catheterisation

Wash hands properly. Put sterile gloves. Clean the genitalia with an antiseptic solution like povidone iodine or cetrimide solution. Insert Xylocaine jelly into the urethra and keep it for a few minutes. Massage the urethra towards the perineum to push the jelly in posterior urethra. In adults a Foley's catheter (14Fr) should be passed while the penis is held taut. Do not use any force. In female patients, the labia should be parted using the middle and index fingers of the left hand and the area is cleaned. Always check the balloon of the catheter before insertion.

If a stricture is not there the catheter will pass freely. Once the urine starts draining, push the catheter for a few more centimetres more into the bladder. Then inflate the balloon with 10ml of distilled water or saline. Connect the catheter to the tubing of the urine collecting bag to avoid ascending infection. Record the volume of the urine drained.

TWOC (Trial without catheter)

If urethral catheterisation is successful then once bladder is decompressed, further treatment can be followed by TWOC which means removing the catheter after 2-3days and then allowing the patient to void. Success rates are about 35%. If catheter has been put in, it should be kept for 2-3days. It maybe associated with haematuria which gets well of its own. There may be urosepsis and urinary leakage around the catheter. These should be explained to the patient.

In case of benign hyperplasia of prostate presenting with acute retention, after decompression of bladder, it is better to try α_1 -blockers and have TWOC, so that the need for emergency surgery is obviated. Urgent prostatic surgery after acute urinary retention is associated with greater morbidity and mortality than elective surgery. In case of chronic retention with overflow, a gradual decompression is preferred.

Most of the times catheterisation is successful, but at times it may fail due to trauma or narrow urethra in a stricture. Occasionally, a large prostatic middle lobe may also prevent the catheter entering the bladder. If a catheter cannot be passed then following plan should be pursued.

Suprapubic Puncture:

Supra pubic puncture with commercially available supracath catheters such as Supra cath plus or plastic Trocar and cannula is easy, provided that the bladder is palpable. The skin, fascia and retropubic space are anaesthetised with 0.5% xylocaine. It is important to first aspirate to confirm that the needle is in the bladder. Put the supra cath into the bladder, remove the trocar, inflate the balloon and secure the catheter in position. If urine cannot be aspirated then

supra cath should not be attempted. If these devices are not available, then an open supra pubic cystostomy should be done to relieve the retention.

Referral Criteria

Most of the conditions are within the domain of any general surgeon, hence does not require referral. But for certain conditions like high volume of benign hyperplasia of prostate, carcinoma prostate, urethral stricture, and blood clots in urinary bladder, patients may have to be referred to a Urologist or a general surgeon trained in Urology. For injury or disease of spinal cord, neurosurgeon or neurologist's help may be sought.

Medico Legal Issues

Before any procedure routine informed consent should be taken. Before catheterisation patient should be counselled that in some patients haematuria may occur and it may not be successful always. Explain other options in case it fails.

Secondary Management (Operative Management):

It depends on the cause and needs a surgeon trained in urology. For secondary management, the nature of procedure, operations and their complications, should be explained to the patient and attendants(s).

Operative management will require following details:

Who does what?

- Surgeon:
 - Diagnosis and Work up.
 - Pre-operative planning.
 - Operative procedure.
 - Post-operative care in conjunction with Anaesthetist and Nurses.
 - Post-operative follow ups.
- Anaesthetist:
 - Pre Anaesthesia check up
 - Performing anaesthesia
- Nurse:
 - Pre/ Intra/ Post-operative comprehensive care
 - Dressing of the wound
- Technician:
 - Pre-operative equipment and drugs to be checked and kept ready

- Assist anaesthetist in the OT
- Assist the surgeon, positioning of the patient.

Resources required for one procedure (patient weight 60 kg)

Human Resources Drugs/ Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1-2
3. Anaesthetist – 1- if any intervention is done under anaesthesia
4. Pathologist – 1 (Service from outside can be availed)
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning Staff – 1

Investigations

1. Complete Hemogram
2. Blood sugar
3. Renal Function Test
4. S. Electrolytes in selected cases
5. Prostate Specific Antigen
6. Plain X-ray KUB, to see calculi.
7. USG of KUB and prostate to assess UB and prostate characteristics, post void volume
8. ECG
9. X-Ray chest
10. Histopathology
11. Pan endoscopy wherever indicated.

Drugs and Consumables

1. OT Table and lights
2. Instrument trolley
3. Anaesthetic Machine, instruments including endotracheal tubes and drugs
4. Monitor
5. Cautery – a basic set
6. Set of surgical instruments
7. Suction
8. Sutures

9. Drains
10. Catheters
11. Antibiotics
12. Analgesic
13. I.V. fluids
14. Dressings

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Acute Scrotum

Definition

The acute scrotum is defined as scrotal pain, swelling, and redness of acute onset- any one or a combination of them.

Introduction: 'Acute scrotum' is like 'acute abdomen' in terms of urgency of examination, need for quick surgical consultation and rapid decision on management strategy. Torsion of testis and incarcerated inguino-scrotal hernia are dire surgical emergencies. In the current era of over emphasis being placed on the investigations for the diagnosis and management, the testicular torsion still remains a clinical diagnosis, and surgical exploration on clinical suspicion is justified in the absence of investigative proof.

Symptoms:

In neonates and children history has to be elicited from the parents.

Onset, duration, and progress of the following symptoms should be carefully elicited.

Pain- in the scrotum, may be referred to ipsilateral lower abdomen /groin. Previous h /o self-limiting episodes of pain needs to be elicited.

Swelling

Redness, ecchymosis

Nausea / Vomiting.

Fever

Urinary complaints- Dysuria, Urethral discharge, Burning micturition, Recent history of urinary tract instrumentation, UTI.

Skin rash on buttocks and the lower limbs.

Trauma, physical exercise, exposure to cold weather

Signs

Signs are difficult to elicit when the anatomical land marks are obscured in the grossly swollen and tender scrotum.

Swelling - scrotal / inguino-scrotal ? redness and tenderness of scrotum – extent and location, unilateral / bilateral. necrosis of scrotal skin, note- gross edema and / tenderness make palpation of deeper structures impossible.

Testes - tenderness of entire testis / localized to upper pole? location of testis (High riding testis), size, and consistency.

Epididymis - tenderness, size, consistency

Spermatic chord - tenderness, size, consistency

Opposite testis - horizontal lie ?

Blue dot sign - s/o infarcted appendix of testis / epididymis is pathognomonic.

Prehn's Sign - Pain not relieved by lifting the scrotum -unreliable sign

Cremasteric Reflex absence - unreliable sign

Ger'sign - retraction of the scrotal skin- unreliable sign

Brunzel's sign- high riding horizontal testis- unreliable sign

Urethral discharge

Petechial rashes on the buttocks and thighs.

Investigations

Choice of investigations depends upon the clinical diagnosis and D/D, the availability of investigative facilities and the urgency of treatment. Tests have their own limitations and none have 100% accuracy.

Imaging investigations

Doppler Ultrasonography - its role and limitations - A definitive diagnosis of complete testicular torsion is made when a central testicular perfusion is visualised on the normal side but is absent on the affected side. This has a Sensitivity of 89.9% and Specificity of 98.8%. Literature is rife with conflicting opinions about the role and reliability of Doppler in the management of acute scrotum. Though the color Doppler technology is advancing, its sensitivity and specificity vary widely depending upon several factors which should be taken into account by the clinician before taking final decision in the treatment of acute scrotum. Availability of Doppler and of the Radiologist during night hours is a constraint in low-resource places. It is observer dependent; weightage assigned to the clinical decision of the experienced clinician is more than the Doppler reports of radiology residents; conversely, experienced radiologists' reports outweigh the surgical residents' opinion. Infant's testes are just 1ml in volume and it is technically difficult to detect vascularity. Doppler examination is difficult in children who may keep moving. An exquisitely tender scrotum may not permit optimum Doppler-probe pressure. Central perfusion of the testicular parenchyma is more reliable indicator of the viability; reactionary hyper-vascularity of peripheral tissues in response to ischemic testis can be misinterpreted. In partial torsion, central perfusion may be preserved which may be misleading; partial torsion may progress to complete torsion(360⁰) in a few hours which is detected only if Doppler is repeated.

Nuclear Scanning With Tc 99 Pertechnetate

CT & MRI

Total and differential leucocyte count.

Urine routine, culture

Urethral discharge for culture

Tests for Urinary Tract Abnormality / anomaly - USG, Micturating / Voiding Cysto Urethrogram, cystoscopy, etc

Nucleic Acid Amplification Test (NAAT)

Mumps IgG/IgM for Mumps Orchitis

Differential Diagnosis and Treatment

There are a number of causes for acute scrotum; their order of preference in the differential diagnosis depends upon the permutations and combinations of symptoms and signs and the appropriate investigation reports. Testicular torsion is the most important one. Appendiceal torsion, idiopathic scrotal edema and acute epididymo orchitis are other common conditions.

Causes of Acute Scrotum

Torsion:

- Torsion testis (torsion of the spermatic cord)
- Torsion of embryonal remnants
 - Testicular appendix (Hydatid of Morgagni)
 - Epididymal appendix
 - Paradidymal appendix- Giralde's organ
 - Vas aberrans of Haller
- Neonatal torsion testis-Intravaginal; extravaginal (prenatal and postnatal)

Ideopathic scrotal edema

Acute infections of scrotal contents and coverings

- Acute epididymitis, epididymo-orchitis, mumps orchitis, abscess
- Furuncle, infected sebaceous cysts
- Fournier's gangrene

Traumatic acute scrotum:

- Intratesticular hematoma, testicular contusion
- Testicular rupture, dislocation
- Spermatic cord injury
- Hematocele

Inguino-scrotal hernia

- Incarcerated, strangulated inguinal hernia, +/- testicular ischemia

Acute scrotum superimposed on pre-existing chronic scrotal lesion:

- Spermatocele, rupture, haemorrhage or infection

- Hydrocele, rupture, hemorrhage or infection
- Testicular tumor - rupture, hemorrhage, infarction

Abdominal diseases presenting as acute scrotum:

- Peritonitis tracking along the patent processus vaginalis

Acute scrotal swelling due to other causes:

- Henoch-Schonlein purpura (vasculitis of scrotal wall)

Common conditions depending on the symptoms:

Pain and swelling: Torsion testis, appendiceal torsion ac.epididymoorchitis, trauma, incarcerated hernia, Fourniers gangrene

Pain , no swelling: Appendiceal torsion , Ureteric colic

Swelling, no pain: Idiopathic scrotal edema

Common conditions depending on age groups:

Newborn infants: testicular torsion, birth injury (esp in breech delivery)

Toddlers: acute epididymoorchitis, idiopathic scrotal edema

Adolescents and teen agers: testicular torsion, appendiceal torsion, acute Epididymitis, testicular tumor

Testicular Torsion

The testis is covered by the tunica vaginalis, creating a potential space around the testis. Normally, the parietal layer of tunica vaginalis is reflected from either side of the epididymis, posterior surface of which is attached to the other layers of the scrotum; therefore the testis can not rotate within the scrotum. In 12% males, due to anomalous high investment of the tunica vaginalis, the testicle hangs like a gong in the bell, known as 'bell clapper testis'. In addition the testis may lie transversely. In this anomaly, which may be unilateral or bilateral, the testis is free to rotate spontaneously on the axis of the spermatic cord within the tunica vaginalis (intravaginal torsion) resulting in testicular ischemia; torsion initially causes venous occlusion resulting in edema, and subsequent arterial occlusion and infarction. The onset of ischemic damage and its extent are directly proportional to the degree of rotation (90^o, 180^o, 360^o, 760^o) and the time lapse before detorsion. 4 complete turns of spermatic cord cause necrosis of testis in 2 hours and one turn causes no ischaemia till 12 hours.

Incidence

Though testicular torsion accounts for about 20% of all acute scrotum, it should receive priority attention in the management of every case. It can present at any age, the peak incidence is between 14 and 16 years and accounts for 90% of acute scrotums in teen age boys and 30% in

pre-pubertal boys. The annual incidence is approximately 1 in 4000 males below 25 years of age. The left side is more commonly affected. Horizontal lie, and cryptorchidism are predisposing factors.

Clinical features

Testicular torsion presents with the rapid onset of severe testicular pain and swelling. The pain may be referred to the groin / lower abdomen and associated with nausea and vomiting. It may be preceded by exercise, trauma or exposure to cold weather. It can also occur during sleep. There may be past h/o similar episodes of self limiting pain due to intermittent torsion followed by spontaneous detorsion.

The classic finding is an exquisitely tender testis. Early in the course, it may be possible to palpate the twisted, tender, edematous cord. Testis may be lying horizontally but tenderness may prevent its demonstration: if horizontal lie of the opposite testis is demonstrable, it strengthens the diagnosis. After 12-24 hours, due to progressive edema and inflammation, all anatomical landmarks are obscured and the entire hemiscrotum becomes a confluent mass indistinguishable from acute epididymo-orchitis. Other signs like absence of cremasteric reflex, non-relief of pain on elevation of the scrotum (negative Prehn sign), retraction of the scrotal skin (Ger's sign), high riding and horizontally placed testis (Brunzel's sign) may be elicited but are not reliable. Importantly, patients have no systemic disturbances, are usually afebrile, free from urinary symptoms with normal urinalysis and white blood counts. (in more advanced cases however, leucocytosis may occur due to inflammation).

Torsion of the undescended testis can manifest as acute groin pain with a tender mass in the groin.

With a high degree of clinical suspicion, one may reasonably recommend surgical exploration without delay.

Doppler- its role and limitations:

Doppler ultrasonography, if readily available, and if the diagnosis is doubtful, is the single most useful test in the management of torsion testes. If Doppler shows decrease or absence of central perfusion of the testicular parenchyma, compared to the contralateral testis, torsion is highly likely. Spiral course of the vessels in the spermatic cord may be seen in USG. Doppler Ultrasonography may also exclude significant testicular trauma, show an inguinoscrotal hernia, and can distinguish epididymitis from torsion by demonstrating edema and increased blood flow to the epididymis along with preserved testicular perfusion.

If Doppler cannot be done on an urgent basis, valuable time should not be lost waiting for it, in the face of strong clinical indication for exploration. This is consensus conclusion of many studies. It is better to undertake some negative explorations and save many testes than to lose many testes in an effort to minimize negative explorations.

Tests such as nuclear scans, CT or MRI, have essentially no role in the contemporary management of the torsion testis.

Counselling notes of medicolegal importance

During the preoperative consent procedure, the following ten points must be informed to the adult patients and the parents / guardians of minor children.

1. Diagnostic dilemma- Testicular torsion Vs other conditions
2. Need for emergency exploration SOS
3. Testis may/may not be salvageable
4. Salvaged testis may develop recurrent torsion and need re-exploration
5. Salvaged testis may/may not survive
6. Salvaged testis may become atrophic, and shrink
7. Salvaged testis has a malignant potential
8. Future possibility of infertility / subfertility
9. Need for fixing the other testis; despite, it may develop torsion.
10. Sutures can be sometimes be felt through the scrotal skin, causing pain or irritation

Operative procedure

Testicular torsion is a true surgical emergency and hence merits urgent surgical exploration interrupting the routine OT list, and without waiting for standard period of starvation. External manual detorsion is painful and not successful.

Scrotal contents are exposed by transverse incision. The testis, epididymis, their appendices and spermatic cord are carefully examined. The viability is assessed by the colour. In those with necrotic testis, orchidectomy is done. In others with doubtfully viable testis, the cord is untwisted completely, and the testis is wrapped in a warm, moist gauze. The contralateral testis is exposed through a separate transverse incision and is fixed. The affected testis is reinspected for signs of improved perfusion. If the testis appears viable it should be replaced in the scrotum and fixed and can be achieved by 3-0 prolene sutures passing through tunica albuginea at two or three points and the adjacent inner layers of the scrotal wall. Alternatively testes can be placed in the subdartos pouch. A Jabolay procedure also fixes the testes securely.

In prepubertal boys, even in delayed explorations, testis can still be preserved in few, though some of them may atrophy later. In post pubertal boys, detorsion of ischemic testis and resultant reperfusion may result in antibodies which may damage the opposite testis.

Outcome of treatment of torsion testis

The testis salvage rate depends on the degree of torsion (90° , 180° , 360° , 720°) and the gap between the time of onset of pain and the time of detorsion; it approaches 80-100% at 6hrs, 20-30% at 12hrs and 0-5% at 24hrs. Salvaged testis may need reexploration and orchidectomy; some gradually undergo atrophy. Fertility may be adversely affected. After orchidectomy silicone prosthesis can be placed in the scrotum for cosmetic and psychological advantage.

"Intermittent" testicular torsion due to 'bell clapper testes' is characterised by recurrent episodes of transient acute testicular pain with spontaneous remission, physical examination and ultrasound findings being normal. Prophylactic elective bilateral orchidopexy is justified in such cases.

Torsion of the embryonal remnants

The embryologic remnants of the Müllerian and Wolffian ducts may enlarge due to hormonal surge during puberty. They are pedunculated and undergo torsion and become ischemic. Torsion of Hydatid of Morgagni is the commonest of all the appendices and is common in prepubertal boys. Clinical presentation is like testicular torsion but the onset is less acute and the pain less severe. The point of maximal tenderness is near the superior pole of testis where a swelling can be palpated in some cases. On transillumination, a bluish shimmering structure (the "blue dot sign") may be visible. The cremasteric reflex is present, and the testis is mobile. Doppler ultrasonography reveals a hyper- or hypoechogenic structure near the superior pole of testis and hyperemia of neighbouring tissues; these torsions are generally treated symptomatically, with bed rest and anti-inflammatory drugs. Excision of the twisted appendix is done if exploration is performed due to diagnostic uncertainty or failure of symptoms to subside.

Neonatal torsion

70% occur prenatally and 30% postnatally. Prenatal torsion is extravaginal and the baby is born with an enlarged, firm, nontender testis with the scrotal skin adherent to it; the testis is not salvageable, and can be left alone. Need for fixing the opposite testis is controversial.

Post natal torsion, extravaginal more commonly than intravaginal, presents with an acute tender swelling of the scrotum and needs urgent exploration through inguinal incision. The

opposite testis should be fixed. Testis of the new born measures hardly 1-2 ml and it is difficult to demonstrate testicular perfusion by Doppler.

Birth injury

The scrotum may be injured especially in difficult breech delivery. The scrotum is swollen, bruised and echymotic. Idiopathic scrotal haemorrhage which can occur in a normally delivered baby mimicks but is distinguished from torsion by a small but separate bruise on the superficial inguinal ring.

Ideopathic scrotal edema

This is a common encounter in the pediatric practice. There is acute onset and rapidly progressive (within few hours) painless, non tender swelling and redness of the scrotum, spreading beyond and around the scrotum, in an otherwise asymptomatic child. USG shows normal testicular blood flow. With symptomatic treatment it subsides within 2-3 days. In toddlers diaper dermatitis should be distinguished from idiopathic scrotal edema. Insect bite also can result in similar scrotal edema.

Acute Infections of scrotal contents and coverings

Acute Epididymitis and Epididymo-orchitis

This is a close D/D for torsion; If diagnosis is confident, the management is essentially nonsurgical. In children congenital anomalies and in older men acquired structural and functional abnormalities in the urinary tract are responsible for spread of coliforms organisms from urinary tract to the epididymis and testis. In younger men sexually transmitted gonococcal and chlamydial epididymitis are more common. Mumps orchitis is seen in post pubertal boys.

The scrotal pain and swelling is usually unilateral and commonly associated with fever. Exposure to STD, urethral discharge, burning micturition, dysuria, other lower urinary tract symptoms, urethral catheterization and h/o surgery on the urinary tract strengthen the diagnosis.

Physical findings range from swelling and tenderness localised to the tail of the epididymis in early cases, to a massively inflamed erythematous hemiscrotum obscuring the anatomical landmarks in advanced cases, closely mimicking torsion. A small reactive hydrocele may be found.

Noninfective acute epididymitis and orchitis is seen in Henoch-Schonlein purpura and Behcet's disease.

Investigations

Doppler ultrasound findings

Early acute epididymitis: Enlarged, hypervascular epididymis and normal or increased testicular blood flow.

Advanced epididymitis commonly associated with orchitis: Massive edema can compress and obliterate the testicular vessels resulting in testicular ischemia mimicking torsion. In either case, the surgical exploration should be undertaken to differentiate and manage these two conditions.

Epididymal/testicular abscess can be detected in the USG.

Identification of microbes-Urethral swab/first catch urine / Mid-stream urine for culture and Nucleic Acid Amplification Test (NAAT)

Tests for anomalies in children – USG, CT, MR urography, Micturating / voiding cysto urethrography

Tests in older men - for prostatic hypertrophy- USG, cystoscopy

Mumps IgM/IgG serology: for mumps orchitis especially in post pubertal boys

Treatment

Once torsion has been excluded, majority of acute epididymo-orchitis are treated with antibiotics, bed rest, scrotal support, elevation, analgesics and NSAIDs.

Empirical antibiotic therapy- to all patients, depending upon clinical features, likely causative organisms, initial lab reports and local antibiotic policy. A 10-day course of Doxycycline 100mg PO BD and Ciprofloxacin 500mg PO BD should cover most possibilities.

When sexually transmitted organisms are suspected - sexual abstinence until they and their partner(s) have received completed treatment.

Culture reports should guide further treatment.

In patients with severe epididymo-orchitis or systemic bacteraemia, parenteral broad-spectrum antibiotics against coliforms and Pseudomonas aeruginosa should be used, eg Cefuroxime 1.5 g TDS and gentamicin 80mg BD until systemic illness subsides. Small epididymal / testicular abscesses are drained; larger ones merit orchidectomy.

Prevention of recurrence of acute epididymo orchitis-The anomalies in pediatric patients and structural and functional pathologies in older men should be treated.

Infections of scrotal coverings

Infections of scrotal integuments presenting as acute scrotum are infected sebaceous cyst, furuncle and necrotising fasciitis (Fournier's gangrene). A normal testis is palpable through the uninvolved scrotal skin.

Fournier's gangrene, is a rapidly progressive necrotising fasciitis of the scrotal and perineal skin and subcutaneous tissues, and is a life threatening infection seen in adult men. The scrotum is swollen, and erythematous. The perineal skin around the scrotum is also inflamed. Within hours the dermal necrosis sets in and progresses to gangrene. There is foul smelling purulent discharge. Surgical emphysema due to gas producing organisms is a common finding. The general condition of the patient is one of overwhelming sepsis. Parenteral broad-spectrum antibiotics covering Gram-positive, Gram-negative, and anaerobes are administered as soon as the diagnosis is made; an empirical combination of antimicrobials are chosen from ampicillin-sulbactam, clindamycin, ciprofloxacin and aminoglycoside and changed over to specific antimicrobials as soon as the culture reports are available. CT may reveal a perianal / periurethral abscess. Urgent and repeated surgical debridement and drainage are done which frequently exposes the normal testes. Cystoscopy and proctoscopy may be necessary to exclude urethral and peranal lesions.

Traumatic acute scrotum

Because of testicular mobility and the cremasteric reflex, isolated scrotal injuries due to blunt and penetrating injuries are less common. Hematocele, testicular contusion, rupture and dislocation can occur depending upon the type and magnitude of the impact. Trauma-induced testicular torsion is likely to be misdiagnosed. Most of the scrotal injuries can be diagnosed with the help of USG; but the associated musculo skeletal injuries to the pelvis, bladder and rectum require CT and MRI. Testicular contusion is conservatively treated; all others often need surgical exploration; injured tissues are either repaired or resected.

Inguino-scrotal hernia

An inguino scrotal swelling due to incarcerated enterocele may also masquerade like an acute scrotum. USG and CT done in selected cases can be helpful. This merits an emergency surgical exploration.

Acute scrotum superimposed on pre-existing chronic scrotal lesions

Rupture, hemorrhage or infection of hydrocele and spermatocele can present as acute scrotum.

Testicular tumors with haemorrhage can present as acute scrotum. If the tumor is clinically suspected and the USG is suggestive, the testis is exposed through transinguinal approach and a high inguinal orchidectomy is undertaken.

Abdominal diseases presenting as acute scrotum

There are isolated reports of abdominal diseases presenting as acute scrotum. In bacterial or biliary peritonitis, the fluid can track along the patent processus vaginalis and result in acute scrotum. The contents of the tunica vaginalis on exploration should point to this possibility.

Scrotal swelling due to other causes:

Henoch-Schonlein purpura (HSP) is a vasculitis of scrotal wall resulting in thickening and erythema associated with purpuric lesions on the buttocks and thighs. Drug induced (Amiodarone) epididymitis are occasionally reported. Scrotal edema as a part of generalised anasarca due to hypoalbuminemia, and due to lymphatic obstruction are easily differentiated from other causes of acute scrotum by their slow and progressive course. Treatment of the underlying cause reduces the scrotal swelling.

Resource persons and responsibilities (for one patient)

Referring doctors

General practitioners, Paediatricians and Physicians, Emergency / casualty Medical Officers.

Responsibilities of the referring doctors is to refer early without delay; to alert the patient / parents about the possible diagnosis, emergency nature of the condition, need to reach surgical centre at the earliest, and the consequences of delay. To document of the same.

Doctors involved in the management and their responsibilities

Radiologist and technician (for imaging).

General surgeon / urologist / paediatric surgeons (for treating).

Medical officer / Assistant surgeon (for assisting)

OT staff nurses (for assisting)

Other OT personale

Anesthesiologist (for pre-anaesthetic check up, administering anaesthesia, and for immediate post anaesthetic care)

Pathologist and lab technicians (for lab tests and histopathological examination) Paediatrician (for pre, intra and post operative fluid management and other medications in neonates, infants and children)

Responsibilities of treating Surgeons

History taking, physical examination, advising appropriate investigations and decision making, counselling and obtaining consent and undertaking Surgery if indicated and at the appropriate time, and providing post operative care. Document.

Equipments and instruments

The Operation theatre, anaesthesia machine and instruments, monitors, electro surgical apparatus, and surgical instruments.

Consumables

Endotracheal tube, sutures: absorbable and non-absorbable (4/0 in children and 3/0 in adults), silk ties, drains, dressing material, gauze pieces, sticking plaster.

Drugs:

IV fluids (NS, RL, 5D and DNS), IV cannula, IV infusion set, Anaesthetic agents, antibiotics (3rd generation cephalosporin, quinolones, imidazole and others as per the sensitivity report), analgesics(iv paracetamol), later oral NSAIDS.

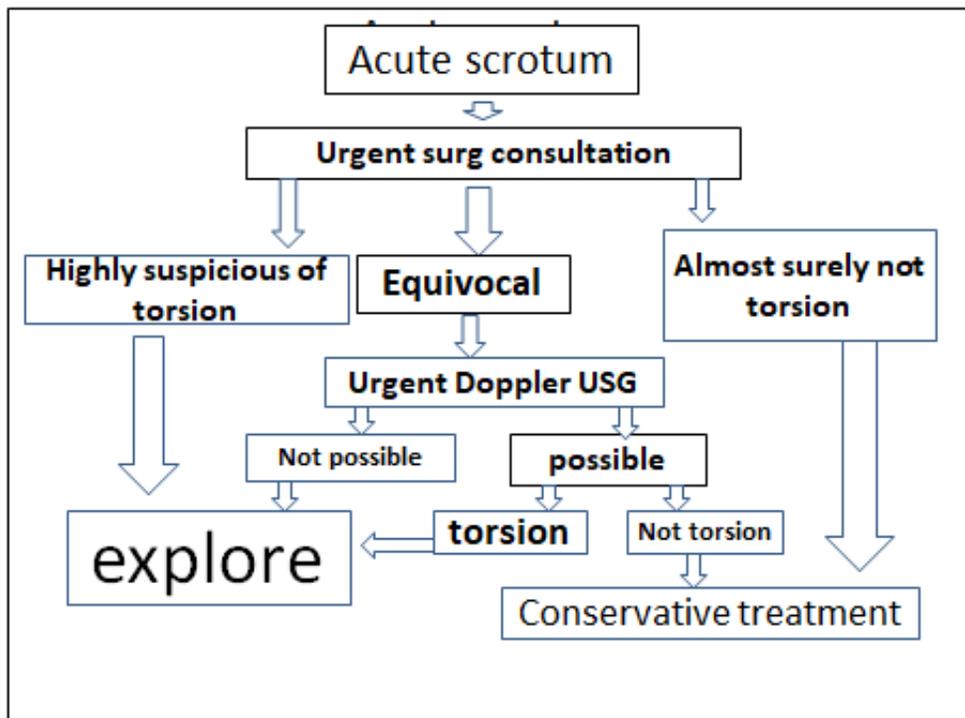
Medicolegal issues:

Acute scrotum, particularly testicular torsion is a sensitive area concerned with 'Consumer Protection Act' because of the diagnostic dilemma leading to delayed treatment and orchiectomy and its consequences. The General practitioners, Casualty medical officers, Paediatricians, General Surgeons, Paediatric Surgeons and Urologists are vulnerable to litigation for alleged failure to provide the 'minimum standard of care' expected of them. Documentation is of utmost importance. All of them should exercise high index of suspicion and document the differential diagnosis. It is important to document the times accurately - time of onset of complaints, examination by the first contact doctor, the Surgeon, the Radiologist, and Surgical exploration. Compliance or otherwise on the part of the patients/ parents should be documented. The testis must be subjected to pathological examination.

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Acute Traumatic Haematuria

Management of Renal Trauma

Mechanism of trauma

Motor vehicle accidents

Fall from height

Assault

Blunt injuries - extent of deceleration in high velocity impact trauma

Penetrating injuries - Trauma to the anterior axillary line is more prone to damage to the renal hilum and pedicle compared to the posterior axillary line, which more commonly results in parenchymal injury.

Clinical Examination

Initial assessment of patient

Flank hematoma

Abdominal or flank tenderness

Ipsilateral rib fractures

Penetrating injuries to the low thorax or flank

Haematuria (with hypotension)

Gross

Microscopic with $>5\text{RBC/HPF}$

The degree of hematuria and the severity of the renal injury do not consistently correlate. So, presence or absence of hematuria should not be the sole determinant in the assessment of a patient with suspected renal trauma.

Investigations

CT using intravenous (IV) contrast and with delayed films to evaluate urinary extravasation is the gold standard in genitourinary imaging in trauma.

Indications for Renal Imaging

1. All penetrating trauma with a likelihood of renal injury (abdomen, flank, or low chest entry/exit wound) who are hemodynamically stable enough to have a CT (instead of going right to the operating room or angiography suite)
2. All blunt trauma with significant acceleration/deceleration mechanism of injury, specifically rapid deceleration as would occur in a high-speed motor vehicle accident or a fall from heights.

3. All blunt trauma with gross hematuria.
4. All blunt traumawith microhematuria and hypotension (defined as a systolic pressure of less than90 mm Hg at any time during evaluation and resuscitation).
Patients with microscopic hematuria without hypotension or acceleration/deceleration injury can be observed clinically without imaging.
5. All pediatric patients with > 5 RBCs/HPF

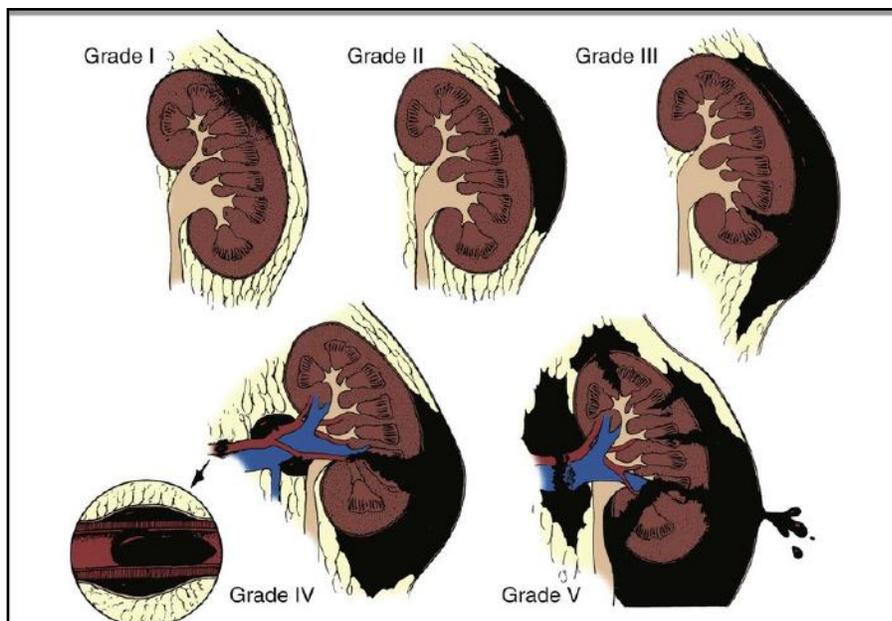
Findings

1. Parenchymal laceration
2. Extravasation of contrast-enhanced urine
3. Degree of retroperitoneal bleeding / haematoma
4. Lack of uptake of contrast material in the parenchyma suggests arterial thrombosis or transection.
5. Associated injury to other organs eg. bowel, pancreas, liver, spleen

CT findings suspicious for significant renal injury include

1. Medial hematoma (vascular pedicle injury)
2. Medial urinary extravasation (renal pelvis or ureteropelvic junction injury)
3. Lack of contrast enhancement of the parenchyma (main renal arterial injury)
4. Active intravascular contrast extravasation (arterial injury with brisk bleeding)

Grading of injury



American Association for the Surgery of Trauma Organ Injury Severity Scale for the Kidney

GRADE*	TYPE	DESCRIPTION
I	Contusion	Microscopic or gross hematuria, urologic studies normal
	Hematoma	Subcapsular, nonexpanding without parenchymal laceration
II	Hematoma	Nonexpanding perirenal hematoma confined to renal retroperitoneum
	Laceration	<1 cm parenchymal depth of renal cortex without urinary extravasation
III	Laceration	>1 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation
IV	Laceration	Parenchymal laceration extending through renal cortex, medulla, and collecting system
	Vascular	Main renal artery or vein injury with contained hemorrhage
V	Laceration	Completely shattered kidney
	Vascular	Avulsion of renal hilum, devascularizing the kidney

*Advance one grade for bilateral injuries up to grade III.

Data from Moore EE, Shackford SR, Pachter HL, et al. Organ injury scaling: spleen, liver, and kidney. J Trauma 1989;29:1664-6.

Management

- **Non-operative management**

It is the standard of care in haemodynamically stable patients

Usually Grade I-III renal injuries

Close observation

Vitals monitoring

Haematocrit monitoring

Bedrest until gross haematuria resolves

No routine repeat CT imaging is required in patients without symptoms (fever, flank pain, dropping hematocrit, increasing hematuria, etc.)

Should bleeding persist or delayed bleeding occur, angiography with selective embolization of bleeding vessels can obviate surgical intervention.

The failure rate of nonoperative management maybe as high as 20% (average ~ 10%), but most patients require only a stent or angioembolization.

- **Operative management**

- **Absolute indications**

- Hemodynamic instability with shock

- Expanding/pulsatile renal hematoma (usually indicating renal artery laceration)
- Suspected renal vascular pedicle avulsion (grade 5)
- Ureteropelvic junction disruption

Relative indications

- Urinary extravasation with significant renal parenchymal devascularization
- Renal injury together with colon/pancreatic injury (these patients have a higher complication rate if their renal injury is not repaired at the time of colon/pancreatic injury, but the renal injury may be closely observed after repair of the enteric injury)
- Delayed diagnosis of arterial injury (which will most likely need delayed nephrectomy).

Surgical exploration of the acutely injured kidney is best done by a transabdominal approach, which allows complete inspection of intra-abdominal organs and bowel.

Renal vessels are isolated before exploration to provide the immediate capability to occlude them if massive bleeding should ensue when the Gerota fascia is opened.

The small bowel is eviscerated and lifted out of the surgical field.

Mid-retroperitoneum is exposed.

Incision is made over the aorta in the retroperitoneum just superior to the inferior mesenteric artery. The incision is extended superiorly to the ligament of Treitz. Exposure of the anterior surface of the aorta is accomplished and followed superiorly to the left renal vein, which crosses the aorta anteriorly.

A vessel loop is placed on the right or left renal vein as necessary.

The right renal vein also can be secured through this incision; but if this proves difficult, reflecting the second portion of the duodenum provides excellent exposure to the vein.

The artery is secured with vessel loops.

The kidney is then exposed by incising the peritoneum lateral to the colon, followed by mobilization off the Gerota fascia.

The Gerota fascia is then opened, and the kidney with injury is completely dissected from the surrounding hematoma.

Should troublesome bleeding develop, the previously isolated vessels can be temporarily occluded with a vascular clamp or a vessel loop tourniquet.

Principles of renal reconstruction

- Complete renal exposure
- Measures for temporary vascular control
- Limited debridement of nonviable tissue
- Hemostasis by individual suture ligation of bleeding vessels
- Watertight closure of the collecting system if necessary
- Possible reapproximation of the parenchymal defect
- Coverage with nearby fascioadipose flaps (Gerota fascia or omentum)
- Liberal use of drains

When polar injuries cannot be reconstructed, a partial nephrectomy can be performed. The open parenchyma should be covered when possible by a pedicle flap of omentum. With its rich vascular and lymphatic supply, omentum promotes wound healing and decreases the risk for delayed bleeding and urinary extravasation. Should it not be available, the use of absorbable mesh may be done.

Reno-vascular injuries

Lacerated main renal vessels may be repaired with 5-0 non-absorbable vascular sutures.

Major vascular injuries → Nephrectomy

Damage Control Surgery

The area around the injured kidney is packed with laparotomy pads to control bleeding, with a planned return in approximately 24 hours to explore and evaluate the extent of injury.

This allows the cold, acidotic, and coagulopathic patient to be stabilized in the ICU before any attempt at potentially lengthy renal reconstruction is attempted.

Damage control may allow patients with complex renal injuries to avoid unnecessary nephrectomy.

Indications for nephrectomy

In an unstable patient, if damage control is not an option, total nephrectomy would be indicated immediately when the patient's life would be threatened by attempted renal repair.

Complications

1. Persistent urinary extravasation resulting in urinoma, perinephric infection, and rarely, renal loss
2. Delayed renal bleeding
3. Perinephric abscess
4. Hypertension

Management of Bladder Trauma

Mechanism of Trauma

The urinary bladder is generally protected from external trauma because of its deep location in the bony pelvis.

Blunt bladder injuries are the result of rapid-deceleration motor vehicle collisions. Many occur with falls, crush injuries, assault, and blows to the lower abdomen. Blunt bladder injuries are commonly associated with other non-urological injuries, most commonly pelvic fracture.

Disruption of the bony pelvis tends to tear the bladder at its fascial attachments

Bone fragments also can directly lacerate the organ.

Other important causes of bladder rupture include penetrating trauma, iatrogenic surgical complications, and spontaneous rupture in patients with a history of neuropathic disease, pre-existing bladder disease, or prior urologic surgery.

Types

Extra-peritoneal

Intra-peritoneal

Extra-peritoneal bladder injury is usually associated with pelvic fracture.

Intraperitoneal injuries can be associated with pelvic fracture but are more commonly due to penetrating injuries or blunt trauma at the dome by direct blow to a fully distended bladder.

Clinical features

Suprapubic pain + Inability to void

Suprapubic tenderness, lower abdominal bruising, muscle guarding and rigidity, and diminished bowel sounds.

Abdominal and pelvic injuries may mask bladder symptoms. Higher suspicion for bladder injury is similarly warranted in patients who are unresponsive because of intoxication or altered sensorium.

Clinical Indicators of Bladder Injury

- Suprapubic pain or tenderness
- Free intraperitoneal fluid on CT or ultrasound examination
- Abdominal distention or ileus
- Inability to void or low urine output
- Clots in urine or clots noted in bladder on CT
- Enlarged scrotum with ecchymosis

Investigations

- X Ray abdomen
- Ultrasonography
- Retrograde cystography
- CT Scan abdomen

After blunt external trauma, the absolute indication for immediate cystography is gross hematuria associated with pelvic fracture— approximately 29% of patients presenting with this combination of findings have bladder rupture.

Relative indications for cystography after blunt trauma include gross hematuria, without pelvic fracture and microhematuria with pelvic fracture.

Penetrating injuries to buttocks, pelvis, lower abdomen, without any haematuria also warrant cystography.

Findings on cystography –

Extraperitoneal – dense, flame shaped collection of contrast in the pelvis

Intraperitoneal – Contrast material outlines loops of bowel and / or the lower lateral portion of the peritoneal cavity.

Because CT is now routinely used to assess trauma patients, concomitant CT cystography is frequently selected as a more efficient means to assess the bladder.

Management

Goals of Treatment –

- Control symptoms
- Repair the injury
- Prevent complications

Immediate catheterization should be performed when blunt bladder rupture is suspected because the most reliable indicator is gross hematuria, which is present in nearly all cases.

If blood is noted at the meatus, or catheter cannot be passed easily, retrograde urethrography should be done, as urethral injury occurs concomitantly in 10-29% cases of bladder injury.

Extraperitoneal - In uncomplicated extraperitoneal bladder ruptures, when conditions are ideal, conservative management with urethral catheter drainage alone is often sufficient. Catheter may be removed after 14 days, after cystographically confirming that complete healing has occurred.

Occasionally, extravasation may persist for several additional weeks, but it resolves with continuation of urethral catheter drainage, after which radiographic confirmation of healing is essential.

In cases where exploration is required, dome of the bladder is opened in midline, bladder is inspected from the inside, ureteral orifices and urethra located, and extraperitoneal injuries repaired from the inside using single layer of interrupted 2-0 or 3-0 absorbable suture.

Bone spicules within the bladder wall may compromise healing. Antimicrobial agents are instituted on the day of injury and continued for at least 1 week to prevent infection of the pelvic hematoma.

Intraperitoneal - All penetrating or intraperitoneal injuries resulting from external trauma should be managed by immediate operative repair.

These injuries are often larger than suggested on cystography and are unlikely to heal spontaneously, and continued leak of urine causes achemical peritonitis. Although most injuries are repaired with open surgery, select patients may undergo laparoscopic repair only if facilities and expertise is available..

Intraperitoneal rupture is closed with 2 layers of running absorbable sutures, after inspecting the bladder from inside and identifying the ureteral and urethral orifices.

Any penetrating injury involving the ureteral orifice or intramural ureter warrants primary closure with stented reimplantation of the ureter. A peri-vesical drain should be employed. In patients with intraperitoneal rupture, antimicrobial agents are administered for 3 days in the perioperative period only. If the bladder has been repaired, a cystogram is obtained 7 to 10 days after surgery. Several more recent studies have shown that suprapubic tube drainage provides no benefit over urethral catheter drainage alone, although maximal urinary drainage using both is recommended when complex injuries are encountered. When concurrent rectal or vaginal injuries exist, the organ walls should be separated, overlapping suture lines should be avoided, and every attempt should be made to interpose viable tissue in between the repaired structures. Fibrin sealant injected over the bladder wall closure may help reduce complications when intervening tissue is unavailable.

Complications

Complications are usually associated with delayed diagnosis or delayed treatment because of misdiagnosis, delayed presentation, or complex injuries resulting from devastating pelvic trauma.

Unrecognized bladder injuries may manifest as acidosis, azotemia, fever and sepsis, low urine output, peritonitis, ileus, urinary ascites, or respiratory difficulties.

Unrecognized bladder neck, vaginal, and rectal injury associated with the bladder rupture can result in incontinence, fistula, stricture, and difficult delayed major reconstruction.

Severe pelvic fractures may cause a transient or permanent neurologic injury and result in voiding difficulties despite an adequate bladder repair.

Referral Criteria:

ICU care may be needed in patients who are haemodynamically unstable.

Blood bank facilities are mandatory.

Patient may have to be referred for CT Scan, if CT Scan facility is not available.

But, patient should not be shifted for CT Scan until he has been resuscitated, stabilized, and is a responder to resuscitation.

Transfer to higher centers may be needed in case of unavailability of expert care. But, transfer should be done only after haemodynamically stabilizing the patient.

Medicolegal Issues

Failure to detect a patient in shock

Failure to resuscitate

Failure to perform necessary diagnostic tests, if facilities are available

Failure to refer a stabilized patient to a higher centre, if expert facility is not available.

Delay in treatment.

Delay in diagnosing complications and taking corrective action.

WHO DOES WHAT?

Surgeon:-	Diagnosis & Work up Pre operative resuscitation in conjunction with Anaesthetist / Intensivist Pre operative planning Operative procedure Post operative care in conjunction with Anaesthetist / Intensivist Post operative follow up
Anesthetist:-	Pre Anaesthesia Check up Part of resuscitation Prforming anesthesia Post op ICU management in conjunction with Surgeon
Nurse:-	Pre/Intra/Postop comprehensive care Dressing of the wound
Technician:-	Pre op equipment and drugs to be checked and kept ready Assist anesthetist in the OT Assist the surgeon, positioning of the patient

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE

Human Resources

1. Surgeon – 1
2. Medical Officer /Assistant Surgeon – 2
3. Anaesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 2
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test
4. Liver Function Test
5. Serum Serology
6. Electrolytes
7. ECG
8. X-Ray – Chest
9. USG
10. CT Scan abdomen with / without retrograde cystogram

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anaesthesia Machine, instruments including endotracheal tubes & drugs
4. Multi channel monitor
5. Set of general surgical Instruments (including bulldog clamps, vascular instruments)
6. Suction apparatus
7. Sutures (including 5-0/ 6-0 nonabsorbable vascular sutures)
8. Drains
9. Catheters
10. Diathermy
11. Antibiotics
12. Analgesics
13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as per decision and expertise of Surgeon.
16. Blood bank facilities

Blunt Trauma Abdomen

Introduction

Identification of serious intra-abdominal injury following blunt trauma is often challenging. Unlike penetrating trauma, wherein bowel injuries are more common, solid organ injuries are more common in a vehicular accident. Many injuries may not manifest during the initial assessment and treatment period. More obvious injuries such as limb injuries are distracting and diagnosis of blunt abdominal injuries which are always hidden and may be missed unless looked for. A high index of suspicion & correlation with mechanism of injury is an essential factor for early diagnosis & treatment.

Indian Incidence

There are no statistics available on this topic.

Symptoms and signs

The initial clinical assessment of patients with blunt abdominal trauma is often unreliable. The signs and symptoms are also absent in intoxicated, unconscious and paralysed patients. Hence it is important to rule out abdominal injury in all such patients and in those injured by a dangerous mechanism e.g. car crash, pedestrian hit by car, fall from height, blunt assault, deceleration injury etc. Abdominal injury should also be ruled out in patients with fractures involving lower ribs, thoracolumbar spine or pelvis.

The most reliable signs and symptoms in alert patients are as follows:

- Pain
- Tenderness
- Febrile Episodes (Fever may occur later when sepsis sets in)
- Gastrointestinal hemorrhage
- Signs of Hypovolemia - Cool, clammy extremities, tachycardia, hypotension
- Evidence of peritoneal irritation - Tenderness, guarding, rigidity

However, large amounts of blood can accumulate in the peritoneal and pelvic cavities without any significant or early changes in the physical examination findings.

On physical examination, the following injury patterns predict the potential for intra-abdominal trauma:

- Lap belt marks: Correlate with small intestine rupture

- Steering wheel–shaped contusions
- Ecchymosis involving the flanks (Grey Turner sign) or the umbilicus (Cullen sign): Indicates retroperitoneal hemorrhage, but is usually delayed for several hours to days
- Abdominal distention
- Auscultation of bowel sounds in the thorax: May indicate a diaphragmatic injury
- Abdominal bruit: May indicate underlying vascular disease or traumatic arteriovenous fistula
- Local or generalized tenderness, guarding, rigidity, or rebound tenderness: Suggests peritoneal inflammation
- Fullness and doughy consistency on palpation: May indicate intra-abdominal hemorrhage
- Crepitation or instability of the lower thoracic cage: Indicates the potential for splenic or hepatic injuries.

Management

Resuscitation/ Primary survey

Resuscitation is performed concomitantly and continues as the physical examination is completed. Priorities in resuscitation and diagnosis are established on the basis of hemodynamic stability and the degree of injury. The goal of the primary survey, as directed by the Advanced Trauma Life Support (ATLS) protocol, is to identify and expediently treat life-threatening injuries. The protocol includes the following:

- Airway, with cervical spine movement restriction
- Breathing and ventilation
- Circulation and control of hemorrhage
- Disability
- Exposure & Environmental control

The abdomen should neither be ignored nor be the sole focus of the treating clinician and surgeon. In an unstable patient, the question of abdominal involvement must be expediently addressed. This is accomplished by identifying free intra-abdominal fluid with diagnostic peritoneal lavage (DPL) or focused assessment with sonography for trauma (FAST). The objective is rapid identification of those patients who need an urgent Laparotomy.

Secondary survey

After an appropriate primary survey and initiation of resuscitation, attention should be focused on the secondary survey of the abdomen. The secondary survey is the identification of all injuries via a head-to-toe examination. For life-threatening injuries that necessitate emergency

surgery, a comprehensive secondary survey should be delayed until the patient has been stabilized.

At the other end of the spectrum are victims of blunt trauma who have a benign abdomen upon initial presentation. Many injuries initially are occult and manifest over time. Frequent serial examinations, in conjunction with the appropriate diagnostic studies, such as abdominal computed tomography (CT) and bedside ultrasonography, are essential in any patient with a significant mechanism of injury.

The evaluation of a patient with blunt abdominal trauma must be accomplished with the entire patient in mind, with all injuries prioritized accordingly.

The initial clinical assessment of patients with blunt abdominal trauma is often difficult and notably inaccurate. Associated injuries often cause tenderness and spasms in the abdominal wall and make diagnosis difficult. Lower rib fractures, pelvic fractures, and abdominal wall contusions may mimic the signs of peritonitis.

In general, accuracy increases if the patient is re-evaluated repeatedly preferably by same surgeon and at frequent intervals. However, repeated examinations may not be feasible in patients who need general anesthesia and surgery for other injuries. The greatest compromise of the physical examination occurs in the setting of neurologic dysfunction, which may be caused by head/spine injury or substance abuse.

The respiratory pattern should be observed because abdominal breathing may indicate spinal cord injury. A sensory examination of the chest and abdomen should be performed to evaluate the potential for spinal cord injury. Spinal cord injury may interfere with the accurate assessment of the abdomen by causing decreased or absent pain perception.

Rectal and bimanual vaginal pelvic examinations should be performed. A rectal examination should be done to search for evidence of bony penetration resulting from a pelvic fracture, and the stool should be evaluated for gross or occult blood. The evaluation of rectal tone is important for determining the patient's neurologic status.

The genitals and perineum should be examined for soft tissue injuries, bleeding, and hematoma. Pelvic instability indicates the potential for lower urinary tract injury, as well as pelvic and retroperitoneal hematoma. Open pelvic fractures are associated with a mortality rate exceeding 50%.

A nasogastric tube should be placed routinely (in the absence of contraindications, eg, basilar skull fracture) to decompress the stomach and to assess for the presence of blood. If the patient has evidence of a maxillofacial injury, an orogastric tube is preferred.

As the assessment continues, a Foley's catheter is placed and a sample of urine is sent for analysis for microscopic hematuria. If injury to the urethra or bladder is suggested because of an associated pelvic fracture, a retrograde urethrogram is performed before catheterization.

With respect to the primary and secondary surveys, paediatric patients are assessed and treated—at least initially—as adults. However, there are obvious anatomic and clinical differences between children and adults that must be kept in mind, including the following:

- A paediatric patient's physiologic response to injury is different.
- Effective communication with a child is not always possible.
- Physical examination findings become more important in children.
- A paediatric patient's blood volume is smaller, predisposing to rapid exsanguinations.
- Technical procedures in paediatric patients tend to be more time consuming and challenging.
- A child's relatively large body surface area contributes to rapid heat loss.

Perhaps the most significant difference between paediatric and adult blunt trauma is that, for the most part, paediatric patients can be resuscitated and treated nonoperatively. Some paediatric surgeons often transfuse up to 20ml/kg of blood products after initial bolus of crystalloid (20ml/kg) in an effort to stabilize a paediatric patient (ATLS guidelines). Obviously, if this fails and the child continues to be unstable, laparotomy is indicated.

Tertiary survey

The concept of the tertiary trauma survey was first introduced to assist in the diagnosis of any injuries that may have been missed during the primary and secondary surveys. The tertiary survey involves a repetition of the primary and secondary surveys and a revision of all laboratory and radiographic studies.

Complications

Complications associated with blunt abdominal trauma include but are not limited to the following:

- Missed injuries
- Delays in diagnosis
- Delays in treatment

- Iatrogenic injuries
- Intra-abdominal sepsis and abscess
- Inadequate resuscitation
- Delayed splenic rupture

In patients that undergo laparotomy and repair, complications are similar to other conditions that require operative intervention.

Commonly recommended studies include serum glucose, complete blood count (CBC), serum chemistries, serum amylase, urine analysis, coagulation studies, blood typing and cross-matching, [arterial blood gases \(ABGs\)](#), blood ethanol, urine drug screens, and a urine pregnancy test (for females of childbearing age).

Serum electrolyte values, creatinine level, and glucose values are often obtained for reference, but typically they have little or no value in the initial management period.

Aggressive radiographic and surgical investigation is indicated in patients with persistent hyperamylasemia or hyperlipasemia, conditions that suggest significant intra-abdominal injury.

All patients should have their tetanus immunization history reviewed. If it is not available, prophylaxis should be given.

Plain Radiography

Although their overall value in the evaluation of patients with blunt abdominal trauma is limited, plain films can demonstrate numerous findings. The chest radiograph may aid in the diagnosis of abdominal injuries such as ruptured hemidiaphragm (eg. a nasogastric tube seen in the chest) or pneumoperitoneum.

The pelvic or chest radiograph can demonstrate fractures of the thoracolumbar spine. The presence of transverse fractures of the vertebral bodies (ie, Chance fractures) suggests a higher likelihood of blunt injuries to the bowel. In addition, free intraperitoneal air, or trapped retroperitoneal air from duodenal perforation, may be seen.

USG

Bedside ultrasonography is a rapid, portable, non-invasive, and accurate examination that can be performed by emergency clinicians and trauma surgeons to detect hemoperitoneum. In fact the FAST examination has virtually replaced DPL as the procedure of choice in the evaluation of hemodynamically unstable trauma patients. The current FAST examination protocol consists of 4 acoustic windows with the patient supine. These windows are pericardiac, perihepatic,

perisplenic, and pelvic (known as the 4 P s). An examination is interpreted as positive if free fluid is found in any of the 4 acoustic windows and as negative if no fluid is seen. An examination is deemed indeterminate if any of the windows cannot be adequately assessed.

Hemodynamically stable patients with positive FAST results may require a Computed Tomography (CT) scan to define the nature and extent of their injuries better. Taking every stable patient with a positive FAST result to the operating room may result in an unacceptably high laparotomy rate and is not recommended.

Hemodynamically stable patients with negative FAST results require close observation, serial abdominal examinations, and a follow-up FAST examination. However, if by mechanism and clinical examination, abdominal injury is strongly suspected, if the patient is intoxicated or has other associated injuries.

Positive FAST in a haemodynamically unstable patient is strongly indicative of intraperitoneal bleeding and an emergency exploratory laparotomy is recommended. Negative FAST in a haemodynamically unstable patient rules out major intraperitoneal bleeding and search must be made for other sources of bleeding in the chest, pelvis, long bones or external wounds by clinical examination, appropriate imaging.

Computed Tomography

Plain CT scan alone does not provide essential information. So it's always to be followed by IV contrast in which arterial, venous and delayed phase of CT scan abdomen should be done to fully evaluate intraabdominal injuries.

Although expensive and potentially time-consuming, CT scanning often provides the most detailed images of traumatic pathology and may assist in determination of operative intervention. CT remains the standard criterion for the detection of solid organ injuries and can influence the management decision. In addition, a CT scan of the abdomen can reveal other associated injuries, notably vertebral and pelvic fractures and injuries in the thoracic cavity.

CT scan, unlike DPL or FAST, has the capability to determine the source of hemorrhage. In addition, many retroperitoneal injuries go unnoticed with DPL and FAST examinations.

Transport only hemodynamically stable patients to the CT scanner. When performing CT scans, closely and carefully monitor vital signs for clinical evidence of decompensation. Preliminary evidence suggests that a flat vena cava on CT scan is a marker for underresuscitation and may be correlated with higher mortality and hemodynamic decompensation.

CT scans provide excellent imaging of the pancreas, duodenum, and genitourinary system. The images can help quantitate the amount of blood in the abdomen and can reveal individual organs with precision. The primary advantage of CT scanning is its high specificity and use for guiding nonoperative management of solid organ injuries.

Drawbacks of CT scan relate to the need to transport the patient from the trauma resuscitation area and the additional time required to perform CT scan compared to FAST or DPL.

Although some pancreatic injuries may be missed with a CT scan performed soon after trauma, virtually all are identified if the scan is repeated in 24-48 hours. For selected patients, endoscopic retrograde cholangiopancreatography (ERCP) may complement CT scan to rule out a ductal injury.

Finally, CT scan is relatively expensive and time consuming and requires oral or intravenous (IV) contrast, which may cause adverse reactions. The best CT imaging requires both oral and IV contrast. Some controversy has arisen over the use of oral contrast and whether the additional information it provides negates the drawbacks of increased time of administration and risk of aspiration. The value of oral contrast in diagnosing bowel injury has been debated, but no definitive answer exists at this time. Oral or rectal contrast is generally not administered unless one is specifically looking for bowel injury.

Nonoperative Treatment

Nonoperative management (NOM) strategies based on CT scan diagnosis and the hemodynamic stability of the patient are now being used in adults for the treatment of solid organ injuries, primarily those to the liver and spleen. In blunt abdominal trauma, including severe solid organ injuries, selective nonoperative management has become the standard of care.

Angiography is a valuable modality in nonoperative management of abdominal solid organ injuries from blunt trauma in adults. It is used aggressively for nonoperative control of hemorrhage, thereby obviating nontherapeutic cost-inefficient laparotomies.

If the decision has been made to observe the patient, closely monitor vital signs and frequently repeat the physical examination. An increased temperature or respiratory rate can indicate a perforated viscus or the formation of an abscess. Pulse and blood pressure can also change with sepsis or intra-abdominal bleeding. Physical examination findings reflecting peritonitis are an indication for surgical intervention.

When can patient be treated non-operatively?

1. Hemodynamically stable

2. CT scan shows solid organ injury (Liver or spleen)[NOM of pancreatic injury needs MRCP and ERCP facility and NOM of renal injury needs facility for ureteric stenting]
3. No evidence of active bleeding / dye extravasation unless there is facility for interventional radiology.
4. No evidence of bowel perforation or ischaemia.
5. ICU observation for 48 hours.
6. Facility for emergency intervention if needed.

Surgical Treatment

Treatment of blunt abdominal trauma begins at the scene of the injury and is continued upon the patient's arrival at the emergency department (ED) or trauma center. Management may involve nonoperative measures or surgical treatment, as appropriate.

Indications for laparotomy in a patient with blunt abdominal injury include the following:

- Signs of peritonitis
- Shock with evidence of intra-abdominal bleed on USG or DPL
- Clinical deterioration during observation - Development of hypotension or signs of peritonitis
- In stable patients, evidence of bowel injury pneumoperitoneum) or ischemia on CT scan (bowel wall thickening, intramural pneumatosis)
- In stable patients evidence of diaphragmatic injury on CT scan
- In stable patients evidence of ongoing bleed on CT scan and non-availability of facility of interventional radiology

Operative treatment is not indicated in every patient with positive FAST scan results. Hemodynamically stable patients with positive FAST findings require a computed tomography (CT) scan to better define the nature and extent of their injuries. Operating on every patient with positive FAST scan findings may result in an unacceptably high laparotomy rate.

When laparotomy is indicated, broad-spectrum antibiotics are given. A midline incision is usually preferred. When the abdomen is opened, hemorrhage control is accomplished by removing blood and clots, packing all 4 quadrants, and clamping vascular structures. Obvious hollow viscus injuries (HVI) are sutured. After intra-abdominal injuries have been repaired and hemorrhage has been controlled by packing, a thorough exploration of the abdomen is then performed to evaluate the entire contents of the abdomen.

After intraperitoneal injuries are controlled, the retroperitoneum and pelvis must be inspected. Do not explore pelvic hematomas. Use external fixation of pelvic fractures to reduce or stop

	Operative procedure Post operative care in conjunction with Anaesthetist/Intensivist Post operative follow up
Anesthetist:-	Pre Anaesthesia Check up Part of resuscitation Performing anesthesia Post op ICU management in conjunction with Surgeon
Radiologist:-	FAST if Surgeon needs help Repeat Ultrasounds CT Scans Special Investigations
Nurse:-	Pre/Intra/Postop comprehensive care Dressing of the wound
Technician:-	Pre op equipment and drugs to be checked and kept ready Assist anesthetist in the OT Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (patient weight 60 kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer /Assistant Surgeon – 1
3. Anesthetist – 1
4. Radiologist – 1 ----Services from outside can be availed
5. Pathologist – 1-----Services from outside can be availed
6. Staff Nurse – 1
7. Technician – 1
8. Nursing Orderly – 1
9. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test in selected cases
4. LFT in selected cases

5. S. Electrolytes in selected cases
6. USG (Consider DPL if USG is not available)
7. ECG
8. X- Ray – Chest
9. CT scan in selected patients
10. Histopathology

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. Tranexamic acid
14. I.V. Fluids
15. Dressings
16. Access to blood and blood product

Burns

The Problem

Burns has always been, and will continue to remain, a significant public health problem in low/middle-income countries. An estimated 163000 deaths occur annually due to burn-related injuries. However the patterns of burn injuries in India are different from what is observed elsewhere in the world.

1. There exists a higher incidence amongst females.
2. India also faced the highest number of self-inflicted burn injuries.
3. There are generally associated with larger body surface involvement.
4. And therefore, with higher mortality rates.

Several studies have shown that age, gender, total body surface area involved are significant predictors of mortality (5). But the delay in accessing and receiving appropriate care is also a statistically significant predictor of mortality (5). Emergency medical services comprising of an unorganised system of ambulances both in the private and public sectors might help in increasing the access to care at least amongst the urban population. An accurate evaluation with appropriate initial management too would go a long way in improving the survival outcome of these patients. This, coupled with appropriate early referral to a specialist, would greatly help in minimising the suffering and optimising the results (8).

Currently most practice guidelines on this topic deal with the management of burn patients in developed countries or by Plastic Surgeons. However in India, it would be a general surgery graduate that many burn patients would encounter in most practice settings in the country. The Association of Surgeons of India has therefore taken upon itself the task of advising the government and providing training and education to its member surgeons practicing in various medical settings in the society. The guidelines have put forth a consensus opinion on recommendations for various aspects of burn care, for the General Surgeon. There are obviously limitations in what can be done for the patient and therefore the guidelines have also provided suggestions as to when the patient should be referred to a higher centre for a Plastic Surgeon's expert opinion.

Upon arrival of a burn patient to the casualty / emergency room, the primary survey should comprise of (6,7):

- Airway management
- Breathing and ventilation
- Circulation and cardiac status

- Disability, neurologic deficit and gross deformity
 - Exposure (completely disrobe the patient, examine for associated injuries and maintain a warm environment)
1. First evaluate the airway to assess if it is compromised or is at risk of compromise.
 - A. If it is compromised, emergency intubation and continued ventilatory support will be required.
 - B. If the airway patent, it is important to decide if the patient is at risk of developing an airway obstruction in the immediate 2-3 days. This risk is seen with the presence of inhalation injury. To arrive at a clinical diagnosis of inhalation injury, ask if the fire was in a closed space, thus predisposing to inhalation injury. Clinical signs of inhalation injury that would suggest that airway obstruction is imminent would include carbonaceous sputum, blistering of the mucosal membrane of buccal cavity, oropharyngeal edema, stridor or hoarseness of voice or altered mental status. Early endotracheal intubation and mechanical ventilation would be indicated if these are present (8).
 - C. Intubation and sedation itself is not without risk and could worsen a patient's condition. Mere facial burns with singed facial or nasal hairs do not indicate inhalational injury and therefore does not warrant an elective endotracheal intubation. For example, flash burns often harm the face but rarely involve the airway. Humidified supplemental oxygen would be sufficient supportive care until upper airway edema subsides (8).
 2. Breathing assessment is done once the airway is secure to assess the patient's ability to adequately ventilate and oxygenate.
 - A. Document the auscultatory findings for bilateral breath sounds, the respiratory rate and the depth of respiration.
 - B. It is important to look for circumferential burns of the trunk or neck that may impair the patient's ability to breathe. Treatment for this would be the performance of a rapid bedside escharotomy (9).
 3. Upon presentation itself, all major burn patients should be placed on a cardiac monitor with a continuous pulse oximeter and BP evaluation (8).
 - A. Peripheral, central and intraosseous routes are available for access and may safely be placed through burnt tissue if necessary. Oral resuscitation is only feasible for burns smaller than 30%. When used in larger burns, vomiting with an increased risk for aspiration exists.

- B. A heart rate of 100–120 beats per minute is considered normal given the increased catecholamine response. A higher rate would therefore indicate inadequate volume resuscitation, pain management or other trauma (6).
 - C. Fluid management based on weight of patient and the burn size could be done after further assessment of the patient is complete (10,11). There is no need to administer any fluid bolus unless signs of hypovolemia (hypotension etc) exist. In fact bolus administration only leads to further exacerbation of edema and should therefore be avoided.
 - D. As part of the circulatory assessment, evaluate the perfusion of all extremities. If there are any circumferentially burnt extremities, then an emergency bedside escharotomy is indicated. Do not wait for disappearance of a palpable distal pulse.
- 4. Disability, deficit and deformity. Evaluate the mental status via the Glasgow Coma Scale (GCS) (9). The possibility of associated injury, substance use, hypoxia, inhalation injury or a pre-existing condition should always be addressed as part of the history of the event.
 - 5. Exposure: The patient must be appropriately disrobed to assess for injury. All jewellery and other accessories should be removed to prevent a tourniquet effect developing (9). Use clean blankets to prevent or limit hypothermia during the examination process. It is meaningless to pour cold water upon wounds as the temperature would have most likely equilibrated by that time the patient comes to the hospital. In any case, ice and cold water can cause vasoconstriction and even hypothermia.

Thereafter, a secondary survey, which includes burn-specific intervention, might be initiated. This involves:

- A. Examination for non-burn related life-threatening injuries.
- B. Imaging, laboratory analyses and adjunctive measures such as urethral catheters, nasogastric tubes, etc. should be completed at this time.
- C. Assessment of the TBSA or Total Body Surface Area involved.
 - 1. During the primary survey only a rough estimate of the area of involvement and depth is required for initiation of the fluid resuscitation. Therefore now, during the secondary survey, a precise assessment is made.
 - 2. Patients at the extremes of age with very thin skin are more susceptible for extensive injuries from a relatively less intense thermal insult (9).

3. The extent of burn is estimated using the Rule of Nines (12) for adults and the Lund-Browder chart (13) for infants and children. The size of the patient's palm, including the fingers, is approximately one percent TBSA (14).

D. Inhalation injury

1. Systemic poisoning due to the inhalation of gases produced by combustion, such as carbon monoxide (CO) and hydrogen cyanide (HCN) is suggested by a decreased level of consciousness together with a history of exposure to fire in an enclosed space. Measurement of blood carboxyhemoglobin (COHb) is the confirmatory test for a diagnosis of carbon monoxide poisoning. A facility that has the capability to analyze arterial blood gases can do this at a little extra cost. Most often the diagnosis is made clinically. With a clinical suspicion of having CO poisoning, the patient should immediately be given high-flow oxygen at a rate of 8–15 L/min for at least 6 hours or longer if symptoms persist. Empirical treatment for hydrogencyanidepoisoning too involves administration of high-flow oxygen. Similarly, the early effects of the inhalation of smoke on the trachea-bronchial tree results in hypoxia, which again is treated by the administration of oxygen. The use of oxygen as an initial treatment of fire victims is therefore potentially life-saving. Intubation and mechanical ventilation would be dictated by the level of consciousness (8).
2. Edema formation in the head and neck may be insidious and can become manifest within up to 24 h or longer post burn. If the patient has not been intubated, it is necessary to watch for any upper airway obstruction developing with time. Moderate elevation of the head of the bed allows gravity to help reduce airway edema by facilitating venous and lymphatic drainage. Clinical features one must look for would include symptoms of hoarseness or stridor, cough, tachypnea, rhonchi on auscultation, and hypoxemia on pulse oximetry. The patient should be given oxygen by mask to maintain adequate arterial oxygen saturation. Suction should be used to keep the airway clear of debris and secretions.
3. Normal chest radiographs do not exclude the diagnosis. The observation of damaged mucosa below the larynx on bronchoscopy is definitive (15).

E. Fluid resuscitation for burn patients.

1. Adult patients with burns > 20% total burn surface area (TBSA), and pediatric patients (including infants) with burns > 10% TBSA, should be formally resuscitated.
2. Resuscitation should always aim at providing just adequate perfusion using the smallest quantity of fluid possible. Over-resuscitation is physiologically detrimental and

can lead to compartment syndrome of the extremities and abdomen or worsen respiratory distress. Under-resuscitation too can further worsen burn shock and lead to organ failure. (10,16).

3. The resuscitation formula most commonly used to guide burn resuscitation is the Parkland formula. Lactated Ringer's solution can be initiated at the rate of 4 mL/kg/% burn over a 24-hour period, with the first half over 8 hours and the next half over the following 16 hours (16). It is important to calculate the 8 and 16 hour periods from the time of the burn injury occurring.
4. Though colloids are suitable for safer resuscitation especially when large volumes are involved, their prohibitive costs have virtually removed them from local standard practice patterns (8).
5. Oral resuscitation :
 - A. Patients with burns < 30% TBSA are candidates for oral resuscitation
 - B. Early oral intake can be used to offset intravenous resuscitation volume requirements for patients with larger burns (10).
 - C. If only oral fluid administration is practical, drinking liquids (typical of the local diet) equivalent to 15% of the body weight every 24 hours is recommended for two days. One level teaspoon of table salt (or the equivalent) must be ingested for each litre of oral fluids. Administering the fluids orally as frequent, small amounts (50 mL or less) allows for oral fluid resuscitation without inducing vomiting.
 - D. Some of the fluids that could be used would include rice water (kanjee) with salt, fresh lime water with salt and sugar, vegetable or chicken soup with salt, lassi (yogurt drink with salt and sugar), sports drink (e.g., Gatorade) with 1/4 tsp salt and oral rehydration solutions (ORT) used for infectious diarrhoea.
6. Monitoring the urine output is still the most sensible and reliable method to ensure adequate resuscitation. A urine output of 0.3–0.5 mL/kg/h in adults and 1.0 mL/kg/h in children (10,16) should be the aim. For the first 3 hours of resuscitation, urine output values may approach anuria, irrespective of the adequacy of fluid resuscitation. This is most often not due to inadequate resuscitation but due to a lag time taken for physiological equilibration. If more aggressive fluid therapy is attempted, it will only lead to fluid overload.
7. Resuscitation formulas serve merely as a guide. Patients should be resuscitated based on their physiologic needs, not entirely from numbers dictated by a formula.
8. Formula instructions further recommend that paediatric patients require more fluid for burns comparable to those of adults due to the increase in body surface area-to-weight ratio (10). Maintenance fluids, including a source of glucose, should be added to the paediatric patient resuscitation fluid as hepatic glycogen stores will be depleted after 12–14 h of fasting (10).

9. For other subtypes of patients, including those with inhalation injuries, electrical burns, exceedingly deep burns (e.g., 4th degree injuries) and those with inadequate or delayed resuscitation, have been shown to demonstrate additional fluid needs (17).
10. The importance of early initiation of tailored resuscitation is thus emphasised.

F. Bedside escharotomies for limbs and trunk

1. May be required immediately after admission or even up to 72 hours after burn injury.
2. In the limbs
 - a. Sluggish capillary filling in the nail beds, taking greater than 3 seconds, is not always diagnostic of hypoperfusion secondary to restrictive circumferential burns. One must ensure there are no systemic causes of distal hypoperfusion, such as hypoxia, decreased cardiac output, hypovolemia, or peripheral arterial constriction (19).
 - b. The presence of distal pulses does not rule out the presence of early compartment syndrome, because the amount of pressure required to reduce arteriolar or capillary filling is much less than that required to cease blood flow in the larger arteries. (8).
 - c. Progressively weakening or absent Doppler signals is definitely an indication for escharotomy (8).
 - d. Pulse oximetry values > 95% suggest adequate distal perfusion, whereas values < 90% indicate a need for escharotomy. Values between 90% and 95% are concerning but require further investigation. (But remember that carbon monoxide poisoning will falsely elevate the oxyhemoglobin level, leading to a false negative finding) (8).
 - e. Direct intra-compartmental pressure measurement, if available, helps in the decision. Compartmental pressure below 25 mmHg is associated with adequate tissue perfusion, while pressure above 40 mmHg is an absolute indication for escharotomy. Pressures between 25 and 40 mmHg require clinical correlation with other findings (18-24).
 - f. In cases of doubt, and to avoid unnecessary escharotomies, first elevate the limbs. Watch for spontaneous resolution of edema by gravity. If the compromise persists, the escharotomy should be performed (18-24).
 - g. Escharotomy incisions should be placed longitudinally along the neurovascular bundles, extending from normal skin to normal skin or from joint above to joint below and up to healthy tissue at the base. On the limbs and fingers they are longitudinal in the medial and lateral mid-axial lines with additional incisions on the dorsum of the hands and feet if they are involved. In Abdominal Compartment syndrome (ACS) though the external restriction by the eschar does not play a major role, escharotomy incisions are made longitudinally with transverse incisions in the upper parts of the thorax and abdomen.
 - h. It is important to keep in mind the main complication associated with this limb and lifesaving procedure, i.e., an inadequate release. (8). Other complications including damage to the underlying structures are infrequent if they are correctly placed. (8).

G. Fasciotomy

1. Rarely indicated as a primary procedure in burns
2. Fasciotomy is more difficult technically than escharotomy. It requires general anesthesia and therefore, is not a bedside procedure.
3. As the deep fascia has to be cut to release the compartments, the potential for injury to the underlying neurovascular bundles is higher.
4. In non-electric burns, escharotomy alone should be performed initially. If clinically the compartment syndrome persists, fasciotomy should be done. Compression of deep structures, such as nerves, may lead to paresthesia and pain on passive muscle stretching is an indication for fasciotomy (8).
5. In electric burns, fasciotomy should be directly done as it also allows direct inspection of muscles for early excision of the necrotic tissue. (8).
6. There is no role for closed fasciotomies in burns; all the fasciotomies should be open (8).
7. Complications of fasciotomy are the same as those of escharotomy but occur much more commonly, particularly the injury to neurovascular bundles and deeper structures (24).

H. Tetanus immunization status should be evaluated and addressed if indicated. Patients who are current with vaccination status (3 doses TT + booster every year) require no further treatment. Burn patients with unknown or inadequate vaccination status should receive TT in addition to tetanus immune globulin (TIG) (27). The minimal risk accompanying vaccination and the benefit-to-harm ratio heavily weighted toward the benefit should be conveyed to the patient's relatives.

I. Abdominal compartment syndrome (ACS) :

1. Suspected clinically when there is an unexplained reduction in minute ventilation, oliguria, or both in a major burns patient who has already received fluid resuscitation beyond the calculated value. (8).
2. The diagnosis can be confirmed by inserting a catheter into the urinary bladder and measuring the intravesical pressure (IVP). The normal IVP is < 5 mmHg but up to 12 mmHg may be acceptable. Values above 25 mmHg necessitate intervention, while values between 12 and 25 mmHg requires close follow-up (25,26). Inserting a venous femoral catheter and observing for any slowing or interruption of flow in the IV fluid also suggests the development of this complication. (8).

J. Wound care

- a. Mechanical cleansing by wound irrigation significantly correlates with decreasing the bacterial count in the wound (30-33). Running tap water, not stored, is the most cost-effective (better than even saline) and should be the rule for burn wound cleansing. Swabbing the wound with antiseptics or antimicrobial agents during cleansing did not offer any benefit with regards to wound contamination, healing, patient satisfaction or cost-effectiveness (34). Instead antiseptics/antimicrobials could be used as topicals after tap water/saline irrigation or after surgical debridement, to prevent the exposed bacteria from entering the blood stream (8).
- b. Management of superficial burns with blisters :
 - 1 Majority of the guidelines and studies suggested de-roofing of the blister and coverage with biologic or modern dressing. However, when the blisters are not impending rupture or impeding movement or dressing (even if they are of considerable size), it is recommended that they be left intact (8). When the wall of the blister is thin and likely to rupture, de-roofing or evacuation is the most appropriate choice. If the blister has been de-roofed, then an interface (tulle with or without paraffin or vaseline) should be applied to the wound, followed by topical antimicrobial cream (water-soluble base) and the wound is then closed with bulky fluffy dressing to ensure as much moisture retention as possible (8).
 - 2 Snipping open of blisters seems to be a valuable and cost-effective technique. As for de-roofing, it does not impede movement. It reduces pain and can provide a temporary "biologic" dressing that would even ensure a reduced ooze from the wound surface. An interface should be applied followed by antimicrobial cream with a fluffy dressing (8).
 - 3 Frequency of change of dressing: It is best to leave the dressing as long as possible over the wound. If however, there is significant soakage of the wound, then the interface can be left behind and the outer layers alone changed. When the patient presents late (more than 24 h after the accident), the dressing should be changed every other day until the absence of infection is assured and the wound has begun healing and then the wound dressing could be left for a longer period (8).
- c. Management of deep partial and full thickness burns :
 1. During the first few hours post burn, wounds are generally sterile or are at the stage of superficial bacterial colonisation. But by day 4-5, extensive bacterial involvement of damaged skin occurs, which progressively worsens to even cause invasion of unburnt tissue by about one week (35-39).

2. In the emergency situation, any moist dressing can be applied to the wound after patient stabilisation (39). The primary goals of further wound dressing are the prevention of desiccation of viable tissue and the control of bacterial growth (39).
3. For deep partial and full thickness burn eschar, tangential excision and grafting is the ideal treatment (8). Until this is done, it is imperative that the wound be kept moist and covered with a closed dressing technique. The eschar then remains soft and protected from infection. Also, heat and fluid loss is reduced. But the closed technique prevents autolysis beneath the eschar and therefore delays its spontaneous separation for several weeks. If the closed technique is not done, these wounds, upon drying out, will be covered with a dry scab, which will make tangential excision difficult.
4. For deep partial and full thickness burn eschars if early excision is not being planned, then they could be dressed by the open technique until eschar separation begins. The eschar separates itself from the bed by autolysis and can be cut away in stages during the regular dressings (40). After removal of the eschar, the wound is treated as a raw area and should be dressed by the closed technique.
5. For facial burns and perineal burns, the open method only is done as the closed technique is often impractical (8).
6. When using non-adherent dressing, use successive strips rather than wrapping it around the wound as it helps minimize the risk of circulatory impairment (41).

When to transfer a burn victim to a higher centre?

It is a well documented fact that improved outcomes for burn victims are seen if they are treated in a facility capable of providing an advanced level of burn care (28). Therefore, it is important to accurately identify those patients with burns who need to be transferred to a Plastic Surgery Center, so that the outcome can be improved. In general, the recommendations in the literature regarding transferring a burn patient to a higher centre is based on the recommendations put forth by the American Burn Association (29).

They are as follows:

1. Partial thickness burns greater than 10% total body surface area (TBSA).
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
3. Third degree burns in any age group.
4. Electrical burns, including lightning injury.
5. Chemical burns.
6. Inhalation injury.

7. Burn injury in patients with preexisting medical disorders which could complicate management, prolonged recovery, or affect mortality.
8. Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit.
9. Burned children in hospitals without qualified personnel or equipment for the care of children.
10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

However, as mentioned before, the pattern of burn injuries in India is very different. Several factors therefore need to be taken into consideration.

1. Additional costs the patient will have to incur would include transport costs, higher costs required for advanced burn care etc. However the cost of inadequate care leading to loss of life or function and its burden on the family too needs to be remembered.
2. Hospitalised burn patients in India would rely heavily on their family for their care. Transferring such a patient to a distant location could jeopardise the quality of care.
3. The larger number of burn patients from the economically weaker sections of the society and the already overburdened tertiary care centres too have to be kept in mind.

Therefore we recommend the following changes in the commonly followed referral criteria.

1. We find no reason to transfer any 2nd degree superficial burns (eg. scald injuries) whatever be the area of involvement to a higher centre as appropriate dressings would be enough to heal the wound in less than 10 days. Even superficial burns involving the face, hands, perineum should heal with regular dressings.
2. To transfer patients with deep burn wounds (2nd, 3rd or 4th degree) for which tangential excision or other forms of surgical debridement are being planned.
3. To transfer patients with inhalation injury.
4. Amongst electrical burn injuries to transfer the high voltage injuries alone. Flash burns and other low-voltage electrical injuries can be managed as other thermal burns.
5. To transfer chemical burn injuries with wounds that will require surgical debridement.
6. To transfer burn injuries of any area and depth if the required supportive facilities are not available (eg. trauma center if concomitant trauma is also present).
7. Additionally we recommend all surgical units to have an association with the nearest Plastic Surgical Centre for guidance and referral. In the era of smartphones and internet, it would be useful for any general surgical service to seek the opinion of a Plastic Surgeon regarding

the depth of burn and other critical factors that would determine the further treatment plan. Many smartphone based applications exists that enable almost instantaneous transmission of photographs of wounds etc. The input he would receive would certainly help the surgeon take a very important decision as transferring or not transferring the patient to a advanced-care facility.

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Chest Trauma

Introduction

The thoracic cavity is the confluence of three major anatomical systems: the airway, upper gastro intestinal and the cardiovascular system. As such, any blunt or penetrating trauma in chest can cause significant disruption to each of these systems that can quickly prove to be life threatening unless rapidly identified and treated. Most common cause of chest trauma is road traffic accident. Official statistics of National Crime record Bureau (NCRB) states that 150,785 persons were killed and 494,624 injured in road traffic crashes in India in 2016. However unreported incidents are fairly high. The Global Burden of Disease (GBD) study estimates that there were 253,993 accidental deaths in India in 2016, almost 68% more than the deaths reported by traffic police [1]. Chest trauma may be isolated or associated with polytrauma and accounts for 25% of mortality in trauma patients[2,3,4] Approximately a third of chest trauma deaths occur before or shortly after reaching the treatment facility [4]. This rate is much higher in patients with polytraumatic injuries. Other causes of blunt and penetrating thoracic injury include being impaled by objects as a result of industrial accidents, falls, collisions, blast injuries, and fragmenting military device, natural disasters like earthquake, landslides. Injury by animals, both blunt and penetrating is rarely reported in English literature, but is commonly seen in patients from rural areas in Indian subcontinent, as animals are frequently used for agriculture and transport [5]

85-90% of chest trauma patients can be rapidly stabilized and resuscitated by a handful of critical procedures while at the same time obtaining history of **mechanism** of injury as in any trauma patient. The current mantra in the advanced care of chest trauma utilizes the concept of "scoop and run" in the treatment of injuries in the field. [6] With the development of modern (civilian) emergency medical services, field care of injured patients has improved. Rapid assessment to identify life-threatening injuries along with key interventions (ie, management of the airway and control of hemorrhage) and avoidance of massive volume increases before rapid transport to the closest appropriate facility is the current standard of care. This is in contrast to the concept of "stay and play," during which trained personnel make major triage and treatment decisions in the field.

Types of chest trauma:

- Blunt Trauma-> Blunt force to the chest wall
- PenetratingTrauma ->Projectiles that enters chest causing small or large hole.
- Compression Injury -> Chest is caught between two objects and compressed.
- Combination of blunt and compression trauma (asshown in the diagram)



In this perspective, management of chest trauma is done systematically assuming that trauma in all the other systems are being managed efficiently.

Management

All trauma patients must be managed in accordance with American College of Surgeons in the Advanced Trauma Life Support (ATLS) guidelines [7,8] which includes ABC with emphasis on maintenance of proper airway, breathing, circulation and management of shock such as tension pneumothorax or hemorrhagic shock

1. Primary Survey and Initial Actions (Major problems should be corrected as they are identified)[7,8]

Approximately a third of all deaths from thoracic trauma occur immediately on or shortly after patient's arrival in emergency room (ER). Hence immediate management is necessary in the surviving patients after evacuation from accident sites, while arranging the transport to hospitals.

A. Initial resuscitation and management includes apart from ABC as in ATLS protocol, early field procedures in the following life threatening injuries in patients as described below briefly:

- Airway obstruction and significant tracheobronchial injuries-> oral suction, endotracheal intubation or needle cricothyroidotomy or tracheostomy [7,8]
- Tension pneumothorax needs a 5-8cm needle puncture in the 2nd intercostal space in mid clavicular line or if admissible an Intercostal water seal drainage (ICD) lower down in the fifth intercostal space (usually at the nipple level), just anterior to the midaxillary line.
- Open pneumothorax causing sucking wound needs placement of an occlusive dressing over the wound to maintain ventilation and if available, seal the occlusive dressing gauge on three sides keeping one edge not sealed to Tape it securely on three sides to

provide a flutter valve type effect and thus allow extrusion of trapped intra pleural air (as shown below)



[8]

- Flail chest needs bulky dressing for splinting of flail chest which can be strapped on area of flail segment during transport.
- Cardiac tamponade needs Needle aspiration

B. During transportation to ER of unstable patients:

- Clothing stuck to the wound should not be removed during transportation.
- Any metallic pin or stick piercing through the chest should not be removed as it may be temporarily tamponading any large bleeder.
 - Trauma bandage and Triangular Bandages are to be applied to splint fractured ribs and clavicles, C-Spine collars to be applied and patient laid on back board.
 - Some apparently stable patients can also present as walk in to ER. C-Spine collars to be applied and patient to be laid on back board, as history of mechanism of trauma may suggest imminent deterioration as in contained great vessel rupture in frontal automobile collision.

C. After arriving in emergency room (ER): Life-threatening thoracic injuries are often identified during repeated primary survey by carefully assessing the patient's ABCs **again**. The injuries to be identified and treated.

- Airway obstruction needs to be provided with a patent airway and ensuring adequate ventilation and normal hemodynamics.
 - Hemodynamically unstable patients: Packed RBC (O-Rh Negative blood transfusion), consideration of surgical intervention.[9]
 - Pneumothorax: Tube thoracostomy by intercostal chest drainage procedure (ICD).
 - Open Pneumothorax: Taping of the wound and tube placement at site separate from injury (shown below [8])
 - Massive Hemothorax: As above, except if greater than 1500 cc of blood obtained on initial chest tube placement or more than 150-200cc/hr x 4 hours, patient needs to go to the operation room (OR) for urgent surgical intervention like thoracotomy.

- Flail Chest: Symptomatic support and analgesics, intubation and ventilation as needed. In extreme cases patient may need cardiothoracic surgical intervention.

- Cardiac Tamponade (more common in penetrating trauma): If subxyphoid pericardiocentesis is used as a temporizing maneuver, the use of a plastic-sheathed needle by Seldinger technique for insertion of a flexible catheter is ideal and if available, under USG guidance [8]. However, if expertise available, a subxyphoid open window drainage in supine position under local anesthesia, with a sterile catheter [4] such as Foleys, or even nasogastric or endoscopic suction cannula tube can be used.

- Blunt Aortic injury with contained rupture: If blood pressure is stabilized, followed by close observation and aortic repair either endovascular or open procedure. If unstable, massive transfusion protocol, transfuse pRBC and emergency aortic repair by direct or prosthetic bypass procedure.[10]

- Some patients with **Potentially life Threatening injuries** presenting with shock or dyspnea, who do not have visible injuries like external bleeding wound or hemoptysis or hematemesis, to qualify for immediate emergency invasive procedure as listed above, need imaging after hemodynamic stabilization before further intervention. They are:

- Contained Aortic rupture
- Significant Myocardial contusion
- Rupture of diaphragm
- Esophageal injury
- Significant Pulmonary contusion

D. Secondary survey and imaging

If the patient's condition is stable at presentation or stabilized after resuscitation with proper airway and circulatory control, secondary survey and the following imaging begins as available.

1DA. Chest X Ray (CXR) taken in supine position and if permissible erect CXRs are more sensitive in detecting a haemo-pneumothorax, but are often inappropriate in the acute trauma patient. Approximately 200mL of blood can be detected on an upright CXR, while a supine film only has a 40%-60% sensitivity in detecting haemothorax [3]. The sensitivity for rib fracture is poor (approx 50%) on plain film.

1DB. Ultrasonography (USG) of thorax and abdomen.

1DC. Computerized tomogram (CT)/contrast enhanced CT (CECT) of thorax and abdomen / contrast angiography.

These will provide information on potentially life-threatening injuries for **specific** management. These injuries can occur as single or part of combined injuries.

A systematic review of a CXR film may reveal suspected and unsuspected injuries, and the presence of any foreign bodies. In some cases, no fracture line is discernible in CXR, which needs CT for further evaluation. Fractures of the bony thorax, including the ribs, clavicles, spine, and scapulae, should be noted. If the impact is moderate, simple rib fracture can result while in case of massive impact, flail chest, organ injury such as pulmonary contusion, esophageal rupture and/or cardiac contusion, great vessel partial or complete rupture or arterial dissection can result. USG of chest or upper abdomen and CT or CECT needs to be carried out for suspected more grievous intra-thoracic injuries after initial actions for stabilization.

1 DA – CXR : to diagnose the followings:

1. Fracture of bony thorax: specific position and extent of fracture may indicate underlying grievous lesions

- Fracture of clavicle is often associated with pulmonary or cardiac contusion.
- Fracture of two or more ribs in 81% will have either a pneumothorax or a hemothorax.
- Fracture of the 1st rib will have significant energy transfer and neurovascular injury to the underlying subclavian vessels and/ or brachial plexus.
- Fracture 4th to 9th ribs will have associated injury to lungs, bronchus, pleura and heart
- Fracture of ribs below 9th rib may have splenic, hepatic or renal injuries,

2. Subtle subcutaneous air or foreign bodies as soft tissues shadow

3. Pneumothorax appears as lucent gas space devoid of pulmonary vessels and pleural line appearing in the image [3].

4. Hemothorax, pulmonary contusion, diaphragmatic hernia will appear as radio opaque shadow in chest cavity.

5. Pneumomediastinum- Mediastinal widening indicate tracheobronchial, esophageal or great vessel injury

6. Cardiac tamponade is evidenced as increased width of the cardiac silhouette.

1DB - Ultrasonography

Ultrasonography has now become fairly routine in the early evaluation of the abdomen and thoracic injuries including pericardium [8,9] Thoracic ultrasound examination is also valid when CT scan is not available .

The focused assessment for the sonographic evaluation of the trauma (FAST) patient endorses four standard viewing ports to quickly access for abnormal fluid collection:

- Right upper quadrant,
- Left upper quadrant,
- Pelvis,
- Subxiphoid.

In addition,

- Extended (E)-FAST, which uses an extension of the right and left upper quadrant views to include the right and left hemithorax (right and left longitudinal thoracic views), respectively, can aid in the diagnosis of hemothorax or pneumothorax.

Of the four views, the subxiphoid view is the most accurate in the hands of surgeons for detecting abnormalities in the trauma setting [4]. It is safe, expeditious, repeatable, and effective even in the hands of surgeons from different specialties.

1DC - CT or CECT: It is not essential for every patient with chest trauma and should never be performed in the hemodynamically unstable state or in the presence of obvious life-threatening injuries. It can be done after primary survey and stabilization of the patient in selected group of patients. Airway should be secured and Chest tubes should be placed prior to the CXR and/or CT scan if there is any clinical evidence of a pneumothorax/hemothorax. CT scans may reveal injuries not seen clearly on plain radiograph: aortic disruption, pneumothorax, pneumomediastinum, hemothorax, or pulmonary contusions, diaphragmatic injuries etc along with detecting lesions in other systems like neck, abdomen and pelvis [11]. However, unnecessary CT leads to greater costs, emergency department time, and patient exposure to ionizing radiation [12]. NEXUS (National Emergency X-Radiography Utilization Study) Chest decision instrument is one such an algorithmic criteria for selective CT of chest for patients with minor injury as judged on CXR, for identification of those who might harbour more serious injuries. There is high sensitivity (>98%) for the prediction of thoracic injury seen on chest imaging (TICI) like CXR with major clinical significance [12] under NEXUS criteria. This is modelled after NEXUS Head CT Decision Instrument for selective CT in patients with blunt head trauma [13] and selective CT for Cervical-spine in cases of blunt trauma of spine under NEXUS C-spine criteria [14]. Low energy fractures of the ribs disproportionately affect the elderly owing to a frail and brittle bone structures. Nonetheless, profound morbidity is observed in the aged from loss of efficient respiratory mechanics, pain-related chest wall splinting, and underlying lung injury. For each additional rib fracture in the elderly, mortality increases by 19% and the risk of pneumonia by 27% [2]. These patients also have a higher incidence of chronic lung disease and cardiovascular disease that can worsen outcomes. An oxygen saturation <92%

on room air, a tidal volume under 1.4 Litre and an incentive spirometry volume under 15 ml/kg are markers of pending respiratory compromise [2]. So, injuries considered as minor or of no clinical significance in younger patients may turn major in the elderly.

1DC1 - NEXUS chest decision instrument Indications for CTis considered in all patients having the following **major** chest injuries**irrespective of age** above and below 60 years where**clinical findings and CXR** suggests the understated pathologies:

- Aortic or great vessel injury CXR signs of blunt aortic injury [2]
 1. Widened mediastinum
 2. Loss of aortopulmonary window
 3. Deviation of trachea to R
 4. Deviation of NGT to R
 5. Thickened paratracheal stripe
 6. Left haemothorax
 7. Left pleural cap
 8. Fracture of 1st or 2nd rib
 9. Polytrauma with loss of consciousness
- Ruptured diaphragm:(pneumothorax can mimic diaphragmatic herniation [3])
- Pneumothorax: Received evacuation procedure (chest tube or other procedure)
- Hemothorax: Received drainage procedure (chest tube or other procedure)
- Sternal fracture: Received surgical intervention
- Multiple rib fracture: Received surgical intervention or epidural nerve block
- Pulmonary contusion: Received mechanical ventilatory assistance including noninvasive ventilation

1DC2. NEXUS chest decision instrument Indications for CT of chest based on clinical findings and CXR and mechanism of injury include the following conditions in **all age** groups:

1. Abnormal CXR finding showing any thoracic injury (including clavicle fracture) as discussed above.
2. Clinically distracting Injury (abdominal injury or lower limb injuries like, long bone fractures with or without large lacerations, degloving injuries which produce pain and acute functional impairment, sufficient to distract the clinician from a reliable thoracic examination to diagnose a concomitant chest injury as in the case of upper torso injury which may be sufficiently painful to distract from a reliable cervical examination for C -Spine clearance).
3. Chest Wall Tenderness, sternum, thoracic spine, or scapular tenderness

4. History of Rapid Deceleration Mechanism (defined as a fall >20 feet [>6.0 meter] or motor vehicle crash >40 mph [>64 km/h])
5. Chest pain
6. Intoxication
7. Abnormal alertness/mental status, [12]

1DC3. NEXUS chest decision instrument Indications for CT in patients above 60 with injuries where **clinical findings and CXR** suggest it as **Minor. But for those those below 60 CT is not** indicated:

1. Pneumothorax - No ICD but observed as inpatient >24 hours
2. Hemothorax- No ICD necessary but observed as inpatient for >24 hours
3. Sternal fracture - No surgery but had in-hospital pain management >24 hours
4. Sternal fracture - No surgical intervention, no inpatient observation (pain as OP)
5. Multiple rib fractures - No surgical intervention, received in-hospital pain management or observation >24 hours
6. Multiple rib fractures-> No surgical intervention, no inpatient observation but pain managed on an outpatient basis.
7. Pulmonary contusion or laceration -> No mechanical ventilatory assistance but observed >24 hours.

1D4.NEXUS chest decision instrument Indications for CT inpatients above 60 years old with injuries which are considered of **no clinical significance** in those **below 60** years age where it is not indicated :

1. Small Hemothorax - No ICD, no inpatient observation, managed on OP basis
2. Small Pneumothorax -> No ICD, no inpatient observation, managed on OP basis
3. Pneumo-mediastinum without pneumothorax -Managed on OP basis.
4. Pulmonary contusion or laceration -> No mechanical ventilatory assistance, no surgical intervention, no inpatient observation and managed on an outpatient basis[12].

The **Minor** categories were generated to account for those instances in which **CT generally visualized minute abnormalities** that resulted in no changes in management[15].

1DD. Echocardiography: Transthoracic echocardiography should be performed if there is suspicion clinically for cardiac contusion any cardiac valvular abnormality, pericardial effusion and tamponade. Transesophageal echocardiography (TEE) can detect aortic dissection and great vessel perforation. A major shift in the diagnosis of aortic and great vessel injuries has occurred in recent times.[16]. Since the late 1990s, CT angiography (CTA) has become the most prevalent diagnostic modality in this patient population.

2. Management of Injuries

Emergency room trauma life support starts with repetition of clinical examination in primary survey together with anamnestic information on the mechanism of thoracic trauma will provide information on potential severity of thoracic injury (16,17,18]. Outlines of management followed by discussion of specific lethal and nonlethal injuries are discussed as below:

2A. Outlines of Management

A. Goals of management:

1. Integrated Trauma team approach
 2. Address Life threatening injuries
 3. Expedite definitive therapy
- Initiate trauma activation system: alert or respond, depending on mechanism and physiological parameters
 - Notify Trauma/CTS/Vascular teams as appropriate
 - Manage in a trauma resuscitation bay with comprehensive noninvasive monitoring

B. Resuscitation for lethal injuries:

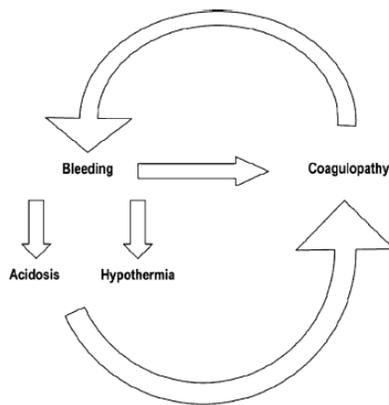
1. Airway & Breathing

- Apply O₂ titrating to Sats \geq 94%
- Perform chest decompression if tension hemothorax and or pneumothorax
 - a. Consider needle decompression
 - b. Urgent thoracostomy and ICD insertion
- Mechanically ventilate with lung protective strategy if respiratory failure evident or imminent – particularly if significant lung contusion or flail segment is present

2. Circulation

- a. Resuscitation with principles of damage control during resuscitation -if there is evidence of uncontrolled hemorrhage while expediting surgical control of bleeding.
 - Activate massive transfusion protocol- 1:1:1 red cells: FFP: platelets
 - Aim is to restore radial pulse or SBP to 80(80bpm/80 mm Hg) in otherwise normal subjects below 60. But in elderly population with history of hypertension, higher systolic BP is maintained.
 - Correction of Coagulopathy and prevention of dilutional coagulopathy by avoiding excessive crystalloid infusion. Current advanced trauma life support (ATLS) guidelines state that blood loss should be replaced with a 3 to 1 ratio of isotonic crystalloid fluid [18].

- Aggressive resuscitation to combat bleeding with a goal to normalize vital signs can result in hypothermia, and dilution, which perpetuate coagulopathy and the “lethal triad” (Re)bleeding, hypothermia and acidosis. Coagulopathy is defined as PT, activated partial thromboplastin time (APTT), or thrombin time (TT) greater than 1.5 times normal. It needs prevention with limited resuscitation with warm crystalloids for prevention of hypothermia. Colloids, pRBC and coagulation factors as needed [18]



- Tranexamic Acid 1g bolus & 1g infusion over 8h if within 3 hrs of injury may be used.

b. Indications for operative thoracotomy

1. Massive HTX

- >1500mL on ICC insertion
- > 200mL/hr following ICD insertion

- Classic indication means an absolute volume of blood loss, however the clinical status of the patient is a more important indication for thoractomy.

2. Cardiac injury +/- haemopericardium

3. Vascular injury at the thoracic outlet

c. Indications for ER thoracotomy

Penetrating chest/epigastric trauma with cardiac arrest with loss of output < 10 mts

C. Specific therapy

1. Bronchoscopy is indicated in all cases of suspected tracheobronchial injury.

In severe injury it can guide the ETT distal to the air leak.

2. Place ICD for hemothorax and/or pneumothorax

- Small asymptomatic pneumothorax identified on CT do not mandate ICD insertion. However any traumatic pneumothorax visualised on CXR should be drained by an ICD.

Size of chest tubes:

- Large bore chest tubes (Ch. 36–40) compared with smaller chest tube (Ch. 28–32) have no advantage in treatment of severely injured patients [17].
 - A drainage tube 20 to 24 F can be used as these are comfortable and does not impinge upon the rib margins in an average Indian patient of both sexes inflicting pain. A larger one can be necessary in larger body mass or when there is repeated collection in bag necessitating frequent cleansing of clots.

3. Surgical fixation of ribs is considered in significant chest injuries – particularly if the chest is flail. There is limited evidence that fixation reduces the duration of mechanical ventilation and its associated complications.

4. Thoracic Endovascular Aortic Repair (TEVAR) for aortic transection

5. Operative intervention is indicated in blunt oesophageal injury/diaphragmatic injury with herniation of intra abdominal organs

D. Supportive therapy

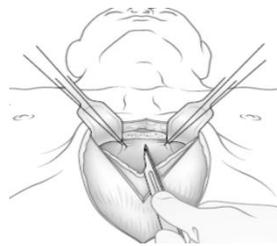
- Keep nil by mouth if awaiting surgical intervention or at risk of ventilatory compromise
- IV fluid at 100mL N/Saline/ hour for an average 50 kg person or Ringers solution
- Adequate analgesia is paramount in the event of multiple rib #s. PCA (with parenteral opioid) should be provided and anaesthetic input for regional blocks considered.
- Chart antiemetics
- Chest physiotherapy
- Keeping the patient warm
- Maintenance of normoglycemia and correction of any acidosis
- Documentation to be completed.

Further management: Patients with significant chest injury should be managed in a HDU or ICU environment.

2B.Potentially Lethal injuries: Without any investigations to resuscitate during primary survey-> Tension pneumothorax, Upper or lower airway obstruction, Open pneumothorax causing sucking wound (discussed above)

• **Lethal injuries:**

- Massive intra thoracic hemorrhage persistent after initial Intercostal chest tube drainage (ICD)
-> Thoracotomy (discussed later)
- Cardiac tamponade -> Pericardiocentesis, operative repair



- Aortic injury-Operative repair(TEVAR/Open repair)
- Cardiac rupture -> operative repair
- Tracheobronchial rupture -> Bronchoscopy, operative repair
- Diaphragmatic herniation repair with associated Intra-abdominal hollow and solid organ-> operative repair.

3. Secondary survey after stabilization and the patient in :

• **Repetition** of clinical examination in **primary survey** during resuscitation and stabilization, together with first hand information on the mechanism of thoracic trauma will provide information on potential severity of thoracic injury[19]. The secondary survey involves further, in-depth physical examination, an upright chest x-ray examination if the patient's condition permits, arterial blood gas (ABG) measurements, and pulse oximetry and ECG monitoring. In addition to lung expansion and the presence of fluid, the chest film should be examined for widening of the mediastinum, a shift of the midline, and loss of anatomic detail.

Multiple rib fractures and fractures of the first or second rib(s) suggest that a significant force has been delivered to the chest and underlying tissues. Ultrasound has been used to detect both pneumothoraces and hemothoraces. However, other potentially life-threatening injuries are not well visualized on ultrasound, making the radiological imaging a necessary part of any evaluation after traumatic injury [8].

- Repeated examination is mandatory to avoid omission of progression of a pneumothorax or hemothorax when ICD may be necessary
- When a small pneumothorax or hemothorax is progressive necessitating ICD
- When the patient is mechanically ventilated.

4. THORACOTOMY

4A. Indications

According to the ATLS guideline [8] this is recommended as follows

- Blood loss over the chest TD >1,500 mL initially but continuous bleeding
- or >200 mL/hour over 2–4 hours; Patients who have an initial output of less than 1500 mL of blood
- The persistent need for blood transfusions as in cases of hemoptysis is an indication for thoracotomy
- Massive subcutaneous emphysema;
- Penetrating chest trauma.

4A1a. Absolute Indications for Resuscitative thoracotomy:

- Unresponsive hypotension (systolic blood pressure [SBP] < 60 mm Hg)
- Traumatic arrest with previously witnessed cardiac activity (before or after hospital admission) after **penetrating** thoracic injuries.
- Persistent hypotension (SBP < 60 mm Hg) with diagnosed cardiac tamponade
- Hemoptysis or endobronchial blood loss in massive lung contusion with endoscopic or radiographic evidence of significant tracheal or bronchial injury.
- Significant impairment of mechanical ventilation due to tracheobronchial tree injury with air-leakage causing hypoventilation.
- Penetrating truncal trauma (resuscitative thoracotomy)
- Vascular injury at the thoracic outlet
- Loss of chest wall substance (traumatic thoracotomy)
- Radiographic evidence of great vessel injury
- Significant missile embolism to the heart or pulmonary artery
- Transcardiac placement of an IVC shunt for hepatic vascular wounds[16]

4A1b. Chronic indications for thoracotomy:

- Nonevacuated clotted hemothorax
- Chronic traumatic diaphragmatic hernia
- Traumatic cardiac septal or valvular lesion

- Chronic traumatic thoracic aortic pseudoaneurysm
- Non-closing thoracic duct fistula
- Chronic (or neglected) posttraumatic empyema
- Infected intrapulmonary hematoma (eg, traumatic lung abscess)
- Missed tracheal or bronchial injury
- Tracheoesophageal fistula
- Innominate artery/tracheal fistula
- Traumatic arterial/venous fistula

4A1c. Relative Indications for thoracotomy :

- Traumatic arrest with previously witnessed cardiac activity (before or after hospital admission) after **blunt** trauma
- Traumatic arrest without previously witnessed cardiac activity (before or after hospital admission) after penetrating chest injuries
- Prehospital cardiopulmonary resuscitation: <10 minutes in intubated patient, <5 minutes in nonintubated patient

4A1d. Contraindications for thoracotomy :

- Blunt thoracic injuries with no previously witnessed cardiac activity
- Multiple blunt trauma
- Severe head injury

4B a. Approach thoracotomy for surgical intervention

- Anterolateral thoracotomy in the 4–6th intercostal space ->necessity for emergency room thoracotomy is extremely rare. However, anterolateral thoracotomy will permit a potentially lifesaving measure (clamping of a great vessel) in an extreme situation before proceeding to the operating theatre.
- ‘Resuscitative’ sternotomy is advocated in penetrating thoracic injury for optimal outcome.
- Minimally invasive surgery or videoassisted thoracoscopic surgery (VATS) -> for major thoracic resections has led to advanced techniques for management of major bleedings in the elective-surgery-patient. VATS as a procedure for pleural space management in the non-critical, non-massive-transfusion patients can be of great assistance [20]. Its value in persistent non major-vessel-bleeding haemothorax in terms of pleural space debridement is undisputable [21]. If suspected on serial Chest x-ray at 3 days after chest tube placed, repeat CT of chest should be performed and video assisted thoracoscopic surgery (VATS) can be performed to fully evaluate if present. In some cases VATS is considered to be related to lower ARDS-rates in comparison to open thoracotomy patients, whereby an obvious bias for the non-massive-injury-patients exist [9]
- “Trapdoor” incision for exposure and control of the distal subclavian artery injuries

- Clamshell (transverse sternotomy and bilateral anterolateral thoracotomy) or hemi-clamshell (longitudinal sternotomy and anterolateral thoracotomy) [9]

- **Video-Assisted Thoracic Surgery**

Role of Video-Assisted Thoracic Surgery in Thoracic Trauma

Indications

- Treatment for ongoing thoracic hemorrhage
- Treatment of retained hemothorax
- Treatment of persistent pneumothorax
- Diagnosis and treatment of diaphragmatic injuries
- Pericardial window for relief of cardiac tamponade
- Management of thoracic duct injuries
- Treatment of post-trauma empyema
- Removal of foreign bodies

- **Relative Contraindications of VATS**

- Coagulopathy
- Prior thoracotomy

- **Absolute Contraindications of VATS**

- Hemodynamic instability
- Suspected cardiac injury
- Suspected great vessel injury
- Inability to tolerate single-lung ventilation
- Inability to tolerate lateral decubitus position

MECHANISM OF CHEST TRAUMA:

- Blunt
- Blast injury
- Penetrating

BLUNT TRAUMA

Results in Chest injuries involving

Soft tissues over the chest wall and intrathoracic lesions ->

- Bruises
- Scratches
- Laceration
- Pneumothorax including tension pneumothorax
- Hemopneumothorax
- Intra thoracic organ injury involving heart, lung esophagus, great vessels

Bony structures

- Rib fractures
- Flail Chest
- Sternal fractures
- Thoracic spine injuries

B 1. Rib Fracture at one end of ribs

- Single or Multiple rib involvement
- Usually after blunt trauma.
- Fracture of first rib to 12 th ribs -> already discussed

Clinical presentation of rib fractures

- Pleuritic chest pain
- Rapid shallow breathing
- Splinting
- Atelectasis
- hypoxemia

Treatment:

• **Chest splinting:** Broken ribs are usually left to heal on their own by active pain management and physiotherapy. Elastic therapeutic taping with an adhesive tape from midline in front to spine at back, can be a temporary practical and cost effective treatment possibility to help control pain and restore respiration in cases of simple single and multiple rib fractures as this reduces grating of fractured ends of ribs. This can reduce pain and possibly render assistance in coughing. This method of fixation however, has a disadvantage, as it definitely reduces ventilation of the half of the thorax in question and in so doing promotes the formation of atelectasis, respiratory insufficiency and pneumonia in 10 % of cases in comparison to 5 % operative fixation group [22]. However taping can assist to mobilize patients. Moreover, malunion and rib crowding is higher in cases of splinting in comparison to surgical fixation

group [22] and hence not recommended except as a temporary method during transportation to trauma centre.

- ICD for associated significant pneumothorax or pleural effusion
- Strong analgesia
- Intercostal Nerve blocks with long acting local anesthesia (LA) like bupivacaine
- Pulmonary toileting by physiotherapy and nebulization -> Patient is asked to cough with a pillow held with hands adjacent to the fractured site and breathe deeply to reduce atelectasis and pneumonia.
- If the patient is stable-> spontaneous ventilation can be managed with analgesic supports and chest toileting.
- If unstable-> Ventilatory support with intermittent positive pressure ventilation (IPPV) which acts as a virtual splint for fractured ribs in patients with flail chest.
- Internal Fixation with plates and screws-> For multiple rib fractures when IPPV is not available

B 2. Fracture of two or more ribs in two or more places (Flail Chest)

Clinical presentation of Flail Chest

- Painful Breathing.
- Paradoxical Chest Movements.
- Rapid, Shallow respiration, Dyspnea, Tachypnea, Tachycardia.
- Bruising/Swelling.
- Crepitus (Grinding of bone ends on palpation).

Diagnosis of flail chest is purely clinical

- CXR is necessary to confirm rib fractures and Arterial blood gas (ABG) to assess severity for initiating mechanical ventilatory support

Treatment of Flail Chest

- ABC's with c-spine control as indicated
- High Flow oxygen and mechanical ventilation if SO_2 is still $<85\%$
- Adequate analgesia (Including opiates)
- Intra-plural local analgesia and intercostal nerve block
- Observe the patient for development of pneumothorax, hemothorax and even worse Tension Pneumothorax

- **Bilateral rib fracture on both sides of a flail sternum** as in an automobile steering wheel thrust over the chest, the patient is to be made to lie prone during transportation to reduce paradoxical sternal movement.

B 3. Isolated sternal fractures is low, and surgical repair is uncommon (<2%). It has been suggested that patients with isolated sternal fractures, a normal echocardiogram, and no elevation of cardiac enzymes in the early hours of injury will have a benign course. Surgical management options include metal plates with or without autologous bone grafts [21,23.26]

B 4.

1. Principal indications for operative fixation of flail rib segments:

- a. Those intubated patients with no possibility of being weaned from the ventilator because of a large unstable flail segment of chest wall requires some forms of fixation to avoid the complications of long term ventilation like tracheal stenosis, pneumonia, tracheostomy.
- b. Patients who require mechanical ventilation with a flail chest (FC) without pulmonary contusion (FC) benefit from surgical fixation. “Pure” FC patients benefited from surgical fixation in terms of separation from mechanical ventilation (6.5 vs. 30 days), while those with PC-FC did not (27 vs. 30 days) [27].

2. Indications for fixations of fractured ribs according to nature of rib fracture

Recommended:

- ≥ 5 rib flail chest requiring mechanical ventilation;
- Symptomatic non-union;
- Severe displacement found during a thoracotomy for another reason.
- ≥ 3 rib flail not requiring mechanical ventilation;
- ≥ 3 ribs with severely displaced fractures bi-cortical displacement
- ≥ 3 ribs with mild to moderate displacement and 50% reduction of expected forced vital capacity percent despite optimal pain management.

3. Absolute contraindications for fixations of fractured ribs :

Contaminated field.

a. **Relative contraindications:**

- Severe lung contusion requiring prolonged mechanical ventilation;
- High cervical spine injury requiring mechanical ventilation [25]

b. Devices:

There are a number of devices which predominately fall into five categories:

- (1) plating with cortical screws (single plate or U-type plate²⁰),
- (2) Metal plating [8]
- (3) Judet struts or Judet-like struts,
- (4) Kirschner wires



- (5) intramedullary rods.

The most common ribs repaired are the 4th through the 10th. These are the most mobile ribs and produce a significant amount of pain. The first through third ribs are more difficult to access and the benefit of the added dissection/trauma may not justify repairing these ribs.

The incision should be minimized by carefully placing the incision in the middle of the segment to be repaired. A skin flap should be raised in order for allowing for maximal movement of the incision to visualize the necessary muscular windows [21, 25].

4. Intra pleural pathology and its management

Assessment after stabilization to detect alteration of old lesions or development of new ones. They include

5A1a. PNEUMOTHORAX - one in which chest wall is intact and air enters the pleural space from injured lung surface). There may or may not be associated rib fractures

- **Simple / Closed Pneumothorax**

Clinical presentation of Simple/Closed Pneumothorax

- Chest Pain
- Dyspnea
- Tachypnea
- Decreased Breath Sounds on Affected Side

Treatment for Simple/Closed Pneumothorax

- ABC's with C-spine control
- Airway Assistance as needed
- Small Pneumothorax is usually small and self-limiting. Treatment is by aspiration for small pneumothorax.
- ICD drainage for significant (more than 20 % of volume of hemithorax)
- Pleurodesis-> by Sclerosigagents like Doxycyclin, Bleomycin, talc, and at a later date for recurrent pneumothorax.
- Provide supportive care
- Monitor for Development of Tension Pneumothorax

• Open Pneumothorax

Open pneumothorax is sucking chest wound in which air enters the pleural space through opening in the chest wall.

- Opening in chest cavity that allows air to enter pleural cavity
- Causes the lung to collapse due to increased pressure in pleural cavity
- Can be life threatening and can deteriorate rapidly.

Clinical presentation of Open Pneumothorax

- Dyspnea
- Sudden sharp pain
- Subcutaneous Emphysema
- Decreased breath sounds on affected side
- Hyper-resonance
- Red Bubbles on Exhalation from wound (a.k.a. Sucking chest wound)

Investigations:

- CXR in erect position
- Smaller pneumothorax may need Expiration CXR or USG thorax and abdomen, CT chest

Management:

- ICD
- Observation for hypoventilation
- Endotracheal Intubation->Gold standard,
- Recurrent or persistent pneumothorax ->Pleurodesis
Surgery -> needed in less than 20% Thoracotomy.

Sucking chest wound caused by

- Full thickness hole in the chest wall, more than 2/3rd of tracheal diameter.

Treatment:

- Occlusive dressing during transportation-> taped on three sides act as oneway valve
Followed by Chest drainage
- Do not clean the wound or remove Occlusive Dressing or any penetrated object stuck in the wound, in emergency.

• Tension Pneumothorax

- Air builds in pleural space with no way for the air to escape
- Results in collapse of lung on affected side that results in pressure on mediastium, the other lung, and great vessels resulting in decreasing venous return =>Shock

Causes:

- Penetrating trauma,
- blunt trauma,
- Iatrogenic tension Pneumothorax

Clinical presentation of Tension Pneumothorax

- Anxiety/Restlessness/Panicky
- Severe Dyspnea
- Absent Breath sounds on the affected side
- Hyper-resonance
- Tachypnea
- Tachycardia
- Poor Color
- Accessory Muscle Use
- Jugular venous distension
- Narrowing Pulse Pressures
- Hypotension
- Tracheal Deviation to opposite side
- Dramatic Presentation.

It's a clinical diagnosis and treatment should not be delayed by waiting for CXR Treatment is by immediate needle decompression by inserting a large-bore (eg. 14 gauge) needle into the 2nd intercostal space in the midclavicular line. It should be inserted at a 90-degree angle to the

chest wall. It can just easily (or sometimes more easily) done by decompressing through the 4th intercostal space in mid-axillary line.

Air will usually gush out. Because needle decompression causes a simple pneumothorax, ICD should be done immediately thereafter with a 20 or 32 F chest drainage tube in the 5th ICS according to build of the subject. These life-threatening injuries and related problems are resolved as they are discovered.

Complications of chest tube drainage

- Hemorrhage
- Infection
- Trauma to the Liver, spleen, Diaphragm, Aorta and **how to avoid** (after a 5cm incision with the knife, and deepening it, insertion of the index finger through the incision is required to palpate the underlying lung and thus avoid injury to the liver or spleen)
- Minor complications like, Subcutaneous emphysema, hematoma
- Improper placement
- Pulmonary edema during rapid decompression of air and/or fluid

CAVEAT: It's advisable not to decompress the chest tube all at once for pneumothorax or hemothorax. Partial occlusion of the chest tube by hemostatic forceps is advisable to reduce the rate of drainage of fluid and thus avoid pulmonary edema.

5A1b. Hemothorax

- Follows Blunt injury / penetrating injury
- Occurs when pleural space fills with blood
- Usually occurs due to lacerated blood vessel in thoracic wall, mainly Intercostal and internal mammary vessels or due to lung laceration by fractured ribs or disruption of any fibrous adhesion bands within the pleura
- As blood accumulation increases, it puts pressure on heart and other vessels in chest cavity

Clinical presentation of Hemothorax

- Anxiety/Restlessness
- Tachypnea
- Signs of Shock may be present
- Diminished Breath Sounds on Affected Side
- Dull percussion note... maybe resonant in supine position.
- Tachycardia

- Flat Neck Veins

Treatment for Hemothorax

- ABC's with c-spine control as indicated
- General Shock Care due to Blood loss
- ICD
- Thoracotomy -> If more than 1500 ml blood drains initially, or ongoing hemorrhage of more than 200 ml/ hr over 3-4 hrs.

5A1c. Pulmonary Contusion

Pulmonary contusion (PC) in turn is the most common injury identified in the setting of blunt thoracic trauma, occurring in 30% to 75% of all cases [24]. Isolated PC may occur consequent to explosion injury, but most patients with multiple injuries have concurrent injury to the chest wall [25]. Conversely, flail chest (FC), the most severe form of blunt chest wall injury with mortality rates of 10% to 20%, is typically accompanied by significant PC [26].

Pathophysiology of a PC is based on hemorrhage into adjacent alveolar spaces rather than injury to the alveolar wall itself. Literally it means crushing and bruising of the lung parenchyma. There may not be external signs of trauma but internally there can be lung hemorrhage and has been confirmed in studies of animals placed at varying distances from explosive charges. High-grade evidence from animal studies indicated that PC is not merely a localized process but probably has global pulmonary and systemic effects relating to cytokine expression and immunosuppression when occurring in a sufficient portion of the lung [27]. Contused lung produces more than its normal amount of interstitial and intra-alveolar fluid and clinical expression takes 48/ 72 hours to develop. Aggressive fluid resuscitation was cited as a key factor in precipitating respiratory failure after blunt thoracic trauma. Further studies during the Vietnam War laid the basis for the current approach in treatment of PC. [26]. In a study of combined pulmonary and chest wall injury Reid and Baird [28] were the first to propose that parenchymal contusion rather than bony thoracic injury was the main factor in respiratory compromise.

- Sudden blow or blunt injury to the chest compression of thoracic cavity and lung followed by an equally sudden decompression as in steering wheel compressive injury. Concussive and compressive force is most important cause.
- The natural progression of pulmonary contusion is worsening hypoxemia for the first 24 to 48 hours.
- Classic symptoms include dyspnea, tachypnea, hemoptysis, cyanosis, and hypotension
- CXR findings not significant initially.
- CT may be confirmatory.

Clinical presentation of Pulmonary contusion

- Hemoptysis
- Dyspnea
- Cough
- Chest wall abrasion
- Echymosis.

Treatment of pulmonary contusion

Obligatory mechanical ventilation in the absence of respiratory failure solely for the purpose of overcoming chest wall instability should be avoided.

The use of optimal analgesia and aggressive chest physiotherapy should be applied to minimize the likelihood of respiratory failure and ensuing ventilatory support.

- Oxygen administration
- Pulmonary toileting
- Fluid restriction and diuresis
- All patients should be observed in a hospital setting because of a tendency of their ventilatory status to deteriorate rapidly except in small contusion in otherwise healthy young persons who can be treated as an out patient.
- Patients with PC-FC requiring mechanical ventilation should be separated from the ventilator at the earliest possible time. Positive end-expiratory pressure (PEEP)/ continuous positive airway pressure (CPAP) should be included in the ventilatory regimen.
- By standard ATLS protocol, patients with significant hypoxia ($P_{aO_2} < 65$ mm Hg, $S_{aO_2} < 90\%$) - Mechanical ventilation.

Complications of pulmonary contusion -> Respiratory insufficiency and secondary pneumonia, ARDS [29]The mortality rate from an isolated pulmonary contusion is low, but when combined with other severe injuries, it rises to as high as 50%

5A1d. Subcutaneous Emphysema

- Air collects in subcutaneous tissues of chest wall and abdomen
- Pressure of air in pleural cavity and mediastinum
- Feels like rice crispies below the skin
- Puffiness can be seen from neck to groin area where it is able to travel from the chest cavity along the fascial planes of abdomen to upper thigh and scrotum
- Usually occurs on the chest, neck and face,

Clinical presentation of subcutaneous emphysema

- Swelling of the neck
- Chest pain
- Neck pain
- Dysphagia
- Wheezing and difficulty breathing.

. Causes of Subcutaneous Emphysema

- Both blunt and penetrating trauma causing airway injury or esophageal injury by fractured rib fragments injuring the pulmonary parenchyma or esophageal injury and leakage.

Diagnosis

- Mainly clinical
- CXR for stable patients
- FAST, CT for severe chest trauma patients after stabilization

Treatment

- Usually benign, no treatment needed.
- If massive -> then ICD. Small cuts in the skin as taught previously are contraindicated
- Catheters in subcutaneous tissues **not to be done**.
- Treatment of the underlying cause is needed.

5A1e.Tracheo-bronchial Injuries

Tracheobronchial injuries are uncommon, but they occur usually after high-energy impact and are associated with trauma to other vital organs. Commonly these injuries are caused by motor vehicle accidents, **where an unrestrained automobile occupant hyperextends the neck, striking it on the dashboard or steering wheel and producing a crushing injury of the cervical trachea**. The second mechanism is a consequence of high airway pressures, like fall from a height, while the third is rapid deceleration. The typical clinical features include respiratory distress, dyspnea, and air leak. Hoarseness or dysphonia is also common, occurring in up to 45% in some series. Persistence of an undiagnosed air leak is life-threatening and may lead to hypoventilation and, ultimately, respiratory insufficiency.

76% of these injuries occur within 2 cm of the main carina, and 43% were located within 2 cm of the right main bronchus [30]

Clinical presentation of Tracheo-bronchial Injuries are:

- Hoarseness, subcutaneous emphysema
- Dyspnea , Pneumothorax , hemoptysis
 - Mediastinal crunch known as Hamman's Sign
 - Intercostal retractions, Respiratory distress
 - Stridor
 - Chest drain reveals a large air leak
 - Collapsed lung may fail to re-expand
 - Diagnosis -> Bronchoscopy, CT

Management of Tracheo-bronchial Injuries

- Priority is to stabilize AIRWAY.
- Intubation of the unaffected bronchus and
- Operative repair.

5A1f.Traumatic Asphyxia

- Results from compression injury to chest cavity as in stampede or house collapse or road traffic accident
- Can cause massive rupture of Vessels and organs of chest cavity.
- Death

Clinical presentation of Traumatic Asphyxia as in cases of chest compression

- Severe Dyspnea
- Distended Neck Veins
- Bulging, Blood shot eyes
- Swollen Tongue with cyanotic lips
- Reddish-purple discoloration of face and neck
- Petechiae

Treatment for Traumatic Asphyxia

- ABC's with c-spine control as indicated
- High Flow oxygen
- Treat for shock
- Treatment of associated injuries

5A1g.Pericardial Tamponade

Cause of Pericardial Tamponade

- Penetrating Trauma

Pathophysiology:

- Blood and fluids leak into the pericardial sac which surrounds the heart. As the pericardial sac fills, it causes the sac to expand until it cannot expand anymore. Once the pericardial sac cannot expand anymore, the fluid starts putting pressure on the heart. Now the heart cannot fully expand and can't pump effectively.
- With poor pumping the blood pressure starts to drop.
- The heart rate starts to increase to compensate but is unable.
- The patient's level of conscious drops, and eventually the patient goes in cardiac arrest.

Clinical presentation of Pericardial Tamponade

- Distended Neck Veins
- Increased Heart Rate
- Muffled heart sound
- High Respiratory Rate
- Poor skin color
- Hypotension
- Beck's Triad-> Low BP, raised venous pressure, muffled heart sounds.
- All the patients with penetrating injury anywhere near the heart + Shock => Always suspect **cardiac injury**.
- Must be differentiated from Tension pneumothorax.
- In case of major bleeding from other site, neck veins may be flat.

Diagnosis:

- Clinical suspicion.
- CXR-> enlarged Globular heart shadow.
- Echo-> Fluid in pericardial sac.
- Central venous pressure-> high
- CT scan

Treatment of Pericardial Tamponade

- ABC's with c-spine control as indicated
- High Flow oxygen.
- Treatment of shock
- Rapid Transport
- Pericardiocentesis

Pericardiocentesis under echocardiographic guidance

- Using aseptic technique, Insert at least 3" needle at the angle of the Xiphoid Cartilage at the 7th rib
- Advance needle at 45 degree towards the clavicle while aspirating syringe till blood return is seen. If available, a sheath with pigtail catheter can be used in a Seldinger technique
- Continue to aspirate till syringe is full then discard blood and attempt again till signs of no more blood drains out. Or attach a three way stop cock to the needle and the other end to a IV infusion set leading to a drainage bottle or container and control drainage by slow decompression
- Closely monitor patient due to small amount of blood aspirated can cause a rapid change in blood pressure

Surgical treatment of Pericardial Tamponade

Minimal approach in subxiphoid area-> under local anesthesia [31]

- 4cm incision through the Xiphisternal attachment
- Separate of the diaphragmatic attachment and incise the parietal pericardium after holding with 2 artery forceps
- Enlarge the opening and introduce one 20 or 24 F chest drainage tube into the pericardial cavity and fix with sutures
- Attach the other end to a water seal bottle

Standard thoracotomy under general anesthesia or VATS

- Left Thoracotomy

5A1h. Blunt Myocardial Injury

- Rarely causes hemodynamic Instability.
- Diagnosis - ECG, Echocardiography, Trans esophageal Echocardiography,
 - Complication - Arrhythmias in 1st 24 hrs.

5A1k. Esophageal injury

- Results from penetrating trauma; blunt injury is rare and can be found in survivors of vehicular run over injury

Pathophysiology of esophageal Injury in blunt trauma - Severe compression injury in chest or upper abdomen with a full stomach causing tear in the gastro esophageal junction. Heimlich maneuver is also a risk factor. Complications:

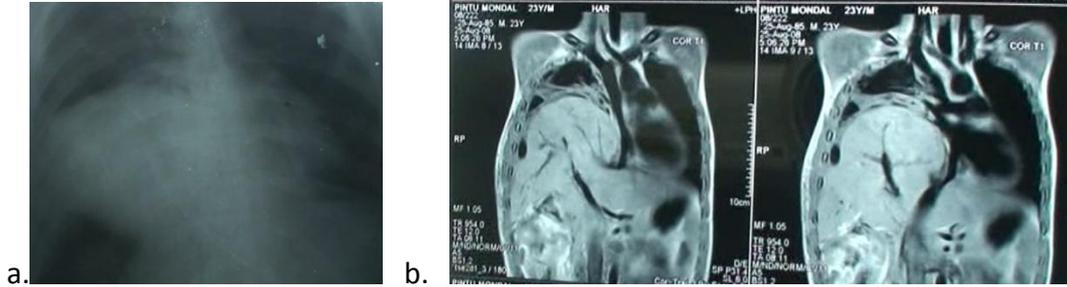
- Air leakage from esophageal tear - Surgical emphysema, pneumothorax and later pleural effusion
- Bacteria and Saliva leakage -Mediastinitis abscess, empyema
- Gastric juice containing gastric acid - acute inflammatory reaction in mediastinum - fluid and electrolyte disturbances, Later - Sepsis - Pneumonia, Pleural collection
- Cardiovascular collapse

Clinical presentation

- Patient can present with odynophagia, mediastinitis, pleural effusion, air in the retro-esophageal space, subcutaneous emphysema and unexplained fever within 24 hours of injury
- CT is helpful to suspect the diagnosis though combination of esophagogram and esophagoscopy confirm diagnosis in more stable patients
- Treatment is operative repair and drainage in recent localized tear
- Mid-esophageal injury => Right thoracotomy.
- Distal esophageal injury => Left thoracotomy.
- In extensive esophageal tear, Esophagectomy along with cervical esophagostomy, closure of esophago gastric junction and feeding jejunostomy with later provision of esophageal enteric bypass

5A1I. Diaphragmatic Rupture

- a. A tear in the diaphragm that allows the abdominal organs enter the chest cavity.
- b. Any penetrating injury to or below 5th intercostal space can cause diaphragmatic penetration & abdominal injury.
- c. Blunt injury to the diaphragm is usually caused by a compressive force applied to the pelvis & abdomen.
- d. More common on Left side. Right sided traumatic diaphragmatic herniation is less common as liver helps protect the right side of diaphragm but not uncommon within the survivors.



- Right sided traumatic diaphragmatic hernia with liver herniated into the right pleural cavity with deviation of mediastinum to left. Mechanism of injury was caused by partial run over injury where the rear wheel of an empty slowly backing medium sized truck ran partially over the right side of abdomen of a young man of 30 and then rolled back. a. The CXR taken on the 1st day was interpreted logically as simple 6th and 7th rib fracture, right lung collapse. After stabilization, a MRI with contrast study was done on the next day which showed that the right lobe had herniated into the right chest cavity. Exploration through a right thoraco abdominal approach confirmed rupture of right lobe of diaphragm which repaired. [32]
- Associated with multiple injury patients
- Usually large, with herniation of the abdominal contents into the chest.

Clinical presentation

- Abdominal Pain
- Shortness of Air
- Decreased Breath Sounds on side of rupture
- Bowel Sounds heard in chest cavity

Diagnosis:

- Chest radiography after placement of a nasogastric tube, Contrast studies of the upper or lower gastrointestinal tract,
- CT scan & diagnostic peritoneal lavage.
- Most accurate evaluation is by video-assisted thoracoscopy (VATS) or laparoscopy.

Treatment of Diaphragmatic Rupture

Operative Repair

Penetrating diaphragmatic injury must be repaired via the abdomen and not the chest, to rule out penetrating hollow viscus injury. Laparoscopy can also be done.[4]

5A1m.Laryngeal Injuries

Blunt trauma to the larynx is therefore rare as larynx enjoys a position of relative protection in the neck but its mortality rate reaches as high as 40%, with most patients dying from the injury at the trauma scene [30].

It is suspected after motor vehicle accidents, hanging attempts, sporting blows such as in karate or soccer, and severe falls.

Clinical features -> laryngeal blunt trauma include hoarseness, pain, skin contusions, cervical emphysema, cervical neck crepitus, dysphagia, and upper airway obstruction.

Risk - laryngospasm and sudden airway obstruction

Diagnosis - Clinical and CT is a sensitive diagnostic test for laryngotracheal injury, and it may be indicated **despite normal flexible laryngoscopy**.

Treatment -Intubation in cases of upper airway obstruction or tracheostomy to be readied before performing endoscopy

5A1 n. Great Vessels injury: The thoracic great vessels consist of the aorta and its major intrathoracic branches, the pulmonary arteries and veins, the venae cavae, and the azygous vein. By far the most lethal injury to any of these is descending aortic injury, which accounts for as many as 40% of fatalities after blunt thoracic trauma, with the majority of deaths at the trauma scene[8] .

Eighty percent of patients with thoracic aorta injury will die before reaching a trauma center. For patients who survive to hospital arrival, 50% will die within 24 hours. This significant mortality rate is related to the high incidence (40%) of severe associated injuries[10]. The risk of rupture of contained blunt trauma thoracic aorta injury (BTAI) is highest within the first 24 hours of injury. For this reason, immediate repair of BTAI was advocated and considered the standard of care for decades. In the 2000 a Level II recommendation was made for prompt repair unless patients “have more immediately life threatening injuries that require intervention such as emergent laparotomy or craniotomy, or if the patient is a poor operative candidate due to age or co-morbidities.”[33]. Delayed repair of BTAI was traditionally reserved for high-risk patients with major associated injuries or severe comorbidities. However, following publication of the 1998 article by Fabian et al[34] the use of **anti impulse regimens** as used in cases of aortic dissection, consisting of betablockers and vasodilators like nitroglycerine or

sodium nitroprusside infusion, became widespread, **delayed repair** of aortic lesion was extended to low-risk trauma patients. However, patient at a high risk of aortic rupture, based on clinical suspicion, imaging characteristics, and/or grade of injury should not be considered for delayed repair. This would include patients with Grade 3 and 4 injuries, which are defined as BTAI with pseudoaneurysm (Grade 3) and BTAI with active extravasation (Grade 4). In addition, clinical or radiographic evidence of pseudo-coarctation may be an indication for urgent repair. Patients who benefit the most from delayed repair are those who have major associated injuries. These patients clearly require resuscitation and treatment of immediately life-threatening injuries before aortic repair. If facilities are available thoracic endovascular aortic repair (TEVAR) is recommended more frequently than open repair as it is a less invasive procedure, carries a significantly lower risk of blood loss, mortality, and paraplegia and a risk of stroke when compared to open repair[10].

Ruptured thoracic aorta: imaging-> a widened mediastinum (>10 cm), loss of aortic knob contour, shift of the endotracheal tube and the trachea to the right, elevation of the left main-stem bronchus, depression of the right main-stem. Again, a high index of suspicion in a patient who has suffered a high-speed collision is critical, because about half of the patients with contained aortic rupture have no external signs of trauma.

i. Physiology:

Should be considered in mechanisms where severe acceleration/deceleration is present

ii. Treatment:

- a) Avoid hypertension, using beta-blocker with vasodilator if necessary (with vasodilator avoid tachycardia)
- b) If shock is present must find and treat other sources, as ruptured aorta is rarely if ever the source of hemorrhagic shock
- c) Once other source of shock confirmed, operative, or more frequently endovascular repair should be performed
- d) Consult cardiothoracic surgery
- e) When definitive repair must be delayed, hypertension should be avoided

6. Blast Injuries

Latest is the blast injuries in crowded places or in isolation during social unrest. The damage from a powerful blast can be particularly severe, as the blast pressure wave imparts a large amount of kinetic energy to a small area. Blast kinetic energy especially affects gas-containing organs, such as the lung, resulting in pulmonary hemorrhage, hypoxia, and shock.

7. Penetrating Trauma

Stab Wounds and Firearm Injuries. The evaluation and management of penetrating trauma to the thorax is best analyzed by anatomic distribution [35]. This includes the chest wall, the great vessels and other major vascular structures, the trachea and major bronchi, the lung parenchyma, the heart, the esophagus and the diaphragm. Puncture wounds can be small and easily missed. The patient must be completely exposed with close inspection of critical areas such as the axilla.

Central wounds and peripheral wounds have different implications. Upper thoracic wounds suggest potential injuries to neck structures while lower thoracic to abdominal structures. Stab wounds differ from gunshot wounds in their potential depth of penetration and degree of damage to surrounding organs. Blast injury sites in chest with sucking wound can be initially covered with adhesive Steri-Drape or a soft Esmark covering with placements of chest tubes inside the thorax to achieve lung inflation and control any air leak. Definitive operation can then be planned.

8. Complications of thoracic trauma

A. common problems after thoracic trauma are listed

Pulmonary

- Atelectasis
- Acute respiratory distress syndrome or acute lung injury
- Pneumonia
- Infarction
- Lung abscess
- Arteriovenous fistula
- Bronchial stenosis
- Tracheoesophageal fistula

Pleural Space

- Empyema
- Bronchopleural fistula
- Organized hemothorax
- Chylothorax
- Fibrothorax
- Diaphragmatic hernias

Vascular

- Thromboembolism
- Air embolism
- Pseudoaneurysm
- Great vessel fistula

Chest Wall

- Hernias
- Persistent pain

Mediastinum

- Mediastinitis
- Pericarditis

Pneumonia is the most common infectious complication after any multiple trauma, particularly those involving the thorax. The incidence increases with the duration of endotracheal intubation, and it is associated with up to 50% of deaths after trauma.

Diagnosis is often difficult, with only half of patients presenting classically with fever, leukocytosis, respiratory distress, and an abnormal chest radiograph. Sputum sampling is essential, however, and in the intubated patient, invasive testing is required, such as transtracheal or bronchoscopic aspiration with bronchoalveolar lavage. Pleural effusions can occur and are seen in 50% of patients with Haemophilus influenzae pneumonia.

Traumatic Asphyxia

Traumatic asphyxia is an uncommon clinical syndrome usually occurring after a severe crushing or compression-related injury to the chest. Symptoms and associated physical findings include subconjunctival hemorrhage, cervicofacial cyanosis resulting in a purple-blue discoloration of the neck and face, facial edema, vascular engorgement of the head, mucosal petechiae, and multiple ecchymotic hemorrhages of the face, neck, and upper chest. The diagnosis is made primarily from the history and physical examination. CXR are usually normal. Prompt establishment of the ABCs of trauma management is critical, with special attention to reestablishing oxygenation and perfusion to ensure a successful outcome. Head elevation should be maintained at 30 degrees. If a patient survives the initial insult, the prognosis is excellent. Skin discoloration and subconjunctival hemorrhage resolves within 3 to 4 weeks.

Referral Criteria

- ICU care will be needed in most patients as the range of injury can be quite exacting on medical resources.
- Patients will often need a quick but thorough assessment, resuscitation, stabilization and subsequent referral to a higher center with cardio-vascular surgery services. [36]

Medicolegal Issues

- Failure to detect / investigate / refer a patient of suspected major intra-thoracic trauma.
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action.

Who does what?

Doctor:

Surgeon:-Diagnosis & Work up

- Pre operative planning if surgery is planned at the center. The services of a specialist cardio-thoracic surgeon may be sought urgently.
- Operative procedure
- Post operative care in conjunction with Anaesthetist/Intensivist
- Post operative follow up

Anesthetist:- Pre Anaesthesia Check up

- Part of resuscitation
- Performing anesthesia with help or advice from cardiacanaesthetist.
- Post op ICU management in conjunction with Surgeon

Nurse:- Pre/Intra/Postop comprehensive care

Dressing of the wound

Technician:- Pre op equipment and drugs to be checked and kept ready

Assist anesthetist in the OT

Assist the surgeon, positioning of the patient

Perfusionist:- Serious injuries may even warrant extra-corporeal bypass

Resources required for one patient / procedure (Patient weight 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 2/3

3. Anesthetist – 2
4. Blood bank officer on-call 1
5. Staff Nurse – 1
6. Cardiac Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG
8. X- Ray – Chest
9. Echocardiogram
10. Contrast-enhanced CT Scan
11. CT Angiography

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of thoracic surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Dressings
15. Extra-corporeal bypass machine on standby.

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Haematuria- Non Traumatic

Introduction

Haematuria, the presence of blood or red blood cells in the urine, is a common clinical presentation, and can sometimes be a sign of occult cancer. This article describes current practice in the management of this condition and suggests modalities for evaluating patients with haematuria.

Methods

A review of contemporary national and learned society guidelines was performed.

Literature review

Visible haematuria or gross haematuria is an alarming symptom. Urine may be discoloured red or brown by blood or blood clot. A drop of blood will colour even a litre of urine. The source of bleeding may be in the kidneys, in the ureters, in the bladder or in the urethra, and from the prostate in men. Whilst infection and nephrolithiasis are common and benign causes of haematuria, the possibility of cancer has to be considered in all patients presenting with this complaint. Even a **single** episode of frank or microscopic haematuria has to be fully investigated (1, 2) (Tables 1, 2).

Microscopic haematuria is a sign rather than symptom, and is not visible to inspection and is defined as 3 or more red blood cells per high power field on microscopy. Asymptomatic microscopic haematuria (AMH) is defined as three or greater red blood cells (RBC) per high powered field (HPF) on microscopic examination of one properly collected urinary specimen in the absence of an obvious benign cause.

It is important to identify those individuals at higher risk for urologic malignancy. Patients with gross haematuria are a higher-risk group than those presenting with microscopic haematuria. Visible haematuria is a presenting sign in more than 66% of patients with urologic cancer. The sensitivity of visible haematuria in revealing malignancy is significant: 0.83 for urothelial carcinoma of the bladder, 0.66 for ureteric carcinoma, and 0.48 in renal cell carcinomas (3). The oncologic significance of visible haematuria is different for males and females. In men ages >60 years, the positive predictive value of visible haematuria for urologic malignancy is 22.1%, and in women of the same age it is 8.3%. Findings from screening studies in asymptomatic microscopic haematuria revealed that, an overall urinary tract malignancy rate of approximately 2.6%, and up to 4% in high risk individual, ranged from 0-25% (4).

Table 1

The causes of Haematuria

Neoplastic

Bladder cancer
Prostate cancer
Ureteral and upper tract cancer
Renal cancer

Structural

Benign prostatic hyperplasia (BPH)
Cystic renal disease
Arteriovenous malformation

Infections

Cystitis
Pyelonephritis
Urethritis
Genitourinary Tuberculosis
Schistosomiasis
Hemorrhagic cystitis

Calculus

Nephroureterolithiasis
Bladder stones

Medical renal disease

Alport syndrome
Benign familial haematuria (thin basement membrane nephropathy)
Nephritis
IgA nephropathy
Systemic lupus erythematosus (SLE)

Iatrogenic

Nephrotoxic/cytotoxic medications
Anticoagulation

Instrumentation of the urinary tract Radiation cystitis

Pseudohematuria

Hematologic

Sickle cell anemia

Coagulopathy

Gynecologic

Placenta percreta

Endometriosis

Idiopathic

Exercise-induced hematuria

Loin pain hematuria syndrome

Other

Renal Vein Thrombosis

Endometriosis

Medication

Food-related

Traumatic

Table 2

The risk factors for Urinary Tract Cancer in patients with Micro-hematuria

Male sex

Age (>35yrs)

Past or current smoking

Occupational or other exposure to chemicals or dyes (polycyclic aromatic hydrocarbons or aromatic amines)

Analgesic abuse

History of gross hematuria

History of urologic disorder or disease

History of irritative voiding symptoms (urgency and frequency)

History of pelvic irradiation

History of chronic urinary tract infection

History of exposure to known carcinogenic agents or chemotherapy, such as alkylating agents (particularly cyclophosphamide)

History of chronic indwelling foreign body

The causes of haematuria

Haematuria can be caused by malignancy, infection, stones, structural abnormalities, medical renal disease, and trauma (5). Coagulopathy caused by clotting disorders or anticoagulation can induce or exacerbate bleeding from underlying urinary tract lesions. Nephrotoxic medications can cause renal inflammation and renal papillary necrosis, whereas other medications can cause bleeding from the bladder mucosa. Instrumentation of the urinary bladder by endoscopes and catheters can cause traumatic bleeding that is generally self-limited.

Exercise-induced haematuria and loin-pain haematuria syndrome, idiopathic conditions of unknown clinical significance are rare benign causes of haematuria and often self-limited.

Gross haematuria should always be distinguished from pseudohaematuria, where blood is originating from a non-urinary tract source or discoloration of the urine by non-heme compounds which give the appearance of haematuria.

Infections

Both upper and lower urinary tract infections are the common causes of visible haematuria and often associated with symptoms of urinary frequency and dysuria. Urinalysis may also reveal leukocytes, nitrite, or leukocyte esterase test positive.

Genitourinary tuberculosis and Schistosomiasis may present with haematuria and sterile pyuria.

Malignancy

Bladder carcinoma is the most common cause of painless gross haematuria in adult >60 years. Urothelial carcinoma can occur anywhere along the urinary tract from renal pelvis to distal urethra. Ninety percent of bladder cancers are urothelial carcinomas and typically present as painless haematuria.

Squamous cell carcinoma and adenocarcinoma are rare types of bladder tumours. Renal cell carcinoma and metastases to the kidney can also cause haematuria, with or without flank pain. Prostate cancer can present with intermittent visible haematuria. Penile cancers with urethral or vascular invasion may cause visible blood in the urine.

Trauma is not covered in this presentation

Urinary stone disease

Urolithiasis, caused by the precipitation of crystals in the kidney or bladder, commonly causes intermittent pain that can be severe, although some patients are asymptomatic. Microscopic haematuria can be seen in up to 85% of patients, but visible haematuria is rare (6).

Structural abnormalities

Benign prostatic hyperplasia (BPH) predisposes men to visible haematuria and clot formation, possibly due to increased density of microscopic vessels in the enlarged prostate. A 1996 cross-sectional study had a self-reported rate of hematuria in 2.5% of patients with BPH (6). After relief of acute urinary retention by catheter decompression, visible hematuria can occur in 2% to 16% of patients (haematuria ex vacuo)(7). Cystic renal lesions such as those in polycystic kidney, medullary sponge kidney, and medullary cystic disease may cause visible haematuria. Vascular malformations and arterio-venous fistulas may spontaneously bleed into the urinary tract.

Medical renal disease

Numerous medical renal diseases cause haematuria. Pathology involving the renal parenchyma and glomeruli may present as haematuria that is characterized by dysmorphic red blood cells and casts of red or white blood cells. Significant proteinuria may be a prominent feature of these disorders, and renal function may be compromised. Referral to a nephrologist is important for the management of this broad category of disorders. In patients with renal failure, heavy proteinuria (>3 g/day), or severe hypertension, a nephrology consultation should be obtained, a prompt treatment or a renal biopsy may be necessary (9).

Benign familial haematuria is caused by a genetic defect that results in thinning of the basement membranes.

Alport syndrome is a hereditary disorder of the glomerular basement membrane caused by abnormalities in type IV collagen, and may progress to renal failure. It presents as proteinuria, haematuria, anterior lenticonus, hearing loss, and hypertension.

Glomerulonephritis (such as IgA nephropathy, post infectious glomerulonephritis, membranoproliferative glomerulonephritis, rapidly progressive glomerulonephritis, and systemic lupus erythematosus [SLE]) require renal consultation and are a spectrum of primary and secondary immune-mediated diseases that cause inflammation of the glomeruli. Proteinuria and renal failure are also present in these disorders to varying degrees.

Glomerular pathology can be an isolated phenomenon or part of a systemic autoimmune process such as SLE.

Hematologic

Sickle cell anaemia can present with urinary symptoms including haematuria, dysuria, and polyuria, with isosthenuria (urine that is not concentrated by the kidneys) on urinalysis.

Patients with coagulopathy may bleed from multiple sites, including the gastrointestinal and genitourinary tract. Patients on anticoagulation therapy may have visible haematuria, but urinary tract bleeding (even in cases of super-therapeutic anticoagulation) almost always represents an exacerbation of an underlying disease process or urinary tract lesion.

Gynaecological causes

Placenta percreta is a form of invasive placental implantation where the myometrium of the uterus is penetrated. From 5% to 7% of patients show this depth of invasion, and in rare cases it extends into the bladder to cause severe haemorrhage and hemodynamic instability. This diagnosis should be suspected in pregnant women with visible haematuria, especially if there is a history of placenta previa or prior caesarean section.

Endometriosis where the ectopic endometrial tissue involves the ureters or bladder may present as flank pain, dysuria, and haematuria that is cyclic in nature.

Iatrogenic

Instrumentation of the urinary tract by endoscopes or percutaneous access can cause self-limited bleeding.

Catheters or the presence of an indwelling ureteral stent or nephrostomy tube can cause urinary tract bleeding.

External beam radiation for pelvic cancers can cause inflammatory radiation cystitis that ranges in severity from microscopic haematuria and urinary frequency to severe haemorrhagic cystitis, incontinence, and bladder necrosis.

Prostate brachytherapy can lead to acute or late complications of haematuria.

Medications that can incite tubular necrosis or interstitial nephritis can cause haematuria. Nephrotoxic medications include aminoglycosides, cyclosporine, and some chemotherapeutic agents. Interstitial nephritis can be induced by penicillin, sulphonamides, and nonsteroidal anti-inflammatory drugs.

Cyclophosphamide is an important cause of haemorrhagic cystitis that may result in severe bleeding.

Anticoagulation with heparin, warfarin, and low-molecular-weight heparin can cause genitourinary tract bleeding. Typical therapeutic levels generally do not cause haematuria unless there is underlying pathology. Further evaluation must be undertaken to rule out important causes of bleeding, such as malignancy.

Idiopathic

Exercise-induced haematuria is a benign, self-limited condition seen in athletes and active people.

Loin-pain haematuria syndrome is a benign entity of unknown aetiology where symptoms of pain and intermittent haematuria predominate. This occurs primarily in women of childbearing age. The clinical significance of this syndrome as a diagnostic entity is a matter of debate.

Pseudo-haematuria

Certain foods and medications can discolour the urine, mimicking haematuria. Medications include phenazopyridine, rifampicin, phenytoin, levodopa, methyldopa, and quinine. Consumption of beets, blackberries, and rhubarb can also discolour the urine.

Urgent Considerations

Emergency action for hemodynamically unstable patients

Life-threatening bleeding from the urinary tract is exceedingly rare but may present in select aetiologies including renal trauma, haemorrhage from arteriovenous malformations or renal masses, placenta percreta, or haemorrhagic cystitis (5). These patients require urgent evaluation in an emergency department or intensive care setting. Hemorrhagic shock from severe bleeding requires aggressive resuscitation with fluids, colloid, or blood products. Emergency exploratory surgery or vascular embolization by interventional radiology may be required for control of bleeding in vascular malformations. Hemodynamically unstable patients with a poor response to resuscitation may require immediate intervention such as surgery or angioembolization in selected situations (10). Catheterization is necessary, particularly if bladder distension with blood and/or urine is present.

Clot retention

The presence of clots in the urine indicates significant haematuria. Blood clots can cause urinary obstruction at the bladder outlet. Irrigation of the bladder and the possible use of continuous bladder irrigation may be necessary to prevent clot retention and obstructive renal failure

Diagnostic Approach (11, 12, 13, 14&15)

Common urological causes of haematuria include urinary tract infection and ureteric and renal stones, but concurrent pathology should be suspected if haematuria is significant or persistent. Importantly, if benign conditions are excluded, and the haematuria continues, further investigation is advised, as this may be the only sign of an underlying genitourinary malignancy. Recommended investigations for haematuria include computed tomography intravenous pyelogram, urine cytology, urine microscopy and culture and blood tests (full blood examination, renal function and, in men, prostate-specific antigen). Patients with risk factors for genitourinary malignancy, macroscopic haematuria or those in whom no cause is found, should be referred to a urological service for further investigation including cystoscopy. Acute urinary retention is a common acute presentation of macroscopic haematuria. This can be managed with continuous irrigation and rarely requires emergency surgical intervention (12, 13).

The evaluation of gross haematuria requires a complete history and physical exam. The urinalysis, a critical component of the workup of visible haematuria, is the initial test and should be performed on a fresh, midstream, and clean-catch or catheterized specimen of urine.

The presence of white blood cells, leukocyte esterase, and nitrites points to infection and this should be confirmed by urine culture and treated with antibiotics. The urine is tested by urinalysis, microscopy, and culture after the completion of antibiotic therapy to ensure resolution of haematuria (11).

Further evaluation is required if there is persistence of haematuria after completion of antibiotic therapy. The presence of significant proteinuria, red cell casts, and dysmorphic red blood cells requires nephrology evaluation for possible medical renal disease. Urine cytology should be performed in older patients with painless, visible haematuria as they are at high risk for malignancy.

A serum creatinine and eGFR is used to assess baseline renal function and suitability for radiographic studies that require intravenous contrast. A complete blood count is helpful for evaluating potential anaemia and for the presence of infection. Other blood tests may be ordered if a coagulopathy is suspected.

Imaging of the upper urinary tract by CT urography is the imaging modality of choice, follows laboratory testing. Finally, urological referral for cystoscopy is necessary to rule out pathology of the lower urinary tract. Further investigations should be carried out in all patients with confirmed haematuria that is not explained by the above evaluations

History

Age: patients ages 35 years or older with visible haematuria are at higher risk for genitourinary tract cancer and require a full evaluation.

Gender: women may have pseudohematuria from recent intercourse, or from menses in premenopausal women.

Women tend to have more urinary tract infections than men. Men have a higher incidence of urinary tract cancer. Young women exposed to dieting agents containing aristolochic acid (Balkan nephropathy) are a special population at risk for upper tract urothelial carcinoma. Pregnant women with prior Caesarean sections are at risk for placenta percreta.

Timing of blood in the urine stream: the timing of haematuria during micturition (initial, terminal, total) is an important clue in localizing the source of bleeding (12).

Blood that appears at the onset of a void, then clears, is called initial haematuria. Terminal haematuria occurs at the end of a void. Initial and terminal haematuria represent bleeding from the urethra, prostate, seminal vesicles, bladder neck or trigone of bladder. Total haematuria, which is present throughout the void, indicates bleeding of bladder or upper tract (kidney or ureteral) origin. Sometimes shape of blood clot can give clue for site of origin, amorphous clots are usually from bladder and vermiform or worm like clots from renal or ureteric origin.

Lower urinary tract symptoms: a personal history of dysuria, urinary frequency, urgency, and urethral discharge points to an infectious or inflammatory process (14). Benign prostatic hyperplasia (BPH) can cause haematuria and obstructive urinary symptoms such as urinary hesitancy, straining to void, and a sensation of incomplete emptying. Urinary stasis, caused by severe BPH, can lead to urinary tract infection and bladder stone formation.

Haematuria alone does not cause pain unless it is associated with inflammation or acute urinary obstruction. Pyelonephritis and renal nephrolithiasis may present as flank pain. Pain from kidney stones often radiates to the groin. Intermittent or total bladder outlet obstruction by a bladder stone or clot can present as suprapubic pain or discomfort.

Recent vigorous physical activity: can cause a self-limited exercised-induced haematuria, but other important aetiologies must be ruled out.

Haematuria can be due to inflammatory or cytotoxic mechanisms. Any history of analgesic abuse should be elicited. The degree of therapeutic anticoagulation should be determined if appropriate.

Exposures such as smoking and industrial chemicals (benzene, aromatic amines): linked to urothelial carcinomas (12).

Peri-orbital and peripheral oedema, weight gain, oliguria, dark urine, or hypertension suggests a glomerular cause.

Recent pharyngitis or skin infection: may suggest post infectious glomerulonephritis.

Joint pains, skin rashes, and low-grade fevers suggest a collagen vascular disorder or systemic lupus erythematosus.

Family history: should include a history of kidney stones, cancer, prostatic enlargement, sickle cell anaemia, collagen vascular disease, and renal disease.

Recent urologic interventions: may cause recurrent haematuria, for example, bladder catheterization, placement of an indwelling ureteral stent, or recent prostate biopsy.

Physical exam

Vital signs: Hypotension and tachycardia are seen in patients who are hemodynamically unstable from acute blood loss. Body core temperature may be elevated in the setting of infection (12)

Pallor of the skin and conjunctiva: often seen in patients with anaemia.

Peri-orbital, scrotal, and peripheral oedema: may indicate hypoalbuminemia from glomerular or renal disease.

Cachexia: may indicate malignancy.

Tenderness of the flank or costovertebral angle: may be caused by pyelonephritis or by enlarging masses such as a renal tumour.

Suprapubic tenderness: can be elicited in the setting of cystitis, whether caused by infection, radiation, or cytotoxic medications (12).

The bladder is not palpable when decompressed: a bladder filled with 200 mL of urine is percussible. In acute urinary retention, usually seen in patients with BPH or obstruction by clots, the bladder is palpable and may be felt up to the level of the umbilicus (12).

An abnormal, nodular, digital rectal exam: may signify prostatic adenocarcinoma or an invasive bladder tumour. An enlarged prostate or enlarged median lobe of the prostate is a sign of benign prostatic hyperplasia.

Palpable adenopathy: either supraclavicular or inguinal, may indicate a neoplastic process.

The presence of a urethral catheter, suprapubic catheter, ureteral stent, or nephrostomy tube may signify an iatrogenic cause of bleeding that is generally benign.

Laboratory evaluation

Urine dip stick analysis must be performed for dark or discoloured urine to differentiate true haematuria from pseudohaematuria caused by medications or foods. False-positive tests may occur in the setting of myoglobinuria or haemoglobinuria, confirmed by the absence of red blood cells on microscopic exam. A low specific gravity is seen in urine that is poorly concentrated due to intrinsic renal disease. Heavy proteinuria (>3 g/day) suggests glomerulonephritis. The presence of nitrite or leukocyte esterase may indicate infection.

Microscopic evaluation of the urine will confirm the presence of red blood cells or casts. Frank haematuria will obscure the microscopic exam with a full field of red blood cells, usually reported as >150 red blood cells/high power field. Red cell casts or dysmorphic red blood cells indicate a tubular/glomerular source of bleeding. Bacteria, white blood cells, and white cell casts indicate a urinary tract infection. Crystals in the urine indicate urolithiasis.

Urine cultures should be performed in patients with clinical evaluation suggestive of infection to identify the cause of a urinary tract infection, and the sensitivity data used to direct appropriate antimicrobial therapy. Urine cultures should be performed on catheterized or clean-catch, mid-stream specimens to avoid contaminated results. A repeat urinalysis should be performed 6 weeks after treatment.

Urine cytology should be sent for patients with any risk factors for urothelial carcinoma.

Complete blood count can be sent to evaluate anaemia in cases of severe bleeding. Leucocytosis supports a diagnosis of infection.

Coagulation studies may be performed if there is suspicion for undiagnosed coagulopathy, disorders of haemostasis, or super therapeutic anticoagulation therapy. In general, these studies do not add to the evaluation of haematuria, and further investigations must be performed to determine the cause of bleeding. Visible haematuria in anticoagulated patients likely signifies underlying pathology. Nonvisible haematuria in anticoagulated patients has an incidence similar to the general population.

Other specific testing may include haemoglobin electrophoresis to diagnose sickle cell disease, or measurement of serum complement levels to evaluate glomerular pathology. Low serum complement levels are seen in post infectious glomerulonephritis, systemic lupus erythematosus nephritis, bacterial endocarditis, and membrano-proliferative glomerulonephritis. A high antistreptolysin O titre suggests a recent streptococcal infection.

Urinary bladder cancer markers (BTA stat, NMP22, uCyt +/-Immunocyt, UroVysion FISH, and microsatellite analysis) are being investigated. However, they are not proven or useful in the primary workup of haematuria. They may have a greater role in surveillance of known cancer patients with equivocal cytology results.

Prostate specific antigen may play a role in assessing the lower urinary tract (e.g., prostate cancer) as a source of visible haematuria.

Imaging studies (16, 17, 18, 19, 20, 21)

Imaging is a key part of the evaluation of haematuria and provides structural and functional information about the renal parenchyma and upper urinary tract. Several modalities are available for visualization of the upper urinary tract, including ultrasonography (US), computed tomographic urography (CTU), magnetic resonance urography (MRU), and intravenous urography (IVU). CTU is the imaging modality of choice, as it provides the greatest anatomic detail and the highest sensitivities and specificities for a range of aetiologies ranging from renal masses to stones to urothelial tumours (16). CTU, compared with IVU, has a superior ability to characterize renal masses, and a higher sensitivity in detecting upper tract urothelial tumours.(17,18).The noncontrast phase of CT can also detect renal stones with sensitivity of 94% to 98%, compared with 52% to 59% for IVU. (19)

An ideal CTU should have 4 distinct phases: a noncontrast phase establishes baseline tissue density and reveals urinary stones, fat, and hematoma; an arterial enhancement phase reveals

inflammatory or neoplastic structures; a corticomedullary phase can show sustained renal tissue changes and damage; and a delayed excretory phase allows for evaluation of the urothelium of the renal pelvis, ureters and bladder (20). When used in the evaluation of trauma, sufficient contrast is necessary to evaluate the injury effectively.

Prior to CTU, patient renal function should be assessed by the evaluating clinician, and a serum creatinine may be ordered to rule out impaired kidney function. The use of iodinated contrast is a well-known cause of acute renal failure, especially in patients with renal insufficiency (2). Clinicians should also be aware of the risks of severe contrast reactions, which are rare but well documented. Finally, clinicians should be aware of the dose of ionizing radiation delivered by each imaging modality, particularly in children and pregnant women (21, 22, 23, 24).

MRU provides less detailed anatomic visualization than CTU, but has the advantage of avoiding ionizing radiation. Useful in case of compromised renal function.

If circumstances preclude the use of both CTU and MRU, combining a noncontrast CT scan or renal US with retrograde pyelography (RGP) provides an alternative evaluation of the upper urinary system.

If urinary calculi are detected in a noncontrast CTU, a plain radiograph of the abdomen (kidneys, ureters, and bladder) should be performed to note the position and radio density of the stones for future follow-up. Often, a CT scout (topogram) is performed at the time of CTU, which can serve this purpose.

CT virtual cystoscopy (CTVC) and MR virtual cystoscopy (MRVC) have been investigated as potential tools in the detection of bladder tumours. Although one meta-analysis shows some improvement in sensitivity and specificity over conventional ultrasonography, the definitive test for the diagnosis of bladder tumour remains cystoscopy.

Nuclear renal scans, arteriography, and voiding cystourethrography are other imaging studies that can be ordered as clinically indicated, but are not part of the initial evaluation.

Special studies

Cystoscopy: during a cystoscopic examination, a rigid or flexible cystoscope is used to evaluate the urothelium of the bladder, prostate, and urethra. The ureteral orifices can be visualized, and upper tract bleeding can be seen as a jet of blood tinged urine or clot emanating from these structures. Because urothelial carcinoma can arise from any portion of the urinary tract mucosa, complete visualization of the bladder, bladder diverticula, and anterior and posterior

urethra is necessary. Prostatic hypertrophy can be seen, and associated varices that may cause bleeding can be visualized. A drawback of flexible cystoscopy is its limited usefulness in the presence of active urinary bleeding.

Retrograde pyelography (RGP): contrast can be injected into each ureteral orifice to opacify the luminal space of the ureter and kidney. In patients who cannot undergo CTU or MRU, RGP is an alternative.

Renal biopsy: may be necessary to determine a medical renal cause of visible haematuria. As certain types of medical renal disease, such as crescentic glomerulonephritis, can quickly progress to renal failure and requires prompt immunosuppressive therapy, an urgent consultation and kidney biopsy by a nephrologist may be necessary.

Asymptomatic microscopic haematuria

There is controversy in the investigation of asymptomatic microscopic haematuria, and various guidelines offer conflicting opinions as to what defines this condition and the extent of investigation deemed essential. Nevertheless this is indeed a common condition in the era of widespread use of anticoagulation for various indications and the clinician should follow the following broad principles (25).

Most authorities agree that a positive dipstick reading merits further evaluation by microscopy before proceeding to other investigations.

The assessment of the asymptomatic microscopic haematuria patient should include a careful history, physical examination, and laboratory examination to rule out benign causes. Once benign causes have been ruled out, asymptomatic microhaematuria should prompt a urologic evaluation.

At the initial evaluation, an estimate of renal function with calculated eGFR and creatinine are essential because intrinsic renal disease may have implications for renal related risk during the evaluation of patients with AMH. Dysmorphic red blood cells, proteinuria, cellular casts, and/or renal insufficiency, or any other clinical indicators suspicious for renal parenchymal disease warrants concurrent nephrology workup but does not preclude the need for urologic evaluation. Microhaematuria that occurs in patients who are taking anti-coagulants requires both urologic and nephrologic evaluation.

The initial evaluation for AMH should include a radiologic evaluation. Triphasic computed tomography (CT) urography (without and with intravenous (IV) contrast) is the imaging

procedure of choice because it has the highest sensitivity and specificity for imaging the upper tracts. For patients with relative or absolute contraindications that preclude use of multi-phasic CT (such as renal insufficiency, contrast allergy, pregnancy), magnetic resonance urography (MRU) (without/with IV contrast) is an alternative imaging approach. If contrast CT is contraindicated, magnetic resonance imaging (MRI) with retrograde pyelograms (RPGs) provides alternative upper tract evaluation. For patients with relative or absolute contraindications that preclude use of both multiphase CT (such as renal insufficiency, contrast allergy) and MRI (presence of metal in the body) non-contrast CT or renal ultrasound (US) with retrograde pyelography provides alternative evaluation of the entire upper tracts.

The use of urine cytology and urine markers is NOT recommended as a part of the routine evaluation of the asymptomatic microhaematuria patient.

For the urologic evaluation of asymptomatic microhaematuria, a cystoscopy should be performed on all patients aged 35 years and older. A cystoscopy should be performed on all patients who present with risk factors for urinary tract malignancies in age <35 years old.

In patients with persistent microhaematuria following a negative work up or those with other risk factors for carcinoma in situ (e.g., irritative voiding symptoms, current or past tobacco use, chemical exposures), cytology may be useful. If a patient with a history of persistent asymptomatic microhaematuria has two consecutive negative annual urinalyses (one per year for two years from the time of initial evaluation or beyond), then no further urinalyses for the purpose of evaluation of AMH are necessary. For persistent asymptomatic microhaematuria after negative urologic work up, yearly urinalyses should be conducted. For persistent or recurrent asymptomatic microhaematuria after initial negative urologic work-up, repeat evaluation within three to five years should be considered.

Discussion

In a resource challenged health care environment the evaluation of haematuria has to be rationalised, and adjusted to available facilities. After a review of guidelines the following issues seem pertinent to the evaluation of haematuria in India.

A) Cost of haematuria evaluation

Cost associated with haematuria evaluation can be substantial and include microscopic urinalysis, renal ultrasonography, cystoscopy, CT urography, urine cytology.

Although cytology still has a role for higher-risk patients with negative or equivocal initial evaluation results, indiscriminate use of these tests, as suggested in some earlier

recommendations, could result in potentially substantial direct and indirect cost and unnecessary anxiety.

The **single most useful** imaging investigation for a patient with haematuria is the contrast enhanced computed tomography with urography (delayed film) of the entire renal tract. Whilst routine urinalysis and ultrasonography are modest in cost and widely available, the CECT KUB or urography is an expensive initial investigations and is not widely available. It is imperative that health care management addresses issues in both the rationalisation of indications and cost of the CT urography.

The costs of the consulting urologist's and nephrologist's evaluation and management services are also a consideration, as well as any additional procedures or tests indicated in the adjudication of uncertain or suspicious findings on the initial evaluation

B) Referral Criteria

In India patients with haematuria in rural areas will usually present to the primary health center / primary care physician/practitioners of alternative medicine. In urban areas, they will very often find their way to a surgical specialist/ medical specialist/hospital emergency (government/private) and sometimes directly to a specialist urologist or even a nephrologist.

The initial evaluation is usually with urinalysis and urine culture and sensitivity and co prescription of an antibiotic in patients with a urinary tract infection. Most patients will undergo an ultrasonography of the renal tract. This is well within the practice realm of most medical practitioners in this country. By and large most guidelines worldwide do not recommend urine cytology and urine tests for tumour markers, and these tests are costly for inclusion into routine haematuria work up in this country.

It is important in our country to then **identify the correct referral pathway**, as available logistics: facilities, economics, expertise may all dictate how the evaluations proceed.

It is important that patients with gross haematuria are then referred to an urologist for further evaluation and specific treatment and an identified cause.

The investigation of asymptomatic microscopic haematuria (AMH) will require history and physical assessment for other potential AMH causes (e.g., infection, menstruation, or recent urologic procedures). Once these are excluded patients are followed up with further urinalyses, and if negative the evaluation may cease.

If there is persistent microscopic haematuria the patient will need renal function testing, imaging and cystoscopy. Nephrology evaluation is indicated if there is proteinuria, abnormal erythrocyte morphology, or other signs indicate medical renal disease. Areas of uncertainty include the age threshold for urologic evaluation (35–50y), the imaging modality of choice (CT for all vs. risk-stratified approach to CT vs. MRI vs ultrasonography for allvs USG with cystoscopy and retrograde pyelography), nephrology evaluation (concurrent vs. sequential, following negative urology evaluation).

Conclusion

Haematuria may be a sign of renal pathology, local infection, or systemic disease. Haematuria can be differentiated into 2 categories: macroscopic haematuria (visible to the naked eye) and microscopic haematuria (> 3 red blood cells/high-powered field on urinalysis). This review outlines the current literature regarding evaluation and management of patients who present with haematuria (Figs. 1, 2, 3).

Figures1, 2, 3: Adapted from Haematuria as a Marker of Occult Urinary Tract Cancer : Advice for High-Value Care From the American College of Physicians Matthew Nielsen, MD, MS, and Amir Qaseem, MD, PhD, for the High Value Care Task Force of the American College of Physicians* *AnnInternMed.* 2016;164:488-497.

Figure 1

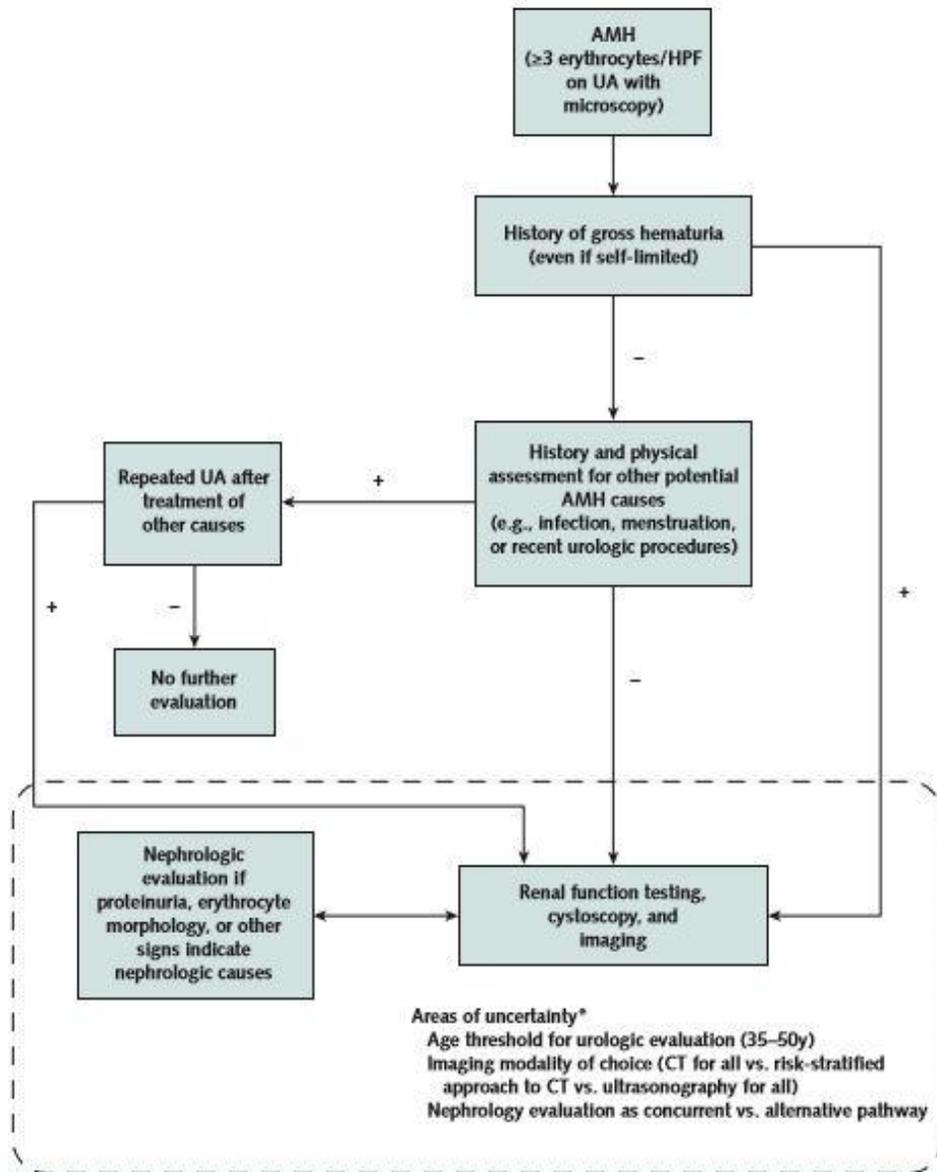
Table 1. Organizational Recommendations for the Initial Evaluation of Average-Risk Patients With Asymptomatic Microscopic Hematuria

Recommendation	Year	Reference	Case Definition				Components of Evaluation	
			Dipstick	Microscopic Urinalysis Results, erythrocytes/HPF	Positive/Total Test Results, n/N	Age Threshold, y	Cystoscopy	Preferred Imaging Method
American Urological Association guideline	2012	12	Inadequate	≥3	1	≥35	All patients	CT urography
American Urological Association best practice policy	2001	16	Inadequate	≥3	2/3	≥40	All patients	CT urography or IVP/ultrasonography
Canadian Urological Association guideline	2008	30	Inadequate	≥2	2	≥40	All patients	Renal ultrasonography
British Association of Urological Surgeons guideline	2008	32	≥1 heme	Not required	2/3	≥40	Not specified	Not specified
Dutch Guideline on Hematuria	2010	31	Inadequate	≥3	2/3	≥50	All patients	Renal ultrasonography

CT = computed tomography; HPF = high-powered field; IVP = intravenous pyelography.

Figure 2

Figure 1. Summary of recommendations for the evaluation of patients with hematuria.



AMH = asymptomatic microscopic hematuria; CT = computed tomography; HPF = high-powered field; UA = urinalysis.
* See Table 1 for more details.

Figure-3

THE EVALUATION OF HEMATURIA AS A MARKER OF OCCULT URINARY TRACT CANCER	
Disease/Condition	Hematuria
Target Audience	Internists, family physicians, and other clinicians
Target Patient Population	Adults with hematuria
Interventions Evaluated	Urine dipstick testing Microscopic urinalysis Cystoscopy Urinary cytology Ultrasonography Computed tomography
Outcomes Evaluated	Cancer detection
Benefits	Reduction in mortality and morbidity associated with possible cancer
Harms	Unnecessary testing Complications related to cystoscopy (pain or infection) Radiation exposure (for computed tomography) Hypersensitivity reactions and contrast nephropathy (for iodinated contrast with computed tomography) Potential association with subsequent unnecessary, invasive, and expensive procedures
High-Value Care Advice	<p><i>High-Value Care Advice 1: Clinicians should include gross hematuria in their routine review of systems and specifically ask all patients with microscopic hematuria about any history of gross hematuria.</i></p> <p><i>High-Value Care Advice 2: Clinicians should not use screening urinalysis for cancer detection in asymptomatic adults.</i></p> <p><i>High-Value Care Advice 3: Clinicians should confirm heme-positive results of dipstick testing with microscopic urinalysis that demonstrates 3 or more erythrocytes/HPF before initiating further evaluation in all asymptomatic adults.</i></p> <p><i>High-Value Care Advice 4: Clinicians should refer for further urologic evaluation in all adults with gross hematuria, even if self-limited.</i></p> <p><i>High-Value Care Advice 5: Clinicians should consider urology referral for cystoscopy and imaging in adults with microscopically confirmed hematuria in the absence of some demonstrable benign cause.</i></p> <p><i>High-Value Care Advice 6: Clinicians should pursue evaluation of hematuria even if the patient is receiving antiplatelet or anticoagulant therapy.</i></p> <p><i>High-Value Care Advice 7: Clinicians should not obtain urinary cytology or other urine-based molecular markers for bladder cancer detection in the initial evaluation of hematuria.</i></p>

HPF – high-powered field.

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Head Injury

Introduction

Head injuries are amongst the most common types of trauma encountered in emergency departments. They are usually associated with road traffic accidents; fall from height, physical assaults or as part of occupational or sports injury. Penetrating head injury can also occur due to assaults using heavy objects, gun shot or blast injury. Depending on their severity, early management steps under the appropriate emergency department settings may be of great benefits to such the patients. Many patients have some form of visible external injury but it is not uncommon to have no external stigmata. In such cases, any injury associated with altered level of consciousness should be ruled out for any associated brain injury.

Head injury may or may not be associated with the primary brain injury. Secondary injuries are sustained during the transport and treatment of patients with primary brain injury. Survivors of brain injury often continue having neuropsychological conditions that impair their daily lives. Hence the primary goal of treatment for patients with a suspected primary brain injury is to prevent secondary brain injury.

Causes of secondary brain injury

Hypotension
Hypoxia
Hypoglycemia
Seizures
Hypertension
Hyponatremia
Hypernatremia
Increased intracranial pressure

Incidence

According to the National Crime Records Bureau 2001, 2710,019 accidental deaths, 108,506 suicidal deaths and 44,394 violence-related deaths were reported in India. But specific number or incidence of Head injury patients in India is not known or published authentically as of now.

Classifications, clinical findings, and indications for imaging

Head injuries can be classified in several ways. The Glasgow Coma Scale (GCS) is being used by most of centres across world for neurological assessment in Head injury patients.

Glasgow Coma Scale:

Feature	Response	Score
Best eye response	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No eye opening	1
Best verbal response	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No verbal response	1
Best motor response	Obeys commands	6
	Localising pain	5
	Withdrawal from pain	4
	Flexion to pain	3
	Extension to pain	2
	No motor response	1

'Minor' head injury is indicated by a GCS score of 13 to 15. Patients with a GCS score of 9 to 12 are categorized as 'moderate'. A GCS score of 8 or less is the accepted definition of coma or 'severe' brain injury. Another classification of head injury uses morphology and divides the head injuries into skull fractures (further divided as vault or basilar) and intracranial lesions (further divided as focal or diffuse). Focal intracranial lesions include epidural, subdural, intracerebral hematomas and contusions. Concussion, multiple contusions, hypoxic/ischemic

injury and diffuse axonal injury comprise diffuse intracranial lesions. Subarachnoid haemorrhages either unifocal or multifocal can be associated with trauma, as an extension of a primary intracerebral hemorrhage. However such morphology based classifications are of little use while appreciating the 'severity' of the patient's head injury.

Recent NICE guidelines for CT Imaging post head injury (2014)

- A. For adults who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:
- GCS less than 13 on initial assessment in the emergency department.
 - GCS less than 15 at 2 hours after the injury on assessment in the emergency department.
 - Suspected open or depressed skull fracture.
 - Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
 - Post-traumatic seizure.
 - Focal neurological deficit.
 - More than 1 episode of vomiting.
- B. For adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury, perform a CT head scan within 8 hours of the head injury:
- Age 65 years or older.
 - Any history of bleeding or clotting disorders.
 - Dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 meter or 5 stairs).
 - More than 30 minutes retrograde amnesia of events immediately before the head injury.
- C. For children who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:
- Suspicion of non-accidental injury.
 - Post-traumatic seizure but no history of epilepsy.
 - On initial emergency department assessment, GCS less than 14, or for children under 1 year GCS (paediatric) less than 15.
 - At 2 hours after the injury, GCS less than 15.

- Suspected open or depressed skull fracture or tense fontanelle. Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
 - Focal neurological deficit.
 - For children under 1 year, presence of bruise, swelling or laceration of more than 5 cm on the head.
- D. For children who have sustained a head injury and have more than 1 of the following risk factors (and none of those in recommendation point C, perform a CT head scan within 1 hour of the risk factors being identified:
- Loss of consciousness lasting more than 5 minutes (witnessed).
 - Abnormal drowsiness.
 - Three or more discrete episodes of vomiting.
 - Dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from a height of greater than 3 meters, high-speed injury from a projectile or other object).
 - Amnesia (antegrade or retrograde) lasting more than 5 minutes.
- E. Children who have sustained a head injury and have only 1 of the risk factors in recommendation (and none of those in recommendation point C) should be observed for a minimum of 4 hours after the head injury. If during observation any of the risk factors below are identified, perform a CT head scan within 1 hour:
- GCS less than 15.
 - Further vomiting.
 - A further episode of abnormal drowsiness.
- F. If none of these risk factors occur during observation, use clinical judgement to determine whether a longer period of observation is needed.
- G. According to the recent guidelines, a provisional written radiology report should be made available within 1 hour of the scan being performed.

Basilar skull fractures are defined as fractures in the skull base and remain among the ones difficult to evaluate and treat. The sphenoid sinus, foramen magnum, temporal bone and sphenoid wings are the most common sites of these fractures. Signs of basilar skull fracture include hemotympanum, CSF otorrhea or rhinorrhea, raccoon eyes, Battle's sign and cranial nerve injuries. Typically from a direct frontal blow, the oculomotor nerve (CN III) in the middle cranial fossa can be affected with a skull base fracture and present, with signs of diplopia and impaired extra-ocular movements. CN VII palsy is also not uncommon in basilar skull fractures.

Head injury frequently involves cervical spine injury necessitating the imaging of the cervical spine. According to the National Emergency X-Radiography Utilization Study (NEXUS) Criteria cervical spine imaging is unnecessary in patients fulfilling all the following five criteria:

- Absence of midline cervical tenderness
- Normal level of alertness and consciousness
- No evidence of intoxication
- Absence of focal neurologic deficit
- Absence of painful distracting injury

Spinal cord injury without radiographic abnormality (SCIWORA) is most often encountered in paediatric population. Numbness, paresthesias, or other non-specific neurologic complaints with normal plain radiographs or CT scans should not be taken lightly and prompt further evaluation with MRI and urgent neurosurgical consultation is indicated.

In case of unclear history of trauma, it is necessary to rule out other causes of similar clinical scenario ischemic stroke, haemorrhagic stroke, venous sinus thrombosis, subarachnoid haemorrhage, CNS infections and non-traumatic / metabolic encephalopathies etc.

Penetrating and blast cranial injuries, though infrequent, pose a surgical challenge to the neurosurgeons. Patients presenting with GCS 6-12 definitely require urgent aggressive management. Recent guidelines suggest aggressive management even with lower scores due to emerging evidences of improved survival, especially in gunshot wounds. Studies suggest more aggressive decompression of the brain, less aggressive debridement and retrieval of deep bone and metal fragments and higher antibiotics like cefazolin.

Paediatric Head injury:

Pediatric craniospinal injuries differ from adult head injuries due different general and CNS anatomy and physiology. In infants and children less than 1 year, due to large size of head, airway management should include one to two inch pad below upper torso to avoid flexion of cervical spine and keep upper airway open. Larger scalp laceration in paediatric patients can cause hypovolemic shock also Children usually present with upper cervical subluxations or dislocations due to higher fulcrum of movement. This fulcrum of movement progressively shifts to lower levels with advancing age, threatening the subaxial spine. Head trauma injures the brain through the thin pliable bone. The bone may or may not fracture. Growing skull fractures and ping pong fractures are other entities common in young age. Due to the thin bones, open fontanelles and sutures, cerebral compliance is more in children. But, when intracranial volume rises acutely, ICP rises to dangerous levels. Thin bones pose difficulty in ICP monitoring. Also the target CPP values differ in children as compared to adults.

Psychiatric symptoms in the survivors of head injury include ailments like Post Traumatic Shock Disorder (PTSD) and post concussive symptoms. Patients present with anxiety, dysphoria, irritability, sleep disturbance, loss of concentration or memory and anger etc. Treatment includes both pharmacological and psychosocial interventions. Cognitive behavioral therapy has a proven efficacy in PTSD and non – PTSD trauma related conditions.

At a Peripheral / Limited resourced centre

Investigations

The basic investigations include:

1. Chest X-ray and x-rays to rule out other concomitant injuries e.g. limb fractures
2. Blood tests including RBS, complete blood count, S electrolytes, S creatinine
3. CT brain plain – if available and as per criteria mentioned above
4. Blood grouping and cross matching for severely injured patients

Primary management

As with any trauma patient, in a patient with head injury there is a need for rapid evaluation. Moreover, the absence of a definitive diagnosis and detailed history/mechanism of injury should never impede the application of essential treatment. As per ATLS Protocols, early primary survey with management of airway, cervical spine, breathing and circulation prevents hypoxemia, hypoperfusion, shock and damage to vital organs like brain, heart, liver and kidneys. Secondary survey helps to systematically rule out all other vital injuries Subsequent to the primary management, identification of a mass lesion that requires surgical evacuation is critical. This is best achieved by an immediate CT scan of the head in hemodynamically stable patients. Obtaining a CT scan should never delay patient transfer to a trauma centre capable of immediate and definitive neurosurgical intervention.

A well established trauma protocol is highly useful in adequate management of head injury patients, especially during the golden hour. Currently prevalent evidence based approach comprises of primary survey which includes ABCDE as described below, adjuncts to the primary survey, secondary survey, re-evaluation and referral.

A: Airway maintenance with cervical spine movement restriction

This involves assessing the patient's airway for its patency and protection. Basic airway opening maneuvers such as chin lift and jaw thrust are applied. It is very important that each patient with head injury is suspected for a possible cervical spine injury. In that case a head-tilt would do furtherharm. Hence a cervical collar is applied as soon as possible and jaw thrust is given for

opening the airway. In presence of gurgling sounds or unclear airway, suction is applied to clear any secretions, bleeding, vomitus or foreign body if. If patient's airway is at risk or GCS is low, a definitive airway such as endotracheal tube (ETT) intubation is required. In case of difficulty or when ETT intubation is contraindicated, surgical airway (needle or surgical cricothyroidotomy) is indicated.

B: Breathing and ventilation

The neck and chest are inspected, palpated and auscultated for tracheal deviation, chest movements, use of accessory muscles, bilateral air entry and any signs of injury. High Flow Oxygen is provided using appropriate mask.

C: Circulation and haemorrhage control

After large bore IV line access, IV crystalloids are given. Anti-fibrinolytic (tranexamic acid) can be added. Shock is suspected in a patient with cold, clammy, pale and peripherally shut down extremities. Volume replacement as per ATLS protocols that is one liter of warmed Ringer Lactate or Normal Saline followed by blood products in class III or IV shock is required. Permissive hypotension is indicated in selected patients especially in penetrating injury.

D: Disability and neurological dysfunction is assessed using GCS scoring, pupils, neurologic examination, lateralizing signs and spinal cord injury assessment.

E: Exposure and environmental control by careful undressing. Avoid hypothermia and undue movement of spine and injured areas.

Adjuncts to primary survey include Xray Chest and Pelvis, Arterial Blood Gas, Electrocardiogram, Ryle's tube and Foley's catheter if indicated and FAST (Focused Assessment by Sonography inTrauma) scan.

Secondary survey: The secondary survey is a thorough head-to-toe assessment of the patient after the primary survey, fluid resuscitation and IV drugs. It also includes a focused history including allergies, medications, past illnesses, possibility of pregnancy, last meal, events leading to the injury, focused physical examination and re-assessment of vital signs.

Re-evaluation and Referral: After the secondary survey, the patient should be re-evaluated with the ABCDE approach and examined for any missed injury (tertiary survey) such as fractures. Constant monitoring is required and rapid transfer to the operating room, or to another center having better tertiary care facilities has to be considered at any time if applicable. Appropriate referral for specialists i.e. neurosurgeon should be sent.

Primary treatment for associated facial injury

Head injury also involves associated facial contusion, abrasions, lacerations and soft tissue injuries. Adequate tetanus toxoid prophylaxis, antibiotic prophylaxis and wound care is warranted according to prevalent protocols. After careful evaluation and primary resuscitation timely reference to facio-maxillary / plastic surgeon is advised whenever indicated.

Primary treatment for cervical spine injury

Cervical immobilisation and imaging are warranted. Once a spinal cord injury (SCI) is identified, urgent Spine Surgeon consultation is recommended. Patient may be shifted to a higher centre with full spine immobilisation. Neurogenic shock is treated with oxygen, IV fluids, and, if necessary, inotropic support.

Control of Intracranial Pressure (ICP)

Hypoxia and hypotension are common causes of secondary brain injury. Mortality increases with the degree of desaturation and the duration of hypoxia. Cerebral Perfusion Pressure (CPP) and mean arterial blood pressure (MAP) are useful indicators for hypoxia and hypotension respectively. CPP is equal to the MAP minus the ICP. Thus, brain perfusion is reduced either by a decrease in blood pressure, or by an increase in ICP. Head injuries may increase ICP by the presence of mass-lesions (e.g. haematoma) and / or edema preventing the free circulation of cerebro-spinal fluid (CSF). Elevation of ICP can reduce cerebral perfusion and cause or exacerbate ischemia.

Various treatments aim to lower ICP:

Hyperosmolar therapy using 20% Mannitol is indicated in raised ICP. Mannitol 0.5-1g/kg boluses can decrease ICP dramatically up to 50% within 30 minutes post administration by reducing cerebral parenchymal cell water. Loop diuretics e.g. furosemide can also decrease ICP. However there is a risk of dyselectrolytemias. Hence electrolytes have to be monitored. Moreover, in patients with GCS <8 mechanical ventilation is indicated and it can be used to not only maintain PaO₂ but also control PaCO₂ by controlled hyperventilation. Controlled hyperventilation aims to reduce ICP thereby augmenting CPP. Therapeutic use of steroids are not indicated in brain injury and may be harmful. In centers where advanced monitoring is available, the ventricular catheter connected to an ICP monitor is the most accurate, low cost, and reliable method of monitoring ICP. It also allows therapeutic CSF drainage. Surgical removal of a portion of the skull, known as decompressive craniectomy (DC), has been performed for the purpose of relieving elevated intracranial pressure with outcome improvement in specific TBI patients but it lacks sufficient evidence to support Level I evidence. Other supportive measures

include prophylactic measures for GI ulcers, deep vein thrombosis, seizures and infection. For thromboprophylaxis, a combination of intermittent pneumatic compression (IPC) with low molecular weight heparin (LMWH) is superior to using either modality alone. IPC is contraindicated in lower limb injuries and LMWH is used with caution in patients at risk of bleeding. However expert opinion is recommended in employing all these treatment options. In view of lack of experience in handling such patients it would be recommended to promptly transfer the patient to a higher centre in haemodynamically stable condition.

Referral criteria

- CT scan not available
- Operation theatre not available
- Neurosurgeon/ Spine surgeon not available
- Polytrauma, especially associated chest trauma
- Lack of expertise in initiating appropriate primary treatment
- Lack of Critical care facility
- In patients with late presentation, persistent unconsciousness or deterioration
- Hemodynamic instability with requirement of mechanical ventilation and ICP monitoring due to persistent intracranial hypertension, despite medical and initial surgical management

At a tertiary care centre

Investigations

- CBC, Renal Function Test with electrolytes, SGPT, BT,CT,PT, APTT
Blood Group & Cross Matching with Blood products availability
- CT scan Brain with Cervical Spine Screening
- MRI scan
- EEG monitoring
- ICP and CPP monitoring

Definitive Management

The diagnosis of head injury relies on brain imaging. Hence when CT or MRI facilities are unavailable it is advisable to promptly transfer the patient to a higher centre. After imaging, based on neurosurgical opinion decompressive craniotomy remains the definitive treatment. Spine surgeon consultation is promptly recommended in case of SCI.

In those patients known or suspected to be on anticoagulants, a repeat CT scan is advised after 6-8 hours or upon clinical deterioration. An INR test should be done as soon as possible and

anticoagulant reversal may be required. Patients on warfarin with intracranial bleeding should receive prothrombin complex concentrate in addition to vitamin K, unless the bleed is extremely small.

Apart from Mannitol, hyperosmolar therapy can be applied using hypertonic saline in raised ICP. Studies have shown hypertonic saline as better than Mannitol in patients having hypotension, hypovolemia and acute kidney injury (AKI). Central venous access is preferable for giving hypertonic saline, and its dosage and serum osmolality should be monitored. Hence it is recommended that hypertonic saline is used only at higher centres.

ICP monitoring is indicated in patients with GCS <8, abnormal CT scan findings, presence of co-morbidities and evidence of hypotension despite initial fluid resuscitation. When ICP monitoring is required the patient is advised for transfer to higher centre. Ventricular catheters and fiberoptic transduced monitors are available but cost may be an issue. ICP should be regulated once pressure exceeds 20 mmHg because sustained pressures greater than 20 mmHg are associated with poor outcomes. CPP should be maintained between 50-70mmHg and can be calculated using ICP and MAP values. Jugular venous oxymetry is recommended in case of therapeutic hyperventilation.

Another treatment option for raised ICP is analgesia, anaesthesia, sedation and neuromuscular blockade. Sedation decreases anxiety, fear, and response to pain, all of which increase ICP. Morphine and Midazolam can be used. Propofol is also useful in moderate doses. For inducing barbiturate coma Pentobarbital is given in a loading dose of 10 mg/kg body weight followed by a maintenance dose of 1 to 2 mg/kg/h. Neuro-muscular blockers can also be used to reduce ICP by relaxation and avoidance of coughing. Obviously higher centre and expert opinion are required since these options carry multiple risks including hypotension and respiratory depression. Concomitant continuous EEG monitoring may be useful.

For intracranial hypertension refractory to initial medical management, therapeutic hypothermia can be an option. Hypothermia decreases the cerebral metabolic rate and oxygen consumption the extent of brain injury. Therapeutic hypothermia is best handled in ICU settings under critical care expertise and hence requires a higher centre.

Complications

- Secondary brain injury
- Dyselectrolytemias
- Sepsis, multi-organ dysfunction syndrome (MODS)
- Cervical spine related complications

Medico-legal issues

- Failure to detect/investigate for head injury
- Missed concomitant injuries
- Delay in referral to higher centre
- No/delayed visit from neurosurgeon
- Delay in definitive treatment
- Delay in diagnosing complications

Minimum required human resources as healthcare professionals and their role

- Nursing staff: Primary resuscitation, wound dressing, nursing care, accompanying the patient during transfer for imaging / to higher centre
- MO / CMO / Emergency Physician: Primary treatment including ABCDE, high degree of suspicion for cervical spine and brain injuries, prompt referral to neurosurgeon.
- Pathologist and their support staff: For reporting patient's blood investigations
- Blood bank staff: For arrangements in case of need for blood products
- Pharmacy staff: For supplying prescribed medications
- Radiologist and their support staff: For conducting and reporting patient's radiological investigations
- Neurosurgeon: Diagnosis, work-up, definitive treatment, high degree of suspicion for complications, explanation and counseling regarding the patient's condition, treatment options and prognosis, operative planning and procedures
- Anaesthetist: Pre-operative checkup, fitness, performing intra-operative anaesthesia, post-operative care.
- OT technicians/nurses: OT assistance, positioning, equipment maintenance and checks
- Support staff: For cleaning and patient transfer including ambulance facilities
- Critical Care Physician: Post-operative care, intensive care
- Other specialist physicians and surgeons: As per patient's co-morbid conditions and requirements e.g. spine surgeon, neurosurgeon, facio-maxillary / plastic surgeon.

Consumables and Drugs

- Stretcher/wheelchair, patient bed, monitor, ECG leads, ECG machine, suctioning facility, oxygen supply, dressing trolley and consumables, Ryle's tube, Foley's catheter.
- IV line, IV set, injections, syringes, needles, pharmacy equipped with medications including tetanus toxoid, IV fluids, antacids, antiemetics, analgesics, antibiotics, specialized drugs as prescribed e.g. antiepileptics, anti-fibrinolytics, hyperosmolar agents, sedatives, etc.

- OT table, lights, instrument trolley, set of surgical instruments, drains, catheters, cautery set
- Anesthetic machine, instruments including endotracheal tubes and related drugs, suction
- ICU bed, ventilator, defibrillator, monitoring of ICP, CPP, EEG

Further Reading:

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Hollow Viscous Injuries

Introduction

Evaluation of patients who have sustained blunt abdominal trauma may pose a significant diagnostic challenge to the most seasoned trauma surgeon. Blunt trauma produces a spectrum of injury from minor, single-system injury to devastating, multi-system trauma. Trauma surgeons must have the ability to detect the presence of intra-abdominal injuries across this entire spectrum. The small bowel is injured in 5-10% of blunt trauma injuries, 30% of stab injuries and 50% of gunshot wounds (1). Patient assessment is often compromised by alcohol or drug intoxication, altered neurological status or injuries to the adjacent structures such as ribs and spine.

Indian Incidence

There are no available statistics on this topic. At our institute, LTMGH, which is a tertiary trauma care center and has about 3000 admissions in trauma ICU annually, incidence of laparotomies for hollow viscus injuries is about 1.6%.

Clinical Presentation

History

Mechanism of injury - (Vehicle collision, intrusion, restraints, deceleration injury etc).

Penetrating injury – type of weapon, distance from the weapon in gunshot, number of stab wounds.

Examination

Inspection:- Abrasions, seat belt sign, penetrating injuries, evisceration, impaled foreign body.

Examination of perineal area for lacerations is a must.

Palpation:- Look for signs of peritonitis ie. tenderness, guarding, rigidity.

Percussion:- Absence of liver and/or splenic dullness.

Auscultation- Absent bowel sounds indicate ileus, difficult to assess in noisy emergency department.

Per Rectal examination – bleeding, laceration

Investigations

1. Chest X-ray – Upright X-Ray chest showing free pneumoperitoneum may indicate stomach or bowel perforation.

2. Diagnostic Peritoneal Lavage (DPL) - DPL is a safe, rapid and accurate method for determining the presence of intra-peritoneal blood in victims of blunt abdominal trauma. The accuracy of DPL is between 92% and 98% (2). The high sensitivity is due to the high false positive rate (3). It is more accurate than CT for the early diagnosis of hollow visceral and mesenteric injuries, but it does not reliably exclude injuries to retroperitoneal structures (4). False positive results may occur in the presence of pelvis fractures. Hemodynamically stable patients with equivocal results are best managed by additional diagnostic testing to avoid unnecessary laparotomies.

3. CT Abdomen - CT scan is the most commonly used diagnostic modality in evaluating the abdomen in hemodynamically stable blunt trauma victims. It is occasionally used in evaluating hemodynamically stable patients with penetrating injuries particularly to the back and flank areas. Findings suspicious for blunt bowel / mesenteric injuries on CT include unexplained intra-peritoneal fluid, pneumoperitoneum, bowel wall thickening, mesenteric fat streaking, mesenteric hematoma, and extravasation of either luminal or vascular contrast (5). The lung window in abdominal CT allows to detect even small quantities of free intra peritoneal air not detectable on X-ray. The accuracy of CT in hemodynamically stable blunt trauma patients has been well established. Sensitivity between 92% and 97.6% and specificity as high as 98.7% has been reported in patients subjected to emergency CT (6). Most authors recommend admission and observation following a negative CT scan (7). Another advantage of CT scanning over other diagnostic modalities is its ability to evaluate the retroperitoneal structures.

4. FAST -The focused assessment by sonography for trauma (FAST) has been a widely used test in the initial evaluation of suspected abdominal trauma. This is not as sensitive as DPL or CT in detecting stomach or small bowel injuries. This is most likely because of the relative inability of the FAST exam to pick up small amounts of free fluid typically found with isolated hollow viscus perforations (8).

5. Diagnostic Laparoscopy (DL) - One of the potential benefits postulated is the reduction of non-therapeutic laparotomies. With modification of the technique to include smaller instruments, portable equipment and local anesthesia, DL may be a useful tool in the initial evaluation of blunt abdominal trauma as well as certain penetrating abdominal trauma. Although there are no randomized, controlled studies comparing DL to more commonly utilized modalities, experience at one institution using mini laparoscopy demonstrated a 25% incidence of positive findings on DL, which were successfully managed non-operatively and would have resulted in nontherapeutic laparotomies (9).

Management of Abdominal Trauma

ATLS Principles - ABCDE

Management

After the initial evaluation and resuscitation of the injured patient, patients with suspected or recognized injury to the stomach or small bowel should undergo immediate exploratory laparotomy through a midline incision. After the initial control of significant hemorrhage, contamination from the GI tract is then addressed. In patients with ongoing hemorrhage temporized by packing, gastric and bowel perforations can be rapidly controlled by placing an atraumatic clamp such as a Babcock clamp on the perforated viscus. Alternatively, these perforations may be controlled by a running closure of the perforation(s) with Polygalactin suture. This is particularly effective if there is significant bleeding from the lacerated stomach/intestine or adjacent mesentery. All injuries identified are repaired as the next step.

In the patient who is hemodynamically stable, definitive repair of hollow viscous injuries is relatively straightforward and based on their severity grade.

Stomach Injury Scale (10)

I	Contusion or hematoma	2
	Partial thickness laceration	2
II	Laceration in GE junction or pylorus < 2 cm	3
	In proximal one-third of stomach < 5 cm	3
	In distal two-thirds of stomach < 10 cm	3
III	Laceration > 2 cm in GE junction or pylorus	3
	In proximal one-third of stomach > 5 cm	3
	In distal two-thirds of stomach > 10 cm	3
IV	Tissue loss or devascularization < two-thirds of stomach	4
	Tissue loss or devascularization > two-thirds of stomach	4

Duodenum Injury Scale (11)

I	Hematoma	Involving single portion of duodenum
	Laceration	Partial thickness, no perforation
II	Hematoma	Involving more than one portion
	Laceration	Disruption <50% of circumference
III	Laceration	Disruption 50 to 75% circumference of D2
		Disruption 50 to 100% circumference of D1, D3, D4
IV	Laceration	Disruption >75% circumference of D2
		Involving ampulla or distal common bile duct
V	Laceration	Massive disruption of duodeno-pancreatic complex
	Vascular	Devascularization of duodenum

Small Bowel, Colon and Rectum Injury Scale (10,11)

I	Hematoma	Contusion or hematoma without devascularization	2
	Laceration	Partial thickness, no perforation	2
II	Laceration	Laceration < 50% of circumference	3
III	Laceration	Laceration > 50% of circumference without transection	3
IV	Laceration	Transection (with extension into perineum for rectum)	4
V	Laceration	Transection with segmental tissue loss	4
	Vascular	Devascularized segment (small bowel, rectum)	4

Stomach

Most intramural hematomas (grades I and II) are repaired with interruptible silk Lembert suture closure after evacuation of the hematoma and hemostasis are obtained. Small grade I and II perforations can be closed primarily in one or two layers. Because the stomach is quite vascular and often bleeds profusely, we prefer a two-layer closure after hemostasis is achieved. A running locked absorbable suture should be used for the inner layer, and interrupted seromuscular sutures of 3.0 or 4.0 silk should be used for the outer layer.

Large (grade III) injuries near the greater curvature can be closed by the same technique or by the use of a GIA stapler. Care must be taken to avoid stenosis in the gastroesophageal and pyloric area. A pyloric wound may be converted to a pyloroplasty to avoid possible stenosis in this area. Extensive wounds (grade IV) may be so destructive that either a proximal or distal gastrectomy is required. Reconstruction with either a Billroth I or II anastomosis is dictated by the presence or absence of an associated duodenal injury. In rare cases, a total gastrectomy and a Roux-en-Y esophago-jejunostomy is necessary for severe injuries (grade V) (10).

Small Bowel

Examination of the small intestine for injury is achieved by evisceration of the small bowel to the right and careful inspection of its entire length. The decision to resect versus repair bowel is made only after careful assessment of the proximity of bowel perforations and the adequacy of the blood supply to the bowel in question and patients overall status. Mesenteric hematomas adjacent to the bowel wall following penetrating injury should be carefully opened and the mesenteric aspect of the bowel inspected for injury. Obvious serosal tears should be closed with interrupted silk Lembert sutures. Grade II injuries are repaired by careful debridement and primary closure. The preferred method is to use a two-layer closure with a continuous Vicryl suture for the inner layer and interrupted silk sutures for the outer layer. Alternatively, a single layer closure with a running or interrupted suture can be used. A transverse closure is preferable because it assures the widest luminal opening. Alternatively, a GIA stapler may be

placed through the injury and a stapled resection and anastomosis performed. Currently used stapling devices have 3 rows of staples on either side and thus do not require oversewing of staple line saving time. Adjacent through and through wounds of the bowel are joined transversely using electrocautery and closed as a single defect. Multiple grade II injuries can usually be closed individually. Small bowel resection for multiple perforations is not recommended unless resection and anastomosis would take less time than closing the perforations individually and the amount of bowel sacrificed is minimal.

Concerns about the mesenteric circulation and residual luminal diameter dictate the treatment of grade III and IV injuries. Injuries to more than 50% of the small bowel circumference should usually be resected because of the high likelihood of luminal narrowing with primary closure. However, grade III wounds that are oriented transversely or in the relative large proximal to mid jejunum may be primarily repaired provided that an adequate lumen (at least 30% of the circumference) is maintained.

Complete transection of the bowel (grade IV) is treated by resection of the injured bowel and its adjacent blood supply followed by anastomosis. Grade V injuries involve small bowel transections with segmental tissue loss or segmental devascularization and requires resection with anastomosis. However if the patient is hemodynamically unstable or if there is gross contamination and sepsis, a stoma is to be preferred over a resection and anastomosis. (10).

Duodenum

Grade I & II – If a duodenal hematoma is diagnosed on CT management is conservative consisting of nasogastric tube suction and TPN. In some cases of failure, operative management may be required. Small hematomas best managed by NG tube suction and distal feeding jejunostomy for enteral nutrition. Large hematomas may require evacuation and meticulous closure of duodenum. In case of lacerations single layer repair with polygalactin interrupted sutures is recommended.

Grade III – Primary repair with pyloric exclusion gastrojejunostomy and feeding jejunostomy is the recommended procedure of choice. Even if a lateral duodenal fistula develops it is a controlled fistula and patient usually tolerates diet n 10-12 days. If primary repair will result in a stenotic lumen then a Roux-en-y end to side duodeno-jejunostomy is done.

Grade IV & V – These injuries involve massive disruption of the duodeno-pancreatic complex and usually damage control surgery is done. Reassessment when patient is stable may warrant a Whipple's procedure (11).

Colon

Primary repair is appropriate for the majority of colon injuries and may be hand sewn single layer anastomosis or stapler anastomosis. However distracting colon injuries, defined as those requiring resection require a 360 degree anastomosis and therefore may increase the likelihood for failure due to the sheer extent of the anastomotic line. Additionally, there may be vascular impairment, as significant mesocolic trauma is almost always present. Finally, the presence of a distracting colon injury is an indicator of the high general severity of injury, which may affect unfavorably the healing of a fresh anastomotic line. In such cases it is prudent to perform a diverting loop colostomy.

Rectum

Intra-peritoneal rectal injuries can be managed like the rest of colon injuries, that is, with primary repair. However, primary repair without a colostomy has not yet been established for extra-peritoneal rectal injuries. The lack of serosal coverage and the difficulty in accessing the low rectal area through a trans-peritoneal approach are routinely cited reasons for adding colostomy as a safeguard of a challenging repair. If the anatomical planes around the rectum are not disturbed by the extensive dissection of trans-peritoneal access, primary repair without colostomy may be safe through the anus (12).

Postoperative management

Mainly consists of ICU care, fluid management and observation of nasogastric suction and drain output. In case of duodenal injuries associated pancreatic contusion may result in pancreatitis which complicates management. Watch for failure of primary repair or anastomotic leaks which may warrant redo laparotomy and stoma formation.

Team responsibilities

Surgeon:- Diagnosis & Work up
 Preoperative planning
 Operative procedure
 Post-operative care in conjunction with Anaesthetist / Intensivist
 Post-operative follow up

Anaesthetist:- Pre -anaesthesia Check up
 Part of resuscitation
 Performing anesthesia
 Post op ICU management in conjunction with Surgeon

Nurse:- Pre/Intra/Postop comprehensive care

Resources required for one patient / Procedure

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 2
3. Anesthetist – 1
4. Pathologist – 1 – Services can be availed from outside
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Renal Function Test
3. LFT in selected cases
4. S. Electrolytes in selected cases
5. USG FAST
6. X- Ray – Chest
7. CT Abdomen

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Dressings

Referral Criteria

ICU care is needed in patients with polytrauma and high grade injuries

Neurosurgical intervention may be required in associated head injury

Medicolegal Issues

- Failure to detect / investigate or refer a patient with hollow viscus injury. Commonly this happens in duodenal and colonic injuries.
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action.

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Intestinal Obstruction

Introduction

Intestinal obstruction accounts for approximately 15 percent of all emergency department visits for acute abdominal pain. Morbidity and mortality associated with intestinal obstruction have declined since the advent of more sophisticated diagnostic tests, but the condition remains a challenging surgical diagnosis.

Pathophysiology

Proximal to the point of obstruction, the intestinal tract dilates as it fills with intestinal secretions and swallowed air. Failure of intestinal contents to pass through the intestinal tract leads to a cessation of flatus and bowel movements. Fluid loss from emesis, bowel edema, and loss of absorptive capacity leads to dehydration. Emesis leads to loss of serum electrolytes, and significant dehydration stimulates renal proximal tubule reabsorption of bicarbonate and loss of chloride, perpetuating the metabolic alkalosis. In addition to derangements in fluid and electrolyte balance, intestinal stasis leads to overgrowth of intestinal flora, which may lead to the development of feculent emesis. Additionally, overgrowth of intestinal flora in the small bowel leads to bacterial translocation across the bowel wall.

Ongoing dilation of the intestine increases luminal pressures. When luminal pressures exceed venous pressures, loss of venous drainage causes increasing edema and hyperemia of the bowel. This may eventually lead to compromised arterial flow to the bowel, causing ischemia, necrosis, and perforation. A closed-loop obstruction may undergo this process rapidly. Intestinal volvulus, the prototypical closed-loop obstruction, causes torsion of arterial inflow and venous drainage, and is a surgical emergency.

Etiology

Adhesions (m.c, 60%)

Neoplasms (20%)

Hernias (10%)

Intussusception (5%)

Volvulus (5%)

Others

Classification-

Dynamic or mechanical obstruction

Adynamic obstruction or paralytic ileus.

Clinical features-

Symptoms-

1. Pain abdomen-colicky and intermittent. Paralytic ileus is painless.
2. Vomiting
3. Abdominal distension-central in small bowel obstruction and peripheral in large bowel obstruction
4. Constipation /obstipation

Signs-

1. Signs of dehydration
2. Abdominal distension, tympanitic note on percussion, borborygmi on auscultation.
3. Strangulation should be suspected when the above features are associated with signs of local or generalized peritonitis wit/without septic shock.

Evaluation

1. History, physical examination, per rectal examination
2. Imaging

Imaging should answer the following questions-

1. Small vs large bowel obstruction
2. Site of obstruction
3. Cause of obstruction
4. Complications like ischemia/gangrene/perforation

Radiography

The initial evaluation of patients with clinical signs and symptoms of intestinal obstruction should include plain upright abdominal radiography. Radiography can quickly rule out intestinal perforation. It accurately diagnoses intestinal obstruction in approximately 60 percent of cases. However, plain abdominal films can appear normal in early obstruction and in high jejunal or duodenal obstruction. Therefore, when clinical suspicion for obstruction is high or persists despite negative initial radiography, non-contrast computed tomography (CT) should be ordered.

Features of small bowel obstruction (SBO) on abdominal radiograph-

1. Dilated small bowel loops >3cm in diameter
2. Multiple air fluid levels
3. Air fluid levels longer than 2.5 cm

4. Paucity of colonic and rectal gas

Computed Tomography

CT is appropriate for further evaluation of patients with suspected intestinal obstruction in whom clinical examination and radiography do not yield a definitive diagnosis. CT is sensitive for detection of high-grade obstruction, and has the additional benefit of defining the cause and level of obstruction in most patients.

Indications of CT in intestinal obstruction

When the diagnosis is in doubt, when there is no surgical history or hernias to explain the etiology, or when there is a high index of suspicion for complete or high-grade obstruction.

In addition, CT can identify emergent causes of intestinal obstruction, such as volvulus or intestinal strangulation.

Features of small bowel obstruction on CT scan-

Criteria	Specific Criteria
Major	Small bowel dilated to 2.5 cm or greater and colon not dilated (<6 cm)
	Transition point from dilated to nondilated small bowel
Minor	Air fluid levels
	Colon decompressed

Enteroclysis

Contrast studies, such as a small bowel follow-through (CT/MR enteroclysis), can be helpful in the diagnosis of a partial intestinal obstruction in patients with high clinical suspicion and in clinically stable patients in whom initial conservative management was not effective. The use of water-soluble contrast material is not only diagnostic, but may also be therapeutic in patients with partial small-bowel obstruction. Contrast fluoroscopy may also be useful in determining the need for surgery; the presence of contrast material in the rectum within 24 hours of administration has a 97 % sensitivity for spontaneous resolution of intestinal obstruction.

Magnetic Resonance Imaging

MRI enteroclysis, which involves intubation of the duodenum and infusion of contrast material directly into the small bowel, can more reliably determine the location and cause of obstruction. However, because of the ease and cost-effectiveness of abdominal CT, MRI remains an investigational or adjunctive imaging modality for intestinal obstruction.

Goals in management for bowel obstruction

1. The goals of initial management are to relieve discomfort and restore normal fluid volume and electrolytes in preparation for possible surgical intervention
2. Triaging the patients who require surgery and who can be given a trial of conservative management.

Components of initial management

1. In general, all patients with mechanical bowel obstruction should be made nil per os (NPO) to limit bowel distension.
2. Mechanical bowel obstruction that is associated with significant distension, nausea, and/or vomiting, nasogastric tube decompression is recommended.
3. **Fluid therapy**-Patients with bowel obstruction can have severe volume depletion, metabolic acidosis or alkalosis, and electrolyte abnormalities. Upon admission, adequate intravenous (IV) access in the form of two large-bore peripheral lines should be obtained for fluid resuscitation. Lactated ringers or normal saline may be appropriate for intravenous fluid therapy. Electrolyte abnormalities should be corrected at the same time.
4. **Rate of replacement** -The rate of correction of volume depletion depends upon its severity. With severe volume depletion or hypovolemic shock, at least 1 to 2 liters of isotonic saline/ RL are generally given as rapidly as possible in an attempt to restore tissue perfusion. Fluid replacement is continued at a rapid rate until the clinical signs of hypovolemia improve (eg, low BP, low urine output, and/or impaired mental status). Further replacement should be guided by urine output (goal -1ml/kg/hour) and CVP.

In comparison, rapid fluid resuscitation is not necessary in patients with mild to moderate hypovolemia. To avoid worsening of the volume deficit, the rate of fluid administration must be greater than the rate of continued fluid losses, which is equal to the urine output plus estimated insensible losses (usually 30 to 50 mL/hour) plus any other fluid losses (eg, gastrointestinal losses) that may be present.

5. **Role of antibiotics** — For uncomplicated small bowel obstruction, antibiotics should not be administered. Although administering broad-spectrum antibiotics is practiced because of concerns for bacterial translocation, data are inadequate to support or refute such a practice. Antibiotics are also warranted for patients with complications (eg, perforation, gangrene) and antibiotic prophylaxis should be administered to those who will undergo operative exploration.

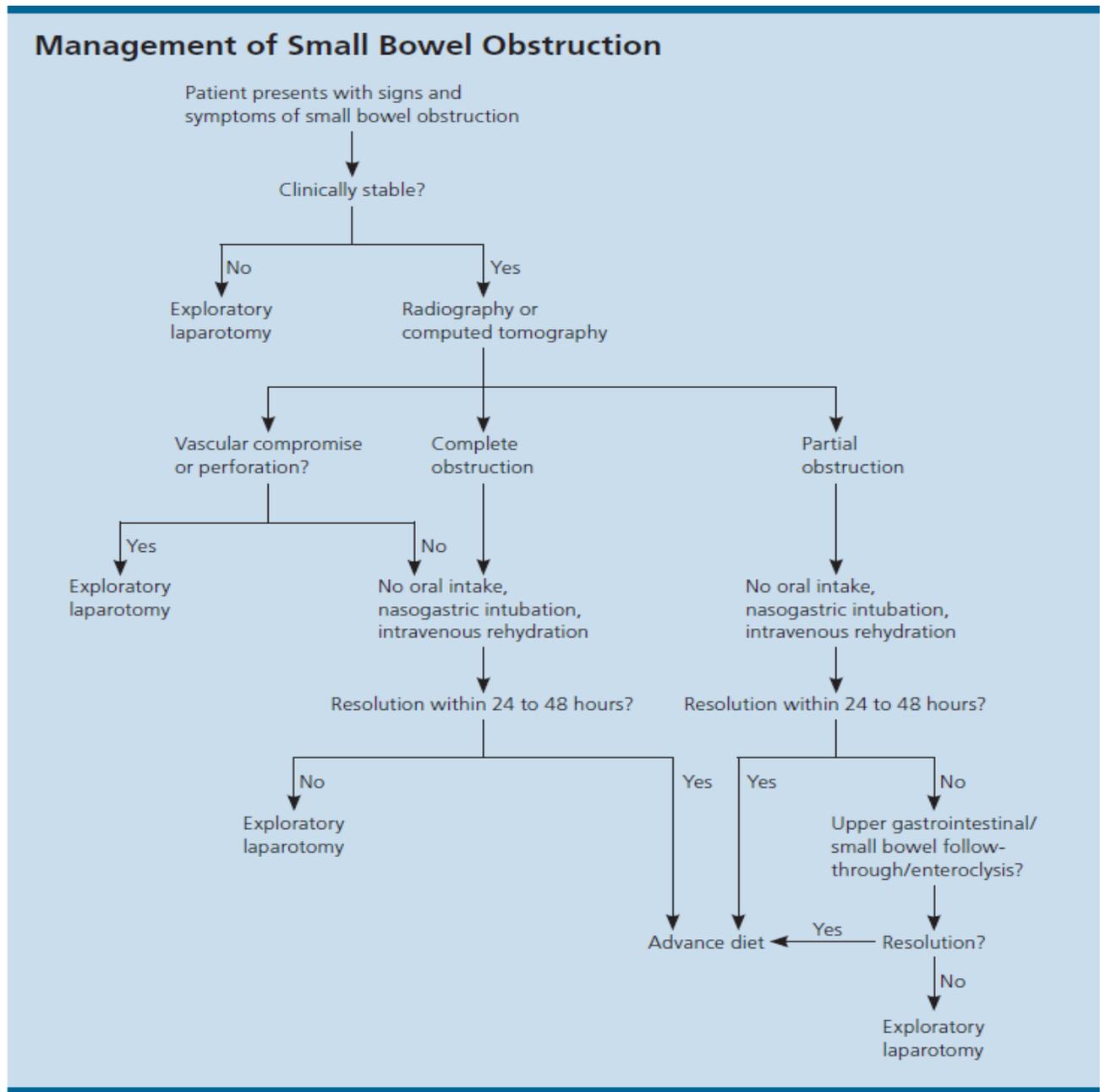
In general, empiric regimens for intra-abdominal infections include antimicrobial activity against enteric streptococci, coliforms, and anaerobes. A combination of 2nd/3rd generation cephalosporin and metronidazole is the preferred regimen.

Indications for Surgical Exploration

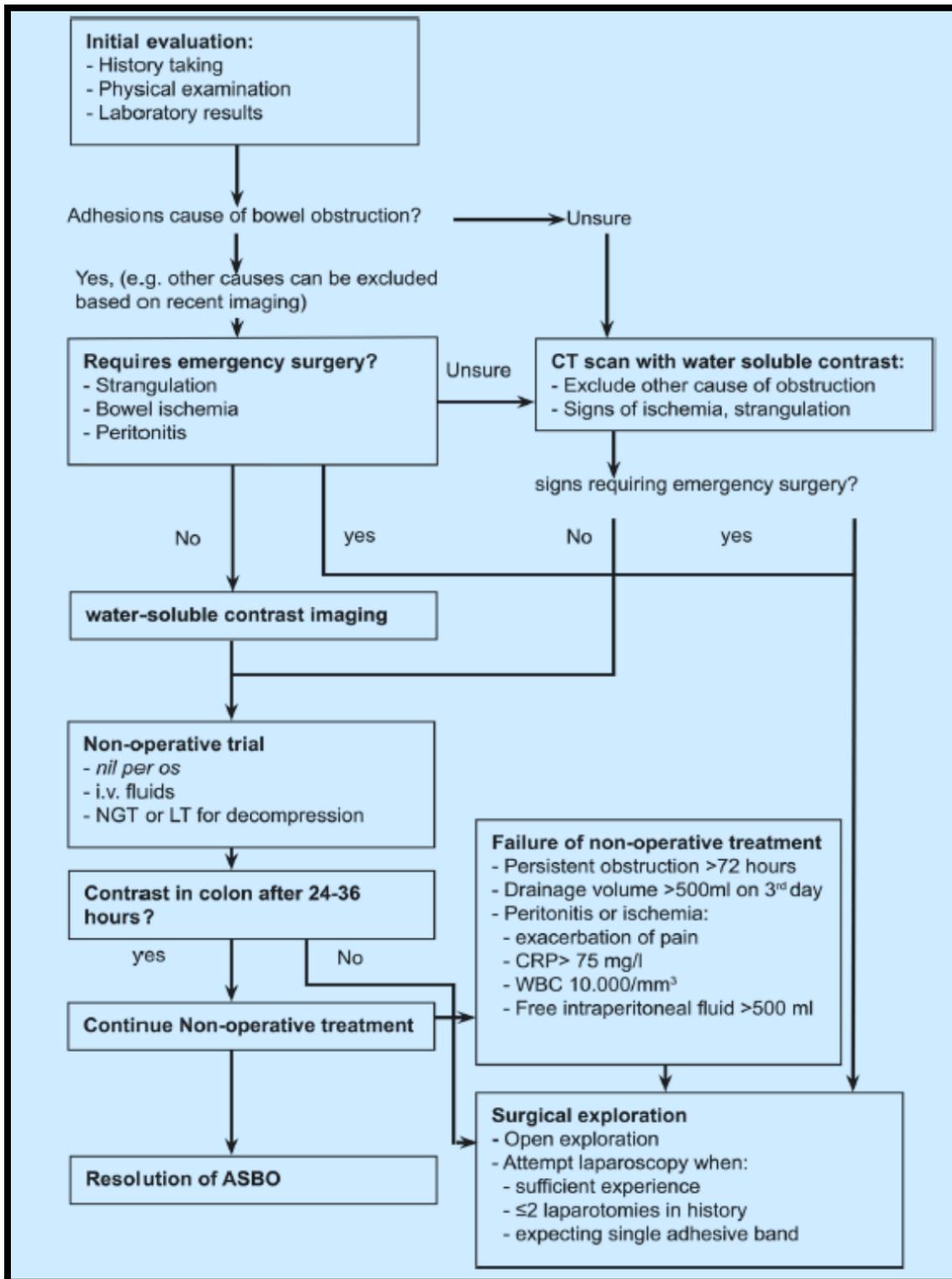
All patients suspected of having complicated bowel obstruction (complete obstruction, closed-loop obstruction, bowel ischemia, gangrene, or perforation) based upon clinical and radiologic examination should be surgically explored.

Management of small bowel obstruction

Algorithm for small bowel obstruction management



Algorithm for adhesive small bowel obstruction



Large bowel obstruction (LBO)

Etiology

1. Colorectal malignancy-90%
2. Colonic volvulus (sigmoid or caecal)- 5%
3. Benign stricture (i.e., diverticular, inflammatory, ischaemic, radiation-induced, or anastomotic)- 3%
4. Rare conditions (e.g., hernia, foreign body, benign neoplasm, gynaecological neoplasm, pelvic abscess, or endometriosis)-2%.

Evaluation

Abdominal radiography, MDCT, and contrast enema are the three commonly used imaging modalities in the evaluation of LBO. Cecum >9 cm and rest of the colon >6 cm in diameter is considered as dilated. The presence of colonic air-fluid levels usually indicates an acute mechanical obstruction.

Xray features of LBO

Diagnosis confirmed by colonic dilation.

Level of obstruction may be determined by a cut-off beyond which the colon or rectum is empty of gas.

Colonic volvulus is identified in 75% of x-rays; characteristic kidney or coffee-bean shape seen with the "apex" locating the origin of volvulus (e.g., sigmoid, caecal).

- Sigmoid volvulus: dilated inverted U-shaped loop of colon projected towards the right side of abdomen; opposing colonic walls produce radio-opaque line; proximal large and small bowel dilation may also be evident.
- Caecal volvulus: dilated right colon rotates to the left side and dilated small bowel may also be present.
- Malignant obstruction: colon typically distended to the point of obstruction with a paucity of distal gas.
- Intramural gas ominously suggests colonic ischaemia.

CT abdomen and pelvis

- Diagnosis of obstruction can be confirmed in >90%.
- May also reveal underlying cause.

- Demonstrates pathology extrinsic to the large bowel and is particularly useful in colorectal carcinoma, where it assesses disease stage.

Flexible/rigid endoscopy

- Main indication is when plain x-ray strongly indicates volvulus. Confirms the diagnosis and is a therapeutic measure.
- Guarded colonoscopy in elderly patients unfit for surgical intervention for planning endoscopic stenting. A biopsy can be taken at the same time to confirm malignancy in the presence of a growth.

Initial management

General principles are the same as that of small bowel obstruction mentioned earlier.

Treatment of common causes of LBO

Colorectal malignancy

Surgical care aims to relieve obstruction and resect the lesion in most cases.

In high-risk surgical patients or those with unresectable disease, a diverting proximal colostomy or ileostomy alone may be performed.

In younger, fitter patients or those with resectable tumours, surgeons may consider primary anastomosis in the right or left colon with or without proximal diverting stoma in the absence of intra-operative hypotension, blood loss, or other complications.

Common scenarios

Right colon growth with obstruction- preferred treatment would be a radical right hemicolectomy with primary anastomosis. In debilitated patients with poor performance score or presence of sepsis or intraoperative hemodynamic instability, simple proximal diversion (ileostomy) would be more appropriate.

Left colon/sigmoid growth-In younger, fitter patients or those with resectable tumours, radical colectomy with primary anastomosis with or without proximal diverting stoma in the absence of intra-operative hypotension, blood loss, or other complications is the procedure of choice.

In unstable patients, Hartmann's procedure is another acceptable option. In unfit and inoperable patients a proximal diversion colostomy or ileostomy should be considered.

Diverticular disease with obstruction

A persistent obstruction merits surgical intervention with either a Hartmann's procedure or a resection and primary anastomosis, with or without a proximal diverting stoma.

Summary of management of intestinal obstruction

Diagnosis

1. Initial evaluation should be complemented with assessment of nutritional status and laboratory tests evaluating at least blood count, lactate, electrolytes, and BUN/Creatinine.
2. Plain X-rays have only limited value in the work-up of patients with small bowel obstruction.
3. CT scan of abdomen and pelvis should be considered in all patients with SBO because it can provide incremental information over plain films in differentiating grade, severity, and etiology of SBOs that may lead to changes in management.
4. Water-soluble contrast study should be considered in patients who fail to improve after 48 hours of nonoperative management because a normal contrast study can rule out operative SBO.

Treatment

1. In the absence of signs that require emergent surgical exploration (i.e., peritonitis, strangulation, or bowel ischemia), non-operative management is the treatment strategy of choice.
2. A trial of non-operative management can be continued safely for 72 h.
3. Laparoscopic adhesiolysis might reduce morbidity in selected cases of adhesive small bowel obstruction that require surgery.
4. Majority of large bowel obstructions require surgical intervention, hence conservative management should be judiciously planned in such cases.

Liver trauma

Introduction

Liver is the most common organ injured in blunt trauma abdomen. Larger size, friable parenchyma, thin capsule and relatively fixed position in peritoneal cavity make the organ more vulnerable to abdominal trauma. Liver trauma management has undergone dramatic change in the last three decades. Improved outcome in patients with liver trauma in was secondary to efficient trauma care system. High resolution imaging and interventional radiology has made non-operative management (NOM) feasible in more than 80% of patients(1).

Epidemiology

Incidence of Road traffic accident (RTA) has increased from 40,000 in 1986 to 85,000 in 2001 (2) and is the most common cause for liver trauma. According to the survey of causes of death (SCD) under the sample registration system (SRS) of India, the rate of injury related deaths increased from 9% to 11% between 1994 and 1998 (3). Tremendous increase in motorization, unsafe environment and less developed trauma care system in India have been the major cause for increase in RTA related deaths. In a retrospective study conducted by Sanjay Kumar et al, liver injury occurred in 7.5% of all trauma admissions and Grade I to III injuries were the most common type of injury(4). Malhotra et al in a study has stated that associated injuries occur in 83% of patients with liver trauma(5). Spleen is the most common associated abdominal organ to get injured. Right lobe is the most commonly injured region in liver because of its large volume. Liver injury following penetrating injury occurs in 16 % to 23% of patients. Despite all available treatment modality, mortality following liver trauma occurs in 3 to 12% of patients mostly secondary to associated injuries(6).

Clinical presentation

Any patient presenting with abdominal trauma along with features of shock, intra-abdominal injury should be suspected. Liver injury should be suspected in a trauma patient sustaining injury to right upper quadrant. Patients may complain of pain in right hypochondrium and/or right chest wall; or right shoulder secondary to irritation of diaphragm. Liver trauma should be suspected when clinical findings of right upper quadrant tenderness, seat belt sign or unstable right lower rib fracture are present. In penetrating injury, major liver trauma can occur without the wound being in close proximity to liver. There may be associated hollow viscus perforation that may present as peritonitis. Hemodynamic stability is the most important to be assessed while receiving liver trauma patients in hospitals as the entire management protocol depends on it.

Investigations

Liver injury should be suspected based on mechanism of injury and physical examination. Imaging like Focussed Abdominal Sonography in Trauma (FAST) for screening and Contrast CT scan (CECT) for definitive management are the mandatory tool in management of liver trauma.

1. FAST:

FAST is the most common initial investigation done especially in unstable patients with abdominal trauma. Liver injury should be suspected in FAST when physician visualise hypoechoic subcapsular rim, intraperitoneal fluid around liver or fluid in Morrison's pouch(7). FAST has replaced diagnostic peritoneal lavage as a mode of screening test in abdominal trauma patients. Hemoperitoneum in presence of liver injury in an unstable patient not responding to initial resuscitation usually ends up in surgical intervention.

2. Contrast enhanced CT scan (CECT):

CECT scan of the abdomen (Triphasic – plain, arterial and portal venous) is the most important investigation in a patient with an abdominal trauma especially with respect to liver trauma in a stable or stabilised patient. CECT scan diagnoses, grades the extent of liver injury as well as diagnoses other organ involvement. The American Association for the Surgery of Trauma (AAST) has graded liver injury based on CT and it has been used universally for stratification of liver injury (Table 1)(8). The predictors of operative intervention in patients with liver trauma in CECT scan include injury extending into major hepatic veins, presence of active contrast extravasation and large hemoperitoneum (9)

Fang et al, in his study of identifying the role of CECT scan in NOM of liver trauma stated that 75% of patients required surgical management when there is arterial blush in imaging. Active contrast extravasation with hemoperitoneum in all six quadrants of the abdomen had a high index for operative intervention (10). Major venous injury should be suspected when the hepatic injury extends into major hepatic veins, IVC or there is non-visualization of major vein in CECT scan. Major hepatic venous injury could also be marker for underlying hepatic arterial injury. In a retrospective study by Poletti et al, requirement for operative management is 6.5 times higher in presence of major venous injury (11).

CECT scan is also useful in assessment of delayed complications in blunt liver trauma, including delayed haemorrhage, hepatic or perihepatic abscess and biliary complications. World Society of Emergency Surgery (WSES) also recommends CECT in the follow up in case of complications although routine CECT in follow up is not recommended (12) .

CECT abdomen though has become an integral part in management of liver trauma it's not without limitations. Contrast induced nephropathy, contrast allergy and less sensitive for diagnosing hollow viscus or diaphragmatic injury are the major limitation. Justin et al in his

review article has stated that the false negative rate in hollow viscus injury was 44.7 to 54.5% and the sensitivity to in diagnosing diaphragmatic injury was as low as 57%.(13)

Table 1. :- **AAST Grading of liver injury (1994 revision)**

Grade		Description
I	Hematoma	subcapsular, 10% surface area
	Laceration	capsular tear < 1 cm parenchymal depth
II	Hematoma	subcapsular 10%–50% surface area; intraparenchymal <10 cm in diameter
	Laceration	Laceration: 1–3 cm in parenchymal depth, <10 cm in length
III	Hematoma	Subcapsular, > 50% surface area; ruptured subcapsular or parenchymal hematoma; Intraparenchymal hematoma > 10 cm or expanding.
	Laceration	Laceration > 3 cm in parenchymal depth
IV	Laceration	Parenchymal disruption involving 25%–75% of hepatic lobe or one to three Couinaud segments in a single lobe
V	Laceration	Parenchymal disruption involving > 75% of a hepatic lobe or more than three Couinaud segments within a single lobe
	Vascular	Juxtahepatic venous injuries (ie, retrohepatic vena cava or central major hepatic veins)
VI	Vascular	Hepatic avulsion

3. MRI Abdomen

MRI has limited value in the evaluation and management of liver trauma and it is advised only in a subset of patients who have contrast allergy or in the presence of compromised renal function

Management:

Isolated liver trauma management has undergone major change with paradigm shift towards non-operative management (NOM). Clinical examination and investigations (FAST and CECT) are the keys to decide on management. However, the primary assessment should prioritize resuscitation and detect injuries that may require immediate intervention to sustain life.

Initial Resuscitation:

Most important parameter in assessing the need for operative management is hemodynamic instability. According to ATLS, a patient with systolic BP < 90 mm of mercury, heart rate > 120

with evidence of skin vasoconstriction, altered consciousness and shortness of breath is defined as unstable patient(14). The fundamental principle in the management of liver trauma patients is to ensure stoppage of ongoing bleeding and to replace the lost volume. The ideal fluid of resuscitation is blood or crystalloids. The overzealous administration of fluids can be detrimental by increasing the blood pressure resulting in dislodging of clots. Schreiber et al in his randomized control trial has recommended that controlled resuscitation is a safe strategy in the managements of polytrauma patients(15).

The treatment strategy depends on the hemodynamics, grade of injury and presence of other abdominal injuries. Continued instability with positive FAST is an indication for operative management. Patients who are stable or stabilized or those who do not have other indication for operative intervention should be offered non-operative management. Operative management may also be required in patients who develop persistent systemic response in the form of fever, ileus, tachycardia etc. This particularly is seen in patients with bile leaking in the peritoneal cavity.

1. Non-operative management

The current shift to nonoperative management in patients with liver trauma was based on the success of similar management in children(16). Improved diagnostics like CECT and better intensive care monitoring have led to a shift to non-operative management of patients with isolated liver trauma. The advantage of NOM in patients with liver trauma include low hospital cost, early discharge, avoiding non-therapeutic laparotomies, reduced abdominal complications and transfusions(1)(17). The practice of NOM has brought down morbidity and mortality in patients with liver trauma(18).

Patient selection, availability of resources like intensive care, blood ,blood products, FAST,CECT scan, interventional radiology facility and good operative back-up decide the success of NOM. World Society of Emergency Surgery(WSES) classifies liver injury into three grades incorporating AAST liver injury grade for the purpose of management(12) (Table 2). Accordingly, all blunt liver trauma patients with hemodynamic stability and absence of other intraabdominal injuries requiring surgery, should undergo an initial attempt of NOM irrespective of the grade of injury. NOM is not the recommended in presence of hemodynamic instability or peritonitis. Gunshot injuries are relative contraindication.

NOM has a success rate of 82 to 100% as per Eastern Association for the Surgery of Trauma (1) (EAST). Non-operative management is not without complications. The most important reason for failure of NOM is hemodynamic instability which occurs of 75% of patients. Various factors have been identified as predictors of failure of non-operative management. These include ongoing fluid resuscitation required to maintain hemodynamic instability, multiorgan injury,

high injury severity score, large hemoperitoneum and contrast extravasation on CECT (19)(17). Complications following NOM are reported to occur in 14-20% of patients(1)(17). These include delayed haemorrhage, bile leak, haemobilia, biliary peritonitis, abdominal compartment syndrome, missed injuries, hepatic abscess and liver necrosis. Higher the grade of injury, more is the chance of complication.

Table 2:- WSES grading of liver injury and management

WSES grade	Blunt/Penetrating / SW/GSW	AAST	Hemodynamics	CECT Scan	First line treatment
Grade 1 (Minor injury)	B/P/SW/GSW	I-II	Stable	No	NOM
Grade 2 (Mod. injury)	B/P/SW/GSW	III	Stable	Yes+Local exploration in SW	NOM(+)-serial clinical / Lab/Radiology
Grade 3 (Severe injury)	B/P/SW/GSW	IV - VI	Stable	Yes	NOM
Grade 4 (Severe injury)	B/P/SW/GSW	I-VI	Unstable	No	OM

B-Blunt, P-Penetrating, SW-Stub wound, GSW-Gun Shot wound, OM - Operative management, NOM-Non-operative management

2. Angioembolization

Arterial angioembolization should be considered as a part of resuscitative and final management protocols. Embolization should be considered as first line intervention in a hemodynamically unstable patient when CT scan shows active arterial bleed (12). The rate of requirement of blood transfusion and infectious complication has been found to be less when embolization was executed as a first line treatment. Angioembolization following initial damage control surgery or definitive surgery is an integral part of “sandwich approach” in patients with liver trauma.

3. Operative management:

Operative management for liver trauma is required in patients having hemodynamic instability. Resuscitation should go hand in hand for successful outcome. The following principles should be kept in mind while operating an unstable liver trauma patient

- a. Primary intervention should be aimed to control bleeding.

- b. Damage control surgery (DCS) in unstable patient should be in the form of perihepatic packing, removal of dead tissue, control bile leak and avoid major hepatic resection
- c. Artery repair should be done if easily possible, otherwise can be managed by embolization/stenting later.

I. Perihepatic packing

Perihepatic packing is the preferred form of DCS in unstable patient. Systematic perihepatic packing followed by sequential angiography called the “sandwich approach” has been recommend for selected patients with liver trauma (20).The most common indications for perihepatic packing includes intraoperative coagulopathy, large subcapsular hematoma, hepatic laceration in the presence of multiple intraabdominal injuries, major hepatic grade injury operated by inexperienced surgical team and or a hospital with limited resources(21).

The following guidelines should be kept in mind during perihepatic packing (21)(22)(23) (Table 4). After stabilizing the patients in intensive care unit, re-exploration should be done around 24 to 48 hours. Early removal may result in bleeding and delaying the removal increases the chance of sepsis. Consideration for early removal of packing is warranted in presence of gross gastrointestinal contamination during initial laparotomy. Standardized techniques of packing have been associated with reduced chance of rebleeding.

Table 4:- Principle to be followed during liver packing

II. Intrahepatic tamponade

1.	Mobilization of liver should be done to estimate the severity of injury and when venocaval injury is suspected (except if retrohepatic venous injury suspected)
2.	Packs should be placed sequentially from 6/7 'O clock to 5'0 clock position
3.	Avoid intrahepatic packing as it may extend injury
4.	In superficial injury if bleeding stopes after 5 to 10 mins of packing, packs can be removed and no further intervention is required
5.	Avoid compression in anteroposterior axis as it may accentuate injury and worsen bleeding
6.	Packing should take the shape of liver. Excessive packing can compress IVC and compartment pressure and may aggravate multiorgan failure
7.	Diaphragmatic side should be free to avoid respiratory compromise

Mesh wrapping has also been advised for perihepatic packing because it doesn't require reintervention and there is no increase in intrabdominal pressure. Two technical points have to be remembered while using mesh as tamponade for liver injury. First, the injured liver has to be covered with enough pressure for a successful tamponade effect. In addition, the mesh should be stabilized after anchoring it to the diaphragmatic crus and the falciform ligament. In addition, the hydrolysed product of the mesh has a bacteriostatic effect, minimizing the risk of infection(24). In spite of the advantages the evidence and recommendation for this method are limited in literature.

Intrahepatic tamponade is usually recommended especially for perforated wound. Best forms of tamponade include balloon tamponade using Senkstacken Blackmore tube, penrose drains or foleys' catheter. Radiopaque material can also be used to confirm device position. Once patient is stabilized devices can be withdrawn.

III. Hepatorrhaphy

Hepatorrhaphy is a technique in which deep parenchymal sutures are placed to bring the injured tissue together to control bleeding vessels and decreasing the dead space. This age-old technique is no more recommended as a form of intervention due to increased risk of ischemia and infection (21).

IV. Hepatotomy and selective parenchymal vascular ligation

This technique is usually recommended in presence of major vascular injuries. Rapid finger fracture technique in addition to intermittent pringle maneuver can help to reach and identify the bleeding vessel. The major drawback of this technique is increased risk of bleeding while trying to reach the target vessel.

V. Liver resection

Non-anatomical liver resection in the form of removal of devitalized tissue is usually done in re-exploration to reduce sepsis and secondary haemorrhage. The line of injury is used as a boundary and standard resection planes are not used in order to avoid removal of excessive tissue. This technique is usually combined with other techniques like hepatotomy and pringle maneuver. In addition to removal of only devitalized tissue the operative time should be kept minimal. Excessive removal of parenchyma is warranted only in selected condition like injury along the right posterior segment where right hepatic vein is commonly injured. In such scenario the injured tract is extended and resected which results in securing the hepatic vein.

Anatomical liver resection in trauma patient can be associated with a mortality >50%(25). Hence such resection should be done only by expert surgeons in advanced hepatobiliary centre. Strong et al in study stated that the overall mortality in liver trauma patients undergoing major resection procedure in specialised centre was 8.1% (26) .

VI. Retrohepatic venous injury

Juxta-hepatic venous injury is potentially lethal. Two types of such injuries have been recognised. Type A is intraparenchymal injuries which bleed through injured liver tissue and Type B injuries in which veins have completely avulsed from IVC. Surgical procedures like total vascular isolation, veno-venous bypass, atriocaval shunting etc are associated with a mortality of 50- 90% (27). Usually such injuries are initially managed with perihepatic packing followed by definitive repair later once patient stabilizes(22).

VII. Liver transplantation

Liver transplant is the last option in the management of liver trauma patients and always done only in specialised centres. The usual indication for liver transplant in the setting of trauma are uncontrollable continuous bleeding after damage control operation, complex liver lacerations not amenable to surgical correction, progressive liver failure following trauma and hepatic necrosis. Two different techniques are being described in literature either one stage or two stage procedure. In one-stage procedure the injured liver is removed with simultaneous transplant which is used in majority of cases. In two-stage approach temporary end to side portacaval shunt is provided, until new organ becomes available. In present era due to adequate experience in liver transplant the mortality following transplant in liver trauma patients has been reduced to 48% from 76% (28).

Multidisciplinary approach

Appropriate care of the trauma patient entails a multidisciplinary effort that requires speed and efficiency. Hepatopancreaticobiliary injuries are often deadly and challenging. Management of patients with liver trauma requires a lot of team work and coordination of trauma surgeons, liver surgeons, intensivists, interventional radiologists, anaesthetists and blood bank staffs. Availability of a multidisciplinary team and specialized facilities under one roof may reduce the morbidity and mortality in patients with liver trauma (29).

Referral guidelines for Liver trauma

Advantages of NOM in patients with liver trauma have been clearly documented in literature. For NOM to be successful, the treating centre should have the feasibility for both multidisciplinary and multimodal approach. A study done by Barrie et al depicted that patients with complex liver trauma managed in advanced hepatopancreatobiliary (HPB) units have reduced risk adjusted mortality rate (30). HPB surgeons having experience in complex haemostatic techniques, training in vascular surgery, in-depth knowledge of the variations of liver anatomy, familiarity with managing post-operative complications were the possible reasons for improved outcome. Availability of interventional radiology, equipped operating room, experienced operative room nursing staffs etc also contributed for reduction in the mortality. Hence patients with moderate to severe injury should be referred to centres of excellence in managing such trauma after initial resuscitation.

Medicolegal aspects in patients with liver trauma

Many surgeons have hesitation of attending trauma patient due to fear of litigation. Though a doctor can choose his patient on his own will, a verdict by the Hon. Supreme court in 2016 has stated that no doctor should refuse treatment in emergency and if refused can be sued for medical negligence (31). The extent of primary care should depend on the qualification of the treating surgeon. A general surgeon with limited available resources should be giving the initial primary resuscitative care to the patient and then refer to advance centre for further care.

Conclusion

Management of liver trauma has undergone rapid changes in the past three decades. The paradigm shift toward non-operative management (NOM) from operative management has resulted in excellent positive outcomes. Operative management if undertaken for complex liver injuries should initially be restricted towards damage control. Close relationship should exist between various teams for better outcome in liver trauma patients. An algorithmic approach for management of liver injury patients as recommended by WSES is shown below (Figure 1)

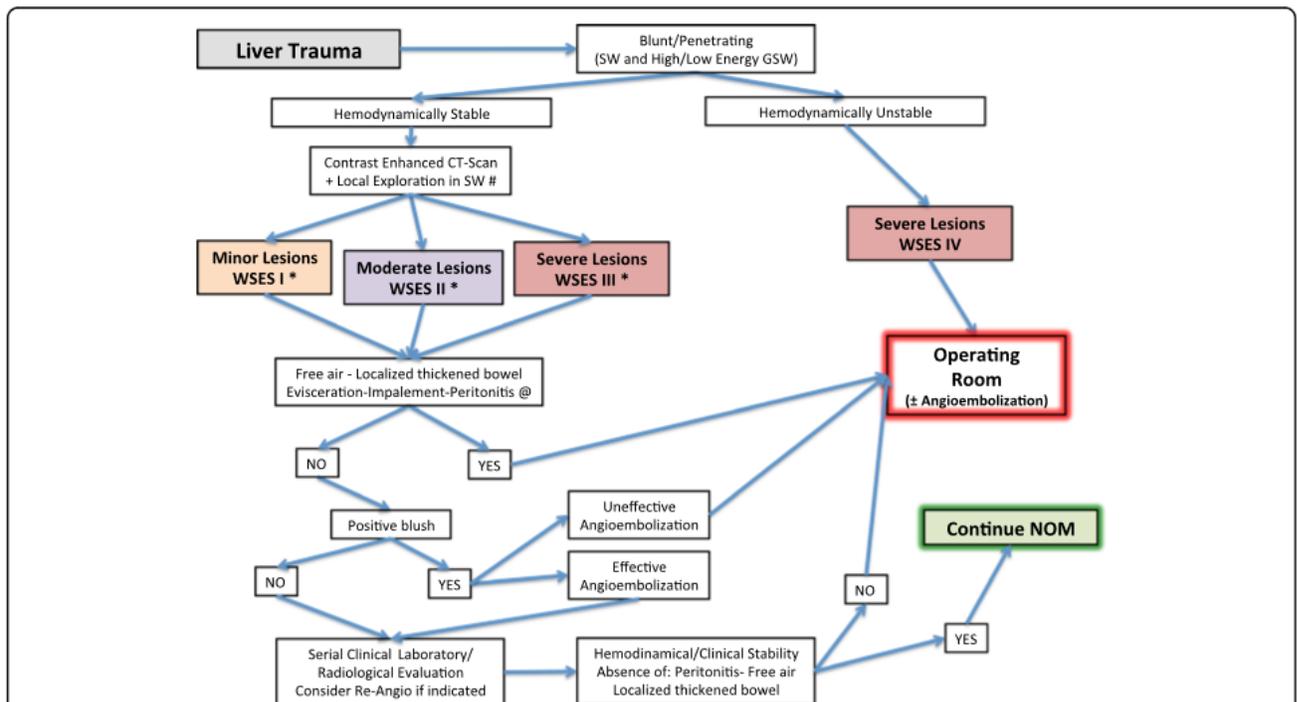


Fig. 1 Liver Trauma Management Algorithm. (SW Stab Wound, GSW Gun Shot Wound; *NOM should only be attempted in centers capable of a precise diagnosis of the severity of liver injuries and capable of intensive management (close clinical observation and haemodynamic monitoring in a high dependency/intensive care environment, including serial clinical examination and laboratory assay, with immediate access to diagnostics, interventional radiology and surgery and immediately available access to blood and blood products; # wound exploration near the inferior costal margin should be avoided if not strictly necessary because of the high risk to damage the intercostal vessels; @ extremely selected patients hemodynamically stable with evisceration and/or impalement and/or diffuse peritonitis with the certainty of an exclusive and isolated abdominal lesion could be considered as candidate to be directly taken to the operating room without contrast enhanced CT-scan)

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Painful Hernia- Irreducibility / Obstruction/ Strangulation

Contents

1. Introduction
2. Inguinal hernia
3. Femoral hernia
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5. Umbilical hernia
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7. Role of mesh in emergency hernia surgery

Introduction

Hernia surgery remains one of the most commonly performed surgical procedures worldwide. Although most repairs are done in an elective setting, a small proportion of hernias can present in emergency with pain, irreducibility, obstruction and strangulation. While a significant number of patients with hernia complain of dull aching or dragging pain, a severe pain is usually due to irreducibility, obstruction, strangulation or ischemia.

Some patients with irreducibility without any obstructive/strangulation may be managed with conservative management and an early elective repair. However, a significant number of these patients will require an emergency surgery to prevent sequelae of obstruction and ischemia.

In the following discussion, we will focus on emergency presentation of hernia due to obstruction/strangulation/ischemia with mention of other causes of painful hernia whenever appropriate.

Emergency hernia surgery is associated with a higher rate of morbidity in the form of recurrence, surgical site occurrence (SSO), surgical site infections (SSI), and mortality than the elective patients (1). This is probably due to poor underlying physiology and adverse hernia factors (2).

Urgent surgery to prevent strangulation vs optimization followed by an early elective surgery remains one of the important considerations in the decision making process in emergency hernia presentations. Timing as well as technique seems to influence outcome.

Literature focusing on emergency hernia surgery is scarce leading to lack of clarity in the management of these patients. The following recommendations are based on evidence available on the subject and may be used for guiding treatment.

Inguinal hernias

It is not uncommon to see groin hernias presenting in an emergency and may require urgent surgery. However, emergency hernia repair is associated with increased morbidity and mortality mostly due to the presence of strangulated or ischemic bowel that may require resection(3).

Clinical and lab evaluation is crucial in irreducible hernias to know if there is any ongoing or impending ischemia and plan further course of action. The following features may suggest ischemia-

1. Bluish/ reddish discoloration of overlying skin
2. Tenderness
3. Tachycardia, tachypnea, hypotension indicating sepsis
4. Lactic acidosis

In the absence of these features, manual reduction of hernia contents may be tried under sedation. If this is successful, an early elective surgery may be planned under controlled conditions. However, most of these hernias will require an emergency surgery.

Open approach remains the commonest strategy in the irreducible groin hernias. The surgery is usually through an inguinal incision. However, a laparotomy may be needed in the following circumstances-

1. Bowel cannot be reduced safely
2. Resection and anastomosis is technically challenging
3. Contents reduce into the abdomen without giving a chance for inspection for viability

Laparoscopic approach can be used in some patients where the abdomen is conducive to a safe pneumoperitoneum and bowel handling(4).

The choice of repairs depends on the degree of contamination, volume of hernia contents and the surgeon's discretion.

In contaminated cases, tissue repair, absorbable/ biological mesh may be used(4)(Grade C recommendation). In large or loss of domain hernias, where postoperative ACS is a possibility, defect may be left open for an elective repair at a later date.

It has been found that there may be no increased risk of mesh related morbidity in emergency surgeries not requiring gut resection(5,6)(Grade C recommendation).

Recently CT and other imaging modalities are increasingly being used in selective cases to plan hernia procedures in emergency settings. Imaging can identify associated hydrocele, obstruction, strangulation and guide the clinician regarding the urgency of intervention. In cases of loss of domain, it may help in predicting abdominal compartment syndrome and plan preemptive action.

Optimization

Patients who present in the emergency frequently may have medical comorbidities as well as metabolic disturbances related to obstruction/strangulation/ischemia that need to be addressed before surgery.

It is important to seek consultation from physicians and specialists for optimization if time permits since this allows the surgery to be done under controlled conditions. For example, some patients may benefit from conservative measures such as nasogastric decompression, fluid and electrolyte correction, chest physiotherapy, DVT prophylaxis and this may buy time for an early elective repair. Patients presenting with metabolic disturbances like acidosis and organ dysfunction secondary to ischemia need consultation and optimization by a critical care team.

Femoral hernias

These hernias frequently present with pain and have a high risk of strangulation. Therefore, an early intervention is advocated(7).

Clinical examination and imaging may be used to confirm the diagnosis. Whenever indicated and available, CT remains the imaging investigation of choice.

Surgical approaches are

1. Open approach- supra inguinal is the commonest route
2. Laparoscopic approach - has only a minimal role

Preoperative optimization stays the same as that for inguinal hernias.

The repair may be with a mesh or anatomical, the latter reserved for contaminated cases.

Ventral Hernias

Emergency management of ventral hernias is challenging for most surgeons. The surgery may be complicated due to hernia factors (large sac, loss of domain, swiss-cheese defect), patient factors (obesity, diabetes, smoking, ischemic heart disease) and wound factors (previous surgery, mesh, sinus, fistulas).

Clinical and radiological (usually CT) evaluation should focus on factors mentioned above and is crucial to the management.

Usually, these hernias are not amenable to manual reduction and frequently require optimization with fluid and electrolyte correction before surgery.

Loss of domain and development of ACS is a major concern in large ventral hernia. These type of patients should be managed in well equipped centres with multidisciplinary approach. The size and position of mesh placement is also varies with the practising Surgeon. Retro rectus placement of mesh with component separation and TARR has shown the best results.

Operative procedures may be open or laparoscopic.

Usually, open surgery is done for majority of these hernias. The procedure typically involves an incision over the hernia sac and may need extensive adhesiolysis, dissecting all the swiss-cheese defects, excision of the previous mesh/ sinus and resection of ischemic gut.

Smaller defects without significant abdominal distension may be managed laparoscopically if facility and expertise is available. The bowels should be reduced carefully, and the defect may be repaired with an underlay mesh. If resection is required, a biological mesh may be preferable(4).(Grade C recommendation) Alternatively, a synthetic or biological mesh can be placed in an extra-peritoneal position such as the on-lay or sub-lay position depending on the grade of contamination and surgeon's discretion.

The strategy to deal with the defect depends on the width, comorbidities, the volume of contents being reduced back in the abdomen and gut resection.

The options are

1. Primary closure without mesh
2. Primary closure with Mesh (onlay/sublay/underlay; synthetic/ biological/ biosynthetic). Macroporous lightweight polypropylene mesh has also shown good results in the presence of infection in some of the published literature(8)(Grade C recommendation).
3. Component separation technique (Anterior/ Posterior) with the intent of defect closure.
4. Bridging repair with mesh (onlay/ sublay/ underlay). If synthetic mesh is used, an omental interposition or an absorbable mesh is preferable to prevent direct contact of bowel with mesh.
5. Flap repairs. It may be prudent to involve surgeons familiar with free and pedicled flap techniques when the requirement for the same is anticipated.

6. The abdomen can be left open with a negative pressure wound therapy and intent for delayed closure. Alternatively, only skin can be approximated leaving the sheath open if there is a possibility of postoperative abdominal compartment syndrome. In instances where the wound/skin can not be closed even after NPWT, and there is doubt regarding patient's fitness for a long flap procedure, skin grafting over a well granulated wound remains an acceptable option with an intent for a delayed definitive repair.

Recent guidelines have acknowledged the limited literature in the emergency management of complicated abdominal wall hernias and therefore the above mentioned principles can only be taken as recommendations(9).

Umbilical hernias

While many of these hernias are relatively straightforward to manage, some may be problematic especially if associated with obesity and cirrhosis.

The latter may benefit from perioperative optimization preferably in a liver unit along with hernia repair(10,11).

Timing of Surgery

Clinicians are faced with the daunting task of deciding between early intervention in strangulated hernias vs delaying surgery for optimization for improved results. It is recommended that patients should undergo emergency hernia repair immediately when intestinal strangulation is suspected (Grade 1C recommendation) (4) in the absence of any prohibitive metabolic disturbances and organ dysfunction/failure. SIRS, CECT, serum lactate, CPK and D-dimer levels are predictive of bowel strangulation. (Grade 1C recommendation)(4).

However, in the presence of only irreducibility and obstruction in the absence of any impending or ongoing ischemia, a preoperative optimization or trial of conservative management may be tried. It may allow for an elective surgery under controlled circumstances with a favorable morbidity and mortality profile.

Mesh in the presence of contamination

There is no controversy in the usage of mesh in clean and clean contaminated cases (CDC wound class 1 and 2).

There exists grade 2C recommendation for contaminated and dirty cases (CDC wound class 3 and 4). WSES guidelines suggest that in stable patients with strangulated hernia with bowel necrosis and/or gross enteric spillage during intestinal resection (contaminated, CDC wound class 3) or peritonitis from bowel perforation (dirty surgical field, CDC wound class 4), a primary

repair is recommended when the size of the defect is small (< 3 cm). When direct suture is not feasible, a biological mesh may be used for repair(4).

A polyglactin mesh can be used as an alternative if biological mesh is not available.

Referral Criteria

- ICU care may be needed in patients who present late with severe electrolyte imbalance, aspiration, sepsis and have other systemic illnesses.

Medicolegal Issues

- Failure to detect / investigate or refer a patient of suspected obstructed/strangulated hernia.
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action.

Who does what?

Surgeon:-	Diagnosis & Work up Pre operative planning Operative procedure Post operative care in conjunction with Anaesthetist/Intensivist Post operative follow up
Anesthetist:-	Pre Anaesthesia Check up Part of resuscitation Performing anesthesia Post op ICU management in conjunction with Surgeon
Nurse:-	Pre/Intra/Postop comprehensive care Dressing of the wound
Technician:-	Pre op equipment and drugs to be checked and kept ready Assist anesthetist in the OT Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (patient weight 60 kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1

6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram
2. Blood Sugar
3. Renal Function Test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG
8. X- Ray – Chest/Abdomen
9. Histopathology if resection of intestine is required.

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopy, the procedure can be done laparoscopically as decided by the Surgeon ensuring that the accepted standard of Care is met.

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Peptic Ulcer Perforation

Introduction:

Lau and Leow have indicated that perforated peptic ulcer was clinically recognized by 1799, but the first successful surgical management of gastric ulcer was by Ludwig Heusner in Germany in 1892. In 1894, Henry Percy Dean from London was the first surgeon to report successful repair of a perforated duodenal ulcer.

Indian Incidence

There are no statistics available on this topic.

Symptoms

1. Sudden, sharp and severe pain in upper abdomen
2. Spreading of pain to rest of abdomen
3. Pain gets worse after oral ingestion or movements
4. Feeling of giddiness and fainting
5. Fever
6. Weakness

Signs

1. Tachycardia
2. Elevated Temp
3. Pallor
4. Reduced abdominal wall movements

Investigations

- a. Haemogram
- b. Liver Function Tests if there is hypovolaemia
- c. Blood sugar
- d. Blood Urea, Serum creatinine
- e. Bleeding time, clotting time and prothrombin time
- f. Xray chest
- g. ECG
- h. USG abdomen If clinical/biochemical/Xray findings are non Diagnostic

Management

Resuscitation

Fluid resuscitation should be initiated as soon as the diagnosis of peptic ulcer perforation is made. Intravenous infusion of fluids is begun and broad-spectrum antibiotics are administered. In select cases, insertion of a central venous line may be necessary for accurate fluid resuscitation and monitoring.

Essential steps include insertion of a nasogastric tube to decompress the stomach and a Foley catheter to monitor urine output.

As soon as the patient has been adequately resuscitated, emergent exploratory laparotomy should be performed.

Conservative Treatment

Wangensteen et al reported that in a patient with perforation but without evidence of pneumoperitoneum, one can safely assume that perforation has sealed off on its own. They advocated a non-operative approach for such patients. However, they too supported operative treatment in patients with perforated ulcer and evidence of pneumoperitoneum.

Berne and Donovan emphasized the use of a water-soluble upper GI study to demonstrate spontaneous sealing of the perforation. They demonstrated that as many as 40% of perforated peptic ulcers had no evidence of leak on upper GI contrast studies. Berne and Donovan concluded that these patients can be observed safely as long as peritonitis does not develop. Mortality rates were 6% and 3% in the operative and nonoperative groups, respectively.

Donovan et al proposed dividing patients based on their *Helicobacter pylori* infection status and recommended nonoperative treatment in all patients except those without *H pylori* infection and those in whom prior treatment of *H pylori* infection had failed.

Despite strong arguments favoring nonoperative treatment of patients with perforated PUD, delaying the initiation of surgery more than 12 hours after presentation was demonstrated to worsen the outcome. Therefore, when definitely indicated, a laparotomy should be performed as soon as possible.

It is also possible to initiate nonoperative management when the patients general condition is too poor to withstand anaesthesia and Surgery. There are many clinical and biochemical criteria which gives predictive outcome of surgery. eg Boey's Criteria

Surgical Treatment

The appropriate surgical procedure depends on the location and nature of the ulcer. Many authorities recommend simple oversewing of the ulcer, with treatment of the underlying H pylori infection or cessation of nonsteroidal anti-inflammatory drugs (NSAIDs) for bleeding PUD.

Additional surgical options for refractory or complicated PUD include vagotomy and pyloroplasty, vagotomy and antrectomy with gastroduodenal reconstruction (Billroth I) or gastrojejunal reconstruction (Billroth II), or a highly selective vagotomy.

The patient is placed in the supine position. A midline incision provides the most expeditious entry into the abdominal cavity. The incision can be extended to the symphysis pubis if necessary.

Once the abdomen is entered, the stomach and duodenum are carefully examined to determine the site of perforation. If the anterior surfaces of the stomach and duodenum show no abnormalities, the gastrocolic ligament is serially divided between clamps to allow entrance into the lesser sac and inspection of the posterior surface of the stomach.

For a perforated duodenal ulcer, the choice of operative procedure depends on variables such as the presence of shock, the presence of life-threatening comorbid conditions, the degree of contamination of the upper abdomen, the amount and duration of perforation, and whether the patient has a history of or currently has intraoperative evidence of chronic peptic ulceration, expertise available at surgery and the facilities for postoperative care.

In the presence of life-threatening comorbid conditions and severe intra-abdominal contamination, the safest technique for an acute anterior duodenal perforation is a simple closure. Reinforcement with a Graham patch, using omentum may be added. Several full-thickness simple sutures are placed across the perforation, using 2-0 or 3-0 absorbable sutures. A segment of omentum is placed over the perforation and tied over.

If contamination of the upper abdomen is minimal and the patient is stable, a definitive ulcer procedure can be performed if there is evidence of a Chronic ulcer perforation and if expertise and facility are available. For a perforated duodenal ulcer, this may include a highly selective vagotomy, a truncal vagotomy and pyloroplasty, or vagotomy and antrectomy.

For a perforated gastric ulcer, the procedure performed depends on the patient's condition. If the patient is moribund, the ulcer is best excised by grasping it with multiple Allis clamps and closing it in two layers or by using a linear stapler. Alternatively, the ulcer can be excised with electrocautery; the defect is approximated with a 2-layer closure with inner continuous 3-0

	Pre operative planning
	Operative procedure
	Post operative care in conjunction with Anaesthetist/Intensivist
	Post operative follow up
Anesthetist:-	Pre Anaesthesia Check up
	Part of resuscitation
	Performing anesthesia
	Post op ICU management in conjunction with Surgeon
Nurse:-	Pre/Intra/Postop comprehensive care
	Dressing of the wound
Technician:-	Pre op equipment and drugs to be checked and kept ready
	Assist anesthetist in the OT
	Assist the surgeon, positioning of the patient

Resources required for one patient / Procedure (Patient weight 60 Kgs)

Human Resources Drugs / Consumables Equipment

- a. Surgeon – 1
- b. Medical Officer /Assistant Surgeon – 1
- c. Anesthetist – 1
- d. Pathologist – 1---- Services from outside can be availed
- e. Staff Nurse – 1
- f. Technician – 1
- g. Nursing Orderly – 1
- h. Cleaning staff-1

Investigations

- i. Haemogram
- ii. Blood Sugar
- iii. Renal Function Test in selected cases
- iv. LFT in selected cases
- v. S. Electrolytes in selected cases
- vi. USG in selected cases
- vii. ECG
- viii. X-Ray – Chest
- ix. Histopathology

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as decided by the Surgeon.

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Perianal Pain

Introduction

Peri-anal pain is the pain in perianal area. These patients report in outpatient department as well as emergency. Pain results from peri anal cellulitis leading on to perianal abscess, ischiorectal abscess, fissure in ano (after defecation), prolapsed thrombosed piles, infected fistula in ano and acute prostatitis. Other rare causes may include faecal impaction, carcinoma anal canal particularly distal to dentate line, carcinoma vulva, proctalgia fugax, carcinoma rectum, solitary rectal ulcer syndrome, chronic proctalgia, Crohn's disease of anorectum, levatorani syndrome, sexually transmitted disease and hidradenitis suppurativa of anal region.

Incidence

There are no statistics available on this topic.

Symptoms

Although there are many causes of peri anal pain but the common symptom is pain in perianal area which may vary from moderate to severe in intensity. Pain of anal fissure is sharp and agonising, starts during defecation and lasts for hours. If pus is present in perianal area, a throbbing sensation is reported. Depending on etiology, patient may have high grade fever with chills. History of bleeding per rectum may be present. In acute prostatitis patient complains of perineal heaviness, retention of urine and pain during micturition and defecation.

Signs

Examination of peri anal and perineal areas may obviously reveal prolapsed thrombosed pile (tender, solid, black or dark purple mass), acute fissure-in-ano, peri anal abscess and infected fistula-in-ano. Typical history, tightly closed puckered anus and inspection of the lower end of fissure is sufficient to diagnose an acute anal fissure. In infected fistula-in-ano purulent discharge will be seen around the anus or from within anal canal. Perianal abscess will present as tender smooth soft swelling in perianal region. A digital rectal examination will reveal tender prostate in case of acute prostatitis, carcinoma anorectum and faecal impaction. Proctoscopy should be done for diagnosing the solitary rectal ulcer.

Investigation

Condition like prolapsed thrombosed piles, perianal abscess and fissure-in-ano do not require any diagnostic investigations. However colonoscopy and biopsy is required for carcinoma anorectum, Crohn's diseases and solitary rectal ulcer syndrome. In modern surgical

practice, MRI is not mandatory in emergency. It may be required later when the drained abscess is not progressing as per expectation and in situations where we realise that drainage was done in a case of high fistula. Sometimes one of the track or part of the track acquires infection and presents as abscess in emergency. CT scan of pelvis would be required in case of carcinoma anorectum and Crohn's disease during complete work up. For acute prostatitis culture and sensitivity of urine and prostatic fluid will be required.

Management

Conservative Treatment

Conservative treatment is required for acute fissure-in-ano, in form of diltiazem 2% cream locally, sitz bath, bran or bulking agents, pain killer and laxative. For thrombosed piles, sitz bath, antibiotic, laxative and anti-inflammatory drugs should be given. For patients of peri anal inflammation, in stage of cellulitis, the problem can be aborted by a combination of pain killer and antibiotics, but once abscess develops, drainage will be required. For fistula-in-ano treatment depends on whether the fistula is high or low but acuteness due to infection is tackled by a combination of antibiotics, anti-inflammatory drugs and sitz bath. Ideal treatment for all types of fistula is the drainage of primary inter-sphincteric infection and primary tracks. For acute prostatitis a suitable course of antibiotics as per results of culture sensitivity will be required.

Surgical Treatment

For perianal abscess, do not wait for fluctuation, do not depend on conservative treatment because the delay in drainage results in chronic infection and destruction of tissue, a cruciate incision is given. Septa are broken and thorough evacuation of abscess cavity is done. Gentle probing is done to find the extent of abscess cavity but probe is not thrust to avoid any false tract formation. Edges of abscess cavity are trimmed so that the abscess cavity is left free for drainage. Severe perianal sepsis with spreading cellulitis, encountered in immunocompromised and diabetic patients is treated by drainage of pus, excision of all dead tissues and aggressive antibiotic therapy. Acute fissure not responding to conservative treatment is subjected to partial subcutaneous lateral internal sphincterotomy. Fistula-in-ano is dealt as per findings of MRI. For conditions like malignancy and Crohn's diseases after ameliorating the acuteness, cases are investigated properly and definitive treatment is given.

Post-operative care and complications

Post-operative care involves sitz bath, regular dressings, antibiotics and pain killers. Retention of urine is a common problem after drainage of perianal abscess. But this usually requires single urinary catheterization.

Complications: there may be recurrence of perianal abscess and fistula-in-ano.

Referral Criteria

Most of the conditions are within the domain of average general surgeon, so do not require referral. But for prostatitis urologist's opinion may be sought. For carcinoma anorectum and Crohn's disease patients may be referred to oncosurgeon or gastro surgeon. For nagging problems like solitary rectal ulcer syndrome, proctalgiafugax and high fistula-in-ano, patients may be referred to a proper qualified colorectal surgeon. Referrals are needed only if the expertise and equipment back up is lacking.

Medicolegal Issues

Most important is proper counselling of patient before surgery and informed consent. Patient should be counselled about recurrence in case of peri anal abscess, fistula-in-ano and fissure. Patient should be told about chances of anal fistula formation after surgery of perianal abscess, which is its known complication. Patient should be thoroughly examined so that condition like malignancy is not missed. In patients where anal dilatation is required, patients should be told about the possibility of anal incontinence.

Who does what?

Surgeon:	Diagnosis and Work up. Pre-operative planning. Operative procedure. Post-operative care in conjunction with Anaesthetist Post-operative followup.
Anaesthetist:	Pre Anaesthesia check up Performing anaesthesia
Nurse:	Pre/ Intra/ Post-operative comprehensive care Dressing of the wound.
Technician: -	Pre-operative equipment and drugs to be checked and kept ready. Assist anaesthetist in the OT Assist the surgeon, positioning of the patient.

Resources required for one patient/ procedure (Patient weight 60 kg)

Human Resources Drugs/ Consumables Equipment

32. Surgeon – 1

33. Medical Officer / Assistant Surgeon – 1

34. Anaesthetist – 1

35. Pathologist – 1 (Service from outside can be availed)
36. Staff Nurse – 1
37. Technician – 1
38. Nursing Orderly – 1
39. Cleaning Staff – 1

INVESTIGATIONS

1. Haemoglobin
2. Blood sugar
3. Renal Function Test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG
8. CT scan/ MRI
9. X-Ray chest
10. Histopathology

Drugs and Consumables

5. OT Table and lights
6. Instrument trolley
7. Anaesthetic Machine, instruments including endotracheal tubes and drugs
8. Monitor
9. Set of surgical instruments
10. Suction
11. Sutures
12. Drains / Catheters
13. Cautery – a basic set
14. Antibiotics
15. Analgesic
16. I.V. fluids
17. Dressings

Pneumothorax

Introduction

The mechanics of ventilation relate to the negative intrathoracic pressure that draws air into the lungs during spontaneous respiration. This negative pressure is best maintained in the pleural space, which is, the potential space between the parietal and visceral layers of the pleura. Collections of air, fluid or blood in the pleural space not only compress the lung tissue, but also cause the pleural pressures to become positive, causing inappropriate ventilation.

The presence of air or gas in the pleural cavity between the lungs and the chest wall, causing collapse of the lung is called pneumothorax. If it is associated with fluid or pus it is called hydro-pneumothorax, or blood, as in chest trauma, it is called hemo-pneumothorax. In this guideline, we will be describing the basic treatment to save the life in patients with pneumothorax.

Any catheter inserted through the chest wall to remove air or fluid from the pleural space may be called a chest tube or Intercostal Chest Drain (ICD)

Causes

Primary Spontaneous Pneumothorax (**PSP**) is usually seen in the less than 40 year olds, with no obvious disease in the lung (no past / present history of lung disease and on chest X-ray no lung pathology is seen). They are generally tall, of thin build and described as having 'Marfanoid features'.

Secondary Spontaneous Pneumothorax (**SSP**) is seen in patients with lung disease like Tuberculosis, COPD (chronic obstructive pulmonary disease) rupture of emphysematous bulle and any other lung disease such as necrotizing pneumonia, or malignancy.

Pneumothorax is also often seen in patients with chest trauma (often, as part of the Polytrauma, may be blunt or penetrating) – These patients almost always need chest drain insertion along with other treatment (will not be discussed in detail here).

Pneumothorax is also seen in patients on ventilators (barotrauma), complication of central line insertion (iatrogenic). Such patients are already in secondary/tertiary care centers (will not be discussed in detail here).

Indian Incidence

Exact incidence of pneumothorax is not known but the problem is *frequently seen in clinical practice*, and is often a life threatening problem. Hence the knowledge of pneumothorax and its initial treatment is essential to all doctors.v

Symptoms

- Sudden, sharp pain in chest
- Difficulty in breathing, both dyspnea and or shortness of breath at rest.
- Cough
- Fever (in secondary spontaneous pneumothorax)

Signs

Low saturation (SpO₂ below 90%)

5 Ts.: Tachypnea, Tachycardia, Tracheal Shift to opposite side, Tympanitic note on percussion and Totally absent (or reduced) breath sounds on affected side.

Investigations

Basic / Essential

- SpO₂ measurement with Vitals (pulse, BP, Temperature and Respiratory rate)
- CXR – (the Chest X'ray) ...best as erect or at least semi recumbent at 60°.
- CBC (complete hemogram), Blood Urea, Serum creatinine, Blood sugar
- ECG (to rule out myocardial infarction)

Advanced / at higher centres

- CT scan of the chest at a later stage before surgery or in complicated patients
- Pulmonary Function Tests - spirometry – to assess lung functional status, especially in SSP
- Arterial Blood Gases

Assessment of size of pneumothorax on Chest X'ray : (British Thoracic Society guidelines)

Small : Small rim of Air around the lung

Moderate : Lung collapsed half way to heart border from the chest wall

Complete : Collapsed lung at the Hilum.

Management

Follow the Airway, Breathing and Circulation (ABC) protocol like in any other emergency. Once the airway is secure, proceed to assess breathing – it is at this stage that the diagnosis of pneumothorax is made. Administer oxygen, establish IV canula access and resuscitate.

Conservative Treatment

Only in patients with PSP and having small pneumothorax (less than 2 centimeters from the chest wall to the collapsed lung may be managed conservatively (just observation, leaked air

gets absorbed with time), or by simple aspiration of the air by a needle/syringe (needle is inserted at 2nd intercostal space at mid clavicular line for tension pneumothorax with orthopnea, not maintaining oxygen saturation and waiting for preparation of ICD insertion). Any patient with tachypnea, tachycardia and difficulty in breathing should be treated by chest drain insertion only.

Intercostal Chest Drain – Difference from other body cavity drains

Chest drainage entails the unidirectional removal of air and fluid from the chest into a collection apparatus.

The Underwater Seal is the most important element in pleural drainage. It is essentially an extension of the chest tube underwater – a low resistance, one way valve for the evacuation of pleural contents.

It works in the following manner.

1. As a conduit for the expulsion of air and fluid from the chest against minimal resistance. When intra pleural pressure raises (e.g., expiration, coughing etc.), air is forced out of the lungs through the mouth, and free contents of the pleural space are forced out through the chest tube and into the underwater seal drainage bottle.
2. As an Anti Reflex Valve. Re-entry of air into the pleural space when intra pleural pressures become negative (e.g. inspiration), is blocked by the underwater seal. Water can be drawn up the tube only to the height equal to the negative intra thoracic pressure (usually up to –20cm of water). So, the apparatus must be kept far enough below the patient to prevent water from being sucked up into the chest (a distance of 1 metre, i.e., 100cm, is sufficient].

The end of the tube must remain covered with water at all times. When a broad based bottle (Tudor-Edwards) and a narrow tube are used, elevation of the water column in the tube will lower the level in the reservoir by only a small amount, keeping the seal intact.

The end of the tube must not be kept too far below the surface of water, because resistance to expulsion of air from the chest is equal to length of tubing underwater. Keeping the tip of the tube 2 – 3 cm below the surface of water should be enough to act as a constant valve.

Intercostal Chest drain insertion - Steps of procedure

Consent

A consent is needed for insertion of chest drain, except in life threatening situations like tension pneumothorax and in management of the Polytrauma patient.

Basic precaution

Clinical examination, correlating with history and recent chest X-ray is mandatory, to confirm side of pneumothorax. The vitals are monitored by a pulse oximeter probe on the finger and administration of oxygen by mask to maintain saturation of 95% & above is preferable.

Position of patient

Supine with head end elevated to about 25°, the arm abducted at shoulder, flexed at elbow, and palm placed below the head, which is turned to opposite side. This exposes the anterolateral chest wall of affected side, and the 'Triangle of Safety' [formed anteriorly by the lateral border of the pectoralis major, laterally by the lateral border of the latissimus dorsi, inferiorly by the line of the 5th intercostal space and superiorly by the base of the axilla].

The 'SAFETY' of the Triangle of Safety relates to

- a) The chest wall is thinnest in this area.
- b) There are NO major nerves or blood vessels running in this area.
- c) The lung beneath always falls away from this area of the chest wall due to any pleural pathology and so there is less risk of injury during insertion.
- d) It is in the most comfortable position for the patient to manage the emerging tube
- e) The resulting scar is cosmetically hidden and acceptable.

Site of ICD insertion: Is in this 'Triangle of Safety' ideal choosing the 5th intercostal space in midaxillary line with arm abducted. It corresponds to level of nipple in men and breast fold in women. (procedure steps and videos are available on YouTube).

Anesthesia:

Premedicate if possible - The drug of preference is (adult male, of 60 kg)

- 75mg dose of Diclofenac Sodium parenteral (IM) about 45 minutes before procedure.
(or a suppository of 100mg Diclofenac Sodium may be used)
- Some people advocate the use of Atropine 0.6mg IM to prevent vasovagal phenomenon – but this is not necessary, if adequate explanation is given, and, enough local anaesthetic is used.

Local Anaesthesia (1% Lidocaine) of about 20ml is used. Use of intravenous sedation (1mg of Midazolam), may be opted for in elective cases and when the patient has a thick chest wall.

ICD Insertion Technique

1. This is a sterile procedure and due aseptic technique is to be used. Ensure privacy. Always have someone assisting you. The area is cleaned and draped.
2. 1% Lidocaine, the local anaesthetic, is administered. After raising a skin wheal at the site of incision, more local anaesthetic is deposited in the subcutaneous tissue and muscle in the line of the expected oblique track of dissection. It is important to infiltrate the posterior periosteum and the parietal pleura. For this, advance the needle tip over the top of the rib (to avoid the intercostal vessels) into the pleural space. As the needle is withdrawn local anaesthetic is instilled on the pleura and the posterior periosteum of the rib. Aspiration of the intrapleural air at the site of instillation of the local anaesthetic is a useful confirmation.
3. While waiting the few minutes for the local anaesthetic to act, use the time to set up the underwater seal drainage system, ensuring length of the tube to be at least 2 – 3 cm under the seal of water in the bottle, and that the correct connectors are available to connect the chest tube to the draining tubes. (Figure 1). Self-contained pleural drainage units are also available and can be used, provided expertise is available. (Figure 3).
4. Using the No. 11 scalpel blade do make a 2 cm incision at the site of local anaesthetic infiltration, down to the subcutaneous fat.
5. Using the artery forceps and blunt dissection, an oblique track is now developed to the upper border of the lower rib of the space of entry. This is done by advancing the artery forceps a short distance with the jaws closed, opening the jaws, withdrawing the instrument, closing the jaws and advancing further.
6. Sutures are now inserted....Use strong Silk (No 1) or Nylon
 - a. A central, deep, vertical mattress stitch is placed and the ends left long with a terminal knot. This suture will be used to close the wound when the drain is removed, [*the sealing stitch*]
 - b. A simple stitch at the medial end of the incision, which will be used to anchor the drain, [*the securing stitch*].
7. Proceed with the blunt dissection. The finger is used to confirm that the track is being formed well. The intercostal muscles and the parietal pleura are now punctured by the artery forceps and the opening widened. There will be loss of resistance and a gush of air. NO FORCE IS TO BE USED AT ANY STAGE. The finger is again used to confirm entry into the pleural space, swept around the margins of the wound to ensure that there is no adhesion of the lung around and that it is safe to inset the tube at this site.

8. The next step of insertion depends on the type of chest tube available.

a. For chest tubes mounted on a central trocar.

The distance of the pleural space from the wound is measured and the chest tube is held firmly just beyond this distance. The tube is guided into the chest through the track created. **NO FORCE IS TO BE USED.** The trocar is NOT meant to penetrate tissues, it is only a guide. Once in the chest, the trocar is withdrawn slightly and the tube fed into the chest, usually antero-apically for pneumothoraces and postero-basally for fluid collections. The trocar is withdrawn and the tube is connected to the underwater seal.

b. For chest tubes that have no central trocar

Either of the following methods of insertion may be followed AFTER the track has been formed by the preliminary steps described above

(i). Using a thick guarded TROCAR and CANNULA, the two are guided through the track created. The trocar is removed and the thumb is used as a temporary seal over the cannula. The chest drain is inserted through the cannula into the chest and the cannula withdrawn over the chest tube, which then quickly connected to the underwater seal drainage. Today, a 10mm Laparoscopic blunt trocar and canula (that used in *open* laparoscopy) could be used for this purpose too.

(ii).The chest drain is grasped at its tip with the artery forceps and together the two are inserted into the chest along the track. Once in the chest, the artery forceps is removed and the tube advanced into the chest suitably

9. Once the tube is connected to the underwater seal, there is an expulsion of intra pleural contents, and, a respiration related swing in the fluid level in the tube under water indicates correct intra pleural location of the tube.

10. The drain is now secured by the simple stitch at one end of the wound, by a series of firm, double knots (at least 3) on either side of the tube. The drain is also fixed to the flank of the patient in mesentery fold of adhesive tape. All joints of the ICD to the draining tubes are fixed by a adhesive tape in the *long axis* of the tubes.

11. The wound area is cleaned and a simple dressing applied. Avoid heavy dressing and strapping of the chest wall. All these hamper breathing.

12. The tube position is confirmed by an *immediate* Chest X'ray.

Post Procedure:

Give analgesics like 1gram of Paracetamol or 50 mg of Tramadol or other appropriate pain killer. Chest X-ray has to be done to confirm the position of the drain and see lung expansion. In patients with secondary spontaneous pneumothorax associated with fluid

(Hydropneumothorax often seen in patients with TB, Empyema) sample of fluid to be sent to lab for appropriate tests. Other treatment, oxygen and antibiotic to be continued as appropriate for the patient.

Management of Chest Drains

1. Analgesia - Chest drains are painful for the patient. Adequate analgesia (orally, parenterally, or even by epidural routes) help the patient co-operate better for the chest exercises and physiotherapy.
2. Breathing exercises and chest physiotherapy are the mainstay for the quick expansion of the lung. Incentive spirometry (e.g., the TRIFLO spirometer) gives the patient the impetus to expand the lung quickly. Upper Limb Movements, especially at the shoulder, and Steam Inhalations also help encourage quick lung expansion.
3. **Nursing Management**
 - i. Patient is to be kept in propped up position (45 to 90 degrees).
 - ii. Check ALL connections are SECURE – All joints to be adhesive taped well. A single layer of tape across the *long axis of each joint* holds better than layers of circular tape over the joint. This prevents disconnection and the subsequent loss of the negative pressure.
 - iii. Always ensure an erect underwater seal bottle and kept at a distance of at least 1 metre (100cm) below the level of the patients chest. The tip of the glass tube that connects to the ICD should be at least 2 cm below the fluid level in the bottle (and NOT more than 7cm below the fluid level).
 - iv. Monitor 4th Hourly (along with vital signs):

Look and Observe the ICD bottle for:

 - i. Swinging / Oscillation of fluid level in glass tube (column movement)
 - ii. Blowing / Air bubbling in ICD bottle: with quiet respiration, and on coughing (air leak)
 - iii. Drainage - type (colour), quantity and amount.
 - Inform if >100ml/hr or if frank blood.
 - v. NEVER lift ICD bottle above the level of the patient's chest, as fluid from the bottle may siphon off into the patient's pleural cavity.
 - vi. Keep 2 clamps (angled) at the Bedside:
 - vii. Do not clamp chest drains : Allow all Nursing procedures / Movement.

Clamp tubes only for tube / bottle related procedures. Eg. To change bottle/tube, empty bottle etc and for accidental disconnection of the tubes at any of the joints.

- viii. Check to AVOID KINKS in the tubes. Teach patient to look for kinks and to avoid sitting / lying on the tubes
 - ix. "MILK" tubes frequently to avoid blockage by fibrin plugs / clots.
 - x. Change connecting tube and bottle at least once every 48 hours, replacing them with sterile equivalents. Wash & disinfect equipment to remove all residue before sterilization.
-
- 4. Suction - When suction is needed, its use is to be correct, to give a constant, low pressure suction, to get out the pleural contents fully, without causing pain to the patient. The drainage system needs a second "suction regulation bottle" (see Figure 2). This is best done under supervision of the Thoracic Surgeon / surgical Team. This is needed when air leak is more, not allowing expansion of lung, particularly in penetrating injury.
 - 5. Serial chest X-rays are needed to monitor the progress of the patient, and confirm the expansion of the lung.

Trouble Shooting Incidents

- 1. Column not oscillating:- This means that the tube is blocked. All efforts must be made to restore patency of the tube by squeezing, milking & even flushing of the drainage tubing. Restoration of patency is confirmed by a respiration related swing in the draining tube.
- 2. Tubes got disconnected. This is no great disaster. Reconnect the tubes and ask the patient to cough. Any air that has entered the chest will be forced out.
- 3. Tube has been pulled out. This needs repositioning of the tube. Using all sterile precautions, a new tube has to be positioned into the chest and be properly secured.
- 4. Leak around the tube. This means that there is a partial block in the draining system. If all blocks have been removed and the leak around the tube still persists, then a single suture may have to be placed along the side of the tube to narrow the wound and seal the leak. Use of tapes and heavy dressings to occlude such leaks is not useful.
- 5. Bottle has broken. This has to be replaced immediately with a fresh bottle with a two hole stop cork, and the underwater seal recreated. Once again, ask the patient to cough. Any air that has entered the chest will be forced out.

Tube Removal

- 1. Timing of tube removal.

This is done on clinical and radiological evidence of complete expulsion of all contents of the pleural cavity with complete expansion of the lung. There should be minimal drainage for 24 hours (< 25 ml/day). There should be no air leak especially when the patient coughs or does the Valsalva manoeuvre. The chest X'ray should confirm complete expansion of the lung. The swing in the fluid level in the tube in the underwater seal bottle should be minimal, relating to the normal negative pressures in the chest during the phases of respiration.

Generally, for pneumothorax, a trial period of tube clamping for 6 hours is done. A repeat chest X'ray is then taken and if this shows complete expansion of the lung, it confirms that the lung leak has sealed and that a proper adhesion between the layers of the pleura has occurred. The tube may be safely removed now.

2. Method of tube removal

Before removal, it is preferred to give the patient a bolus dose of analgesia. Removal of the tube is with a "4 hand procedure", and hence always have someone else also assisting you. It is a sterile procedure. The securing stitch is cut loose while the tube is being supported. The mattress (sealing) stitch previously inserted and kept long, is now made free and the ends held ready to tie a knot. The patient is then instructed to cease respiration in full expiration, and the tube is gently eased out while simultaneously the knot is tied to close the track. A soft dressing is applied. A chest X'ray is taken 4 hours later to confirm there has been no introduction of air into the chest during this removal. Should the stitch break or cut through, simply compress the oblique track and apply an occlusive dressing.

3. A Chest x'ray is repeated after the removal to confirm that there has been no entry of air into the chest and that the lung continues to remain fully expanded

QUESTIONS ASKED

1. What size drains to use?

The best drain is one that is as large a tube as will pass comfortably through the intercostal space. By a rule of thumb, in an adult patient, to drain air, 24F to 28F size is adequate, but to drain fluid, a size of 32F to 36F may be necessary.

2. Can we clamp drains?

The answer is an emphatic **NO**. The resultant pneumothorax can cause more problems to the patient. All connections have to be checked to be secure and then the patient can be subject to all nursing procedures, movement and physiotherapy with NO clamps on the tube.

3. When do we clamp drains?

Drains are clamped only in situations of:

- i). When there is to be a change of the draining tubes and underwater seal bottle.
- ii). Just prior to tube removal, as a trial of clamping for 4 – 6 hours, to confirm that the air leak has stopped.
- iii). In the accidental event of a tube disconnection with loss of the underwater seal.

4. Can the patient be moved?

Yes, the patient can be moved around as usual. All connections have to be checked to be secure and the underwater seal bottle has to be kept erect and at a level of about 1 metre below the site of entry of the chest tube into the chest.

5. What suction pressure to apply?

As a general rule, suction pressures need to be kept between 10-20 cm of water (2 – 3 kPa). While up to 25cm of water suction pressure is needed for massive air leaks, 5 cm suction pressure is sufficient to help drain fluid contents out of the chest. When suction is to be used, a 'suction regulator bottle/chamber' is needed in the drainage apparatus, to ensure continuous low pressure suction in the chest cavity.

6. How long do we leave the drains?

Apposition of the two layers of the pleura is essential to seal air leaks and reduce the drainage. All air leaks will eventually stop if the lung can be kept fully expanded constantly. This may take as much as up to 4 – 6 weeks. If the air leak persists after 6 weeks, the case needs review by a thoracic surgeon. If there is significant discharge, but the lung seems to be adherent, conversion to open tube drainage may be needed.

7. Is there an alternative to the underwater bottle drainage?

Yes, there are. These are artificially made one way valve systems.

i) The flutter valve (HEIMLICH)

This is a one way system created by the use of a plastic diaphragm, which allows air to escape from the chest and yet maintain expansion of the lung. It is attached to the chest drain and strapped to the patient's side, allowing greater mobility of the patient and can be used for pneumothorax only.

This method of dressing is required in penetrating injury. After putting dressing on wound, dressing covered with micropore on three sides. Open side pushes air out during inspiration. It does not allow air to suck during expiration. This flutter valve dressing is kept while patient is having tension pneumothorax, while preparing for ICD insertion.

ii) The intercostal drainage bag.

This is a plastic bag built around a tube that reaches to the bottom of the bag. The bag is then filled with fluid to the prescribed level and this acts as the underwater seal. The tube, which is about a meter long, is connected to the intercostal tube. This bag can now collect up to 200ml of drainage, before the contents have to be drained and fresh fluid poured in to recreate the underwater seal. The bag can be strapped to the thigh of the patient and must be kept erect always.

Complications of Chest Drains

A. Of insertion

1. Thoracic visceral injury

Injudicious use of force to push the chest tube into the chest may cause injury to the lung parenchyma and the heart and great vessels. The KEY to safe insertion is, NEVER to use force. A few minutes spent in dissecting the track, checking with the finger to ensure that the track is being well formed and guiding the tube into position are the essentials to avoid visceral injury.

2. Abdominal organ injury

This is due to improper site selection – the liver on the right and the spleen on the left can be injured by tubes being inserted in lower intercostal spaces. It is important to realize that in the presence of pleuro-pulmonary disease, there may be loss of volume on that side of the chest. This causes the diaphragm to rise and render the upper abdominal organs vulnerable to injury by the chest tubes.

3. Subcutaneous Emphysema.

This is often seen with chest drains. Some air can leak through the track and cause this to happen. Provided a patent chest tube is present, with minimal resistance to the outflow of air from the tube, the subcutaneous emphysema will cease. Moreover, even the very gross forms of subcutaneous emphysema DO NOT cause any respiratory embarrassment.

B. Of management

1. Blocked tubes due to poor position.

Sometimes the tube gets trapped in the major fissure of the lung. This will need withdrawal of the tube and reinsertion.

2. Cardiac Dysrhythmias.

The tube may abut against the mediastinum and occasionally cause cardiac irregularities. If these do not settle with a withdrawal of the tube by 2 – 3 cm, a reinsertion by a separate skin site may be needed. Medical management of the arrhythmia is needed too.

3. Persistent pneumothorax.

Check for

- any obstruction in the drainage system and clear it.
- any leak in the drainage system and seal it.
- If no leak or obstruction, apply a suction of up to 25 cm of water on the drainage system.

4. Failure of the lung to re expand fully.

5. This is rarely due to blockage of the tubes and change of tubes seldom helps. The common causes of non expansion of the lung are:-

- i). Bronchial blockage leading to collapse, usually by retained sputum. Bronchoscopy will help clear secretions, and rule out other causes of bronchial obstruction like, tumour.
- ii). The presence of a fibrinous “peel” (cortex) over the lung. This is the thickened visceral pleura over the collapsed lung tissue and is usual in the cases of delay in treatment of an empyema. A decortication is the best way to deal with this problem.

C. Miscellaneous complications

Rare complications relating to chest tubes are

- i). Infections, can range from wound infection to empyemas. They reflect improper asepsis, and incorrect management of the chest drain
- ii). Re-expansion pulmonary oedema, seen when large effusions are drained in a short period of time and is best prevented by gradual decompression.

When to seek for specialist help:

After 72 hours (3 days), if the chest drain continues to bubble air and / or the lung fails to expand, as seen on the serial chest Xrays, and in most cases of SSP, the patient must be referred to the regional specialist like – thoracic surgeon / cardiothoracic surgeon / chest physician or surgeon with interest in treating these conditions.

Indications for Surgery:

In recurrent PSP or in the first episode of PSP if the lung doesn't expand fully or persistent BPF beyond 2-3 days and certain professionals like pilots, deep sea divers or mountain climbers. In patients with SSP treatment is essentially based on the underlying lung diseases and most patients may require some form of intervention such as pleurodesis or surgery beyond chest drain insertion. It is advisable to do CT scan of the chest before surgery as it helps in planning the surgery better. The chance of recurrent pneumothorax is as high as 25-60% and hence the need for surgery.

Surgery: Thoracoscopy (VATS) excision of BPF or bullae or cavity is preferred as this approach, in the hands of experienced surgeon is better tolerated, and helps in faster recovery. Thoracoscopy surgery reduces the chances of recurrent pneumothorax to less than 5%. Thoracotomy is known to reduce the risk of recurrence to less than 1% and is considered in patients who had failed thoracoscopy (VATS) and often primarily in patients with secondary spontaneous pneumothorax (SSP). The choice of intervention is based on the expertise and experience of the surgeon, anesthetist and other facilities in the center.

Who does what?

Doctor:- MBBS with interest in trauma care &/emergency medicine (preferable to have done ATLS (Advanced Trauma Life Support) or ECLS (Emergency Care Life Support) or ACLS/BLS (Cardiac Life Support or Basic Life Support) OR Surgeon (MS or MCh qualification)

Surgeon / Doctor:- Diagnosis & Work up
Pre operative planning
Operative procedure
Post operative care in conjunction with Physician/Intensivist/Anaesthetist
Post operative follow up

Anesthetist:- Pre Anaesthesia Check up
Part of resuscitation
Monitoring and support of patient, while surgeon instills the Local anaesthetic and does procedure.
Post op ICU management in conjunction with Surgeon

Nurse:- Pre/Intra/Postoperative comprehensive care of patient,
Patient's needs AND care of the ICD.

Technician:- Dressing of the wound
Pre op equipment and drugs to be checked and kept ready
Assist anesthetist in the OT
Assist the surgeon, positioning of the patient

Chest physiotherapist:- Essential to encourage the patient to do the incentive spirometry and clear the respiratory passages of retained secretions / sputum.

Resources required for one patient / procedure (Patient weight 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Medical Officer / Surgeon – 1
2. Anesthetist – 1 (not a must for chest drain insertion)
3. Staff Nurse – 1
4. Technician – 1
5. Chest physiotherapist - 1
6. Nursing Orderly – 1
7. Cleaning staff-1

Investigations / Ancillary support services needed to do

1. Haemogram
2. Blood Sugar
3. X- Ray – Chest – done in Erect position is the best
4. Pulmonary Function Tests.
5. Microbiology for analysis and cultures of ICD drainage fluids, especially in SSP
6. ECG
7. Renal Function Test in selected cases
8. LFT in selected cases
9. S. Electrolytes in selected cases
10. USG in selected cases

Drugs & Consumables

1. SpO2 + Vital Signs Monitor
2. Set of surgical Instruments see list below
 - a. A laid out sterile table with
 - Sterile drapes
 - 2 Bowls (for antiseptic and local anaesthetic)
 - Sterile syringes (10 ml x 2)
 - Appropriate needles [21G & 23G, 1½” are the best]
 - Sterile pots to collect samples, if any.
 - Sterile surgical swabs for use
 - Scalpel with No. 11 Blade

Curved Haemostats x 2 (Spencer-Wells / Kellys)

Toothed, tissue (thumb) forceps

Scissors (Suture cutting)

Strong suture with needle.....Either of

i) Large, curved, cutting needle with Nylon

ii) Large, curved cutting needle with No. 1 Silk

Needle holder

Clamp for the drain

Trocar (Blunt) and canula

b. A good spot light

c. Drainage Apparatus

Appropriate sized chest drains – for adults, 24F and larger

Underwater seal system – bottles, tubing, connectors, sterile water

d. Drugs and medical Items

i. Local Anaesthetic (prefer 1% Lidocaine)

ii. Antiseptic solution for surface cleaning (Liquid Povidone Iodine)

iii. Antibiotics

iv. Analgesics / Sedatives

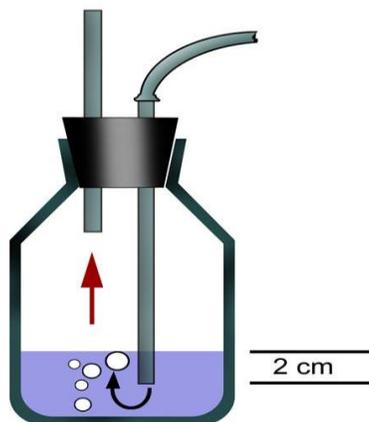
v. Inj Atropine 0.6mg amp for use, if needed for vaso-vagal syncope during procedure

vi. IV Canula (20F), IV Drip set, I.V. Fluids

vii. Dressings, adhesive tape

viii. Waste disposal bins

Figure: 1. The underwater drainage bottle



FROM PATIENT ICD

Figure 2: Use of suction regulator bottle to ensure continuous low pressure negative suction on the ICD.

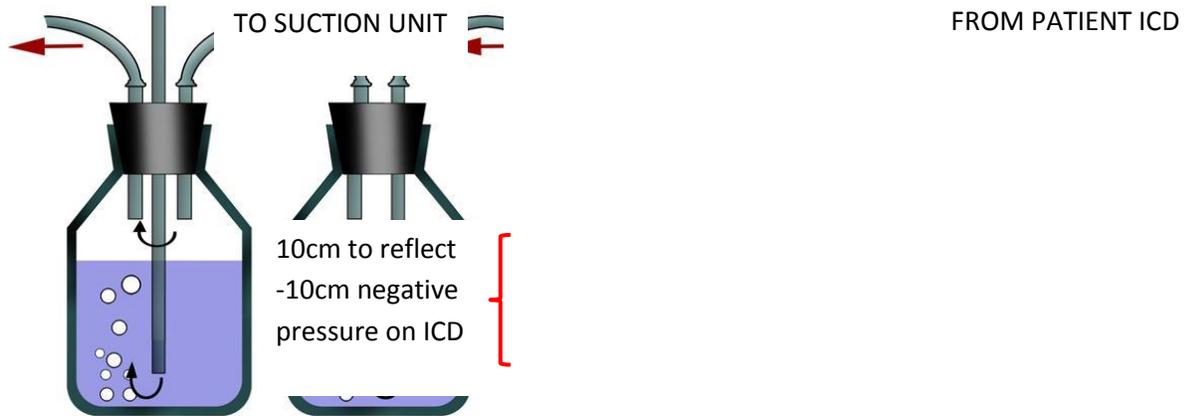
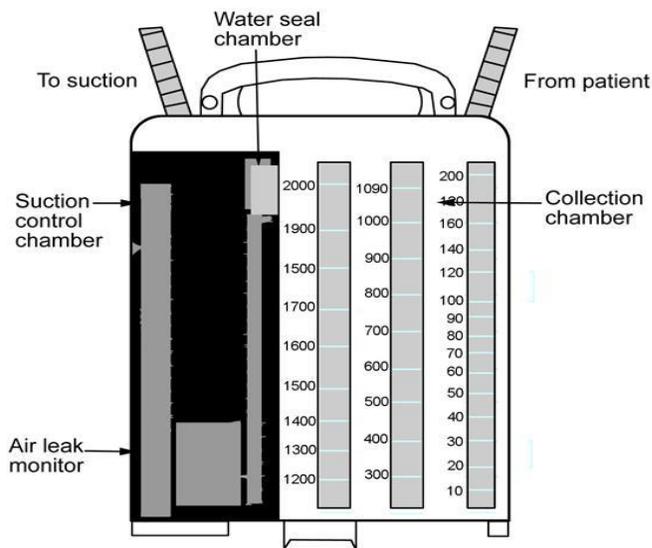


Figure 3: Chest drain apparatus: schematic view multipurpose model



A Simple Algorithm for Management Options for Pneumothorax

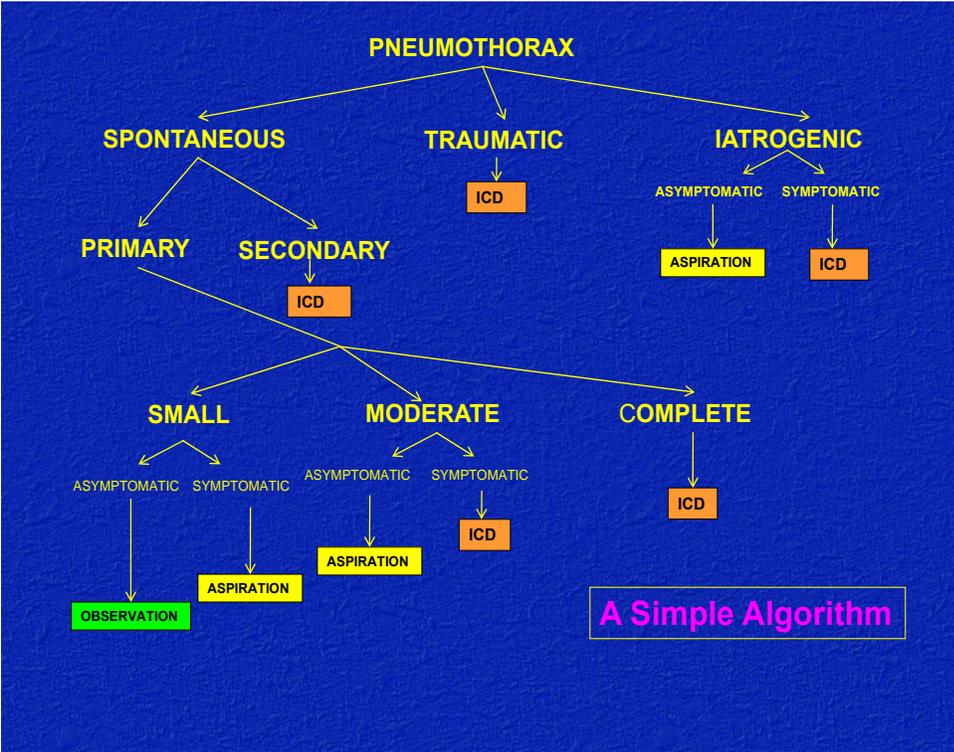


Figure 4: Triangle of Safety

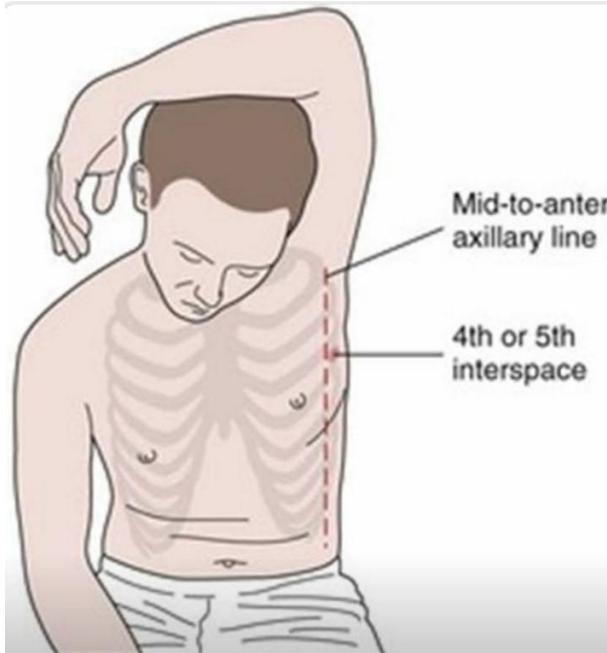


Figure 5: Chest drain apparatus: schematic view multipurpose model



Figure 6: Chest drain

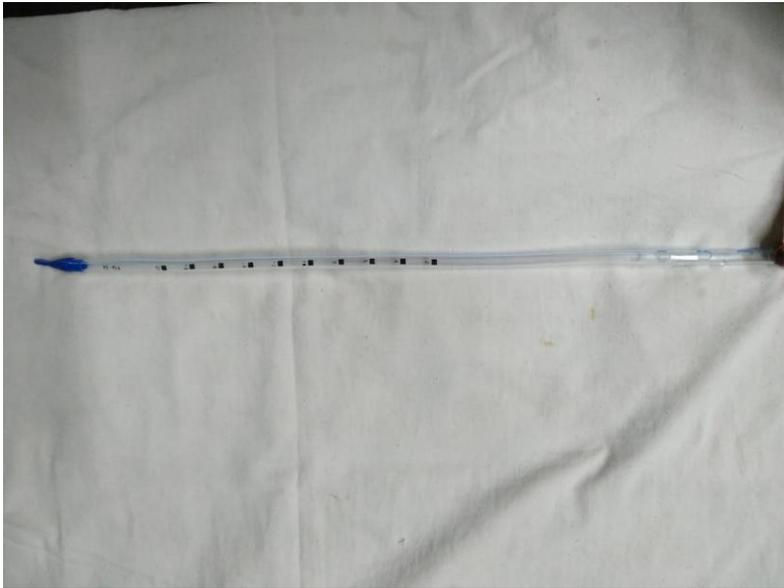
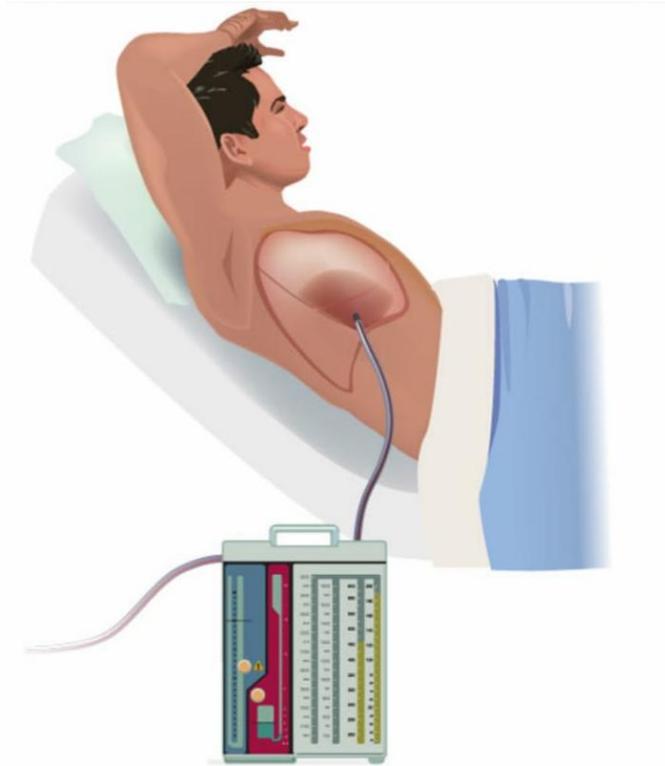


Figure 7: Chest drain in position



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Primary Care of Trauma: ABCDE

Introduction

The goal of initial assessment (Primary Survey) is to rapidly assess the patient for life threatening injury and to simultaneously carry out resuscitation of vital functions of the patient so as to restore circulating volume and to maintain tissue perfusion. This is to be performed rapidly in a sequential manner, incorporating the 'ABCDE' of trauma care.

Scope of these guidelines:

These guidelines are applicable to:

- a) Patients of all age groups
- b) In-hospital care
- c) Primary, Secondary and Tertiary levels of care

Recommendations

A: Airway maintenance and restriction of cervical spine movement

B: Breathing and Ventilation

C: Circulation and Hemorrhage control

D: Disability (Neurological status)

E: Exposure/ Environment control

Quickassessment:

Ask the patient his/her name and what had happened. An appropriate response suggests:

1. Airway is not compromised as there is ability to speak clearly.
2. Breathing is not compromised as there is ability to generate enough air movement to permit speech.
3. Circulation is sufficient enough to maintain brain perfusion and the level of consciousness adequate to recollect what has happened.

A: Airway maintenance and restriction of cervical spine movement

1. Inspect and clear airway of secretions, foreign bodies, fracture fragments by suctioning or with gloved finger.
2. Perform Jaw thrust or Chin lift maneuver and assess if it suffices to maintain adequate airway.

3. Establish a definitive airway (cuffed, secure tube in trachea) if there is any doubt regarding patients ability to maintain adequate airway.
4. Protect the cervical spine at all times during airway assessment by using a cervical collar or by manual in-line stabilization.
5. All injured patients (even if there is no airway compromise) should receive supplemental oxygen (11-15 Liters/minute) through face mask.

B: Breathing and Ventilation

1. Assess breathing by inspecting the neck for prominent veins, tracheal position, chest movement with respiration (including paradoxical movement), deformity of the chest wall, followed by palpating for tenderness, evidence of fracture ribs, subcutaneous emphysema, followed by percussion to assess for air and blood in thoracic cavity, followed up with auscultation for adequacy of air entry and for muffled heart sounds.
2. If there is evidence of tension pneumothorax, immediate needle decompression should be done before proceeding further. The site of needle insertion is anterior to the mid-axillary line in adults, and in the 2nd intercostal space at the mid-clavicular line in children.

C: Circulation and Hemorrhage control

1. Assess adequacy of circulation by pulse rate and pulse volume, blood pressure, skin temperature, level of consciousness and urine output.
2. Vascular access should be established using large bore peripheral venous cannula, introduced in the ante-cubital fossa on both sides, for intravenous fluid, blood and blood product administration. Blood samples for baseline blood investigations should be withdrawn at the time of insertion. The investigations should include blood grouping and typing, hemoglobin percentage, blood sugar and creatinine. If the patient is in shock, 1 Litre of warm Crystalloid should be rushed over 20-30 minutes, the response assessed and to guide further management.
3. Control external bleeding by applying direct pressure, point compression of the proximal vessel, or by applying tourniquet.
4. Clinically assess potential areas of internal bleeding such as thorax, abdomen, retro-peritoneum, pelvis and long bones.
5. If clinical evaluation suggests internal bleed at these sites, appropriately evaluate radiologically by Chest X-ray, Pelvis X-ray, eFAST or by DPL.
6. If there is internal bleeding, appropriate immediate intervention such as intercostal drainage tube insertion, pelvic stabilization by binder or stabilization device, extremity splint application must be carried out. Initiate preparation for appropriate definitive surgical intervention, if anticipated or if required.

7. If resuscitating within 3 hours of injury, administer 1 gram bolus of Tranexamic acid intravenously, followed by 1 gram intravenous bolus after 8 hours.

D: Disability (Neurological status)

1. Rapidly assess level of consciousness, pupillary size and reaction, and presence of lateralizing signs.
2. Objective assessment for level of consciousness and head injury should be done utilizing the Glasgow Coma Scale (GCS).
3. If there is clinical evidence of primary head injury, initiate preparation or further evaluation and intervention.

E: Exposure / Environment control

1. Completely remove all clothing worn by patient by undressing or cutting off, to facilitate a thorough examination.
2. Prevent hypothermia after completion of examination by covering the patient completely with a warm blanket. Additionally use a warming device, if required.
3. Warm intravenous infusion fluids, before starting infusion.
4. Start these maneuvers aggressively if the patient is already hypothermic.

A note on Primary Survey in Children

Initial assessment (Primary survey) and management is as in adults *except*

- a) The Broselow Pediatric Emergency tape can be used to measure the height and to estimate drug doses and the size of equipment such as tubes.
- b) Fluids are to be administered as boluses of 20 ml/kg body weight.
- c) Blood transfusion is to be carried out earlier than in adults.
- d) Cuffed ET tubes are to be used.

Adjuncts to the Primary survey and Resuscitation

- a) Pulse Oximetry
- b) Electrocardiography
- c) Urinary Bladder catheter
- d) Ryle's tube
- e) Radiological assessment

a) Pulse Oximetry

The pulse oximeter sensor should be placed on the finger/toe/ear lobe. Continuous monitoring of heart rate and oxygen saturation should be done.

b) Electrocardiography

Continuous monitoring of cardiac activity for Dysrhythmias, ST segment changes and Pulseless Electrical Activity should be done.

c) Urinary Bladder catheter

Insertion of an indwelling urinary bladder catheter is required in all hemodynamically unstable patients. The color of urine at the time of insertion should be noted, and a urine sample should be sent for routine analysis. If there is hematuria, further evaluation for renal injury should be performed. The urine output should be periodically noted.

Transurethral insertion of indwelling urinary catheter is contraindicated in patients with suspected urethral injury evidenced by blood at the tip of the external urethral meatus, scrotal hematoma, or a high riding prostate on rectal examination.

d) Ryle's tube

A Ryle's tube should be inserted when gastric decompression is required, when there is risk of aspiration of gastric contents, and to detect upper gastrointestinal tract bleed.

Insertion by the nasal route is contraindicated in patients with clinically suspected maxillofacial fractures and base of skull fracture. In patients with such injuries, insertion of Ryle's tube should be performed by the oral route.

e) Radiological assessment

Are to be used judiciously, and are not to be done if it will delay emergent intervention or transfer to another level of care.

Chest X-ray: Antero-Posterior view is to be done when blunt traumatic injury to thoracic organs is suspected

Pelvic injury: Antero-Posterior view is to be done when pelvic fracture or pelvic bleed is suspected.

eFAST: To be performed when blunt abdominal or thoracic injury are suspected.

DPL: To be performed when eFAST is not available, in hemodynamically unstable patient with clinical suspicion of blunt intra-abdominal injury.

Proceeding to Secondary Survey

The more detailed secondary survey is done only after the primary survey (ABCDE) is completed, appropriate resuscitative interventions are carried out and vital parameters have improved. The secondary survey includes 'AMPLE' history (A: Allergies, M: Medications, P: Past illnesses/Pregnancy, L: Last meal, E: Environment/ Events leading to the injury), a detailed head to toe examination, and log roll to examine the back.

Who does what?

Surgeon: Diagnosis and Work up.
 Pre-operative planning.
 Operative procedure if any.
Anaesthetist: Part of the resuscitating team
Nurse: Comprehensive care
 Dressing of the wound.
Technician: - Assist Doctors & Nurses

Resources required for one patient/ procedure (Patient weight 60 kg)

Human Resources Drugs/ Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anaesthetist – 1
4. Staff Nurse – 1
5. Technician – 1
6. Nursing Orderly – 1
7. Cleaning Staff – 1

Investigations

1. Haemoglobin, PCV, Grouping & Cross matching
2. Blood sugar
3. X-Ray chest– AP/C-Spine/Pelvis
4. FAST / eFAST
5. Alcohol/drug levels if indicated
6. ECG

7. CT scan
8. MRI
9. LFT in selected cases
10. Renal Function Test in selected cases
11. S. Electrolytes in selected cases

Drugs and Consumables

1. OT Table and lights
2. Instrument trolley
3. Anaesthetic Machine, instruments including endotracheal tubes and drugs
4. Monitor
5. Set of surgical instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. fluids
14. Dressings

Severe soft tissue infections, Cellulitis

Introduction

Skin and soft tissue infections (SSTIs) are a group of a broad spectrum of clinical entities ranging from uncomplicated cellulitis to the life threatening necrotising soft tissue infection (Table 1).

Table 1 Definition of skin and soft tissue infection

Cellulitis	Acute infection of skin involving deep dermis and subcutaneous fat
Erysipelas	More superficial infection of the skin, involving the lymphatics; characterized by a tender, erythematous plaque with well-demarcated borders
Folliculitis	Superficial infection of the hair follicle with purulence in the epidermis
Furuncle	Infection of the hair follicle with associated small subcutaneous abscess
Carbuncle	A cluster of furuncles
Cutaneous abscess	Localized collection of pus within dermis and deeper skintissues
Pyomyositis	Purulent infection of skeletal muscle, often with abscessformation
Impetigo	Superficial infection of the skin characterized by pustules or vesicles that progress to crusting or bullae
Ecthyma	A deeper variant of impetigo; begins as vesicles/pustules and evolves into “punched-out”–appearing ulcers
Gas gangrene	Necrotizing infection involving muscle; also known as clostridial myonecrosis
Necrotizing fasciitis	Aggressive infection of the subcutaneous tissue that spreads along fascial planes

SSTIs may affect any part of the body but are common in the extremities, perineum and the abdominal wall. Due to the lack of reliable microbiological and laboratory diagnostics, they pose a challenge to the clinician, who has to efficiently and effectively identify those cases requiring immediate intervention from those less severe cases. The high prevalence of drug resistant

microorganisms and the increasing number of immunocompromised patients add to the difficulty in treating these patients.

The primary motive of this chapter is to create a comprehensive clinical approach in managing patients with SSTIs. Because bacterial infections are the most common cause of SSTIs, this chapter does not include a discussion on SSTIs due to viruses, fungi and parasites.

Epidemiology

According to Singh B et al, 41% of patients of SSTIs in their study were in the age group of 41-60 years. Males were more commonly affected than females and abscess was the most common clinical manifestation of SSTIs. *S aureus* was the most common organism to be cultured followed by *E. coli*.¹²

Risk Factors for Cellulitis and Soft Tissue Infections

1. Disruption of the skin barrier:
 - Traumatic: Lacerations, recent surgery, burns, abrasions, crush injuries, open fractures, injection drug use, human and animal bites, insect bites
 - Nontraumatic: Ulcers, tinea pedis, dermatitis, toe web intertrigo
2. Impaired vascular supply /drainage
 - Axillary and pelvic lymph node dissection
 - Saphenous vein harvesting
 - Lymphedema
 - Obesity
 - Chronic venous insufficiency
 - Peripheral artery disease
3. Conditions that predispose to infection
 - Diabetes
 - Cirrhosis
 - Neutropenia
 - HIV
 - Transplantation and immunosuppressive medications
 - History of cellulitis

Pathogenesis:

The development of an SSTI starts with the break in the skin barrier and depends on three steps - adherence of the bacteria to host cells, tissue invasion and secretion of toxins. There are two types of toxins- endotoxins and exotoxins produced by the bacteria. Endotoxins are the

lipopolysaccharide chains present in the gram negative bacterial cell walls. When released in large quantities they lead to rapidly progressive SSTIs and sometimes leading to septic shock, DIC and MODS. Exotoxins are proteins that are actively secreted by the bacteria. They cause tissue damage by enzymatic degradation, cellular dysregulation or pore-formation of the cells. Superantigens are exotoxins secreted by *S aureus* and *S pyogenes*. They activate the T cells by binding on their receptors and cause massive release of cytokines leading to an amplified inflammatory response leading to the precipitation of Toxic Shock Syndrome.

Classification: Several systems of classification are used.

The Infectious Diseases Society of America (IDSA) classifies SSTIs into five categories-

- 1) Superficial uncomplicated infection (includes impetigo, erysipelas and cellulitis),
- 2) Necrotizing infections,
- 3) Infections associated with bites and animal contact,
- 4) Surgical site infections and
- 5) Infections in the immunocompromised host.

Eron et al classified SSTIs based on the severity of local and systemic signs and the presence or absence of comorbid conditions. This system is organized into classes of infection:

Class 1: patients with SSTI, but no signs or symptoms of systemic toxicity or co-morbidities.

Class 2: patients are either systemically unwell with stable co-morbidities or are systemically well, but have a comorbidity (diabetes, obesity) that may complicate or delay resolution.

Class 3: patients appear toxic and unwell (fever, tachycardia, tachypnea and/or hypotension).

Class 4: patients have sepsis syndrome and life threatening infection, eg necrotizing fasciitis.

The World Society of Emergency Surgery (WSES) expert panel classified soft tissue infections using the classification system mentioned below.

Surgical Site infections

Incisional

– Superficial

– Deep

Non-necrotizing SSTIs

Superficial infections (Impetigo, erysipelas, cellulitis)

Simple abscess, boils and carbuncles

Complex abscesses

Necrotizing SSTIs (NSTIs)

Necrotizing cellulitis
Necrotizing fasciitis
Fournier's gangrene
Necrotizing myositis

Superficial infections may be either a superficial spreading infection or inflammation within the epidermis and dermis that may be treated with antibiotics alone or a well circumscribed abscess that may be treated by drainage alone. Most of these infections are caused by Gram positive bacteria and are treated by antimicrobials against them.

NSTIs are life-threatening, invasive, soft tissue infections caused by aggressive, usually gas-forming bacteria. Delay in diagnosis and treatment leads to high mortality. NSTIs are classified into three types according to the bacteria causing the infection; type 1 – poly-microbial, type 2 – monomicrobial pathogenic β -haemolytic Streptococci or CAMRSA, type 3 – mono-microbial secondary to a variety of pathogenic bacilli.

Clinical Presentation:

Table 2: Clinical features- General Clinical features

- Fever
- Tachycardia
- Hypotension (depending on the severity of infection)
- Raised local temperature
- Pain, sometimes disproportionate to clinical signs suggesting NSTI
- Redness
- Dysfunction
- Mental obtundation (in severe infections)

Specific Clinical Features:

Impetigo

- Initially vesicular, later crusted, superficial infection of the skin.
- Most commonly caused by Staphylococcus
- Nasal colonization can act as a source of Staphylococcal impetigo.
- Starts on exposed surfaces appearing as small vesicles that pustulate and then ruptures readily.

- The purulent discharge dries & forms a characteristic golden yellow, stuck on, crusts.
- Pruritus is common and scratching of lesions results in spreading of infection.
- Healing generally occurs without scarring.

Erysipelas

- Superficial infection of skin, with prominent lymphatic involvement.
- Commonly caused by group A streptococcus.
- More common in infants, young children and older adults.
- Lower extremities are more commonly affected.
- Predisposing factors include lymphoedema, venous stasis, obesity, paraparesis, diabetes mellitus, alcohol abuse and nephrotic syndrome.
- It is painful lesion with a bright red, edematous, indurated appearance with an advancing raising and sharply demarcated borders.
- Fever is usually present.
- Cellulitis, abscess and necrotizing fasciitis are complications

Cellulitis

- It is an acute spreading infection that involves subcutaneous tissue.
- Most commonly caused by group a streptococcus and staph aureus.
- Break in the skin barrier is the most common cause.
- May also develop due to the spread of adjacent infections like osteomyelitis.
- Rapidly intensifying pain and redness is a common presentation.
- Fever and lymphadenopathy are usually present.
- The borders in cellulitis are not well demarcated.
- Bacteraemia, thrombophlebitis, especially in older people, and local abscess formation may develop.

Necrotising Soft tissue infection (NSTI)

- Pain out of proportion to physical examination findings
- Edema, induration, or pain beyond area of apparent skin involvement
- Violaceous blisters or bullae
- Pale or mottled skin
- Anesthesia of the skin
- Crepitus
- Skin necrosis or ecchymosis
- Rapid progression

- Failure to respond to initial antibiotics
- Systemic toxicity
- Multiorgan failure
- Incision into necrotic soft tissues yields no bleeding

Differential Diagnosis:

Differential diagnosis of SSTIs is based on the specific anatomical site affected.

Face	Upper extremity	Chest and Abdomen	Lower extremity
Acne	Thrombophlebitis	Drug reactions	Deep vein thrombosis
Drug reactions	Contact dermatitis	Foreign body reactions	Gouty arthritis
Relapsing polychondritis	Envenomations	Familial fever syndromes	Pseudogout
Herpes zoster	Sweet’s syndrome,	Eosinophilic cellulitis	Relapsing polychondritis
Psoriasis	Gouty arthritis	H. zoster infection	Erythromelalgia
	Pseudogout	Carcinomaerysipeloides	
	Erythromelalgia		
	Familial Hibernian fever		

Diagnosis:

The diagnosis of SSTIs in most of the cases is clinical. Laboratory investigations are used to confirm the diagnosis and to find out the etiology.

Table 3: Lab and imaging studies to evaluate SSTIs

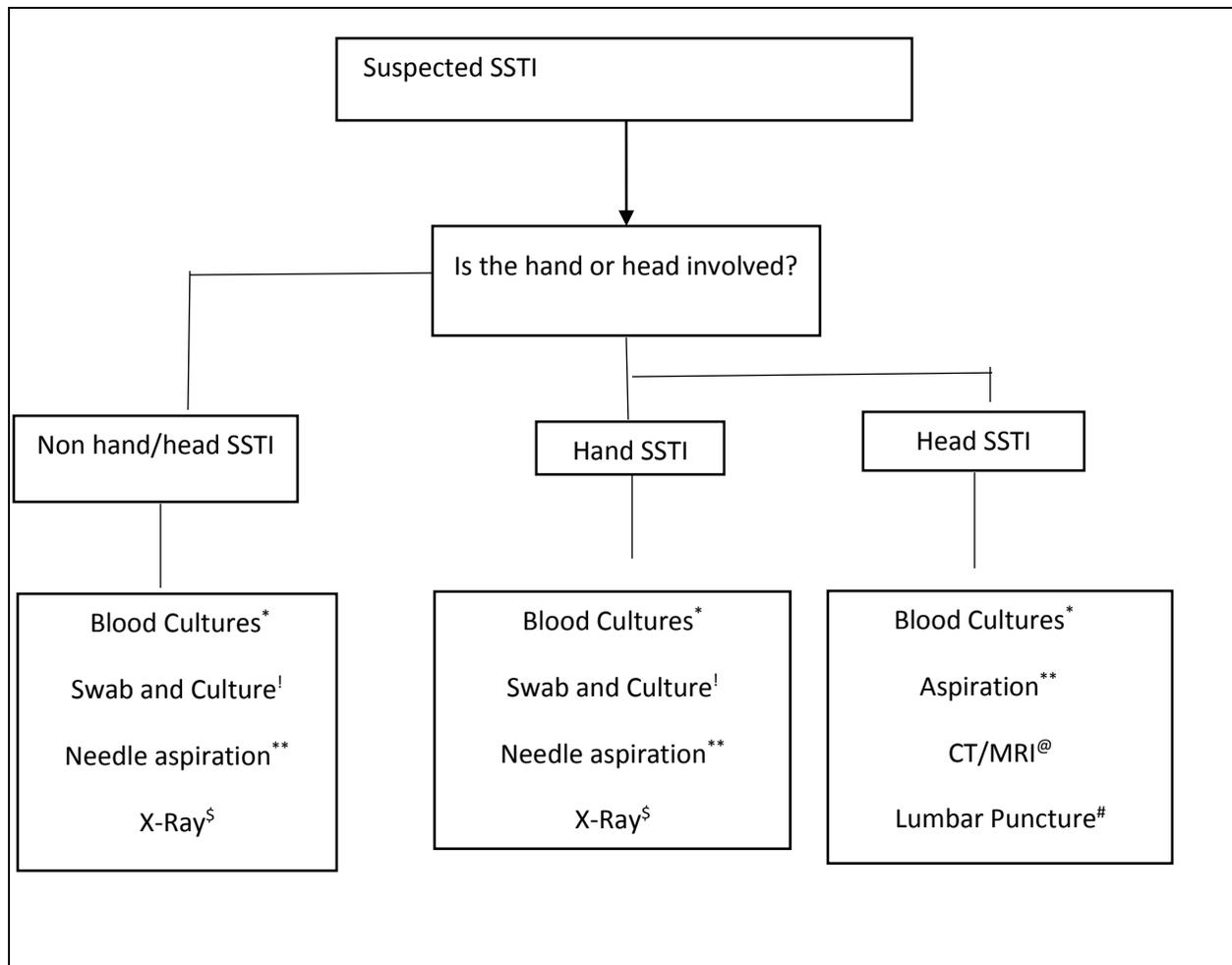
Complete blood count and differential count	Elevated leukocyte count nonspecific but suggestive of infection; markedly elevated counts with left shift suggestive of deep-seated or aggressive infection. Thrombocytopenia suggestive of more severe
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	infection. Hemoconcentration and leukemoid reaction have been described in <i>Clostridium sordellii</i> infection, and <i>Clostridium perfringens</i> infection has been associated with hemolysis
Lactate	Elevated levels indicate presence of tissue/organ hypoperfusion (e.g., sepsis) and/or possible tissue necrosis
Creatine phosphokinase	Elevated levels indicate myonecrosis and may indicate necrotizing fasciitis. Also frequently elevated in <i>Vibrio vulnificus</i> infection
C-reactive protein	Nonspecific, may help risk-stratify suspected NSTI
Gram stain and culture	Should be obtained to guide therapy in purulent infections and if surgical debridement is performed
Radiography	May show gas in tissues planes but poorly sensitive for identifying NSTI
CT with contrast	Useful for identifying NSTI when the diagnosis is uncertain
MRI	Indicated for pyomyositis and osteomyelitis; poor specificity for NSTI
Ultrasonography	Useful for identifying drainable fluid collections/abscesses

Algorithm of the diagnostic approach is depicted in Figure 1.

Figure-1- Diagnostic evaluation of skin and soft tissue infections (SSTIs). As clinically indicated; Ulcerated lesions should be cleaned and debrided before having wound base swabbed; Most useful if vesicle/bullae or fluid abscess present; Seek out bone trauma and air fluid levels; Indications-neurological deficits, vision nonassessable, proptosis/deteriorating acuity or colour/bilateral edema/ophthalmoplegia, no improvement after 24 h and swinging pyrexia not resolving within 36 h (for head only); Only if central nervous system involvement suspected and intracranial pressure excluded. CT - Computed tomography; MRI Magnetic resonance imaging.

(V Ki and C Rotstein, Bacterial skin and soft tissue infection in adults: A review and their epidemiology, pathogenesis, diagnosis, treatment and site of care. Can J Infect Dis Med Microbiol: 19;2 2008)



Estimating the risk of SSTIs:

The Laboratory Indicator for Necrotizing fasciitis (LRINEC) score for differentiating between necrotizing and non-necrotizing STIs given by Wong et al is useful only in the setting of a diagnosed or strongly suspected severe SSTI.

Table 4: LRINEC Scoring

Value	LRINEC score, points
C-reactive protein, mg/L	
<150	0
>150	4
WBC count, cells/mm ³	
<15	0

15–25	1
>25	2
Hemoglobin level, g/dL	
>13.5	0
11–13.5	1
<11	2
Sodium level, mmol/L	
>135	0
<135	2
Creatinine level, mg/dL	
<1.6	0
>1.6	2
Glucose level, mg/dL	
<180	0
>180	1

The total score range is 0-13. If the score is <5, the risk of NSTI is <50%, If it is 6-7, risk is 50-75% and if the score is >8, risk increases to >75%. The advantage of this tool is that it is based on laboratory variable that can be easily estimated in any hospital.

Eron et al also formulated a system of severity stratification based on the clinical features of the lesion and the patient.

Class 1: patients with SSTI, but no signs or symptoms of systemic toxicity or co-morbidities.

Class 2: patients are either systemically unwell with stable co-morbidities or are systemically well, but have a comorbidity (diabetes,obesity) that may complicate or delay resolution.

Class 3: patients appear toxic and unwell (fever, tachycardia, tachypnea and/or hypotension).

Class 4: patients have sepsis syndrome and life threatening infection, eg necrotizing fasciitis.

The disadvantage of this system is that as the clinical features of SSTIs is sometimes ambiguous, it is not very effective in application.

Management:

The principles of treatment of SSTIs are source control, antimicrobial therapy, support, and monitoring.

The following recommendations have been made by the Infectious Diseases Society of America (IDSA) guidelines- 2014:

1. Treatment of Impetigo and Ecthyma

- Bullous/non-bullous impetigo - topical mupirocin or retapamulin twice daily(bid) for 5 days
- Oral therapy for ecthyma or impetigo should be a 7-day regimen with dicloxacillin or cephalexin. When MRSA is suspected or confirmed, doxycycline, clindamycin, or sulfamethoxazole-trimethoprim (SMX-TMP) is recommended.
- Systemic antimicrobials should be used for infections during outbreaks of poststreptococcal glomerulonephritis to help eliminate nephritogenic strains of *Streptococcus pyogenes* from the community

2. Treatment for Purulent SSTIs (Cutaneous Abscesses, Furuncles, Carbuncles, and Inflamed Epidermoid Cysts)

- Incision and drainage is the recommended treatment
- The decision to administer antibiotics directed against *S. aureus* as an adjunct to I & D should be made based on the presence or absence of systemic inflammatory response syndrome (SIRS).
- An antibiotic active against MRSA is recommended for patients with carbuncles or abscesses who have markedly impaired host defenses and in patients with SIRS

3. Treatment for Recurrent Skin Abscesses

- Recurrent abscesses should be drained
- Send Culture and treat with a 5- to 10-day course of an antibiotic active against the pathogen isolated.
- Consider a 5-day decolonization regimen twice daily of intranasal mupirocin, daily chlorhexidine washes, and daily decontamination of personal items such as towels, sheets, and clothes for recurrent *S. aureus* infection.
- Evaluate adult patients for neutrophil disorders if H/O recurrent abscesses

4. Treatment of Erysipelas and Cellulitis

- For cellulitis without systemic signs of infection, an antimicrobial agent that is active against streptococci should be started. (penicillin, amoxicillin, amoxicillin-clavulanate, dicloxacillin, cephalexin, or clindamycin)
- For cellulitis with systemic signs of infection systemic antibiotics are indicated.
- For patients whose cellulitis is associated with penetrating trauma, evidence of MRSA infection elsewhere, nasal colonization with MRSA, drug abuse, purulent drainage, or SIRS, vancomycin or other antimicrobials effective against both MRSA and streptococci is recommended.
- In severely compromised patients, broad-spectrum antimicrobial coverage may be considered.

- Vancomycin plus either piperacillin-tazobactam or imipenem-meropenem is recommended as a reasonable empiric regimen for severe infection.
- The recommended duration of antimicrobial therapy is 5 days, but treatment should be extended if the infection has not improved within this time period.
- Elevation of the affected area and treatment of predisposing factors, such as edema or underlying cutaneous disorders, are recommended.
- In lower extremity cellulitis, clinicians should carefully examine the interdigital toe spaces because treating fissuring, scaling, or maceration may eradicate colonization with pathogens and reduce the incidence of recurrent infection.
- Outpatient therapy is recommended for patients who do not have SIRS, altered mental status, or hemodynamic instability.
- Hospitalization is recommended if there is concern for a deeper or necrotizing infection, for patients with poor adherence to therapy, for infection in a severely immunocompromised patient, or if outpatient treatment is failing.

5. Management of Surgical Site Infections

- Suture removal plus incision and drainage should be performed for surgical site infections.
- Adjunctive systemic antimicrobial therapy is not routinely indicated, but in conjunction with incision and drainage may be beneficial for surgical site infections associated with a significant systemic response such as erythema and induration extending >5 cm from the wound edge, temperature >38.5 C, heart rate >110 beats/minute, or white blood cell (WBC) count >12 000/ μ L.
- A brief course of systemic antimicrobial therapy is indicated in patients with surgical site infections following clean operations on the trunk, head and neck, or extremities that also have systemic signs of infection.
- A first-generation cephalosporin or an antistaphylococcal penicillin for methicillin-susceptible *Staphylococcus aureus* (MSSA) or vancomycin, linezolid, daptomycin, telavancin, or ceftaroline where risk factors for MRSA are high (nasal colonization, prior MRSA infection, recent hospitalization, recent antibiotics) is recommended.
- Agents active against gram-negative bacteria and anaerobes, such as a cephalosporin or fluoroquinolone in combination with metronidazole are recommended for infections following operations on the axilla, gastrointestinal (GI) tract, perineum, or female genital tract.

6. Treatment of Necrotizing Fasciitis, Including Fournier Gangrene

- Prompt surgical consultation is recommended for patient with aggressive infections associated with signs of systemic toxicity or suspicion of necrotizing fasciitis or gas.

- Early and complete surgical debridement is important.
- Empiric antibiotic treatment should be broad (eg, vancomycin or linezolid plus piperacillin-tazobactam or plus a carbapenem, or plus ceftriaxone and metronidazole), as the etiology can be polymicrobial (mixed aerobic-anaerobic microbes) or monomicrobial (group A Streptococcus, community-acquired MRSA) (strong, low).
- Penicillin plus clindamycin is recommended for treatment of documented group A streptococcal necrotizing fasciitis

Management of Pyomyositis

- Vancomycin is recommended for initial empirical therapy. An agent active against enteric gram-negative bacilli should be added for infection in immunocompromised patients or following open trauma to the muscle.
- Cefazolin or antistaphylococcal penicillin (eg, nafcillin or oxacillin) is recommended for treatment of pyomyositis caused by MSSA.
- Early drainage of purulent material should be performed.
- Repeat imaging studies should be performed in the patient with persistent bacteremia to identify undrained foci of infection
- Antibiotics should be administered intravenously initially, but once the patient is clinically improved, oral antibiotics are appropriate for patients whose bacteremia cleared promptly and those with no evidence of endocarditis or metastatic abscess. Two to 3 weeks of therapy is recommended.

Table 5: Antimicrobials Commonly Used for Oral Therapy for Cellulitis and Soft Tissue Infections

Agent	Usual Dosage	Comments
Streptococci <ul style="list-style-type: none"> • Amoxicillin • Penicillin VK 	500 mg PO TID 500 mg PO QID	Useful for <i>Pasteurella</i> species, better bioavailability than penicillin Narrow spectrum; frequent dosing
Streptococci and MSSA <ul style="list-style-type: none"> • Amoxicillin-clavulanate • Dicloxacillin • Cephalexin 	875/125 mg PO BID 500 mg PO QID 500 mg PO QID	Includes anaerobic coverage Frequent dosing Frequent dosing
MRSA (and streptococci /MSSA) <ul style="list-style-type: none"> • Clindamycin • Doxycycline 	300 mg PO BID 100 mg PO BID	Greatest association with secondary <i>Clostridium difficile</i> infection

<ul style="list-style-type: none"> • Trimethoprim-sulfamethoxazole • Linezolid • Tedizolid • Delafloxacin 	<p>1 double-strength tablet PO BID</p> <p>600 mg PO BID 200 mg PO BID 450 mg PO BID</p>	<p>Causes photosensitivity; fewer clinical data Can cause hyperkalemia; use caution in patients with impaired renal function or on angiotensin-converting enzymeinhibitors/angiotensin-receptor blockers Risk for serotonin syndrome with concomitant selective serotonin reuptake inhibitors; bone marrow toxicity with prolonged use Expensive; less risk for thrombocytopenia and drug interactions than linezolid Limited clinical experience</p>
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Table 6: Antimicrobials Commonly Used for Parenteral therapy Therapy for Cellulitis and Soft Tissue Infections

Agent	Dose	Comments
Streptococci <ul style="list-style-type: none"> • Penicillin G • Ceftriaxone 	<p>2/4 millioU IV4–6 h</p> <p>1–2 g IV 24 h</p>	<p>First line for group A streptococci</p> <p>Good gram-negative coverage (does not cover <i>Pseudomonas</i> species or ESBL–producing Enterobacteriaceae)</p>
Streptococci and MSSA <ul style="list-style-type: none"> • Cefazolin • Nafcillin 	<p>1 g IV every 8 h</p> <p>1–2 g IV every 4 h</p>	<p>Has some gram-negative coverage</p> <p>More adverse reactions than cefazolin</p>
MRSA (and streptococci/MSSA) <ul style="list-style-type: none"> • Vancomycin • Daptomycin 	<p>15 mg/kg IV 12 h</p> <p>4 mg IV 24 h</p>	<p>Requires monitoring of troughs; can cause "red man syndrome" and nephrotoxicity</p> <p>Higher dose required for bacteremia, requires monitoring of creatine phosphokinase levels</p>

<ul style="list-style-type: none"> • Linezolid 	600 mg IV 12 h	Risk for serotonin syndrome with concomitant selective serotonin reuptake inhibitors; bone marrow toxicity with prolonged use
<ul style="list-style-type: none"> • Clindamycin 	600–900 mg IV 8 h	Greatest association with secondary <i>Clostridium difficile</i> infection; increasing rates of resistant <i>Staphylococcus aureus</i>
<ul style="list-style-type: none"> • Ceftaroline 	600 mg IV 12 h	Gram-negative coverage similar to ceftriaxone
<ul style="list-style-type: none"> • Dalbavancin 	One dose of 1500 mg IV over 30 min	Single-dose regimen found to be noninferior to 2-dose regimen; limited clinical experience
<ul style="list-style-type: none"> • Oritavancin 	One dose of 1200 mg IV over 3 h	Artificially prolongs prothrombin time and partial thromboplastin time; limited clinical experience

Team Responsibilities:

SSTIs are best managed by a multidisciplinary team. Each team member contributes to the better outcome of the patient. The team can consist of:

1. A physician or an infectious disease specialist who is also the primary physician of the patient.
2. A surgeon if surgical debridement or Incision and drainage of the abscess is required.
3. A diabetologist, if the patient has diabetes mellitus, to monitor and control blood sugar.
4. An intensivist in case the patient is critically ill and requires ICU care
5. A clinical epidemiologist who will help in preventing outbreaks or spread of infection
6. A microbiologist to help us isolate the offending organism.
7. A clinical pharmacologist who is aware of the local antibiotic resistant patterns and availability and who can help the physician plan the antimicrobial therapy of SSTIs.
8. A plastic surgeon for reconstruction, if required, after debridement, once the acute condition of the patient resolves.

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Small Bowel Perforation

Introduction

Small bowel perforations are serious complications of variety of systemic as well as small bowel diseases. It usually leads to generalized peritonitis that needs quick diagnosis and early surgical intervention. However, many patients may present late in a state of pre-established sepsis and multi-organ failure due to missed or delayed diagnosis. Despite best of intensive care, antimicrobial therapy and surgical intervention, these cases have unacceptably high morbidity and mortality.

Etiology

The spectrum of etiology of small bowel perforation in India is quite different in comparison to Western countries. In India as well as other developing countries, typhoid fever is the commonest cause of small bowel perforation followed by tuberculosis, non-specific perforations, abdominal trauma, intestinal obstruction, and round worm infestation. In developed countries, the common causes of small bowel perforation are Crohn's disease, trauma, ischemic enteritis, foreign bodies, radiotherapy, drugs, malignancies and congenital malformations.

Indian Incidence

The incidence of small bowel perforation is much higher in India in comparison to the Western world. In a review of 15 large series reported from India and other Asian countries, small bowel perforation is the second most common cause (6–42 % cases) of secondary peritonitis after gastroduodenal perforation.

Symptoms

Majority of the patients in India are young males and present with pain abdomen, distention, nausea, vomiting, altered bowel habits (usually obstipation), and fever. Abdominal pain may be acute or insidious. Initially, the pain may be dull and poorly localized due to involvement of visceral peritoneum and later progresses to steady, severe, and more localized pain once parietal peritoneum is involved. Other specific features depend upon underlying etiology.

Signs

The patient may have features of localized or generalized peritonitis depending upon the stage of presentation.

On general physical examination, there is fever, tachycardia, tachypnea and hypotension.

On abdominal examination, there is distension, tenderness and rigidity often with masked liver dullness and absent bowel sounds.

The patients having altered mental status are indicative of evolution to severe sepsis.

Investigations

In endemic areas, the diagnosis of perforation peritonitis due to small bowel perforation is primarily a clinical diagnosis. The investigations aid in the diagnosis but no single investigation is diagnostic.

Hematological investigations reveal polymorphonuclearleucocytosis, electrolyte imbalance (hypokalemia, hyponatremia), raised blood urea and creatinine, and metabolic acidosis.

Chest X-Ray in erect posture usually shows pneumoperitoneum.

Abdominal ultrasound has the advantage of being portable. The limitations are bowel gas interference, abdominal distension and patient discomfort. The findings suggestive of small bowel perforation are presence of free intra-peritoneal fluid, extra-luminal air and inflammatory changes adjacent to a thickened small bowel segment. However, ultrasound can be omitted if X-ray is showing pneumoperitoneum.

Triple contrast CT scan (oral, rectal and intravenous) using water soluble dye can be done in hemodynamically stable patient. In emergency, it is not required routinely and is indicated only if diagnosis is doubtful on the basis of clinical examination and X-ray. It picks up even small amounts of extra-luminal air and oral contrast leakage into the peritoneal cavity. It also provides anatomical details of the intestinal wall; detects secondary signs of underlying bowel pathology, surrounding mesentery and lymph nodes.

Laparoscopy is gaining wider acceptance in emergency surgery both as diagnostic and therapeutic modality in specialized centers.

Management

Resuscitation

All patients are resuscitated pre-operatively with intra-venous fluids (2-3 liters of Ringer's lactate) along with nasogastric aspiration and urethral catheterization for monitoring of urine output. The broad spectrum antibiotics covering gram positive, gram negative and anaerobes are started. Parenteral analgesics for pain control and proton pump inhibitors/H₂ receptor

antagonists are given as prophylaxis for stress ulcers. The electrolyte and acid-base imbalance, if any, is corrected. As soon as the patient is adequately resuscitated, emergency exploratory laparotomy should be performed.

Surgical Treatment

Midline laparotomy is performed and the site and cause of perforation are identified and treated accordingly. The peritoneal fluid is sent for culture and sensitivity. After managing the small bowel perforation, the peritoneal cavity is irrigated with warm saline till effluent is clear and single or multiple drains are put in the peritoneal cavity. The laparotomy wound is closed either en-mass or in layers.

The appropriate surgical procedure depends on the site and number of perforations, condition of underlying bowel, severity of peritonitis and general condition of the patient. Various surgical options are:

Primary repair of perforation is performed in cases with single perforation having healthy looking small bowel and good general condition of the patient. The margins of the perforation are freshened and sent for biopsy. The perforation is then closed transversely either in two layers (inner continuous absorbable suture and outer interrupted silk suture) or in single layer of extra-mucosal interrupted silk sutures.

Segmental resection and anastomosis is done in cases having multiple perforations. Both the bowel ends should be healthy and vascular and general condition of the patient should be good.

Primary ileostomy with or without resection of diseased bowel is done in cases with delayed presentation having hemodynamic instability and generalized peritonitis. Such cases usually have severe fecal peritonitis, inter-loop abscess, grossly inflamed gut with multiple perforations and poor mesenteric circulation. In such cases there is high risk of anastomotic leak and its consequent morbidity and mortality. Therefore, diverting ileostomy is a much safer option that serves as a lifesaving procedure.

In some cases with delayed presentation, closure of laparotomy wound is very difficult due to oedema of the bowel wall and mesentery that occurs due to severe peritonitis and rapid fluid infusion during resuscitation. The forced closure of the abdominal wall is likely to cause intra-abdominal hypertension (IAH) and consequently modify pulmonary, cardiovascular, renal, splanchnic, and central nervous system functions. Such patients are managed with open laparotomy wound called "laparostomy." Temporary closure of the abdomen is achieved using simple gauze packing, impermeable and self-adhesive membrane dressing, absorbable or non-

absorbable meshes, plastic bag or vacuum-assisted closure (VAC) devices. Most of these patients also need postoperative ventilatory support for variable periods.

After recovery of the patient, ileostomy closure is done as an elective procedure after 6-8 weeks.

Drainage of peritoneal cavity in cases unfit for anesthesia and surgery. It is done under local anaesthesia in moribund patients as a lifesaving procedure. Single or multiple tube drains are put in right/left upper quadrant and pelvis and attached to drainage bags.

In post-operative period, the patient is monitored for recovery as well as detection and management of complications if any. The broad spectrum antibiotics, analgesics and proton pump inhibitors are continued in the post-operative period. The urethral catheter is removed once patient is ambulatory. The Ryle's tube is removed and patient is allowed orally once bowel sounds appear and patient passes flatus.

Post-operative complications

Wound sepsis

Intra-abdominal abscess

Paralytic ileus

Peritonitis

Faecal fistula due to anastomotic leak

Pneumonitis

Small bowel perforation – specific causes

Apart from the general description mentioned above, the specific features of individual causes of small bowel perforation and their management are as follows:

Typhoid ileal perforation

Typhoid fever is a major health problem in third world countries and occurs due to *Salmonella typhi* infection. The cases of typhoid perforation typically present in emergency with history of constant, high grade fever for the past 2-3 weeks. It is followed by sudden onset central abdominal pain that is severe in intensity and gets generalized all over the abdomen along with distension abdomen, bilious vomiting and obstipation. On examination, there are features of perforation peritonitis.

The pre-operative diagnosis of typhoid perforation in endemic areas is primarily clinical based on history of prolonged fever and clinical findings suggestive of peritonitis.

Hemogram shows anemia, leucopenia with neutropenia in typhoid fever. However leucocytosis occurs once there is ileal perforation.

Widal test is positive from 7th to 10th day in 25 - 75% cases. Thus positive Widal test is useful for the diagnosis, but negative test doesn't rule out the diagnosis.

Blood and stool cultures can pick up the organisms but these are usually negative since majority of the patients have already taken antibiotics for persistent fever.

Intra-operative findings of inflamed, edematous distal ileum with single or multiple oval perforations on anti-mesenteric border of the gut along with fecal peritonitis in endemic areas almost confirm the diagnosis of enteric perforation.

Biopsy of excised specimen may show features of typhoid perforation.

Based on operative findings, various surgical options for the management of enteric perforation are:

Primary closure is usually recommended for single perforation in a fit patient. The necrosed edges of the perforation are excised and simple transverse closure of the perforation is done in one or two layers. Many a times, pre-perforation lesions are seen adjoining to the site of perforation that should be prophylactically buried using Lambert's sutures on the surrounding sero-muscular bowel wall or may be by resection and anastomosis.

Laparoscopic treatment of typhoid perforation with primary closure is also being done successfully by experts in selected cases.

Resection-anastomosis for multiple perforations.

Right hemicolectomy is performed in cases where terminal ileum and cecum are involved with gangrenous changes and multiple perforations.

Ileostomy is done in moribund patients with multiple perforations.

Re-perforation at the site of repair or perforation from another ulcer occurs in 4-15% cases that usually presents with peritonitis and fecal fistula. In such situation, peritoneal drainage is done to remove the feco-purulent material and once the patient is stabilized, ileostomy with peritoneal lavage is done as a life saving measure.

The **post-operative mortality** of enteric perforation is higher in developing countries ranging from 9.9%-62%.

Tubercular small bowel perforation

In India, after enteric perforation, abdominal tuberculosis is the second commonest cause of small gut perforation and accounts for 5-12% of all gut perforations. The gastro-intestinal tuberculosis usually begins with direct ingestion of infected material. The most common site of involvement is ileocecal region where granuloma formation, fibrosis and stricture occurs over a period of time. The perforation usually occurs as a complication in long standing cases of stricture. Its usual site is within or proximal to the stricture and it presents with localized or generalized peritonitis depending upon severity of obstruction, size of perforation and extent of adhesions. In such cases, past history of subacute intestinal obstruction and evidence of tuberculosis on chest X-ray with pneumoperitoneum are important clues for the diagnosis. On CECT abdomen, apart from pneumoperitoneum, presence of enlarged mesenteric lymph nodes with central caseation in endemic areas is highly suggestive of tubercular perforation.

On exploration, intestinal resection and anastomosis should be preferred over primary closure of the perforation because of high risk of leak in primary closure cases.

If there are multiple strictures far apart from the site of perforation, they may be managed with a separate resection and anastomosis or treated with **stricturoplasty**. In stricturoplasty, a 5-6 cm long incision is made along the anti-mesenteric side in the strictured area of the small gut and closed transversely in two layers.

In cases of distal ileal perforation with ileocecal tuberculosis, **conservative ileo-cecal resection** with a 5 cm margin on both sides and end to end anastomosis is preferred over right hemicolectomy. Moreover, by-pass procedure like **ileo-transverse anastomosis** is no longer done in such cases due to risk of complications like intestinal obstruction, fistulae, and blind loop syndrome.

In patients with generalized peritonitis, dense adhesions (abdominal cocoon) and poor general condition, **diverting ileostomy** with or without resection of the diseased segment should be done.

Histopathological examination of the biopsy specimens (small gut, lymph node, omentum) obtained during laparotomy reveal caseating granulomas and acid-fast bacilli. The presence of multiple drug resistant TB (MDR-TB) in the region may dictate the use of Xpert MTB/RIF if facilities exist, to assess drug sensitivity /resistance as per the WHO guidelines.

The patients are given conventional anti-tubercular therapy for at least 6 months. The treatment consists of initial 2 months of rifampicin, isoniazid, pyrazinamide and ethambutol /streptomycin followed by 4 months of rifampicin and isoniazid. Pyridoxine should always be

added to prevent peripheral neuropathy due to isoniazid toxicity. Second line chemotherapy is necessary for a longer period if one or more of these first-line drugs cannot be used because of intolerance or drug resistance. The second line drugs include fluoroquinolones, amikacin, kanamycin, azithromycin and clindamycin.

The reported mortality rate in tubercular gut perforation is very high ranging from 25 to 100%. The factors associated with high mortality include old age, cachexia, delayed operation (36 hours), multiple perforations, multiple strictures, primary closure of the perforation, anastomotic leakage and steroid therapy.

Non-specific perforation

The small bowel perforations are labeled as 'non-specific' when these can't be classified on the basis of clinical features, serology, culture, operative findings and histopathology into any specific disease such as typhoid, tuberculosis or malignancy. Most of the cases are reported from the Asian countries and its incidence is next to typhoid perforation and is closely followed by tubercular small bowel perforation. The operative findings are similar to typhoid perforation and management is also same as that of typhoid perforation.

Other intestinal infections

Other intestinal infections that can rarely cause small bowel perforation are **worms** (*Ascaris lumbricoides*, *Taeniasolium*, *Enterobius vermicularis*, *Trichuris trichura*), **Entamoeba histolytica**, **Clostridium difficile**, **histoplasmosis** and **Cytomegalo virus** infection.

In worm infestation causing small bowel perforation, treatment is emergency laparotomy and resection anastomosis of the involved gut segment followed by deworming drugs. In case of round worms, the bunch of worms is gently milked out of the enterotomy site before anastomosis.

Traumatic small bowel perforation

Small bowel perforation may occur following blunt or penetrating abdominal trauma. The patients usually complain of continuous abdominal pain following trauma. The diagnosis is made on clinical signs of peritonitis and presence of pneumoperitoneum on chest X-ray.

Focused Assessment with Sonography in Trauma (FAST) is an initial step in assessment of hemodynamically unstable patients with abdominal injury. It detects free intraperitoneal fluid in 91-100% cases but identifies only 8% of cases of small bowel perforation with direct sonographic evidence.

The **treatment** is emergency laparotomy. However, in a case of polytrauma, priority of the treatment for small bowel perforation should be lower than other life-threatening injuries.

Delayed presentation: In blunt abdominal trauma, if there is mesenteric tear, it may result in gradual ischemia of adjacent small bowel and delayed perforation that may present as late as 2 weeks to 3 months after injury. The diagnosis in such cases is very difficult and needs high suspicion. The diagnosis of mesenteric hematoma is initially picked up on CECT abdomen. Most of the times, hemodynamically stable and asymptomatic cases can be managed conservatively. However, such cases need constant clinical monitoring and serial imaging in form of X-ray, ultrasound and repeat CECT abdomen if indicated. If delayed perforation is diagnosed and the condition of the patient is deteriorating, an urgent exploration is indicated.

Acute mesenteric ischemia

Acute mesenteric ischemia can lead to intestinal necrosis and small bowel perforation. It mostly affects elderly patients with severe co-morbidities. It usually has sudden onset, having non-specific symptoms and there is rapid clinical deterioration. To begin with, there is severe abdominal pain that persists beyond 2-3 hours but physical findings in the abdomen are unremarkable. The absence of clinical findings is usually responsible for delay in the diagnosis. The patient may also complain of nausea, vomiting, anorexia, diarrhea and fever. Hematochezia is reported to occur in about 15% of the cases. In delayed cases; gangrenous changes set in leading to small bowel perforation and peritonitis. The patient develops tachycardia, hypotension along with distension, tenderness and rigidity of the abdomen and absence of bowel sounds.

Lab investigations are not very helpful in making the diagnosis and are primarily meant for exclusion of other causes of acute abdomen. Plain X-ray abdomen usually has non-specific findings but presence of free air makes the diagnosis of gut perforation. Contrast enhanced **CT scan of the abdomen** is investigation of choice. The findings suggestive of the diagnosis include focal bowel wall thickening, lack of bowel wall enhancement, sub-mucosal haemorrhage, air in portal venous system, intra-mural gas and free air in the peritoneal cavity. **CT angiography** can clearly delineate pathology in mesenteric vessels.

After resuscitation, exploratory laparotomy is done. The gangrenous bowel is resected and based on the general condition of the patient, diverting stoma or primary anastomosis is done. The aim of resection is to conserve as much bowel as possible. In cases with extensive bowel involvement, second look laparotomy after 24 hours should be considered with the aim to preserve the bowel with doubtful viability.

Despite improvement in diagnostic and therapeutic modalities, mortality of acute mesenteric ischemia is about 60%.

Small bowel obstruction

Sometimes, delayed and neglected cases of acute intestinal obstruction can have small bowel perforation and present with features of peritonitis. Various causes are adhesions, bands, inter-loop abscess, internal herniation, intussusception, obstruction (gall stone ileus).

Small bowel tumors

A variety of small bowel tumors can present with spontaneous perforation and majority of them are malignant in nature. These are lymphomas, gastro-intestinal stromal tumors (GIST) and metastatic deposits.

Crohn's disease

The incidence of free perforation in Crohn's disease is 1-3% in Western countries and it is very rare in India.

The patient is usually a known case of Crohn's disease and has sudden worsening in the clinical course with abdominal signs of generalized peritonitis. A high index of suspicion is required for making the diagnosis. Plain X-ray abdomen (erect film) may rarely show free air under the diaphragm. CECT abdomen demonstrates extraluminal air or leaking oral contrast with typical findings of active Crohn's disease in form of thickened small bowel loop with multilayer enhancement and hyper-vascularity at its mesenteric side.

On exploration, debridement and simple suture of the perforation should not be done due to high rate of morbidity and mortality. For ileal perforation, limited resection of the most severely affected bowel segment with primary anastomosis is the treatment of choice. In moribund patients with generalized peritonitis, proximal diverting ileostomy should be done. For jejunal perforations, resection of the diseased loop and end-to-end anastomosis is done. Temporary jejunostomy is avoided due to serious metabolic problems associated with it and greater safety of jejunal anastomosis due to better healing. The mortality rate of free perforations in Crohn's disease has decreased from 41% to 4% ever since the simple suture modality is replaced with resection.

Diverticular disease

Perforated Meckel's diverticulum usually presents as acute abdomen mimicking acute appendicitis. The diagnosis is usually made at operation and it is managed with diverticulectomy or segmental resection and end to end anastomosis.

Jejuno-ileal diverticulosis can rarely perforate causing localized peritonitis because of their location on mesenteric border that readily gets sealed. The treatment is segmental intestinal resection with primary anastomosis including non-inflamed diverticula.

Drugs causing small bowel perforation

Various drugs that are known to cause small bowel perforation due to their side effect are steroids, non-steroidal anti-inflammatory drugs (NSAIDs), potassium chloride tablets, cocaine, oral contraceptives and cancer chemotherapy.

Iatrogenic small bowel perforation

In **laparoscopic surgery**, small bowel perforation is likely to occur during creation of pneumoperitoneum by Veress needle or while blind insertion of first trocar. Sometimes bowel injury might occur during cautery dissection due to inadvertent contact of diathermy to the adjoining gut wall in a direct or indirect manner. The small bowel injury is usually identified during surgery and is managed with primary repair with good outcome. However, if it is missed during surgery, the diagnosis might be difficult in postoperative period, because the features of the ensuing peritonitis are obscured by postoperative pain. Once diagnosed, the treatment is primary closure of perforation after freshening the perforation margins or gut exteriorization depending upon condition of the patient and severity of peritonitis. The mortality of bowel perforation during laparoscopy is reported to be 3.6%.

Abdominal drains can rarely cause small bowel perforation. In the post-operative period, the patient having abdominal drain in situ may complain of high-grade fever with pain abdomen. On examination, there can be features of localized or generalized peritonitis. The small bowel contents coming through the drainage tube make the diagnosis obvious. Ultrasonography of the abdomen may reveal collections of mixed echogenic fluid. A fistulogram through the drain reveals that the tip of the drain had entered the gut.

In patients without signs of peritonitis, discontinuation of the vacuum in suction drain and withdrawal of tube drain invariably leads to healing of perforation site. The patients with generalized peritonitis need repeat laparotomy for management of perforation. It is recommended that to avoid this complication, drains should be placed carefully and removed early after the drainage has decreased.

During **unsafe abortion**, small bowel perforation can occur due to rupture of posterior vaginal wall by operating instrument that damages the adjoining pelvic viscera. The diagnosis is based on clinical findings of peritonitis and X-ray abdomen showing pneumoperitoneum. After resuscitation, early surgical intervention in form of resection/repair of the injured bowel is

done. The awareness and early diagnosis of this clinical entity is of paramount importance in avoiding high morbidity and mortality.

During **double balloon enteroscopy**, done for the diagnosis of obscure intestinal bleeding, two balloons are alternatively inflated and can cause small bowel perforation.

Referral Criteria

ICU care may be needed in patients who present late with severe sepsis and have other systemic illnesses.

Patients with anastomotic leak or persistent sepsis following laparotomy may need ICU care.

Medicolegal Issues

- Failure to detect / investigate or refer a patient of suspected small bowel perforation.
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action.

Who does what?

Doctor:

Surgeon:-
Diagnosis & Work up
Pre operative planning, resuscitation
Operative procedure
Post operative care in conjunction with Anaesthetist/Intensivist
Post operative follow up

Anesthetist:-
Pre Anaesthesia Check up
Part of resuscitation
Performing anesthesia
Post op ICU management in conjunction with Surgeon

Nurse:-
Pre/Intra/Postop comprehensive care
Dressing of the wound

Technician:-
Pre op equipment and drugs to be checked and kept ready
Assist anesthetist in the OT
Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (Patient weight 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram, BT, CT
2. Urine examination
3. Blood Sugar
4. Blood urea, serum creatinine
5. LFT in selected cases
6. S. Electrolytes
7. X- Ray – Chest
8. X ray abdomen standing and lying views in selected cases
9. USG abdomen in selected cases
10. CECT abdomen in selected cases
11. ECG
12. Histopathology

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Defibrillator
6. Set of surgical Instruments
7. Autoclave
8. Suction
9. Sutures
10. Drains
11. Catheters
12. Cautery – a basic set
13. Antibiotics

14. Analgesic
15. I.V. Fluids
16. Dressings
17. Blood transfusion in selected cases
18. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as decided by the Surgeon in selected cases.

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Splenic trauma

Introduction

Spleen is the most commonly injured organ in abdominal trauma. Incidence of splenic injury is up to 60% in Blunt Abdominal Trauma. The first case of traumatic rupture of the spleen was published by Eisendrath in 1902. At the beginning of the nineteenth century, the surgical treatment consisted of splenectomy, which was performed to avoid exsanguinations but operative morbidity and mortality were high. In the mid 20th century advancements in surgical and anaesthesia techniques resulted in improvement in both morbidity and mortality after splenectomy. In the 1960's post-splenectomy sepsis was recognised as a real and frequent occurrence and attempts were made towards preservation of the spleen. Hence, in the last 3-4 decades the treatment has shifted towards organ preservation, non-surgically and surgically.

Indian incidence

There are no statistics available on this topic.

Setting

Trauma to the abdomen is the commonest setting. Blunt trauma, most commonly RTA (road traffic accident) is responsible for majority of splenic injuries. Falls, assault and warfare & civilian bombing are other scenarios where the spleen can be injured. Penetrating injuries are a less common cause. Splenic injury can be isolated or present with other solid or hollow organ injuries. A diseased spleen is more susceptible to rupture even with trivial trauma

Symptoms

- History of trauma
- Pain in the abdomen, may be localised to the left upper abdomen in isolated splenic injury
- Difficulty in breathing may be present if there are rib fractures associated
- Feeling of giddiness and fainting
- If there is associated head injury with altered sensorium, these symptoms may be masked or absent

Signs

- Tachycardia
- Hypotension

- Pallor
- Abdominal distension
- Tenderness in the left upper quadrant
- Signs of peritonism in case of hemoperitoneum
- Ecchymosis in the left flank and lower chest
- “Seat-belt” ecchymosis
- Signs of fracture of left lower ribs
- Kehr’s sign – pain referred to the left shoulder that increases with inspiration

Investigations

Diagnostic

- DPL (Diagnostic Peritoneal Lavage) – time tested, rapidly performed and inexpensive. Still as sensitive as FAST, but invasive
- USG (FAST, Focused Abdominal Scan for Trauma) – for demonstrating free fluid in the abdomen. Very sensitive but poor for delineating organ-specific injury
- Contrast CT Abdomen with arterial and venous phases reliably demonstrates solid-organ injuries. Splenic contrast “blush” is specific for active bleeding. Can also pick up other associated organ injuries.
- X-ray Chest, Angiography, MRI, Radionuclide scans rarely add value

For preparation for management

- Hemogram with serial Haemoglobin
- Blood group and cross match
- Blood sugar
- Blood Urea, Serum creatinine
- Bleeding time, clotting time and prothrombin time

Management

Initial management

Initial management of the patient should proceed on the lines of ATLS protocol. Securing an i.v. access, urinary catheter and insertion of a nasogastric tube should be done in patients with a suspicion of major intra-abdominal trauma after excluding urethral rupture and cervical spine & head injuries. Intravenous infusion of fluids is begun and broad-spectrum antibiotics administered. In select cases, invasive monitoring of arterial and venous pressure may be required.

Hemodynamically unstable patients should be taken to OT if there is a strong suspicion of intra-abdominal injury and FAST is “positive”. Stable patients can be considered for non-operative management if the conditions are favourable.

Grading of splenic trauma

Grading of splenic trauma is based on the CT findings

Table-1.

American Association for the Surgery of Trauma (AAST) Spleen Injury Grading System.

Grade^a	Description of Injury	
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1cm parenchymal depth
II	Hematoma	Subcapsular, 10%-50% surface area Intraparenchymal, <5 cm in diameter
	Laceration	Capsular tear, 1-3cm parenchymal depth that does not involve a trabecular vessel
III	Hematoma	Subcapsular, >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma Intraparenchymal hematoma \geq 5 cm or expanding
	Laceration	>3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularisation (>25% of the spleen)
V	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury with devascularized spleen

^aAdvance one grade for multiple injuries up to grade III.

Non-operative Management (NOM)

The first reports of salvage of spleen in patients of splenic injuries appeared in the late 1960's and early 1970's from pediatric surgeons who used non-operative management & splenorrhaphy and various other techniques to achieve splenic salvage. It is estimated that more than 80% of all splenic injuries can be successfully managed non-operatively.

The aim of non-surgical management in splenic trauma is to preserve function and reduce the morbidity and mortality associated with surgery. It is associated with a lower rate of non-therapeutic laparotomies, lower rate of blood transfusion, reduced overall morbidity and mortality rates, and lower hospital costs.

Inclusion criteria:

1. Patients with stable hemodynamic signs
2. Stable hemoglobin levels over 12-48 hours
3. Minimal transfusion requirements (2 units or less)
4. CT scan injury scale grade of I through III without a blush
5. Relatively younger patients (though older patients have also been shown to benefit)

Patients with other abdominal injuries may be considered for operative intervention despite the above criteria being satisfied.

Patients on anti-coagulants or anti-platelet drugs may be at an increased risk for delayed bleeding.

With the availability of emergency angio-embolisation, even higher grade injuries with active bleeding may also be considered for non-operative management.

Principles of Management and Requirements

1. Hospitalisation in intermediate or intensive care units
2. Continuous monitoring of vital signs, urine output
3. Relative rest
4. Frequent monitoring of hemoglobin levels
5. Follow-up series of abdominal examinations, clinical and radiological
6. Access to blood transfusions
7. Availability of CT in the hospital premises
8. Continued presence of surgeons and/or interventional radiologists

Non-operative management has to be done on the “Threshold of the OT”

Indications of abandoning Non-operative Management

1. Signs of persistent bleeding; fall in Hemoglobin, development or worsening of abdominal pain and peritonism
2. Hemodynamic instability unresponsive to fluid and blood administration
3. Discovery of other injuries like bowel injuries
4. Fever
5. Discovery of vascular complications like pseudo-aneurysms or arterio-venous malformations on follow-up CT

Complications

1. Late-onset bleeding
2. Need for transfusion
3. Undetected intra-abdominal injuries
4. Pseudoaneurysms
5. Pseudocysts
6. Splenic abscesses

Risk factors of failure of Non-operative Management

1. Injury grade
2. Presence of significant hemoperitoneum
3. Contrast extravasation on imaging studies
4. Arterial hypotension upon admittance
5. Associated brain injury
6. The need for blood transfusion

Criteria for discharge

1. Stability of hemoglobin levels
2. Absence or abatement of symptoms
3. Accepting oral feeds and passing motions

Surgical Treatment

Indications

1. Hemodynamically unstable patient with a suspicion of intra-abdominal bleeding
2. Presence of other associated injuries requiring surgery

3. Failure of Non-operative Management
4. Non-availability of facilities for Non-operative Management like CT scan, ICU

Surgical Options

1. Splenectomy
2. Splenorrhaphy
3. Splenic Mesh
4. Partial Splenectomy

Method

The purpose of surgery in splenic trauma is primarily hemostasis. The fastest and the most classic is splenectomy. If the patient is stable and expertise is available, spleen preserving methods can be used.

The patient is placed in the supine position. A midline incision provides the most expeditious entry into the abdominal cavity. The incision can be extended to the symphysis pubis if necessary.

Once the abdomen is entered, hemoperitoneum is evacuated rapidly and packs are placed in all four quadrants of the abdomen for tamponade. The liver and spleen are palpated by the right hand of the surgeon. Once a splenic injury is identified, the next step is to compress the spleen by the left hand to provide tamponade in order to arrest bleeding, which is very effective. The next step is to mobilise the spleen and to deliver it into the wound, usually along with the distal pancreas.

For mobilizing the spleen the posterior attachments of the organ have to be divided with either sharp or blunt dissection. Usually, in cases of severe injuries to the spleen, most of the attachments are already divided by the injury or hematoma, and some blunt dissection with the right hand is sufficient to free the spleen from its posterior attachments and allow it to be delivered into the wound.

Once the spleen is delivered, the hilum is compressed at the tail of pancreas by fingers or by the application of an intestinal clamp. This maneuver effectively arrests bleeding and frees the surgeon for exploration of the rest of the abdomen. The rest of the abdomen is systematically explored for other injuries and bleeding arrested wherever necessary. Once all bleeding is arrested the anesthetist is asked to resuscitate and stabilize the patient before further steps are executed.

Once the patient is stable the splenic artery and vein are identified in the hilum and ligated and divided. Thereafter, the short gastric vessels are carefully divided and the spleen is removed. Often the short gastric arteries have been avulsed and the greater curve of the stomach needs to be inspected and hemostasis has to be achieved. The tail of pancreas is in close proximity to the splenic hilum and may be injured by the trauma or during mobilization. Hence, the tail of pancreas needs to be inspected and over sewn if necessary.

In less emergent situations, splenorrhaphy is the preferred method of surgical care. Multiple techniques are described, but they all attempt to tamponade active bleeding either by partial resection and selective vessel ligation or by putting external pressure on the spleen via an absorbable mesh bag or by suture repair of the spleen over pledgets or omentum. Both "make it yourself" and commercial products are available for this purpose. In patients with capsular injury, the electrocautery or argon beam coagulator device may provide adequate hemostasis and allow for splenic preservation.

The decision to place a drain after splenectomy is made on a case to case basis with most cases with no pancreatic injury or residual ooze not benefitting from a drain.

Post Operative Care & Complications

The nasogastric tube can be removed on postoperative day 1 or 2, depending on the return of GI function, and diet can be slowly advanced. Early mobilisation, chest physiotherapy and early removal of Foley catheter (once the patient is hemodynamically stable and is producing adequate urine) expedites recovery and reduces complications.

Surgical complications include

1. Pneumonia (30%)
2. Wound infection
3. Acute Gastric Dilatation
4. Bleeding from the short gastric vessels
5. Pancreatic fistula
6. Sub-phrenic collections, infected and sterile

Angio-embolisation

The indications of embolisation include

Absolute

1. Grades IV and V in the absence of other injuries in a stable patient
2. Extravasation of perisplenic contrast.

Relative

1. Grades I, II and III in the presence of signs of contrast extravasation on CT
2. Associated splenic intravascular injury (pseudoaneurysm or arteriovenous fistula)
3. Moderate hemoperitoneum
4. Decreased hemoglobin levels during conservative management.

Endovascular embolisation can be either proximal or distal. The proximal approach is faster, simpler and associated with less failure of conservative treatment; additionally, it presents fewer complications than the distal approach.

Complications

1. Bleeding associated with undiagnosed pseudo-aneurysms or re-bleeding
2. Undetected associated injuries (for instance, pancreatic or diaphragmatic)
3. Sepsis or splenic abscesses
4. Splenic atrophy or infarction
5. Acute pancreatitis (especially associated with proximal angio-embolisation)
6. Arterial iatrogenic injury
7. Nephropathy secondary to the contrast medium
8. Deep vein thrombosis
9. Migration of the embolic material
10. Hematoma
11. Paralytic ileus
12. Thrombocytosis

Immunisation

All patients should be routinely vaccinated for Haemophilus and Meningococcus species before discharge to prevent OPSI (Overwhelming Post Splenectomy Sepsis) which occurs in 3-5% patients after splenectomy. Patients should also be warned about their increased susceptibility to Malaria.

Referral Criteria

- ICU care may be needed in patients who in patients who have experienced prolonged hypotension resulting in Acute Kidney Injury and ischemic injury to other organ systems and Multi-organ Dysfunction Syndrome.

- Patients with late-onset complications may need referral for ICU care and for the services of an interventional radiologist

Medicolegal Issues

- Failure to provide primary care and resuscitation to a victim of trauma
- Failure to detect / investigate or refer a patient of suspected splenic injury
- Failure to detect other associated injuries
- Iatrogenic injuries caused during surgery
- Delay in treatment
- Delay in detecting complications and taking corrective action
- Failure to report the case to appropriate Police authorities as all of these are medico-legal cases

Who does what?

Doctor:

Surgeon:	Diagnosis & Work up Pre operative planning Operative procedure Post operative care in conjunction with Anaesthetist/Intensivist Post operative follow up
Anesthetist:	Pre Anesthesia Check up Part of resuscitation Delivering anesthesia Post op ICU management in conjunction with Surgeon
Nurse:	Pre/Intra/Post-op comprehensive care Dressing of the wound
Technician:	Pre op equipment and drugs to be checked and kept ready Assist anesthetist in the OT Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (patient weight 60 kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer /Assistant Surgeon – 1
3. Anesthetist – 1

4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Hemogram
2. Blood Sugar
3. Renal Function Test in selected cases
4. LFT in selected cases
5. S. Electrolytes in selected cases
6. USG in selected cases
7. ECG
8. X-Ray – Chest
9. Histopathology
10. CT Scan if available

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anesthetic Machine, instruments including endotracheal tubes & drugs
4. Patient Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic
13. I.V. Fluids
14. Blood products
15. Dressings
16. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as decided by the Surgeon in stable patients.

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Stridor

- 1.1 Definition and mechanism
- 1.2 Distinguishing supraglottic and and tracheal obstruction
- 1.3 Causes of stridor
- 1.4 Treatment flow chart
- 1.5 Further investigations
- 1.6 Definitive treatment

1.1 Definitions:

- Stridor: It is a high pitched breath sound caused due to turbulent air flow through the larynx or proximaltracheobronchial obstruction.It can be
 1. Inspiratory stridor : obstruction in supraglottic or pharynx
 2. Expiratory stridor : obstruction of thoracic trachea or upper bronchus
 3. Biphasic stridor :obstruction in glottic, subglottic or cervical trachea
- Wheeze: Continuous, coarse, whistling sound produced due to narrowing of lower airway. It is predominantly expiratory in nature
- Rattling: Inspiratory coarse sound felt by placing hands over chest. Casused due to tracheo-bronchial secretions.

1.2 Distinguishing between stridor due to supraglottic and tracheal obstruction:

Supraglottic obstruction	Tracheal obstruction
Inspiratory in nature	Expiratory in nature
Voice is muffled	Voice is normal
Dyspnoea is less severe	Dyspnoea is more marked
Cough is less marked	Deep barking or brassy cough is present

1.3 Causes of stridor

- Supra glottic cause
 1. Nose: Choanal atresia
 2. Tongue: Macroglossia, vascular malformations, lymphangiomas, neoplastic conditions. Base of the tongue lesions are responsible for stridor.
 3. Mandibular malformations which predisposes to falling back of tongue. eg. Micrognathia, Pierre-Robin Syndrome.
 4. Pharynx: Adenotonsillar hypertrophy, retropharyngeal abscess, neoplasia
- Glottic (laryngeal)
 1. Congenital: Web, malacia, cysts, stenosis
 2. Traumatic: Injuries to larynx, foreign bodies, laryngeal edema due to endoscopy or prolonged intubation.
 3. Inflammatory: Acute epiglottitis, laryngotracheitis, diphtheria
 4. Neoplastic: Vascular malformations, papillomatosis, carcinoma
 5. Neurogenic: Laryngeal palsy which may be due to external compression, post thyroid surgery
 6. Miscellaneous: Tetanus, tetany, laryngismus stridulus
- Sub glottic (tracheal and bronchial)
 1. Congenital: Atresia, stenosis, and tracheomalacia
 2. Traumatic: Foreign body, stenosis due to prolonged tracheal intubation.
 3. Inflammatory: Tracheobronchitis
 4. Neoplastic: Tracheal tumors
- Lesions outside respiratory tract
 1. Foreign body in the esophagus
 2. Retropharyngeal and retroesophageal abscess
 3. Large neck mass causing extrinsic compression

1.4 Treatment guidelines: Based on the principles of life support.

Step 1 : Airway clearance : This includes clearing the airway and removing any foreign body by sweeping movement with gloved fingers and oropharyngeal suction.

Step 2 : Initiate breathing : Oxygen (humidified if possible). In centres where heliox (mixture of helium and oxygen) is available, heliox may be used.

Step 3a Circulation : If there is swallowing problem initiate IV Dexamethazone at a dose of 10 mg IV stat followed by 4 mg IV 6 hourly

Step 3b : If patient is able to swallow initiate oral steroids (Dexamethazone at 8 mg twice daily).

Step 4 : Nebulization with salbutamol as and when required.

Step 5 : Add Omeprazole or Pantoprazole oral / IV depending on the patients condition.

Step 6 : If condition does not improve and patient continues to have

1. Progressive airway obstruction
2. Decreasing levels of consciousness
3. Advanced respiratory obstruction at admission

Endotracheal intubation is to be done (**Call for help from Anaesthesiologist / critical care specialist if needed**)

It is mandatory that intubation set with umbo bag is kept ready while managing a case of acute stridor.

Step 7 : If intubation efforts fail, emergency surgical measures are to be considered (**cricothyrotomy, tracheostomy**). **Call for help from General Surgeon and ENT specialists**

It is mandatory that cricothyrotomy and tracheostomy set are to be kept ready while managing Acute Stridor.

Once the patient airway and breathing control is achieved proceed to evaluate the cause.

Step 8 : Investigate for the cause of stridor

- History tick box for evaluation of stridor
 1. Is the event acute onset?
 2. If acute, is there a history of upper resp tract inf. preceding onset of the condition?
 3. Is there a history of exposure to any known allergens?
 4. Is there a history of intubation very recently?
 5. Is there a history of neck surgery?
 6. Is the history chronic?
 7. If chronic, what is the age of onset/duration of stridor(birth, infancy, adult onset)
 8. Is the symptom intermittent, what are the aggravating factors?
 9. Does the patient give history of reflux symptoms?
 10. Does the patient give history of weight loss and asthenia?
 11. Does the patient give history of voice change?
 12. Does the patient give history difficulty in swallowing and recurrent aspiration?

- Physical examination
 1. Look for movement of accessory muscles of respiration: Indicates severe respiratory distress
 2. Distinguish between stridor and wheeze.
 3. Distinguish between inspiratory/expiratory/biphasic wheeze
 4. Examination of nose, tongue, jaw, pharynx and larynx (indirect laryngoscopy. Seek help from ENT specialist)
 5. Look for associated fever which indicates infective cause.
 6. Monitor SpO2 closely

AT THIS POINT THE PATIENT MAY BE TRANSFERRED TO HIGHER CENTRE ONCE STABILIZED (distress subsides, haemodynamically stable based on clinical and objective investigations)

At transfer: Put detail note about the findings at presentation and the treatment provided.

1.5 Further investigations: to be done at centres with optimum infrastructure and critical care facilities

1. Flexible fiberoptic laryngoscopy under topical anaesthesia
2. Soft tissue lateral and PA view of neck and X-ray chest PA and lateral view to diagnose foreign body of the airway.
3. CT scan with contrast to exclude compressive lesions and vascular anomalies
4. Direct laryngoscopy/bronchoscopy under GA: Such procedure is diagnostic and therapeutic. In some cases esophagoscopy may be done if larynx and bronchus is found to be normal.
5. In rare circumstances contrast esophagogram when esophageal stenosis/stricture or fistula is suspected.
6. Angiography may be needed when vascular malformations or abnormal vascular anatomy is suspected.

1.6 Definitive treatment:

Treatment of the etiology is best done in higher centres with appropriate investigation, critical care infrastructure and therapeutic facilities. Such centres are expected to have complete multidisciplinary set up (paediatric medicine and surgery, general surgery, otorhinolaryngology, respiratory medicine and oncology).

Upper GI Bleeding

Introduction

Upper GI bleeding is still having significant mortality at around 10-15% and the mortality hasn't changed much over years despite advances in intensive care, therapeutic endoscopy etc...

Upper GIT is defined as the part of gastro intestinal tract up to ligament of Treitz i.e., 2nd part of duodenum. Indian guidelines are not available. Hence ASI has taken initiatives to form guidelines in the management.

India is a vast country with very diverse facilities and infrastructures at different places varying from very basic in rural areas to very advanced state of art hospitals in major cities. Hence, generalised guidelines applicable to whole India are impossible. At the same time, major hospitals with good facilities cannot cater to huge number of Indian patients who are in villages and suburban areas. Affordability and accessibility are other factors. So, we have to divide the health care centres to different levels depending upon infrastructure and facilities available with them.

Level 1: Basic infrastructure with facilities for resuscitation, patient monitoring are available. Some areas may also have diagnostic UGI endoscopy and/or blood transfusion facilities.

Level 2: Above plus facilities for some therapeutic endoscopy, better patient monitoring with ICU, accessibility to blood component therapy, drugs like Octreotide, Terlipressin etc... are available.

Level 3: All facilities for all advanced therapeutic endoscopy, interventional radiology, major operations, high dependency ICU, services of gastroenterologists etc... are available. There is no strict separation: there may be overlap of facilities. E.g., A level 1 centre may have facility for therapeutic endoscopy but no ICU.

Surgeons should be aware of the facilities available and limitations while handling cases.

Recommendations

1. Initial resuscitation with crystalloids solutions with continuous monitoring should be done at all levels. If Hemoglobin level is less than 7 G%, blood transfusion should also be given with a view to keep Hb level between 7-9 G%. Clinical risk stratification with scoring system should be done initially so as to predict the necessity of specialised treatment plan and outcome. It should be again done after endoscopy. Low risk patients who improve rapidly with conservative management and bleeding stops could be

managed conservatively. UGI endoscopy should be done as early as possible. See C & E below.

2. Patient should be referred to higher level centres if
 - a. Patient continues to bleed
 - b. High risk
 - c. Facilities for blood transfusion and UGI endoscopy are not available
3. Patient is evaluated for requirement of intervention. Patient is managed appropriately with endoscopic procedures or surgery when indicated. Other modalities if available (like arterial embolisation, plasma coagulation, TIPS etc...) may be used when indicated (See below).
4. Surgeon should always consider risks involved, infrastructure and facilities available, his own expertise, time required to shift the patient to higher level centres etc... while managing the case. If higher level centre is far away early decision to shift is advisable.

Explanation and background knowledge

A. Symptoms

Main symptoms: Hematemesis, Hematochezia, Malena

Associated symptoms of the condition causing bleeding: pain abdomen, vomiting, jaundice, abdominal distension etc...

Symptoms of consequences: giddiness, anuria/oliguria etc..as a sequelae to severe bleeding and shock

B. Examination:

Assess the severity of bleeding. Look for tachycardia, hypotension, urine output, CVP monitoring etc...

Risk assessment: Scoring system: Rockall scoring system is simple and can be followed even in periphery. It has pre endoscopy score and post endoscopy score. If facility for endoscopy is not available, pre endoscopy score can be used.

Clinical Risk Stratification

Glasgow Blatchford score should be used pre endoscopy or where there is no endoscopy facility.

Glasgow-Blatchford Score	
Admission risk marker	Score component value
Blood Urea (mmol/L)^[5]	
6.5-8.0	2
8.0-10.0	3
10.0-25	4
>25	6
Haemoglobin (g/dL) for men	
12.0-12.9	1
10.0-11.9	3
<10.0	6
Haemoglobin (g/dL) for women	
10.0-11.9	1
<10.0	6
Systolic blood pressure (mm Hg)	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥100 (per min)	1
Presentation with melaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

In the validation group, scores of 6 or more were associated with a greater than 50% risk of needing an intervention.

Score is equal to "0" if the following are all present:

Hemoglobin level >12.9 g/dL (men) or >11.9 g/dL (women)

Systolic blood pressure >109 mm Hg

Pulse <100/minute

Blood urea nitrogen level <6.5 mg/dL

No melena or syncope

No past or present liver disease or heart failure

Initial treatment and resuscitation

- Immediate assessment of hemodynamic status should be done. If unstable, immediate initial replacement by crystalloids should be initiated later with blood and blood products as required. Close Monitoring of vitals is mandatory along with SPO2
- Use of Clinical scoring system Glasgow-Blatchford Score (GBS), Score between 0-1 need not be admitted but should be warned of rebleeding.
 - Supplemental oxygen if required
 - Pharmacotherapy should be initiated with bolus dose of Omeprazole / Pantoprazole 80 mg followed by 8 mg / hour infusion for 72 hours, followed by 40 mg BD dose. It should be stopped if the diagnosis of non ulcer bleed is established.
 - If variceal bleeding is suspected, Broad spectrum Antibiotic course should be initiated eg: Ceftriaxone combined with vasoconstrictor pharmacotherapy (Terlipressin – 2mg iv 4-6th hourly initially, followed by 1mg iv 6th hourly or somatostatin as an initial bolus of 250 g followed by a 250 g/h or Octreotide – 100mcg iv stat followed by 50mcg/hour infusion in that order of preference. They should be continued until haemostasis is achieved or for up to 5 days)
 - If patient is on Warfarin, Stop Warfarin and give Inj. Vitamin K. Prothrombin complex infusion should be considered if available.
 - Blood and Blood products transfusion to maintain hemoglobin 7 -9 G% (PRBC/Blood), PT/INR below 1.5 (Fresh Frozen plasma), Fibrinogen above 1 G/L (Fresh frozen plasma) and platelet count above 50000 (Blood/PRBC/Platelet).
 - Use of Clinical scoring system Glasgow-Blatchford Score (GBS), Score between 0-1 need not be admitted but should be warned of rebleeding.
 - If patient is on anticoagulants, they should be stopped in consultation with cardiologists/hematologists

- If patient is on antiplatelets, consider the risk of stopping the drug. Weigh risk benefit ratio, if necessary in consultation with cardiologist.
- Antiplatelets in Peptic ulcer bleeding -Decision can be taken after risk stratification based on endoscopic findings and after weighing risk benefit ratio, if necessary in consultation with cardiologist. See notes below. If given as a primary prevention, stop the drug. If given as a secondary prevention, start as early as possible along with a PPI. Low dose aspirin can be restarted after 7 days of stoppage of bleeding.
- NG or orogastric lavage is not required in patients with UGI bleeding for diagnosis, prognosis, visualization, or therapeutic effect.
- Anti H pylori treatment should be instituted in peptic ulcer bleeding.

Notes: Risk stratification for those who are on antiplatelets.

Risk Stratification*	Antiplatelets used for Primary prophylaxis**	Antiplatelets used for Secondary prophylaxis***
High risk stigmata F1a, F1b, F2a, F2b	Stop: Re-evaluate risk. Re-start after ulcer healing, if clinically indicated	If on Aspirin alone: Stop: Re-start after 3 days: Re-evaluate with endoscopy If on Dual Antiplatelets Therapy (DAPT): Continue with low dose aspirin without interruption. Early cardiologist's consultation. Re-evaluate with endoscopy
Low risk stigmata F2c, F3	Stop: Re-evaluate risk. Re-start at discharge, if clinically indicated	If on Aspirin alone: Continue If on Dual Antiplatelets Therapy (DAPT): Continue without interruption

Risk Stratification using Forrest's classification:

- I a – Active spurting
- Ib - Oozing
- IIa - Visible Vessel
- IIb - Adherent clot
- IIc - Black spot in ulcer crater
- III - Clean base ulcer

Primary Prophylaxis = Given prophylactically in patients without cardiovascular disease

Secondary prophylaxis = Given prophylactically in patients with cardiovascular disease

VI. INVESTIGATIONS

- Haemogram
- Liver Function Tests
- Blood sugar
- Blood Urea, Serum creatinine
- Bleeding time, clotting time and prothrombin time
- ECG
- USG abdomen
- Upper GI Endoscopy
- Others as required

Endoscopy

- Endoscopy should be done early (less than 12 hours) in high risk patients to get early control of bleeding. Following cases are considered high risk,
 - a. Tachycardia and hypotension continues in spite of adequate resuscitation or under treatment in hospital.
 - b. Vomits blood in hospital under treatment
 - c. There is contra indication to stop anticoagulants
 - d. Prokinetics (Metoclopramide/Erythromycin): IV prokinetic (Erythromycin 250 mg or Metoclopramide 10-20 mg IV) 30 to 90 mins before UGI endoscopy.
 - e. Timing of Endoscopy
 - i. Within 6 hours of admission if hemodynamically stable (Pulse <100 Systolic BP>100 mm) and ongoing bleeding.
 - ii. If hemodynamically stable without evidence of ongoing bleeding: Within 12 to 24 hrs is acceptable.
- Endotracheal intubation is required, if there is respiratory insufficiency, altered mental status or ongoing hematemesis.

Comprehensive Risk Stratification after endoscopy:

Rockall Score (2)

Variable[2]	Score 0	Score 1	Score 2	Score 3
Age	<60	60- 79	>80	
Shock	No shock	Pulse >100 BP >100 Systolic	SBP < 100	
Co-morbidity	Nil major		CHF, IHD, major morbidity	Renal failure, liver failure, metastatic cancer
Diagnosis	Mallory-Weiss	All other diagnosis	GI malignancy	
Evidence of bleeding	None		Blood, adherent clot, spurting vessel	

Interpretation

Total score is calculated by simple addition. A score less than 3 carries good prognosis but total score more than 8 carries high risk of mortality.

Its prediction is moderately accurate at high risk and accuracy is low when used for prediction of low risk patients.

Patients with high risk and limited facilities to treat should be referred immediately to higher centres after initial resuscitation and treatment.

Endotherapy

Therapeutic single- or double-channel endoscopes with large-diameter suction channels - allow rapid removal of fresh blood from the GI tract during endoscopy. Water pump is useful to flush out the blood/clots for better visualisation.

Upper GI bleeding can be classified into

1. Variceal bleeding
2. Non-variceal bleeding

Treatment of acute variceal bleeding

If there are facilities for therapeutic endoscopy and the treating person is having adequate expertise, following are the options:

Esophageal varices;

1. Sclerotherapy - intravariceal-less commonly used now a days.
2. Band ligation
3. Cyanoacrylate Glue therapy in select cases of active bleed

Gastric varices; Cyanoacrylate Glue therapy

If endotherapy fails then TIPS should be considered in select patients and facility is available.

If above facilities are not available, Sengstaken Blackmore tube should preferably used and transported to higher centre if bleeding continues.

Treatment of duodenal ulcer bleeding

Endotherapy for high risk group for rebleeding or active bleeding should be considered. Following are the options:

1. Injection methods (Adrenalin): Preferably should be combined with another method.
2. Thermal methods (Coagulation)
3. Mechanical methods (Hemoclip)

Notes:

- * It is preferable to use dual mode of endotherapy for Duodenal ulcer.
- * Endotherapy is not necessary in ulcers with clean base or pigmented spot.
- * If an adherent clot is there, general recommendation is that the clot should not be removed.
- * If a visible vessel is there in ulcer bed, it should be controlled by endoscopic methods, even if it is not bleeding.
- * Dieulofoy lesions: Thermal methods or hemoclips or a combination of the two can be used. If fails, therapeutic embolisation is to be done. If a facility for embolisation is not available or embolisation fails then surgery is indicated.
- * If esophageal varices are detected with bleeding stigmata or varices with no other attributable cause of bleeding, they should be treated with above methods.
- * PPI cover should be continued in all patients.
- * If patient continues bleed after initial endotherapy, second attempt should be done.
- * If second attempt also fails to arrest bleeding, other options should be considered like Surgery or arterial embolisation depending on facilities.

Immediate post-endoscopic management

Normal diet

Oral PPI once daily

Others: Warfarin – withhold for as long as safely possible to help allow the ulcer to heal. In patients requiring aspirin – low dose (75mg/day) can be started around 7 days.

Repeat endoscopy: indications

Peptic ulcer - repeat UGI Endoscopy after 6-10 weeks of acid-suppressive therapy – to confirm healing/ rule out malignancy

If H pylori present and treated – repeat evaluation to confirm eradication

Follow up

Endoscopic therapy should be repeated every 2 – 3 weeks until obliteration

First surveillance EGD performed 1 to 3 months after obliteration

Then every 6 to 12 months to check for variceal recurrence.

Mallory-Weiss tear:- Most of them settle with conservative management. Risk is low and can be treated adequately at primary care level.

Angiography and Therapeutic Embolisation:

If facilities are available for embolisation, it can be used in selective patients where 2 attempts at endoscopic therapy fail to control bleeding.

Indications for surgical intervention

All conservative and endoscopic methods have failed to arrest the bleeding.

1. Hemodynamic instability (requiring > 4-6 units of blood transfusion: Younger patients more and elderly lesser number of units of blood: decision taken more readily in older patients, because younger patients can withstand more hemodynamic instability)
2. Failure of endoscopic procedures to stop bleeding
3. Recurrent bleeding after 2 endoscopic therapy attempts
4. Shock with ongoing bleeding/recurrent bleeding
5. Continued slow bleeding requiring repeated transfusion

In these conditions, the surgeon should consider his own expertise in handling these cases and the facilities available (like ICU, Intensivist, blood transfusion facilities etc...). If in doubt, refer the patient to a centre where better facilities are available and better results can be expected. Mortality in re bleed group is 30-40%. Hence, surgeon should be aware of this risk and should have no hesitation to refer the patient to higher centres, if facilities are limited. Type of surgery depends on the etiology and is beyond the scope of this discussion.

Medico legal Issues

- Failure to detect / investigate or refer the patient in appropriate time
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action.
- Not following the guidelines, e.g., not giving blood transfusion even when Hb% is below 7G%, delaying endoscopy, keeping the patient even after 24 hours when facility for upper GI endoscopy not available etc...

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VOMITING

Introduction

Vomiting is the oral expulsion of gastrointestinal content due to gut & thoraco abdominal wall contraction. It is a complex act which is coordinated by brain stem on activation by afferent pathway & effected by motor response in gut, pharynx & somatic musculature.

Pathophysiology of vomiting

The afferent pathway includes-

1. Gastric irritants stimulate gastro-duodenal vagal afferent nerve, and bowel obstruction or mesenteric ischemia activates nasogastric afferents, thereby activating 5HT receptors.
2. Blood borne stimuli activates area postrema in medulla known as chemoreceptor trigger zone (CTZ) which activates neurotransmitters like 5HT₃, M₁, H₁ & D₂ receptors.
3. Motion sickness & inner ear disorders act on labyrinthine pathway which activate vestibular nerve, act on CTZ by muscarinic M₁ & Histaminergic H₁ receptors.

Afferent pathway signals are integrated in brain stem. The Nucleus Tractus Solitarius, Dorsal Vagal, & Phrenic nuclei in brain stem are involved in the pathway. Also, the medullary nuclei which regulate respiration & nuclei controlling pharyngeal, facial & tongue movement are also involved in causing vomiting.

Signal from brain stem is relayed to efferent motor pathway. This consists of both autonomic & somatic systems. This includes phrenic nerve to diaphragm, spinal nerve to abdominal & intercostal muscle, autonomic fibres to gut, viscera & voluntary muscle of pharynx & larynx. Integrated co-ordination of these finally result in intestinal contents being expelled from mouth as vomitus.

Causes of vomiting

Surgical:

Abdominal

- Inflammatory
 - Cholecystitis
 - Pancreatitis
 - Appendicitis
 - Perforation Peritonitis

- Duodenal ulcer
- Obstructing disorders
 1. Gastric outlet obstruction
 - a. Pyloric stenosis
 - b. Gastric malignancy
 - c. Duodenal malignancy
 2. Small bowel obstruction
 3. Large bowel obstruction
 - a. Volvulus
 - b. Intussusception
 - c. Benign Lesion
 - d. Malignant Lesion
 - e. Adhesion-----rare in the large bowel
 4. Superior mesenteric artery syndrome
- Biliary colic
- Altered sensorimotor function
 1. Intestinal pseudo obstruction
 2. Gastro oesophageal reflux
- Postoperative vomiting
- Renal causes
 - Ureteric colic

Extraabdominal Causes: Raised intracranial Pressure

Causes of vomiting in new born

1. Atresia & stenosis – Duodenal / Jejunal / ileal Colonic atresia
2. Mid gut volvulus
3. Meconium ileus
4. Hirschprung's disease
5. Imperforate anus- rare symptom unless neglected
6. Necrotising enterocolitis
7. Incarcerated inguinal hernia

Medical

- Labyrinthine disease
 1. Motion sickness
 2. Labyrinthitis
 3. Malignancy

- Intracerebral disorder
 1. Hydrocephalus
 2. Haemorrhage
 3. Malignancy
 4. Abscess
- Cardiopulmonary disease
 1. Myocardial infarction
 2. Cardiomyopathy
- Psychiatric illness
 1. Anorexia
 2. Bulimia nervosa
 3. Depression
- Drugs
 1. Cancer chemotherapy
 2. Antibiotics
 3. Cardiac antiarrhythmic
 4. Digoxin
 5. Oral hypoglycemics
 6. Oral contraceptives
- Metabolic disease
 1. Pregnancy
 2. Uremia
 3. Ketoacidosis
 4. Thyroid & parathyroid disease
 5. Adrenal insufficiency
- Toxins
 1. Liver failure
 2. Ethanol
- Enteric infections(food poisoning)
 1. Bacterial
 2. Viral
- Altered sensorimotor function
 1. Chronic nausea vomiting syndrome
 2. Cyclic vomiting syndrome
 3. Cannabinoid hyperemesis syndrome
 4. Rumination syndrome
- Antidepressants
 1. Parkinson's therapies

2. Smoking cessation agents

Management of vomiting

The main aim of management depends upon-

- a. Assessment of severity of the condition
- b. Correct hypovolemia, electrolyte imbalance and ketosis
- c. Provide symptomatic relief to break the cycle of vomiting and prevent further vomiting.
- d. Treatment of specific cause

8. Assessment of severity of the condition

- When a patient presents in A&E, the first aim is to assess the severity of dehydration that the patient has incurred due to vomiting and start resuscitation simultaneously.

Mild dehydration	Moderate dehydration	Severe dehydration
May have no symptoms Mild thirst Concentrated Urine	Significant thirst Oliguria Sunken eyes Dry mucous membranes Weakness Postural hypotension (>20mmHg drop in systolic BP/>30mmHg in Hypertensives/ 10mmHg drop in diastolic BP after 3 mts of standing)	Significant thirst Tachycardia Low pulse volume Cool extremities Reduced skin turgor Marked hypotension Confusion Oliguria/Anuria

- Once severity of dehydration has been assessed, fluid resuscitation needs to start as mentioned later in the section.
- Thereafter, history of the present illness needs to be taken. History consists of number of episodes, character of vomiting, content of vomitus and associated symptoms to point towards the pathology. Vomitus can be feculent in cases of intestinal obstruction, bilious in cases of acute cholecystitis, biliary colic. Vomitus can be semi digested food particle in case of gastric outlet obstruction, ureteric or renal colic and can be watery in cases vertigo.
- Associated symptoms like pain abdomen, chest pain, dizziness, obstipation will help in diagnosis & further management. General & systemic examination will further guide about the causes of vomiting. Systemic examination consists of CNS to rule out vestibular disorder, intra-cerebral causes, motion sickness, meningitis etc. Examination of abdomen done to rule out causes like peritonitis, cholecystitis, acute appendicitis, ureteric or renal colic, obstructed or strangulated hernia.

- The investigations to be sent are –
 - Blood- CBC, Serum electrolytes, Serum Amylase and Lipase, LFT, Serum Creatinine, Blood urea, CRP, ABG.
 - Total leucocyte count raised in acute cholecystitis, acute appendicitis
 - Electrolyte disturbances and renal impairment may result from excessive fluid losses and may be especially important in older patients.
 - Radiological Investigations – Plain X-ray abdomen & chest, Ultrasonography of abdomen and pelvis in the emergency. Following these, if patient persists to have vomiting, further investigation with CECT / MRI abdomen and pelvis can be done.
 - X ray Abdomen helps to rule out intestinal obstruction & perforation.
 - Urgent ultra sound abdomen to rule out acute cholecystitis, acute appendicitis, renal colic, pancreatitis, etc.
 - Endoscopic evaluation – can be done in persistent vomiting not responding to initial management and where there is suspected mechanical obstruction in the upper GI system.

9. Correct hypovolemia, electrolyte imbalance

▪ Fluid management

- For both mild and moderate dehydration consider a trial of oral rehydration combined with an anti-emetic. Severe dehydration is the result of large fluid losses and may be complicated by electrolyte and acid base disturbances which require treatment and observation over a prolonged period.
- If dehydration subsides continue with ORS and clear fluids. Avoid solid food until vomiting episode has passed.
- Guide to Urine output monitoring
 - Normal urine output - 800-2000ml in 24 hrs (1-2ml/kg/hr)
 - Oliguria - <400ml-500ml in 24 hrs (0.5ml/kg/hr) – Adult
 - <1ml/kg/hr -- infant
 - <0.5ml/kg/hr – children
 - Anuria - <100ml in 24hr
- Resuscitation of severe dehydration
 - 100ml/kg in 3.5hrs Ringer Lactate (Normal Saline if RL not available) divided into 30ml/kg in 1hr followed by 70ml/kg in next 2.5hrs.
 - Maintenance fluid is given 30-40ml/kg in 24hrs Intravenous fluids continued till signs of dehydration and vomiting subsides.
- In all cases of intravenous fluid replacement, details of fluid balance should be recorded. Observation and reassessment of hydration status at regular intervals will allow calculation of fluid volume requirements and reduce risks of fluid overload.

10. Provide symptomatic relief to break the cycle of vomiting and prevent further vomiting

- **Role of naso gastric tube**

NG tube is used in intestinal obstruction, gastric outlet obstruction, persistent vomiting e.g severe acute pancreatitis with persistent ileus & gastric distension. NG tube should be placed till vomiting subsides or intestinal obstruction is relieved. Early enteral feeding should be the aim and regular assessment for scope of NG tube removal should be done.

- **Pharmacotherapy**

Along with the fluid management and NG tube, there may be need for pharmacotherapy directed at specific causes as mentioned in the table below.

Postoperative vomiting	Ondansetron(5HT3 antagonist) Granisetron (5HT3 antagonist)	4–8 mg q4/8 hrs oral or i.v 1–2 mg q24 hrs o/iv/tderm.
Toxin induced vomiting	Prochlorperazine (antidopaminergic) Thiethylperazine (antidopaminergic)	5/10 mg q6/8 hrs iv/im/oral 10mgq6-8hrs oral, im, rectal
Metabolic vomiting	Prochlorperazine (antidopaminergic) Thiethylperazine (antidopaminergic)	5/10 mg q6/8 hrs iv/im/oral 10mg q6-8hrs oral,i.m,rectal
Drugs induced vomiting	Prochlorperazine (antidopaminergic) Thiethylperazine (antidopaminergic)	5/10 mg q6/8 hrs iv/im/ oral 10mg q6-8hrs oral/im/rectal
Motion sickness	Dimenhydrinate(antihistaminergic) Meclizine (antihistaminergic)	25/50 mg q6/8 hrs oral/iv/ im 25–50 mg q24 hours oral
Vestibular disfunction	Dimenhydrinate(antihistaminergic) Meclizine (antihistaminergic)	25/50 mg q68hrs oral/iv/im 25/50 mg q24 hours oral
Cyclic vomiting syndrome	Amitriptylin(tricyclic antidepressant) Nortriptylin (tricyclic antidepressant)	1mg/kg/day titrated to oral 10-25mg daily oral
Chemo & Radiotherapy induced vomiting	Ondansetron (5HT3 Antagonist) Granisetron(5HT3Antagonist) Aprepitant(NK1Antagonist) Dexamethasone(corticosteroids)	4–8 mg q4–8 hrs oral /iv 1/2 mg q24 hrs o/iv/trans dermal 80/125 mg q24 hours oral 4/8 mg q4/6 hrs oral/im/iv
Gastroparesis / intestinal obstruction	Metoclopramide Domperidone	10/20 mg q6/8 hrs oral/im/iv 10 mg q8/24 hours oral

Treatment of specific cause

Once the acute management for vomiting has been done and the patient stabilized, the treatment of specific surgical cause should be the aim depending on need in emergency, semi-emergency or elective situation.

Cholecystitis	Cholecystectomy
Appendicitis	Appendicectomy
Perforation	Omental patch repair as emergency procedure
Pancreatitis	Conservative management
Pyloric stenosis	
Duodenal /jejunal / ileal/ colonic atresia Hirschprung's disease	Resection & anastomosis
Meconium ileus	Bishop-koop operation
Imperforate anus	Anoplasty
Gastric malignancy	Total gastrectomy / Subtotal gastrectomy
Duodenal obstruction & periampullary carcinoma	Pancreatico duodenectomy(whipple's) gastro jejunostomy
Adhesion	Initial conservative management for 72 hrs if not relived then surgery (release of adhesion)
Volvulus	Derotation of rotated segment
Intussusception	Hydrostatic reduction using warm saline or barium sulphate solution under fluoroscopy Operative laparotomy +/- resection & anastomosis

Who does what?

Doctor:

- Surgeon: Diagnosis & Work up
 Pre operative planning
 Operative procedure if needed
 Post operative care in conjunction with Anaesthetist/Intensivist
 Post operative follow up
- Anesthetist: Pre Anesthesia Check up
 Part of resuscitation
 Delivering anesthesia
 Post op ICU management in conjunction with Surgeon
- Nurse: Pre/Intra/Post-op comprehensive care
 Dressing of the wound
- Technician: Pre op equipment and drugs to be checked and kept ready
 Assist anesthetist in the OT
 Assist the surgeon, positioning of the patient

Resources required for one patient / procedure (patient weight 60 kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer /Assistant Surgeon – 1
3. Anesthetist – 1
4. Pathologist – 1---- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

- i) Hemogram
- ii) Blood Sugar
- iii) Renal Function Test in selected cases
- iv) LFT in selected cases
- v) S. Electrolytes in selected cases
- vi) USG in selected cases
- vii) ECG
- viii) X-Ray – Chest
- ix) Histopathology
- x) CT Scan if available

Drugs & Consumables

- b. OT Table & lights
- c. Instrument trolley
- d. Anesthetic Machine, instruments including endotracheal tubes &drugs
- e. Patient Monitor
- f. Set of surgical Instruments
- g. Suction
- h. Sutures
- i. Drains
- j. Catheters
- k. Cautery – a basic set
- l. Antibiotics
- m. Analgesic
- n. I.V. Fluids

- o. Blood products
- p. Dressings

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