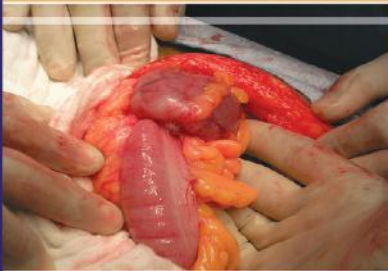


S Devaji Rao

GENERAL SURGICAL EMERGENCIES



JAYPEE

General Surgical Emergencies

General Surgical Emergencies

S Devaji Rao MS MNAMS FICS

Teaching Faculty

National Board of Examinations

Senior Consultant in General Surgery

Surgical Gastroenterology and Surgical Oncology

St Isabel's Hospital, Mylapore, Chennai, Tamil Nadu, India

Chennai Meenakshi Multispecialty Hospital Ltd, Chennai, Tamil Nadu, India



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Jaypee Brothers Medical Publishers (P) Ltd.

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd.
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
Email: jaypee@jaypeebrothers.com

Overseas Offices

J.P. Medical Ltd.
83 Victoria Street, London
SW1H 0HW (UK)
Phone: +44-2031708910
Fax: +02-03-0086180
Email: info@jpmedpub.com

Jaypee-Highlights Medical Publishers Inc.
City of Knowledge, Bld. 237, Clayton
Panama City, Panama
Phone: +507-301-0496
Fax: +507-301-0499
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Jaypee Brothers Medical Publishers (P) Ltd.
17/1-B Babar Road, Block-B, Shaymali
Mohammadpur, Dhaka-1207
Bangladesh
Mobile: +08801912003485
Email: jaypeedhaka@gmail.com

Jaypee Brothers Medical Publishers (P) Ltd.
Shorakhute, Kathmandu
Nepal
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Email: jaypee.nepal@gmail.com

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Printed at

Dedicated to

My teachers

My parents

Uma Bai and Siva Rao

My beloved wife

Kalpana

My daughters

Bhavna and Kirthana

Preface

Many textbooks are available dealing with the details of emergency surgical conditions. Not many are available dealing exclusively with the emergency conditions, especially those related to surgery. Not that every patient coming to the casualty at odd hours with an acute symptom has a life-threatening emergency, but it becomes necessary to comfort the patient at the first instant, followed by a quick diagnosis of the clinical condition. It is also imperative that the impending emergency should be identified by the clinician, so that a catastrophe is avoided. The classical example of such confusing situation is the acute pancreatitis. This can present with a variety of symptoms which will not give a lead towards diagnosis, but the whole clinical picture establishes after some time, and takes the patient through a very tough morbid situation, and may also end in death.

This concise book has been designed in such a way that the common emergency conditions are detailed to the extent so that surgical students or the casualty doctors or young surgeons may not miss the diagnosis. It cannot replace the many well-established textbooks, but it gives sufficient information for a clinician to manage the emergency.

Color photographs are useful add-ons to these chapters, which will make the reader remember the information for a long time.

Every new doctor who is resident in the casualty and the intensive care unit will face with a variety of clinical problems, and I hope the handbook will come handy.

I also hope that I have hit the required details at the right level for the young surgeons.

S Devaji Rao

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My special thanks to my daughters Dr Bhavna for going through the script and making corrections, and Dr Kirthana for drawing the diagrams.

My very special thanks goes to my wife Kalpana for the tolerance during the preparation of this manual.

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Section I

Introduction

1. Introduction

Introduction

Emergencies are defined as the clinical situations which require urgent care. Not necessarily they should have a life-threatening pathology, but even a simple innocuous pathology can create an emergency situation, such as a passing calculus in the ureter can cause such a pain which will upset the patient, the relatives and friends, and warrant emergency care and also confuse the attending doctor till he makes a final diagnosis.

Clinical diagnosis of such emergencies may be difficult in some patients forcing the clinician to run through all the investigations to make the final diagnosis. A well informed medical student or a young surgeon will take it right, will not upset himself, comfort the patient and go through the clinical examination and administer the right treatment.

In some emergencies like hemorrhagic conditions, history taking cannot be done in detail due to paucity of time, and the treatment should get initiated at the earliest, probably in the intensive care unit. Failure to recognize and manage such critical illnesses, may lead to pathophysiological changes leading to multiple organ dysfunction. In such a situation, intensive care plays a very important role. The role of intensive care includes:

- Resuscitation and stabilization
- Optimization to prevent organ failure
- Decision regarding complex surgery
- Support of failing organs
- Recognition of futility.

In well developed hospitals, the critical care is managed at two levels, namely, intensive care units (ICU) and high dependency units (HDU), only the availability of ventilators in the ICU differentiating the both. In the ICU, some patients may require one patient – one nurse combination.

Critically ill patients can be classified according to the level of medical and nursing care needed.

- Level 0: Patients who can be managed by ward based care
 - Level 1: Patients who can be managed by ward based care with advise or support from the critical care team
 - Level 2: Patients requiring more advanced levels of monitoring or intervention, such as management of single failing organ system
 - Level 3: Patients requiring advanced respiratory support and management of multiple organ failure.
- ◆ For a good outcome, the first step is to make a proper diagnosis, followed by the necessary investigations and sensible management.

Section II

Assessment

2. Assessment of Surgical Emergencies

Assessment of Surgical Emergencies

As soon as the patient is wheeled into the hospital or the casualty, it is required to determine whether the patient's condition warrants emergency care or not. Though there are parameters which help to determine this, many times, experience matters. For example, a patient with acute pancreatitis may not look very sick after an episode of acute abdominal pain. The following measures will help determine the seriousness of the patient.

1. Make the patient lie down comfortably.
2. Elicit a quick history.
3. Make a thorough clinical examination.
4. Come to a quick working clinical diagnosis.
5. Ask for essential meaningful investigations.
6. Admit the patient where thought to be required.
7. Put the patient in intensive care whenever required.
8. Collect investigation reports at the earliest.
9. Start emergency treatment.
10. Ask for expert opinions.

MAKE THE PATIENT LIE DOWN COMFORTABLY

It is necessary to make the patient lie down, as a walk in patient may collapse suddenly (e.g. septic shock, hemorrhagic shock).

ELICIT A QUICK HISTORY

A good history will establish the diagnosis in majority of cases.

However, when the patient is too sick, a quick questioning is only possible. They are:

- A – allergies
- M – medications
- P – previous medical history, pregnancy
- L – Last meal
- E – Events leading to admission.

If possible, and if there is enough time, full history should be elicited in the following:

- Chief complaint
- History of present illness
- Previous history of present complaint
- Past medical history
- History of drug intake and allergies
- Social and personal history
- Family history
- History of immunization.

Make a Thorough Clinical Examination

General Examination

- Skin
 - ❖ Pallor (e.g. anemia)
 - ❖ Yellow discoloration (e.g. jaundice)
 - ❖ Bluish discoloration (e.g. cyanosis)
 - ❖ Pigmentation (e.g. von Recklinghausen's disease)
 - ❖ Eruptions (e.g. macule, papule, vesicle, bulla, etc.)
 - ❖ Nodules (e.g. Heberden's nodes of osteoarthritis)
 - ❖ Visible veins (e.g. visible neck veins of cardiac overload, caput medusae of portal hypertension).
- Eyes
 - ❖ Orbital region
 - Prominence of eyeballs (e.g. exophthalmos of thyrotoxicosis)
 - Sunken eyeballs (e.g. dehydration)

- ❖ Eyelids
 - Puffiness (e.g. trauma, renal pathology)
 - Color (e.g. black eye of head injuries)
- ❖ Conjunctivae
 - Color (e.g. pale in anemics)
- ❖ Sclera
 - Color (e.g. yellow in jaundiced patients)
- ❖ Cornea and iris
 - Opacities and ulcers (e.g. trauma, infections)
- ❖ Pupils
 - Size, shape, symmetry and reaction to light (e.g. changes in cranial nerve lesions)
- Oral cavity
 - ❖ Lips
 - Color and texture (e.g. cracked lips of exposure to extreme cold)
 - Pigmentation (e.g. telangiectasia)
 - ❖ Gums
 - Color (e.g. blue line of lead poisoning)
 - Texture (e.g. inflammation due to gingivitis)
 - Pigmentation (e.g. hemorrhages due to leukemia)
 - ❖ Teeth
 - Color and pigmentation (e.g. mottled yellow color of fluorosis)
 - Number and health status of the teeth (e.g. Hutchinson's teeth of congenital syphilis)
 - ❖ Tongue
 - Color (e.g. blue color of central cyanosis)
 - Texture (e.g. smooth tongue of vitamin B₁₂ deficiency)
 - Ulcer (e.g. aphthous ulcers)
 - Altered movements (e.g. hypoglossal nerve palsy)
 - ❖ Hard palate and soft palate
 - Color (e.g. anemia)
 - Cleft (e.g. cleft palate)
 - Ulcer (e.g. malignancy).

Halitosis (Bad breath) may be due to poor oral hygiene or even oral pathologies, e.g. oral cancers or bronchopulmonary pathologies like lung abscesses.

- Hands and feet
 - ❖ Hands of the patient should be examined carefully, like
 - Shape of the hands (e.g. characteristic shape of Dupuytren's contracture, tetany)
 - State of the joints (e.g. deformed joints of rheumatoid arthritis)
 - Shape and color and deformity fingers (e.g. clubbing or nicotine staining of chronic smokers)
 - Nails (e.g. koilonychia of iron-deficiency anemia)
 - Abnormal movements of fingers (e.g. tremors of thyrotoxicosis)
 - ❖ Feet of the patient are generally tucked under the bedclothes and their examination should not be forgotten. Examination may reveal
 - Edema, unilateral (e.g. filarial leg) and bilateral (e.g. hypoproteinemia)
 - Skin changes (e.g. pigmentation or ulcers due to ischemia)
 - Ulcers (e.g. trophic ulcers)
- Look for (jaundice, anemia, cyanosis, clubbing, oedema, lymphadenopathy – JACCOL)
- Vitals (pulse, respiration, blood pressure)
- Peripheral examination (warm/cool, capillary refill)
- Jugular venous pressure
- Mental test score or GCS (Glasgow Coma Scale), if the patient appears confused or impaired consciousness).

Systemic Examination

- Site of interest
- Other systems.

Come to a Quick Working Clinical Diagnosis

- A quick working clinical diagnosis should come to the mind of the clinical examiner
- He should also keep the various differential diagnosis in mind, so that diagnosis can be revised at anytime, without bias.

Ask for Essential Meaningful Investigations

- It is essential to ask only for meaningful investigations
- If investigation packages are available in the hospital, it is worthwhile to ask for it which may include the required investigations also
- If asking for this package will delay the results, it is appropriate to ask for the relevant ones first, followed by the rest.

The blood tests in general should include:

- Complete blood count (CBC)
- Urea, creatinine and electrolytes
- Blood sugar (random)
- Serum amylase, lipase
- Liver function tests
- Coagulation profile
- Blood grouping and Rh typing
- Arterial blood gas analysis (ABG).

The radiological tests should include:

- Chest X-ray (erect)
- Abdominal X-ray (supine and erect)
- Ultrasound scan
- CT.

Admit the Patient where Thought to be Required

No time should be wasted in deciding where to admit the patient. The best bed available is the best choice.

Put the Patient in Intensive Care Whenever Required

When in doubt about the seriousness, admit the patient in the intensive care.

Collect Investigation Reports at the Earliest

In the commotion during the emergency, it is not unusual to forget to collect the reports. The clinician should stay composed, and should remember to collect all the reports, which will help in the management.

Start Emergency Treatment

- It is always wise to start an intravenous line for control of lifeline. The choice of fluid depends on the situation (e.g. normal saline in diabetics, glucose solutions in hypertensives, plasma expanders in shocked patients)
- Antibiotics may be needed if infective pathology is suspected
- Oxygen by nasal mask is a safe additive
- Urethral catheterization and hourly output determination is necessary.

- Start emergency treatment if required even in the casualty, and history taking can be followed later
- While examining a patient in emergency, site of interest should be examined first to save time so that appropriate treatment is started.

Ask for Expert Opinions

- It is always necessary to obtain expert opinions to manage the situation
- Multiple opinions may be necessary, but it should not hurt the sentiments of the experts involved
- Intravenous fluids (rate and nature) and antibiotics will vary according to the laboratory reports and revised diagnosis
- Colloids may be necessary and should be adequately given at appropriate times
- The average daily water requirements for adult are 30 to 35 ml/kg + 500 ml/day/degree of pyrexia (above 37°C)
- Normal adult requirements for electrolytes are 1 mmol/kg each of Na, K, Cl
- Normal fluid requirements for children depend on weight
 - ❖ <10 kg : 100 ml/kg/day
 - ❖ 10 to 20 kg : 1 liter + 50 ml/kg/day
 - ❖ >20 kg : 1.5 liters + 25 ml/kg/day.

Section III

Critical Care

3. Critically Ill Patient and Critical Care
4. Shock
5. Acute Respiratory Distress Syndrome

Critically Ill Patient and Critical Care

A critically ill patient requires high level monitoring, especially when circulatory/respiratory support is required, or if more than one organ is failing. The intensive care is started early enough to be able to reverse the condition.

CARDIAC SUPPORT

It depends on the Hb, perfusion pressure, ventilation and gas exchange. For this to be achieved, the following are required:

- Arterial lines (beat to beat BP indication, easy access for arterial blood sampling)
- Central venous pressure monitoring (measures intravascular volume, and the right heart preload)
- Pulmonary artery catheterization (measures cardiac output, ensures optimal fluid resuscitation and helps in the use of vasoactive drugs).

CVP monitoring indicates only the right heart filling or preload, is not a reliable measure of left ventricular preload. Pulmonary artery catheterization is useful for monitoring the left ventricular filling.

RESPIRATORY SUPPORT

Respiratory support is needed when respiratory failure occurs, due to inadequate exchange of oxygen or CO_2 to meet metabolic needs, which is determined by the lack of improvement with oxygen therapy or the patient is tiring with an increasing pCO_2 . The methods by which respiratory support can be given are:

- Continuous positive airways pressure (CPAP) in a spontaneously breathing patient. This is indicated when:
 - ❖ The patient is tiring with rising $p\text{CO}_2$
 - ❖ The patient is unable to maintain their own airway
 - ❖ Noninvasive ventilation is contraindicated
 - ❖ Need for endotracheal suction
- Noninvasive positive pressure ventilation (NIPPV) is an alternative via a face mask.

INOTROPIC SUPPORT

This is needed when the patient is not able to maintain the blood pressure and urine output, by normal crystalloid support. Different inotropic agents are used in various circumstances:

- Noradrenaline: Dose 0.01 to 0.4 mcg/kg/min IV infusion — acts on α_1 -receptor causing vasoconstriction, in sepsis increases the renal blood flow and enhance urine production
- Adrenaline: Dose 0.01 to 0.30 mcg/kg/min IV infusion
 - ❖ In low doses: Acts on β -receptors (causes increase in heart rate and contraction)
 - ❖ In high doses: Acts on α -receptors (causes increased peripheral resistance)
- Dobutamine: Dose 1 to 25 mcg/kg/min via central vein—predominantly acts on β_1 (increases the heart rate and force of contraction) and also on β_2 - and α_1 -receptors (decreases peripheral and pulmonary vascular resistance)
- Dopamine: Acts directly on α , β and dopaminergic receptors and indirectly by releasing noradrenaline
 - ❖ Low dose: 0.5 to 2.5 mcg/kg/min (renal dose—increases renal and mesenteric blood flow. \uparrow renal flow results in \uparrow GFR and \uparrow renal sodium excretion)
 - ❖ Moderate dose: 2.5 to 10 mcg/kg/min (cardiac dose—stimulates β_1 -receptors causing \uparrow myocardial contractility, stroke volume and cardiac output)
 - ❖ High dose: >10 mcg/kg/min (cardiac plus—stimulates α -receptors causing \uparrow SVR, \downarrow renal blood flow and \uparrow potential for arrhythmias)
- Dopexamine: Dose 0.25 to 0.5 mcg/kg/min—a synthetic analog of dopamine with β_1 activity with no α activity.

Shock

Shock is defined as inadequate organ perfusion and tissue oxygenation.

It is classified as:

- Hypovolemic shock
- Septic shock
- Anaphylactic shock
- Cardiogenic shock
- Neurogenic shock.

HYPOVOLEMIC SHOCK

This occurs due to loss of intravascular volume (blood or fluid), which results in activation of sympathetic nervous system, which causes tachycardia and vasoconstriction of skin, muscle and GI system, so that blood is directed to vital organs, e.g. brain, heart, etc.

The vasoconstriction of renal and splanchnic circulation causes renal failure, GI sloughing and hemorrhage. When shock persists, the perfusion of brain suffers causing confusion and aggression. The hyperventilation causes respiratory alkalosis, which is overtaken by metabolic acidosis due to poor tissue perfusion and anaerobic metabolism. The hypovolemic shock is classified depending on various factors (Table 4.1).

Table 4.1: Classification of hypovolemic shock

Class	Blood loss	Pulse rate (per min)	Blood pressure	Urine output (ml/hr)	Resp rate (per min)	Consciousness level	Treatment
I	<15% (up to 750 ml)	60–100	No change	>30	<20	No change	Crystalloid/colloid
II	15–30% (750 – 1500 ml)	>100	Pulse pressure reduced	20–30	20–30	Anxious	Crystalloid/colloid
III	30–40% (1500 – 2000 ml)	>120	BP fall	5–15	30–40	Confused	Blood
IV	>40% (>2000 ml)	>140	BP significant fall	Anuria	>40	Lethargic	Blood

Treatment of Hypovolemic Shock

- Control of hemorrhage
- Restoration of fluid volume.

- A fluid challenge of 1 to 2 liters should be given for adults and 20 ml/kg for children
- Rapid restoration of pulse and BP indicates a loss of <20 percent
- If the response is transient in spite of resuscitation, it is better to intervene surgically.

SEPTIC SHOCK

Shock occurring due to severe sepsis called systemic inflammatory response syndrome (SIRS).

Symptoms and Signs

- Heart rate (>90/min)
- Temperature (>38 or 36)
- Respiratory rate (>20/min)
- Leukocyte count (>12000/cmm)

Note: In the absence of infection, SIRS can be said to exist when two of the above criteria.

Pathogenesis

This results due to severe infection and inflammation, mediated by acute phase cytokines, which have a generalized effect distant to the site of original insult. Leukocytes adhere to endothelial cells via adhesion molecules leading to changes in vascular permeability and edema.

Treatment

- Resuscitation
- Identification of the source of sepsis
- Treatment of focus of sepsis (antibiotics/drainage of pus)
- Critical care management
 - ❖ Fluids
 - ❖ Oxygen administration
 - ❖ Vasopressors
 - ❖ Steroids
 - ❖ Activated protein C
 - ❖ Hemofiltration.

ANAPHYLACTIC SHOCK

Incidence and Etiology

This is a hypersensitivity reaction.

The usual causes are:

- Drugs
- Blood transfusion
- Radiological contrast.

Pathogenesis

It is mediated by immunoglobulin E (IgE).

Symptoms and Signs

- Urticarial rash
- Wheeze due to bronchospasm
- Tachycardia
- Fainting
- Rhonchi
- Hypotension.

- Stop the cause if found
- Airway management and oxygen
- IV access and fluids
- Adrenaline (1 ml in 1:1000 IM)
- Antihistamine (IV)
- Bronchodilators.

CARDIOGENIC SHOCK

Occurs due to pump failure, commonly after myocardial infarction.

NEUROGENIC SHOCK

Occurs after spinal cord injury.

Pathogenesis

Occurs due to disruption of autonomic nervous system control over vasoconstriction, which results in a decrease in peripheral vascular resistance and blood pressure, with resultant bradycardia. Temperature control may also be lost.

Treatment

- Ventilation
- Fluid management
- Inotrope support.

Acute Respiratory Distress Syndrome

Incidence and Etiology

- Acute respiratory distress syndrome (ARDS) indicates the acute diffuse pulmonary inflammatory response to either direct or indirect insults from extrapulmonary pathology
 - ❖ Direct—via airway or injury to chest (e.g. aspiration, toxic gases, pneumonia)
 - ❖ Indirect—blood-borne insults (e.g. sepsis, polytrauma, severe burns, drugs)
- Frequently associated with multiple organ (kidney, liver, intestines) dysfunction.

Pathogenesis

- Pathogenesis is complex
- There is transudation of fluid in the lungs, thickening of alveolar capillaries resulting in ventilation/perfusion mismatch. The pulmonary artery wedge pressure remains normal, distinguishing it from pulmonary edema.

Clinical Presentation

They are not specific, but share many things with other pulmonary pathologies.

Relevant Investigations

- Blood gas analysis (PaO_2 / FiO_2 of less than 200 mm Hg)
- Chest X-ray shows bilateral diffuse infiltrates
- Pulmonary artery wedge pressure (less than 15 mm Hg).

22 Treatment

Current treatment is supportive and no specific therapy exists to modulate the sequence of events of ARDS:

- Monitoring
 - ❖ Monitoring of all vitals
- Ventilatory management
 - ❖ Mechanical ventilation to permit adequate oxygen uptake
- Nonventilatory management
 - ❖ Treatment of underlying risk factors
 - ❖ Enteral feeding
 - ❖ Maintenance of hemodynamic stability and cardiac output.

Section IV

Trauma

6. Polytrauma
7. Head Injuries
8. Facial Injuries
9. Spine and Spinal Cord Injuries
10. Thoracic Injuries
11. Abdominal Injuries
12. Urological Injuries
13. Male Genital Injuries
14. Female Genital Injuries
15. Hand Injuries

Polytrauma

INTRODUCTION

Management of a patient with polytrauma depends on proper and systematic clinical evaluation, which identifies immediate and potentially life-threatening conditions before the limb threatening ones, but does not omit the latter. This usually starts with a call from ambulance control.

The first couple of hours following injury are critical, as the patient is very vulnerable. This critical time period is called 'golden hour', which is usually spent in the place of accident or in the emergency department, making this period extremely crucial.

The trauma life support program consists of:

- Preparation
- Primary survey and resuscitation
- Secondary survey
- Continuous monitoring and evaluation.

PREPARATION

Prehospital Communication

A warning from the ambulance control ideally from the scene of accident provides essential information so that receiving personnel are ready to receive the patient of trauma, which provides assessment and treatment without delay. The essential prehospital information are:

- Nature of injury
- Mechanism of injury

- Number, age and sex of the casualties
- Consciousness level of the casualties
- Airway, breathing and circulatory status of casualties
- Treatment provided
- Estimated time of arrival
- Treatment options required.

TRAUMA RECEPTION TEAM

The make-up of this team varies between hospitals depending upon the resources and the time of the day. The trauma team should consist of:

- Trained medical personnel
- Supportive trained paramedical personnel
- A team leader who should assign specific tasks to each person.

The trauma reception team undertakes the following tasks:

Team Leader

- Coordinates the tasks assigned to the team members
- Questions ambulance personnel
- Assimilates clinical findings
- Determines investigations in order of priority
- Liaises with relatives and provide information to trauma team
- Liaises with specialists who are called.

Team Members

- Manage the airway
- Clear the secretions and intubate if necessary
- Manage circulation
- Establish infusion line quickly
- Takes blood for investigations
- Supportive measures
- Connect to monitors
- Urinary catheterization
- Jugular vein catheterization where necessary.

Receiving the Patient

Receiving and transferring the patient (Fig. 6.1) is very crucial and requires five people to do the job, which should be a well practiced procedure in order to protect the spinal cord if it is intact, and to prevent further injury if it is already compromised. During the transfer, the patient's head and neck are stabilized by one member of the team, three others lift from the side and the fifth member replaces the ambulance trolley with the resuscitation trolley.

Primary Survey and Resuscitation

The activities listed below are carried out simultaneously if there are enough personnel, if not should be done in alphabetical order (ABCDE).

- Airway and cervical spine control
- Breathing
- Circulation and hemorrhage control
- Disability
- Exposure.

Airway and Cervical Spine Control

A cervical spine injury should be assumed if the patient has been the victim of significant blunt trauma or if the mechanism of injury indicates that the cervical region may have been damaged. One member of the team needs manually to immobilize the cervical spine while talking to the patient which also assesses the airway.



FIG. 6.1: Patient with polytrauma

- If the patient is able to give a logical answer in a normal voice, the airway is assumed to be patent and the brain adequately perfused.
- If the patient gives an impaired or fails to reply, the airway could be obstructed, and immediate measures should be taken:
 - ❖ A simple chin lift will help in relieving the obstructing soft tissue usually the tongue
 - ❖ Saliva, blood, vomitus, tooth or other foreign bodies should be removed
 - ❖ Suction through rigid suction tube is necessary to remove secretions
 - ❖ In patients who vomit and regurgitate, head end of the bed should be dropped 20 degrees, allowing the secretions to drip down and facilitate removal by suction
 - ❖ A nasogastric tube may be inserted to aspirate the stomach contents and prevent further vomiting.

When it is established that the airway is clear and patent, 100 percent oxygen is provided via mask or by endotracheal intubation. Pulse oxymeter is connected to maintain good SaO₂, if needed with ventilatory support.

The neck should be examined for the following five signs which could indicate the presence of immediately life-threatening thoracic conditions (Table 6.1).

When below given signs in Table 6.1 are checked, the neck may be immobilized with appropriate collar if the patient is not restless, in a restless patient, semi-rigid collar is accepted.

All the multiple injured patients, particularly those who have injuries above the clavicle or a change in level of consciousness, should be treated as though they have a cervical spine injury, until it is ruled out.

Breathing

The clinical examination of chest consists of:

- Inspection
 - ❖ Marks and wounds
 - ❖ Respiratory rate
 - ❖ Inspiratory effort
 - ❖ Symmetry of chest movements

Table 6.1: Signs of life-threatening thoracic conditions

	<i>Signs</i>	<i>Conditions</i>
1.	Swellings and wounds	Vascular and airway injury
2.	Distended neck veins	Cardiac tamponade, tension pneumothorax
3.	Tracheal deviation	Tension pneumothorax
4.	Subcutaneous emphysema	Pneumomediastinum
5.	Laryngeal crepitus	Fracture of laryngeal cartilage

- Percussion
 - ❖ Assess ventilation at the periphery
- Auscultation
 - ❖ Assess defects in air movement.

The respiratory rate and effort are sensitive indicators of underlying lung pathology, and requires to be monitored and recorded at frequent intervals.

Circulation and Hemorrhage Control

Bleeding can result due to:

- Fractures of long bones
- Vascular injuries
- Soft tissue injuries

Application of pressure is the best way of management of hemorrhage, as tourniquets increase intraluminal pressure of the vessels, distal ischemia and tissue necrosis. Application of tourniquet is undertaken in select situations, and when used the time of application of tourniquet has to be noted so that neighboring soft tissue is not jeopardized.

Recognition and Assessment of Hypovolemia

The assessment of hypovolemia has to be done in a systematic manner:

- Skin – color, clamminess and capillary refilling
- Vital signs – heart rate, blood pressure and pulse volume
- Consciousness level

Isolated determinations of above parameters is unreliable as various organs try compensate at early stages, especially at extremes of age.

Common sites of occult bleeding are:

- Chest
- Abdomen and retroperitoneum
- Pelvis
- Long bone fractures
- External bleed into splints and dressings.

Once any overt bleeding is controlled, it is necessary to maintain the circulatory volume. This is done by administering a warm crystalloid followed by blood transfusions, maintaining the radial pulse and blood pressure. This administration of fluids is done by peripheral venous cannulation, and when not possible done through central venous catheterization.

- Vital signs return to normal after less than 2 liters of fluid are administered, when the lost blood is less than 20 percent of the blood volume
- Transient responders who are actively bleeding or recommence bleeding during the resuscitation, and the improved vital signs deteriorate indicating loss of over 30 percent of the blood volume
- Little or no response indicates that the loss is more than 40 percent or no hypovolemia.

Disability

Disabilities when occur in a patient with trauma shows seriousness. It can occur in:

- Hypoxia
- Hypovolemia
- Hypoglycemia
- Increased intracranial pressure.

Exposure

The patient's clothes have to be removed by cutting through the seams so that there is minimal patient movement. All clothes are removed only after adequate intravenous access is established, as a rapid removal of tight trousers can precipitate sudden hypotension due to the loss of the tamponade effect in a hypovolemic patient.

Once stripped, trauma victims should be kept warm with blankets when not being examined. Now the patient is rolled on and the spine examined from base of skull to the coccyx, with a rectal examination.

What to look for during rectal examination in a trauma victim:

- Is the sphincter tone present?
- Is the rectal wall breached?
- Is the prostate in a normal position?
- Is there blood on the examiner's finger?

SECONDARY SURVEY

Once the immediately life-threatening conditions are recognized and treated, the whole of the patient is assessed. This requires head to toe, front to back assessment along with detailed medical history and appropriate investigations.

If the patient deteriorates at any stage, the airway, breathing and circulation must be immediately re-assessed as described in the primary survey.

CLINICAL EVALUATION

Eliciting History

The history is elicited quickly and in concise manner, concentrating on:

- Nature of injury
- Mechanism of injury
- Treatment provided
- Medical history.

Physical Examination

The physical examination should be done in a systematic manner.

Scalp

The scalp is examined for:

- Lacerations
- Swellings
- Depressions

The examination is carried out by inspection and palpation (by running the fingers on the scalp).

- It should be remembered to examine the occipital region, which is usually missed out
- Blind probing should be avoided as it may further damage the underlying structures
- In children, bleeding from scalp lacerations can cause hypotension and efforts to stop bleeding (application of pressure, applying self-retaining retractors) have to be taken immediately.

Neurological State

The neurological state of the patient by the Glasgow Coma Score, the papillary responses and the presence of lateralizing signs should be assessed. This examination should be done frequently so that any deterioration is detected early.

Base of Skull

Since the skull base lies along a diagonal line running from the mastoid to the eye, the signs of a fracture in this region are also found along this line.

Signs of a base of skull fracture

- Bruising over the mastoid (Battle's sign)
- Panda's eyes
- Blood and CSF rhinorrhea
- Blood and CSF otorrhea
- Hemotympanum
- Scleral hemorrhage with no posterior margin
- Subhyoid hemorrhage.

- Battle's sign and Panda's eyes appear after about 12 to 36 hours, and is not a reliable sign in a resuscitation room.
- CSF rhinorrhea may be missed as it is invariably mixed with blood.

Neck

If any deformity is found, it is necessary to splint the neck preferably with a collar.

Eyes

- Hemorrhage
- Foreign bodies (including contact lenses)
- Papillary response and corneal reflexes (in unconscious patient)
- Visual acuity (in conscious patient).

Face

The face is examined by systematic inspection and palpation, and look for:

- Soft tissue injuries
- Fractures of midface
- Fractures of mandible
- Missing or lost teeth.

- Midface fractures may be associated with fractures of base of skull
- Mandibular fractures can cause airway obstruction due to loss of stability of tongue.

Thorax

The thorax is examined for

- Soft tissue injuries
- Fractures of clavicles and ribs
- Crepitus (e.g. surgical emphysema)
- Movements of chest (e.g. flail chest).

Abdomen

The abdomen is examined in a very systematic manner, without forgetting to examine the pelvis and perineum. Percussion is an ideal way to determine the injuries of liver and spleen. In penetrating injuries, if bowel is exposed, it should be covered immediately with sterile cloth.

Urine output measurement is a good indicator in a shocked patient, and this requires catheterization for accurate measurement. Transurethral catheterization without any difficulty may indicate that there is no severe urethral disruption. If the patient is not able to urinate, and catheterization is not possible, severe urethral injury should be suspected, and suprapubic catheterization should be done. The signs of urethral injury in a male are:

- Bruising around the scrotum
- Blood at the urethral meatus
- High-riding prostate.

Extremities

The limbs are examined in the traditional manner of inspection, palpation and active and passive movement.

- The fractures should be assessed and the blood loss estimated
- Distal loss of arterial pulsation may be due to profound shock or arterial injury.

DEFINITIVE CARE

Once the patient is adequately assessed and successfully resuscitated, he is moved to the next stage for definitive care, but they are vulnerable during transfers as monitoring becomes difficult during this time.

Before transfer, it is necessary to:

- Close an open wound with sterile pads
- Correct the deformity and splint the limb.

Note:

- Intra-abdominal bleed should be suspected if the patient is hemodynamically unstable, especially if the lower six ribs are fractured or there are marks on the abdominal surface
- In unexplained hypotension, pelvic bone injuries should be suspected. Compression or distraction test is useful
- Clinical examination may not be reliable in neurologically unstable patients, and investigations like ultrasound and CT or MRI may be needed. Diagnostic peritoneal lavage may be relied upon in certain situations like massive hemoperitoneum.

Head Injuries

Head injuries are classified into:

- Brain injury
- Fractures of skull
- CSF fistula
- Injuries of cranial nerves
- Vascular injuries (rare)
- Scalp injuries.

BRAIN INJURY

For the purpose of understanding the pathophysiology, brain injury is subdivided into:

- Primary brain injury and
- Secondary brain injury

Primary brain injury is further classified into:

- Concussion brain
- Diffuse axonal injury
- Primary brainstem injury
- Contusions and lacerations of brain

These injuries occur depending on the:

- Severity of impact
- Direction of impact force

- Movement of head:
 - ❖ *Type of injury*: closed or penetrating injury. Mild force causes **concussion** of brain characterized by transient loss of consciousness, Post traumatic amnesia, confused state and patient recovers completely
 - ❖ Severe force causes **Diffuse Axonal Injury** characterized by prolonged unconsciousness and neurologic deficits
 - ❖ More severe force causes **Primary Brain stem Injury**. With extremely severe impact force, fatal injury occurs with death at the accident spot itself
 - ❖ To-and-fro movement of head shakes various parts of the brain within the skull causing different injuries: gray matter moves over white matter; subcortical white matter moves over basal ganglia; brain surface hits against the rough floor of cranial fossa floor and sharp edges of falx and tentorium leading to **contusions of brain**. When the pia-arachnoid is torn it is termed **laceration**.
- With impact force/acceleration-deceleration force, brain moves within the cranial cavity—cerebrum moves over brainstem leading to deformation of neuronal-synaptic membranes at central reticular core.

Secondary Brain Injury

- Hypoxic-Ischemic injury to brain because of impaired breathing, aspiration, airway obstruction, chest injury, cervical spine injury, hypotension, etc.
- Intracranial hematomas (viz., acute extradural hematoma, acute subdural hematoma, acute intracerebral hematoma) or expanding contusions and brain edema can cause brain shifts and increased intracranial pressure
- Metabolic abnormalities of glucose, sodium, acid-base, etc.

Acute extradural hematoma Vs Acute subdural hematoma:

- Acute extradural hematoma occurs at the site of impact and hence the scalp injury or fracture skull will give clue to the location of extradural hematoma
- Bleeding is usually from the middle meningeal branches or from fracture edges
- Acute subdural hematoma is 6 to 8 times more common than extradural hematoma and is often due to acceleration and deceleration injury
- Subdural hematoma can be contracoup, i.e. its location can be diagonally opposite to the site of scalp injury. The bleeding is from the cortical veins
- Due to associated severe primary brain injury, prognosis is in general poor compared to isolated extradural hematoma.

- Skull fractures are classified into:
 - ❖ **Fractures of skull vault** which can be:
 - Linear fracture (Fissured fracture)
 - Depressed fracture
 - ❖ **Fractures of skull base** which can involve:
 - Anterior cranial fossa
 - Middle cranial fossa
 - Posterior cranial fossa
- They can be also be classified into:
 - ❖ Simple (or closed) fractures
 - ❖ Compound (or open) fractures
(depending on the absence or presence of communication to atmospheric air through breach of skin or air containing cavities in the skull)
- Fractures of skull vault will indicate the location of underlying extradural hematoma when present
- Compound depressed fractures are potential source for spread of infection leading to brain abscess, meningitis, epidural /subdural abscess
- Depressed fractures of vault can lead to direct brain contusions and hematomas.

Skull fracture indicates that the force of injury was severe but does not indicate the severity of brain injury.

Symptoms and Signs

- Fractures of anterior cranial fossa (Fig. 7.1) can cause:
 - ❖ Ecchymosis of the upper eyelids appearing after about 24 hours
 - ❖ Subconjunctival hemorrhage in the upper eyelid in anterior skull base fractures is pointed towards the cornea and posterior limit will not be seen
 - ❖ In contrast in “Black Eye” where the injury is directly over orbit, the ecchymoses occur in both upper and lower eyelids and appear within minutes of injury
 - ❖ The subconjunctival hemorrhages are actually conjunctival in plane and will move with conjunctiva
- Cranial nerve palsies occur in fractures of the base of skull across or near the cranial nerve exit foraminae:
 - ❖ Fracture of temporal bone can explain 7th and 8th nerve palsies
 - ❖ Fracture of superior orbital fissure can explain spread of injuring force close to 3rd, 4th and 6th nerves
 - ❖ Fractures near jugular foramen can cause lower cranial nerve palsies
- CSF rhinorrhea occurs in fractures involving cribriform plate or paranasal sinuses



FIG. 7.1: Fracture of anterior cranial fossa

- CSF otorrhea occurs in fractures of temporal bone Eustachian tube CSF rhinorrhea. CSF leaks have the risk of meningitis
- Pulsatile proptosis in head injury can be due to carotico-cavernous fistula or more commonly comminuted fracture of orbital roof with normal pulsation of brain being transmitted to orbit. In carotico-cavernous fistula bruit may be present over orbit.

Note: Cranial nerve palsies can occur without skull fracture, e.g. olfactory nerve injury or optic nerve injury.

Relevant Investigations

X-rays (AP, lateral and oblique views) and CT of skull (Figs 7.2A to D) are diagnostic.

Treatment

- Closed depressed fractures over forehead may need elevation for cosmetic purposes
- Compound fractures will need debridement to prevent brain abscess.



FIG. 7.2A: CT—Fracture of frontal bone



FIG. 7.2B: CT—Fracture

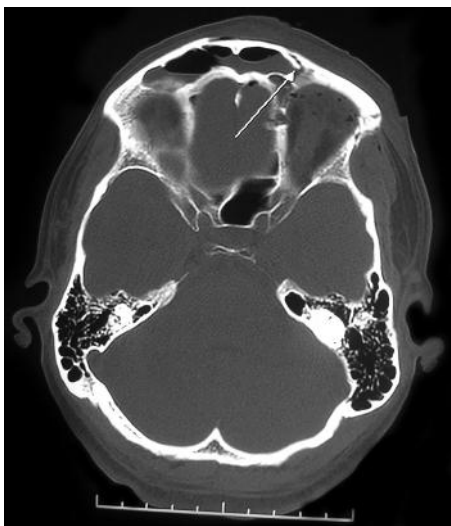


FIG. 7.2C: CT—Fracture of frontal sinus



FIG. 7.2D: CT—Subdural hematoma

CSF FISTULAE

Incidence and Etiology

Leak of CSF is caused by:

- ❖ Fractures involving cribriform plate or paranasal sinuses can lead to CSF rhinorrhea
- ❖ Fractures of temporal bone can lead to CSF otorrhea and through Eustachian tube CSF rhinorrhea.

Complications: Meningitis

Symptoms and Signs

Watery discharge is pathognomonic of CSF leak.

Relevant Investigations

CT of paranasal sinuses after intrathecal contrast is useful.

Treatment

- Lumbar puncture and CSF drainage intermittently for 10 – 20 days
- Surgical repair is necessary if leak persists.

SCALP INJURIES

Scalp injuries indicate the sites of impact force.

They are of three types:

1. Contusions
 2. Lacerations
 3. Hematomas
 - ❖ Subpericranial—Confined to an area of one cranial bone and fixed
 - ❖ Subaponeurotic or subgaleal—More diffuse and extend beyond the margins of bones
 - ❖ Subcutaneous—Superficial and moves with the scalp.
- Fractures beneath scalp injury in unconscious patients suggest the possibility of underlying extradural hematoma
 - Scalp injury over occipital region may give clue to posterior fossa hemorrhage
 - The center of scalp hematomas may liquefy in the center after a few days and often give a false impression of depressed fracture to the palpating finger.

- Bleeding from an open wound
- Swelling in the head with or without external injury to the scalp.

*Treatment*

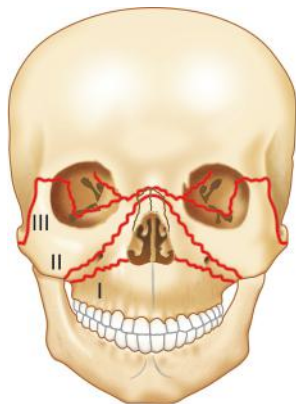
Immediate suturing of the wound, as the vessels are prevented from normal contraction by fixation of their walls to fibrous stroma of scalp.

Note: When there is scalp laceration, before wound closure, depressed fracture has to be excluded clinically under aseptic precautions.

Facial Injuries

FRACTURES OF MIDFACIAL SKELETON

- The fracture of facial skeleton is usually due to direct injury
- Types of fractures of midface are:
 - ❖ Floor of the orbit (Blowout fracture)
 - ❖ Zygomatic
 - ❖ Nasal
 - ❖ Maxillary [LeFort classification (Fig. 8.1)]
 - LeFort I (low level fracture): This is horizontal fracture above the level of the nasal floor (Guerin's fracture, floating fracture)



LeFort classification of maxillary fractures

- I - Low level
- II - Pyramidal (or) subzygomatic
- III - High transverse (or) suprazygomatic

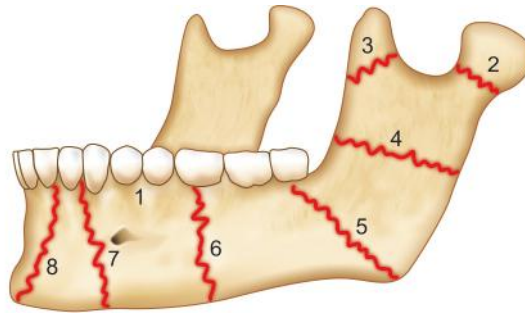
FIG. 8.1: Fractures of maxilla

- LeFort II (pyramidal or subzygomatic fracture): The fracture runs from the thin middle area of the nasal bones down either side crossing the maxillary processes into the medial wall of each orbit
- LeFort III (high transverse or suprazygomatic fracture): The fracture runs from near the frontonasal suture transversely backwards, parallel with the base of the skull and involves the full depth of the ethmoid, including the cribriform plate.

FRACTURES OF MANDIBLE

Fractures of the mandible (Fig. 8.2) can be divided according to the anatomical location into 8 types:

1. Dentoalveolar
2. Condylar
3. Coronoid
4. Ramus
5. Angle
6. Body
7. Parasymphysis
8. Symphysis



Mandibular fractures

- | | |
|------------------|------------------|
| 1. Dentoalveolar | 5. Angle |
| 2. Condylar | 6. Body |
| 3. Coronoid | 7. Parasymphysis |
| 4. Ramus | 8. Symphysis |

FIG. 8.2: Fractures of mandible

The mandibular fractures can be:

- Unilateral
- Bilateral
- Multiple
- Comminuted.

Clinical Presentation

History

- History of injury and hearing or feeling of a bone crack
- The nature of impact (direct violence, indirect violence or excessive muscular contraction) should be determined.

Symptoms

- Pain and loss of function
- Diplopia and enophthalmos are present in LeFort III type
- But loss of function may not be a feature of impacted fracture.



FIG. 8.3A: Ecchymosis and nasal bleed in injury of midface



FIG. 8.3B: Subconjunctival hemorrhage

- Tenderness of the bone on applying pressure
- Swelling and ecchymosis (Fig. 8.3A)
- Nasal bleed (Fig. 8.3A) and CSF rhinorrhea are common in associated base of skull fractures
- Subconjunctival hemorrhage occurs in orbital bone fractures (Fig. 8.3B)
- Blebs on the skin overlying the fractured site
- Deformity: This is elicited by inspection, palpation and measurement. This is the most important sign of a fracture. The deformity in case of a flat bone is either depression or elevation and in case of long bone this is angular, lateral, longitudinal or rotatory. Hard palate is deformed in Le Fort I fracture (Fig. 8.4)
- Dental alignment is lost in displaced fractures (Fig. 8.5).



FIG. 8.4: LeFort I fracture



FIG. 8.5: Displaced mandibular fracture



FIG. 8.6A: X-ray—Fracture of ramus of mandible



FIG. 8.6B: X-ray—Fracture of condyle of mandible



FIG. 8.6C: X-ray—Zygomatic fracture



FIG. 8.7: CT—Fracture of orbital bones

- Abnormal mobility: This is present in case of impacted fractures
- Crepitus: Also present in case of impacted fractures and should be elicited gently
- Loss of function: For example, if the maxilla or the mandible is fractured, the patient may not be able to masticate properly.

Relevant Investigations

X-rays (Figs 8.6A to C) and CT (Fig. 8.7) are diagnostic.

Treatment

- Undisplaced fractures need conservative management
- Displaced fractures require surgical treatment, fixation with plates and screws (craniomaxillofacial plating) and interdental wiring, with the aim to restore precise anatomical alignment.

Spine and Spinal Cord Injuries

INJURIES TO BONY AND LIGAMENTOUS SPINE

Incidence and Etiology

- When an overwhelming force strikes and compresses the spine, the bone crumbles resulting in fracture of spine
- The type of the vertebral fracture depends on the direction of injuring force and the curvature of spine at the time of injury
- With increasing force of injury, two or three columns of spine get injured leading to instability and dislocation
- Generally compression forces cause fracture while distraction forces cause ligament injury
- The injuring forces can cause injury to spinal cord/root.

Symptoms

- When the spinal cord is not affected, pain on movement, stiffness and tenderness are the symptoms
- When the spinal cord is affected, neurological deficit occurs, depending on the level and completeness of damage to the cord
 - ❖ Limb paralysis like paraplegia or quadriplegia may be the presenting symptoms
 - ❖ Respiratory, circulatory and urinary bladder dysfunctions may be superadded.

Signs

Clinical examination is to determine the level and type of injury

- External injuries give clue to the mechanism of injury (e.g. chin injury may indicate hyperextension injury, occipital injury may indicate flexion-distraction injury, vertex injuries may indicate vertebral compression).

Relevant Investigations

X-rays (Figs 9.1A and B), CT (Fig. 9.2) and MRI are essential to assess the extent of injury.

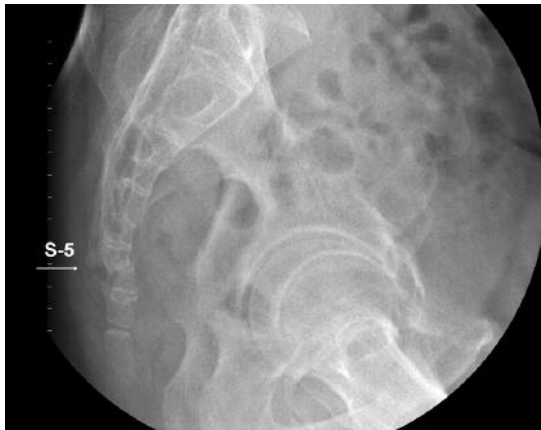


FIG. 9.1A: X-ray—Fracture of 5th sacral vertebra



FIG. 9.1B: X-ray—Compression fracture vertebra



FIG. 9.2: CT cervical spine—Fracture body of C3 vertebra

- Rest to the injured area may be sufficient with analgesic support. Many of them recover completely
- Surgical decompression and stabilization are mandatory for incomplete cord injuries. Though, the procedures are useful for bony stability in complete cord injuries, the neurologic recovery is poor.

INJURIES OF THE SPINAL CORD

Incidence and Etiology

- During the injuries of the spinal cord, the neurons suffer primary damage on impact and are prone to secondary injuries from hypoxia, hypotension, hematoma and swelling
- When the damage is severe, spinal cord function at and below the level of the lesion is abolished
- Spinal cord injuries are usually associated with injuries of vertebral column, usually fracture or fracture-dislocation
- The mechanisms of injury to the spinal cord /root are by:
 - ❖ Primary injuries: The primary injuring force itself can cause violent movement and stretching or disruption of cord with or without abnormal bony displacements
 - ❖ Secondary injuries: The fractured segments of vertebrae, extruded disk, infolded ligaments and intraspinal hematomas can cause secondary compression of cord or roots causing neurologic deficit.

Symptoms and Signs

- Paralysis occurs below the level of the lesion, e.g. thoracic cord injury leads to paraplegia and cervical cord injury leads to quadriplegia
- Flaccid paralysis with no sensation indicates complete lesion with a poor prognosis
- Signs suggestive of spinal cord injury are given in Table 9.1
- In an unconscious patient, some symptoms suggest spinal cord injury (Box 9.1).

Table 9.1: Spinal cord injuries and their signs

Complete cord injury	Total paralysis with loss of sensation below level of injury
Cord hemisection (Brown-Sequard syndrome)	Ipsilateral paralysis with contralateral loss of sensation below level of lesion
Central cord syndrome	Greater motor loss in the upper limbs than the lower limbs Variable sensory loss below level of lesion
Anterior cord syndrome	Paralysis, loss of pain/temperature sensation below level of lesion Proprioception and vibration preserved

Box 9.1: Signs suggestive of spinal cord injury in an unconscious patient

- Diaphragmatic pattern of breathing
- Unexplained hypotension
- Flaccid paralysis
- Reduced anal tone
- Urinary retention/Priapism

Relevant Investigations

X-rays and CT are useful in diagnosis.

Treatment

- The treatment should focus on maintaining stability of the vertebral column either by external or internal fixation
- Recovery is variable and rehabilitation should start early for better results
 - ❖ Establish IV access and give good volume load to support blood pressure
 - ❖ Vasopressors may be needed to maintain circulation
 - ❖ Tracheal intubation and assisted ventilation may be needed to support ventilation
 - ❖ Nasogastric intubation may be needed for gastric decompression
 - ❖ Urethral catheterization is needed.

Thoracic Injuries

RIB FRACTURES

Incidence and Etiology

- This constitutes the most common chest injury
- Minor fractures are those confined to one or two ribs
- Mechanism of injury
 - ❖ Upper rib injuries involve major energy transfer and are often associated with injuries to major vessels, brachial plexus and tracheobronchial tree
 - ❖ Fractures of lower ribs are frequently associated with liver and splenic injuries
 - ❖ Rib fractures in the elderly can occur after relatively low energy transfers as the bones are of low density and chest wall compliance is poor.

Fractures of brittle ribs of elderly patients cause very little underlying injuries, whereas, flexible rib injuries of younger individuals cause severe injury without obvious fractures.

Symptoms

Severe pain on deep inspiration and coughing, poor inspiratory effort, and progressive atelectasis and pneumonia due to underlying lung contusion.

Signs

- Crepitus, and bony tenderness
- Skin bruises should give the suspicion
- The hallmarks of rib fractures are intense pain, poor inspiratory effort, and progressive atelectasis and pneumonia due to underlying lung contusion.

- Apical rib fractures are associated with injury to great vessels
- Mid zone rib fractures are associated with pulmonary contusion
- Basal rib fractures are associated with abdominal visceral injuries (liver, spleen).

Relevant Investigations

- Chest X-ray shows the site and number of fractures (Figs 10.1A and B), underlying pleural and lung injuries
- Chest CT (Figs 10.2A and B) gives clearer view of fractures.

Treatment

Centers around pain management:

- Oral and parenteral analgesics
- Intercostal nerve blocks
- Epidural analgesia especially in elderly or patients undergoing abdominal surgeries.



FIG. 10.1A: X-ray—Fracture rib



FIG. 10.1B: X-ray—Fractures of lower ribs

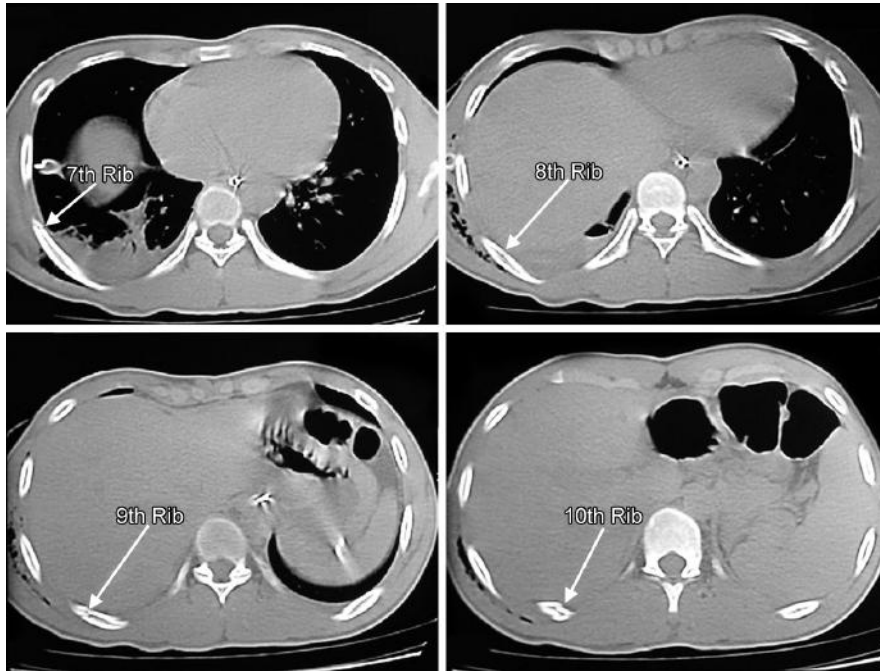


FIG. 10.2A: Chest CT—Fractures of 7, 8, 9 and 10th ribs



FIG. 10.2B: CT—Laceration of liver and perisplenic collection

FLAIL CHEST

Incidence and Etiology

- When three or more ribs are fractured, each in more than one place, producing a free floating section of the chest wall with or without separation of the costochondral junction it is called a flail chest (Fig. 10.3)
- The flail segment interferes with the ventilatory function by ineffective chest wall motion (paradoxical movement) i.e. movement inward with inspiration and outward with expiration, producing pain and splinting and thereby a fall in tidal volume, hypoxia and hypercarbia.

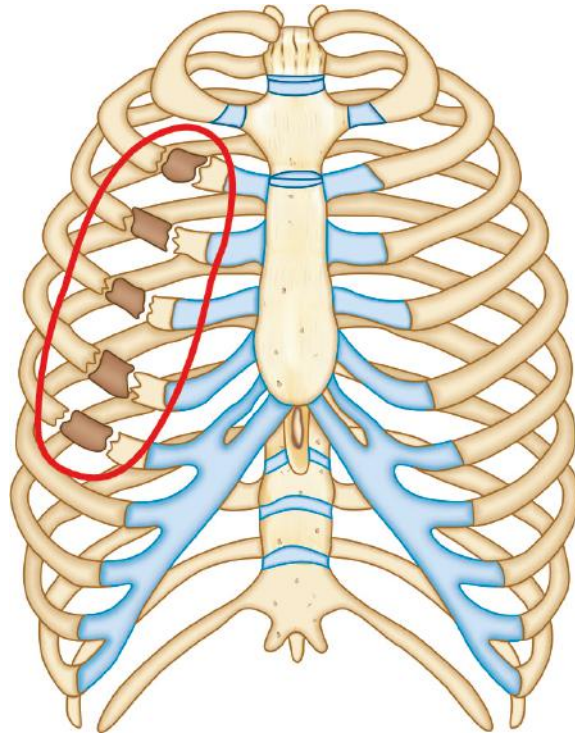


FIG. 10.3: Flail chest

- Other causes for flail chest are:
 1. Traumatic disruption of ligaments and cartilages of ribs—not seen on X-ray.
 2. Destruction of ribs from malignant disease, e.g. multiple myeloma.
 3. Metabolic disease—osteitis fibrosa cystica.
 4. Nonclosure of median sternotomy wound.

Symptom

Dyspnea.

Sign

Paradoxical respiration and hypoxia.

Relevant Investigations

- Chest X-ray—to assess fracture, lung injury, hemopneumothorax
- Arterial blood gas analysis—to aid treatment of respiratory insufficiency (ventilation perfusion mismatch).

Treatment

Treatment of flail chest is shown in Table 10.1.

Table 10.1: Treatment of flail chest

<i>Segment of flail</i>	<i>Respiratory distress</i>	<i>Respiratory function</i>	<i>Treatment</i>
Small	No	Good	Pain relief and observation
Moderate	Severe	Moderate	Mechanical ventilation and analgesics
Large	Severe	Bad	Chest wall reconstruction and mechanical stabilization

STERNAL FRACTURE

Incidence and Etiology

- Occurs mostly at the manubriosternal junction and is associated with very high velocity trauma
- Injury to aorta, esophagus, bronchi, myocardium and spine need to be kept in mind.

Symptom

Severe pain over the anterior chest wall.

Sign

Crepitus over the fracture site is characteristic.

Relevant Investigations

Chest X-ray lateral view and CT (Fig. 10.4) demonstrate the fracture.

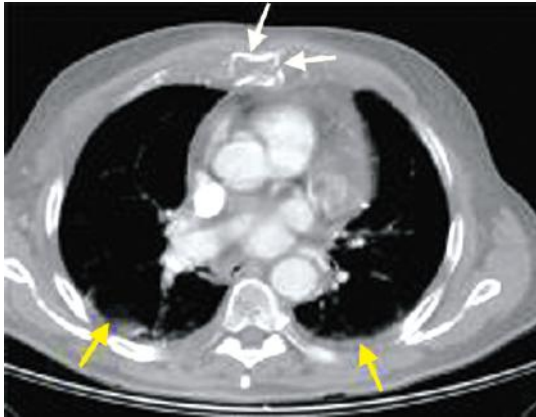


FIG. 10.4: CT—Fracture sternum

Treatment

- Sternal fractures can be managed conservatively with pain relief
- Rarely, in case of persistent chest wall instability, fixation may be necessary.

PNEUMOTHORAX

Incidence and Etiology

- Defined as air in the pleural cavity
- The types (Fig. 10.5) are:
 - ❖ *Closed pneumothorax*: Air in the pleural cavity and has no external communication (e.g. rupture of emphysematous bulla) or from outside
 - ❖ *Open pneumothorax*: Air in the pleural cavity has external communication (e.g. penetrating chest wall injury or rib fracture)

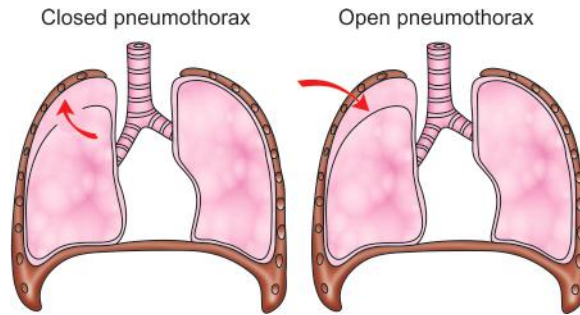


FIG. 10.5: Types of pneumothorax

- ❖ *Tension pneumothorax*: Continued entry of air into the pleural cavity, increasing the intrapleural pressure above the atmospheric pressure, which results in the shift of the mediastinum away from the side of injury.

Symptoms

Chest pain, dyspnea and tachycardia.

Signs

- On examination the neck veins are distended, the trachea and apex beat are shifted away from the side of tension, breath sounds become distant or absent on the side of tension, due to the presence of air between the chest wall and the lung substance
- The chest on the affected side is more resonant (DD—hemothorax, hydrothorax) on percussion.

Relevant Investigations

- Chest X-ray (Figs 10.6A to C) is conclusive, with shift of mediastinal structures away from the side of the pathology, with air shadow lateral to the lung parenchyma on the side of the pathology
- CT (Figs 10.7A and B) is diagnostic.

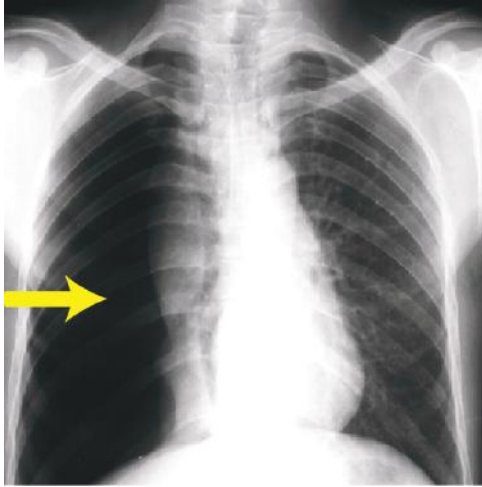


FIG. 10.6A: Chest X-ray—Right pneumothorax

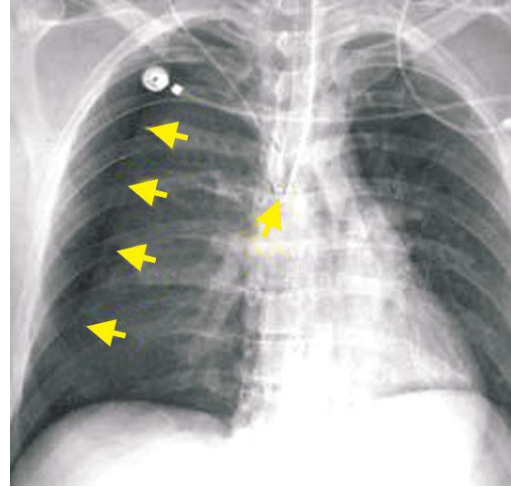


FIG. 10.6B: Chest X-ray—Right pneumothorax in patient on ventilator



FIG. 10.6C: Chest X-ray—Tension pneumothorax

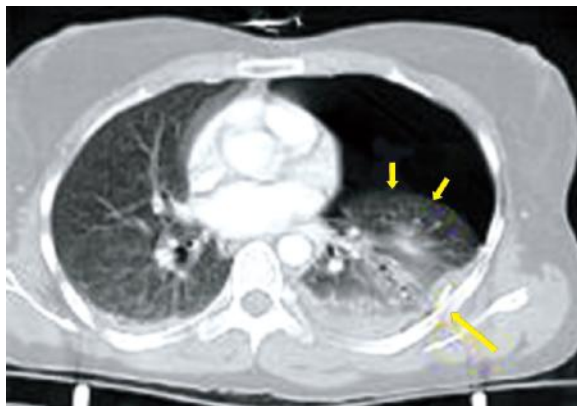


FIG. 10.7A: CT—Left pneumothorax with fractures of ribs

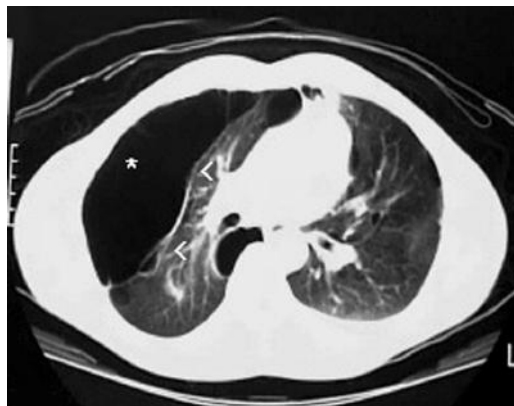


FIG. 10.7B: CT—Right tension pneumothorax

Treatment

- *Open pneumothorax*: The external wound is closed with a tape to convert it into a closed variety, supported by intercostal drainage
- *Closed and tension pneumothorax*: Simple aspiration of air from the pleural space followed by tube thoracostomy.

- Large chest wall wounds more than 75 percent the diameter of trachea allow preferential air entry through the chest wall. Any attempt to ventilate leads to movement of air in and out of the defect. No ventilation is achieved, and severe respiratory compromise occurs
- All traumatic pneumothoraces should be drained
- Massive air leaks may require bronchoscopy to exclude bronchial rupture
- Bronchial rupture should be suspected in the presence of deceleration injury, mediastinal widening, hemoptysis, first rib and clavicular fractures.

SURGICAL EMPHYSEMA

Incidence and Etiology

Defined as air in the subcutaneous tissues due to the air entry from the injured lung or external injuries like fractured rib.

Pathogenesis

The entrapped air gradually spreads along the fascial planes into the neck, mediastinum but, rarely down into the scrotum producing, a pneumoscotum.

Symptoms and Signs

Subcutaneous tissues appear swollen and crepitus is a classical finding.

Relevant Investigations

- X-ray is diagnostic and reveals the air shadow in the subcutaneous plane, and also the underlying cause (e.g. rib fracture)
- CT (Fig. 10.8) is diagnostic.

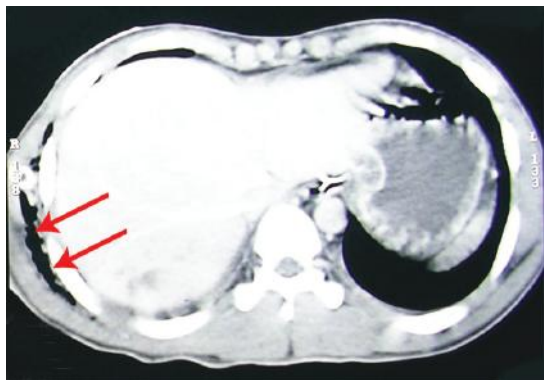


FIG. 10.8: CT—Right sided surgical emphysema with rib fracture

Treatment

- Small emphysema resolves spontaneously
- Hemodynamic instability warrants surgical intervention
- Treating the underlying cause.

HEMOTHORAX

Incidence and Etiology

Defined as blood in the pleural space, which is usually due to external (blunt or penetrating) trauma.

Symptoms

Chest pain, dyspnea and tachycardia.

Signs

- Tachycardia
- Neck veins are distended
- Trachea and apex beat are shifted away from the side of tension
- The chest on the affected side is less resonant or dull (DD—pneumothorax) on percussion
- Breath sounds become distant or absent on the side of lesion, due to the presence of blood between the chest wall and the lung substance
- Hypotension and shock may be evident depending on the amount of blood loss (bleeding from lung parenchyma is usually small but those from the intercostals and internal mammary arteries may be large).

Relevant Investigations

- Chest X-ray (Fig. 10.9A) is conclusive, with shift of mediastinal structures away from the side of the pathology, with haziness with obliteration of costophrenic angle
- CT (Fig. 10.9B) is diagnostic.



FIG. 10.9A: Chest X-ray—Left hemothorax

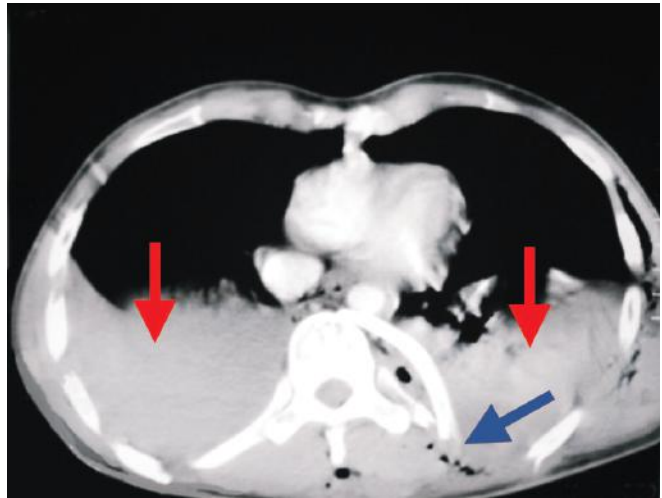


FIG. 10.9B: CT chest—Bilateral hemothorax (Red arrows) with left 10th rib fracture (Blue arrow)

Treatment

- Small collections of blood may be aspirated under aseptic conditions
 - Large collections need intercostal drainage
 - Thoracotomy is needed for massive bleeds (initial bleed of more than 1 – 1.5 liters), or continued bleeds of more than 200–300 ml/hr, to control the source of bleeding.
- Early drainage is the key to success in the management of hemothorax
 - Once clot gets established, thoracotomy is needed
 - While draining hemothorax, it is advisable to use large drains (28 – 32 F)
 - Initial drainage of >600 ml or continued drainage of > 150 ml/hr will need thoracic surgical referral.

PULMONARY CONTUSION/LACERATION

Incidence and Etiology

- While contusions of the lungs are produced by blunt chest injury with hemorrhage and edema in the lung parenchyma, lung lacerations are due to penetrating injuries
- There may be associated injury to larger airways:
 - ❖ Blunt injury usually produces injuries within 2.5 cm of the carina
 - ❖ Penetrating injuries may be at any level.
- Alveolar microhemorrhages are responsible for the poor ventilatory status
- Tracheobronchial injuries may coexist.

Complications: Mediastinal emphysema in case of major airway injury and hemopneumothorax in case of peripheral bronchial injuries.

Symptoms and Signs

- Dyspnea, tachycardia and chest pain
- Small lacerations produce no symptoms.

Relevant Investigations

- Chest X-ray is diagnostic, which shows vague opacification in the injured area (usually within 1-2 hours of injury)
- CT (Fig. 10.10) and MRI are conclusive
- Bronchoscopy is needed to evaluate tracheobronchial injuries.



FIG. 10.10: CT chest—Bilateral pulmonary contusion

Treatment

- Most lacerations of the lung resolve spontaneously and need no treatment
- Large lacerations may require resuscitation and tube thoracostomy
- Bronchial injuries involving more than 1/3rd circumference of the bronchus require surgery
- Mechanical ventilation is warranted when there is profound V/Q mismatch.

INJURIES OF THORACIC AORTA

Incidence and Etiology

- Thoracic aorta is vulnerable for injuries at its three fixed sites:
 - ❖ Annulus
 - ❖ Ligamentum arteriosum—isthmus
 - ❖ Aortic hiatus
- The most common sites being:
 - ❖ The ascending aorta proximal to innominate artery
 - ❖ The descending aorta at the point beyond the origin of left subclavian artery (ligamentum arteriosum)
- Mechanism of injury:
 - ❖ Penetrating: More common and may involve other great vessels
 - ❖ Blunt: Rapid deceleration produces shear at the fixed portions of the aorta, or by anteroposterior compression.
- Almost 80 percent die on the way to the hospital.

Symptom

Severe respiratory distress.

Signs

- Features of massive hemothorax
- Signs of shock may be evident
- Neurological signs of cord compression may be present.

Injuries distal to innominate artery may show pseudocoarctation syndrome (upper extremity hypertension and hypotension and low pulse in lower limbs).

Relevant Investigations

- Chest X-ray and CT (Fig. 10.11) are diagnostic with the following findings:
 - ❖ Widening of superior mediastinum
 - ❖ Depression of left main stem bronchus
 - ❖ Loss of aortic knob
 - ❖ Massive hemopneumothorax
 - ❖ Look for associated 1st rib fracture, flail chest, sternal fracture and fracture of thoracic spine.
- Arteriography is useful for definitive diagnosis of location and extent of injury.

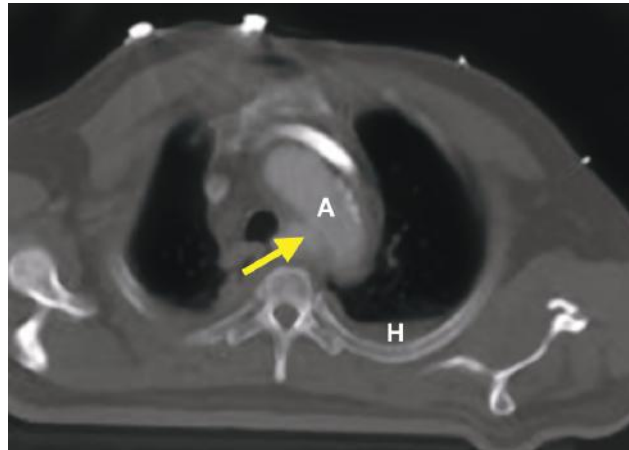


FIG. 10.11: CT chest—Aortic rupture with upper rib fractures

- Resuscitation is the primary and emergent treatment
- Resection of damaged segment of aorta and repair/interposition grafts is curative.

◆ INJURIES OF MYOCARDIUM

Incidence and Etiology

- Myocardial injuries are caused by:
 - ❖ Penetrating injuries (e.g. gunshot or stab injuries) where the outcome of injury depends on size of pericardial defect
 - ❖ Blunt injuries
 - Rupture into the pericardium producing pericardial tamponade
 - Myocardial contusion
 - Arrhythmias.
- Commotio cordis is the condition of sudden cardiac death or near sudden cardiac death after blunt, low-impact chest wall trauma in the absence of structural cardiac abnormality. Ventricular fibrillation is the most commonly reported induced arrhythmia in commotio cordis.
- Blunt impact injury to the chest with a baseball is the most common mechanism and does not result solely from the force of a blow as it is not seen in association with any rib or sternal fracture. It is largely the result of the exquisite timing of the blow during a narrow window within the repolarization phase of the cardiac cycle, 15 to 30 msec prior to the peak of the T wave. Survival rates for commotio cordis are low, even with prompt CPR and defibrillation.

Symptoms

Dyspnea and cyanosis.

Signs

- Examination reveals, distension of jugular veins, hypotension and narrowing pulse pressure and distant heart sounds (Beck's triad)
- The jugular venous distension raises paradoxically on deep inspiration (Kussmaul's sign), because the increased venous return cannot be accommodated within the constricted heart
- Signs of shock may be evident
- Pulsus paradoxus is a cardinal sign (drop in systolic BP >10 mm during inspiration due to CO₂ absorption).

Relevant Investigations

- Chest X-ray is contributory with cardiomegaly
- ECG may remain normal for over 36 hours but exhibit features of ischemia, dysrhythmia later
- ECHO is done to assess regional wall motion abnormalities
- CPK-MB values show elevation (check values at admission, 24–48 hours).

Treatment

- Cardiac monitoring and resuscitation are important
- Cardiac tamponade warrants pericardiocentesis/subxiphoid pericardial window
- Thoracotomy is done to create an opening of pericardial sac.

ESOPHAGEAL INJURIES

Incidence and Etiology

- Esophageal injuries are caused by:
 - ❖ *Penetrating injury* may occur at any level and are should be suspected when the injury crosses the midline (e.g. in sword swallows as circus act), during esophagoscopy
 - ❖ *Blunt injury*: Usually following severe blow to the sternum or epigastrium. The common site of injury is at the lower 1/3rd esophagus.

Symptoms

Fever, dyspnea (due to mediastinitis or mediastinal emphysema) or tachypnea.

Signs

Features of surgical emphysema (spread of mediastinal emphysema to neck, face and chest wall) may supervene, with signs of hypoxia about 3–4 days later.

Relevant Investigations

- Chest X-ray may reveal
 - ❖ Pneumomediastinum
 - ❖ Air in the prevertebral space
 - ❖ Left pleural effusion
 - ❖ Hemo or pneumothorax in the absence of rib fracture.



FIG. 10.12: Gastrograffin swallow—Leaking dye in esophageal perforation (penetrating injury)

- Gastrograffin swallow may show the leak (Fig. 10.12)
- Esophagoscopy may show the injury
- Intercostal drainage shows particulate food matter, and show air leak during both phases of respiration.

Treatment

- Intercostal drainage is mandatory
- Early operative repair of the esophageal tear is necessary.

DIAPHRAGMATIC INJURIES

Incidence and Etiology

- Injuries of diaphragm are caused by:
 - ❖ Blunt injuries produce large radial tears and herniation of abdominal viscera into the chest
 - ❖ Penetrating injuries are small initially and enlarge over a period of time.

Symptoms and Signs

Diagnosis is difficult unless the tears are large and allows herniation of abdominal contents into the chest cavity—dyspnea and tachycardia.

Relevant Investigations

- Chest X-ray is contributory and may show:
 - ❖ Bowel loops in the chest (in herniation of small bowel)
 - ❖ Double shadow overlying the diaphragm
 - ❖ Nasogastric tube in the chest—in patients with gastrothorax.
- Contrast studies and CT of chest are conclusive.

Treatment

Surgical repair with or without mesh is mandatory.

Abdominal Injuries

Abdominal injuries can be of two varieties. They are:

1. Closed injuries (caused by blunt injuries)
2. Open injuries (caused by penetrating or stab injuries).

CLOSED INJURIES

- Caused by a blunt force exerted suddenly on the abdomen, such as:
 - ❖ Fall from a height
 - ❖ Blow with a fist
 - ❖ Injuries inflicted by heavy weapons like crowbars, poles, sticks
 - ❖ Run over injuries in road traffic accidents
 - ❖ Hitting against the steering wheel during sudden deceleration or braking of a speeding vehicle (in the absence of seat belt).
- They may lead to compression of intra-abdominal organs against the vertebral column causing rupture of:
 - ❖ Solid organs
 - ❖ Hollow organs
 - ❖ Mesentery
 - ❖ Detachment of gut from the mesentery
 - ❖ Contusion of abdominal wall.

OPEN INJURIES

- Caused by any sharp instrument like knife, flying objects like bullets, missiles, pieces of wood or glass.
- The incriminating agents enter the abdominal cavity taking with them some infection resulting in peritonitis.
- The points of entry and exit of the agent will indicate the direction and the possible organs injured.
 - ❖ Hollow organs may perforate and infect the peritoneal cavity (due to contamination by the contents of the organ—feces, urine, intestinal or gastric contents), or bleed (either into the organ or outside or both).
 - ❖ Solid organs cause hemorrhage (inside the organ or outside or both), and large bleeds cause shock and sometimes death.

Symptoms and Signs

- General features:
 - ❖ *Symptoms*: Weakness, clouding of thoughts and speech, air hunger, restlessness, decreased or absent urine output
 - ❖ *Signs*: Pallor, tachycardia, hypotension, subnormal temperature.
- Local features: Depend on the organ involved and the severity of damage.

INJURIES OF LIVER

Incidence and Etiology

The liver ranks high on the list of intra-abdominal organs involved by injury:

- Blunt injuries are more common than the penetrating injuries, due to increase in motor traffic moving at high speeds, and are associated with fracture of lower ribs on the right side. The dome of the liver is involved with anterior-posterior tears, more on the right lobe (7:1).
- Spontaneous rupture of liver is seen in:
 - ❖ Primary carcinoma in adults
 - ❖ Trauma during birth in children (postmature babies) being delivered per vaginam.

Liver injuries (Fig. 11.1) are classified into:

- Transcapsular (blood and bile will seep into the peritoneal cavity)
- Subcapsular (collection of blood between the capsule and the liver parenchyma mostly on the superior surface of liver)
- Central (interruption of liver parenchyma leading to intrahepatic hematoma, abscess and hematuria).

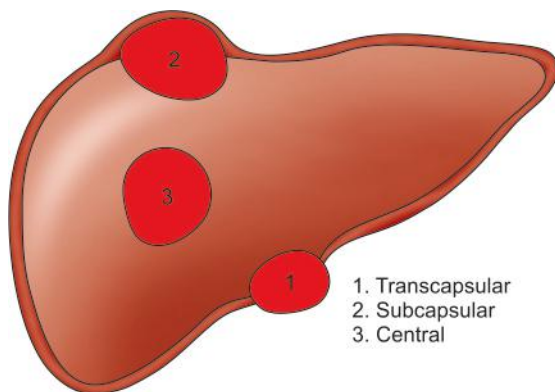


FIG. 11.1: Types of liver injuries

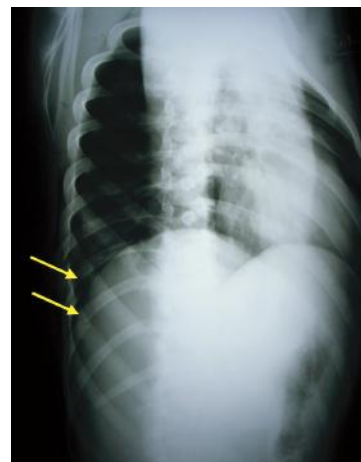


FIG. 11.2: X-ray showing lower rib fracture

Symptoms and Signs

- *Transcapsular*: Symptoms and signs of shock and peritoneal irritation (pain right upper abdomen with reference to the right shoulder, guarding and rigidity of the right hypochondrium, absent bowel sounds, shifting dullness)
- *Subcapsular*: Local tenderness and increase in area of liver dullness
- *Central*: Signs of shock may be present with hemobilia and hematemesis.

Relevant Investigations

- Plain chest X-ray (Fig. 11.2) will demonstrate fracture of lower ribs on the right side
- Plain X-ray abdomen may show haziness in the area of the liver with elevation of right dome of diaphragm
- CT (Figs 11.3A and B) and MRI are useful in localizing the damaged areas of liver and collections of blood or bile
- Peritoneal tap may be useful in identifying bile leaks
- Colloidal gold Au^{198} or Technitium-sulfur colloid $\text{Tc}^{99\text{m}}$ scans are useful during active bleeding.

Treatment

- Correction of shock
- No surgical intervention is needed for small injuries

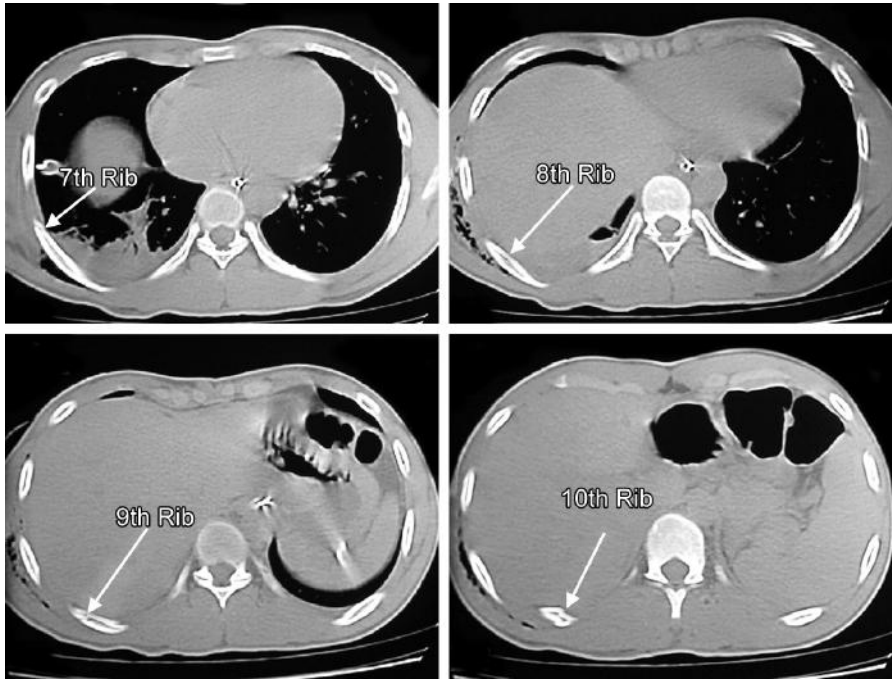


FIG. 11.3A: CT—Right lower rib fractures

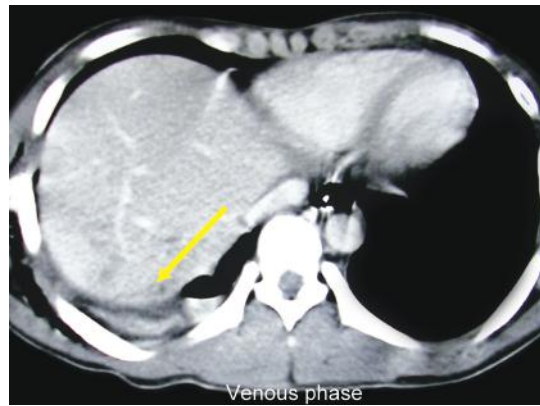


FIG. 11.3B: CT—Liver injury with hemoperitoneum

- Early surgical intervention, sometimes amounting to hepatectomy, is needed for large injuries associated with vascular or biliary tract injuries.
- Post-traumatic liver hemorrhage is amenable to radiological intervention (embolization).

INJURIES OF SPLEEN

Incidence and Etiology

- Spleen is the intra-abdominal organ most frequently injured by blunt trauma, usually by thoraco abdominal injuries associated with fractures of left lower ribs.
- The causes of splenic injuries are:
 - ❖ Blunt injuries of lower chest and abdomen (e.g. automobile accidents—may be associated with rib fractures, lung injuries, fracture of spine, intra-abdominal organs)
 - ❖ Transabdominal and transthoracic penetrating injuries (e.g. gunshot or knife injuries—may be associated with injuries to left kidney, colon, pancreas, vascular structures of mesentery)
 - ❖ Operative injuries (e.g. during operations on adjacent viscera—gastrectomy)
 - ❖ Spontaneous rupture or minor trauma (e.g. enlarged spleen of malaria).
- Splenic injuries may be:
 - ❖ Linear or stellate lacerations (due to excessive traction on the capsule during operations)
 - ❖ Capsular tears (due to excessive traction on the capsule during operations)
 - ❖ Subcapsular hematoma (due to excessive traction on the capsule during operations)
 - ❖ Puncture wounds (caused by penetrating injuries)
 - ❖ Intrasplenic hematomas (caused by penetrating injuries)
 - ❖ Rupture of parenchyma (due to extreme friability and vascularity of the organ).

Symptoms

Symptoms and signs of shock and peritoneal irritation.

Signs

- Pain left upper abdomen with reference to the left shoulder (Kher's sign)
- Guarding and rigidity of the left hypochondrium
- Absent bowel sounds
- Shifting dullness

- A mass of percussible area of fixed dullness in the left hypochondrium (Ballance's sign)
- Lacerations may be seen on the left lower chest.

- The clinical presentation of splenic injury is of three types:
 1. *Acute*: Immediate presentation of symptoms
 2. *Delayed*: A quiescent period of 7 days to less than 2 weeks, between the injury and intraperitoneal bleeding (Latent period of Baudet), probably related to a temporary tamponade of a minor laceration or the presence of a slowly enlarging subcapsular hematoma which eventually ruptures
 3. *Occult*: Mild symptoms caused by rupture of organized intrasplenic or parasplenic hematoma.

Relevant Investigations

- Plain chest X-ray chest will demonstrate fracture of ribs on the left side
- Plain X-ray abdomen may show:
 - ❖ Haziness in the area of the spleen
 - ❖ Elevated immobile left dome of diaphragm
 - ❖ Medial displacement of gastric shadow with indentation
 - ❖ Obliteration of psoas shadow
 - ❖ Widening of space between splenic flexure and the preperitoneal pad of fat.
- CT (Figs 11.4A to C) and MRI are useful in localizing the damaged areas of spleen and collections of blood
- Peritoneal tap may be useful in identifying blood in the peritoneal cavity.

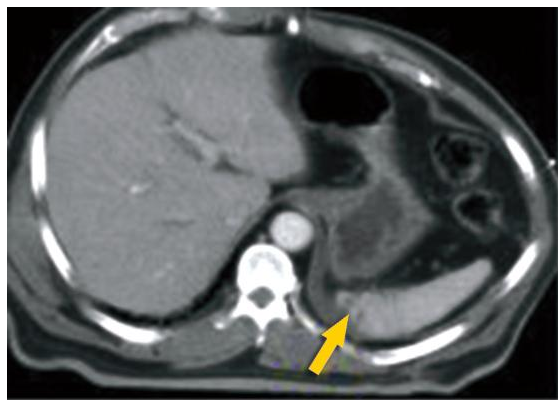


FIG. 11.4A: CT—Splenic injury with lower rib fracture

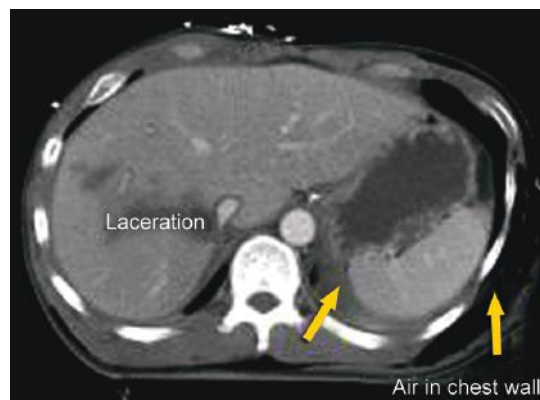


FIG. 11.4B: CT—Laceration of liver and perisplenic collection



FIG. 11.4C: CT—Combined renal and splenic injuries of automobile accident

Treatment

- Once the diagnosis is made the operation should not be delayed
- Splenectomy is the recommended treatment, regardless of the type and the extent of the injury.
- Following splenectomy, to prevent the life-threatening infections (pneumococci), long-term prophylaxis with penicillin (minimum of 2 years), with immunization against *Pneumococcus*, *Meningococcus* and *Hemophilus influenzae* is required.

INJURIES OF MESENTERY

Incidence and Etiology

- Mesentery consists of its arteries and veins and they may be injured by either penetrating or non-penetrating abdominal trauma
- In most cases, associated organ injuries are found
- Isolated injury to mesenteric vessels is rare.

Symptoms

Depending on the size of the vessel lacerated, the rapidity of bleeding and associated organ injury, the patient will present with signs of shock, abdominal pain and distension.

Sign

On examination, tenderness is marked.

When the bleeding occurs within the layers of mesentery, the clinical signs evolve slowly and the viability of the bowel is threatened.

Relevant Investigations

- Plain X-rays may show air under the diaphragm when the bowel is perforated by the injury
- Peritoneal tap may be useful in establishing the bleeding.

Treatment

- Repair of the torn mesentery is required
- Resection and anastomosis is required for nonviable intestine.

INJURIES OF DUODENUM

Incidence and Etiology

- Duodenum can be injured both by penetrating and nonpenetrating abdominal trauma
- The duodenum can rupture:
 - ❖ Intraperitoneally and cause immediate chemical irritation of the peritoneum due to the highly alkaline duodenal content
 - ❖ Retroperitoneally (more common with blunt trauma such as steering wheel injuries).

Symptoms and Signs

- Intraperitoneal rupture:
 - ❖ Abdominal pain, fever and distension with vomiting
 - ❖ Examination shows marked tenderness in the upper abdomen and later signs of generalized peritonitis.
- Retroperitoneal rupture:
 - ❖ Pain in the epigastrium and back, with pronounced vomiting
 - ❖ Testicular pain is a common feature of retroperitoneal rupture of duodenum.

Relevant Investigations

- Plain X-ray of abdomen may show air under the domes of diaphragm (intraperitoneal rupture) or large accumulation of air above the right kidney (retroperitoneal rupture). Diagnostic accuracy can be increased

by injecting air in the Levine's tube to increase the air collections. Water soluble dye injections in the tube can make the diagnosis more precise

- CT with contrast is conclusive
- Paracentesis may show bile-stained fluid, if the rupture is intraperitoneal.

Treatment

- Simple suturing may be adequate in many cases
- Supplementary gastroenterostomy is required for large tears
- Rarely, even a pancreatoduodenectomy may be necessary for extensive trauma involving the periampullary region.

INJURIES OF SMALL INTESTINE

Incidence and Etiology

- Injuries to the small intestine are more common than injuries to the duodenum and large intestine, the usual mechanism being the blunt trauma crushing the bowel against the vertebral column, more commonly the duodenojejunal flexure and the ileocecal junction, the fixed parts
- Blunt injuries cause slow necrosis of bowel and leak occurs late
- Signs and symptoms develop late—2 to 3 days later, depending on the size of the damage and leak of contents
- Penetrating injuries can also cause small bowel trauma, but less commonly, probably due to its sliding away from a knife because of its great mobility
- Associated mesenteric tears are common.

Clinical Presentation

- Abdominal pain, distension and vomiting
- Tenderness and guarding are pronounced around the damaged bowel and the patient may point it (Pointing sign).

Relevant Investigations

- Plain X-rays may show air under the domes of the diaphragm
- Paracentesis will show bile-stained fluid.

Treatment

- Simple suturing is done for simple tears
- Resections are required for large tears with nonviable bowel
- Peritoneal toileting is mandatory under cover of antibiotics.

INJURIES OF LARGE INTESTINE

Incidence and Etiology

- Large bowel injuries can be caused by penetrating and nonpenetrating injuries
- Ruptures may be:
 - ❖ Extraperitoneal for ascending and descending colon injuries, whereas
 - ❖ Intraperitoneal, when the injuries are of transverse and sigmoid colons
- Patient develops septic complications quickly as the large fluid leak is fecal and infected
- Delayed presentation is not uncommon as in small bowel injuries

Symptoms

- Abdominal pain, vomiting and distension
- High grade fever occurs due to fecal contamination.

Signs

Signs of peritonitis.

Relevant Investigations

- Plain X-rays may show air under the domes of the diaphragm (intraperitoneal ruptures)
- Paracentesis will show feculent fluid.

Treatment

- Early laparotomy is required
- Closure of tears with proximal diversion is necessary
- Peritoneal toileting is mandatory under cover of broad-spectrum antibiotics.

Incidence and Etiology

Occurs when the intra-abdominal pressure rises above the venous pressure.

This can be caused by:

- Blood
- Free gas
- Free fluid
- Tissue or splanchnic edema
- Organomegaly.

Pathogenesis

Occurs due to underperfusion of intra-abdominal organs (e.g. gut, kidneys and liver).

Symptom

Falling urinary output.

Signs

Tense and quiet abdomen.

Relevant Investigations

Measurement of intra-abdominal pressure by connecting the urinary catheter to a pressure transducer (>20 mm Hg suggests abdominal compartment syndrome).

Treatment

Surgical exploration

-
- Patients may bleed torrentially when the abdomen is opened followed by hemodynamic instability, following visceral reperfusion
 - Assisted ventilation may be required for many days.

Urological Injuries

RENAL INJURIES

Incidence and Etiology

- Kidneys are the most common organs to get injured in the urological system
- Injuries to the kidneys may be caused by:
 - ❖ Closed blunt abdominal trauma such as road traffic accidents, during active sports and may be associated with lower rib fractures
 - ❖ Penetrating injuries by bullets, knives are also causes of renal trauma
- Renal trauma is of three types:
 - ❖ Minor trauma (e.g. renal contusion, subcapsular hematoma)
 - ❖ Major trauma (e.g. deep cortical lacerations, extravasation of urine)
 - ❖ Renal vascular trauma (e.g. avulsion of renal pedicle).

Symptom

Hematuria is the most common symptom.

Signs

- Bruising over the ribs posteriorly or evidence of penetrating injury
- Tenderness and guarding in the loin and expanding mass
- Signs of shock may be seen in major trauma.

- Plain X-rays may show fractures of lower ribs
- CT is the investigation of choice and accurate assessment is possible (Figs 12.1 and 12.2)
- Renal arteriography may be needed in cases where CT is not contributory, especially with renal vessel injuries.



FIG. 12.1: CT—Injury of right kidney

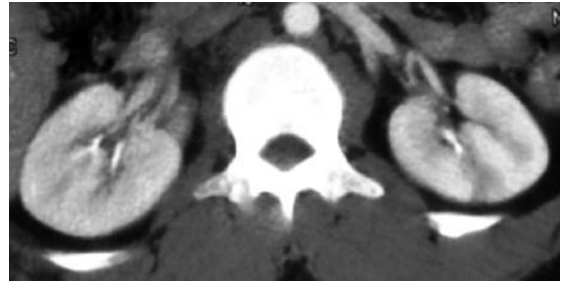


FIG. 12.2: Contrast enhanced CT—Left renal injury

Treatment

- Any patient with renal injury should rest in bed and all urine samples should be examined for blood
- Surgical exploration is warranted in closed injuries, when there is retroperitoneal bleeding, expanding loin mass, urinary extravasation, evidence of nonviable renal parenchyma and renal pedicle injuries
- Exploration of abdomen is required for penetrating injuries for assessment of other injuries and kidney should only be explored when the condition warrants.

URETERIC INJURIES

Incidence and Etiology

- Most common cause of ureteric injuries is during abdominal and pelvic operations
- Urinary leak occurs around the 5th postoperative day
- The operations associated with ureteric injuries are given in Table 12.1.

Table 12.1: Causes of ureteric injuries

Operations		
<i>Gynecology</i>	<i>General surgery</i>	<i>Urology</i>
Hysterectomy (abdominal or vaginal) – open or laparoscopic	Sigmoid colectomy	Excision of bladder diverticula
Ovarian cystectomy	Abdominoperineal resection	Ureterolithotomy
Anterior colporrhaphy	Surgery for aortic aneurysm	Ureteroscopy

Nature of Injuries

- Complete ligation of one or both ureters, leads to increase in intraureteric pressure, kidneys stop secreting urine, with resultant dilatation of pelvis and atrophy of kidney
- When the obstruction is incomplete (inclusion in a stitch), secretion continues to be normal, hydronephrosis and infection occur
- When the ureter is divided or suffers crushing injury, urine leaks into the retroperitoneum or peritoneal cavity which may result in a urinary fistula
- The injury may be recognized at the time of surgery
- If bilateral ligation is not recognized during surgery, it manifests as uremia.

Symptom and Sign

The clinical presentation varies according to the nature of injury (Table 12.2).

Table 12.2: Ureteric injuries and their clinical presentations

	<i>Nature of injury</i>	<i>Clinical presentation</i>
Symptoms	Bilateral ligation	Anuria in the immediate postoperative period
	Unilateral ligation	No symptoms/loin pain/fever
Signs	Division	Urine leak through the drain/wound/vagina
	Retroperitoneal urine leak	Abdominal distension and paralytic ileus
	Intraperitoneal urine leak	Abdominal distension and paralytic ileus with peritonitis and sepsis



FIG. 12.3: IVU—Leakage of contrast from damaged ureter

Relevant Investigation

IVU (Fig. 12.3) or contrast enhanced CT will demonstrate the nature of obstruction and urinary leak.

Treatment

- If injury is recognized during surgery, it should be rectified immediately:
 - ❖ Spatulation and end-to-end anastomosis is done, when the length is not lost
 - ❖ Incomplete damages to the ureter may be treated by stenting and spontaneous healing may be expected.
 - ❖ Surgical corrections (ureteroureterostomy, ureteroneocystostomy) are necessary to restore the anatomy, before renal function is compromised.

URINARY BLADDER INJURIES

Incidence and Etiology

- Urinary bladder is partially extraperitoneal and is also closely covered by the parietal peritoneum, and because of this anatomy, it can rupture:
 - ❖ Extraperitoneally (80%)
 - ❖ Intraperitoneally (20%)

Table 12.3: Etiologic factors of bladder injuries

Etiology (Trauma)	
<i>Extraperitoneal rupture</i>	<i>Intraperitoneal rupture</i>
Fracture pelvis	Blunt abdominal trauma with full bladder
Prostatic surgery	Penetrating injury
Difficult pelvic surgery	Injuries during therapeutic and surgical endoscopy

- Common in patients with abdominal trauma, who have a full bladder and are in a drunken state. The etiologies of these types vary. They are tabulated in Table 12.3.

Symptoms

- Severe lower abdominal pain with anuria
- Passage of blood stained urine is a common symptom.

Signs

- Distended abdomen with or without signs of peritonitis
- Catheterization is easy but no urine is drained, as there is leak
- Extraperitoneal leak is associated with pelvic fracture and causes tender suprapubic thickening.

Relevant Investigations

- Plain X-ray of abdomen will show ground glass appearance of the lower abdomen
- IVU, retrograde cystography (Figs 12.4A and B) or CT cystography are helpful in identifying the rupture and leak.

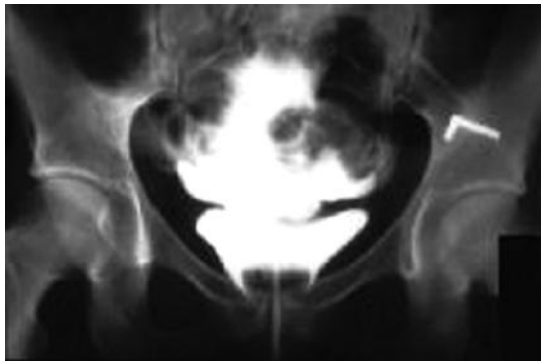


FIG. 12.4A: Cystogram—Intraperitoneal rupture of urinary bladder



FIG. 12.4B: Cystogram—Extraperitoneal rupture of urinary bladder

- Intraperitoneal rupture of bladder requires surgical exploration and repair
- Extraperitoneal rupture is managed by drainage of retropubic space with suprapubic drainage of bladder.

◆ URETHRAL INJURIES

Incidence and Etiology

- Male urethra is more prone to injury, and is commonly caused by instrumentation
- Pelvic fracture is another cause of urethral injuries
- Treated or untreated, urethral injuries may end in strictures
- Urinary incontinence and impotence are other complications.

Symptom

Blood at urethral meatus, hematuria and or anuria.

Sign

Distended bladder with tenderness on pelvic bones at the region of fracture, with perineal hematoma.

In rupture of membranous part of urethra, prostate is high riding.

Relevant Investigation

Retrograde urethrography (Fig. 12.5) will be informative (catheterization should be avoided).



FIG. 12.5: Retrograde urethrography showing leak of contrast through urethral fistula

Treatment

- Anterior urethral injuries:
 - ❖ *Complete*: Primary repair
 - ❖ *Incomplete*: Catheterization and allow it to heal spontaneously
- Posterior urethral injuries: Suprapubic drainage and urethral catheterization by rail-road method initially and delayed perineal end-to-end urethroplasty.

Male Genital Injuries

INJURIES OF PENILE SKIN

Incidence and Etiology

- In adults, the injury to the penile skin occurs due to:
 - ❖ Direct injury during a fall
 - ❖ Assault or entrapment of clothing in machinery
 - ❖ In people who indulge in perverted sex or during masturbation with metal objects.
- Children can injure the penis by pranky acts and cause damage to the penile shaft and skin (Fig. 13.1A).

Symptom

Profuse bleeding from the wound.

Sign

The penile skin is lacerated in an irregular fashion (Fig.13.1B)

Relevant Investigation

No specific investigation is necessary.

Treatment

Immediate surgical repair is necessary.



FIG. 13.1A: Gangrene of glans penis due to tying of hair around it



FIG. 13.1B: Laceration penile skin
(*Courtesy: Dr MG Rajamanickam*)

FOREIGN BODIES IN PENILE URETHRA

Incidence and Etiology

Insertion of foreign bodies into the urethra is an unusual but a documented activity, especially by those who are mentally disturbed and in those involved in perverted sex. A wide variety of objects have been documented to be in the urethra, such as batteries, safety pins, telephone wire, ballpoint pen, etc.

Symptoms

Lower urinary tract symptoms (LUTS) – Irritation, pain, and hematuria. Urinary retention is possible in total obstruction of the urethra.

Signs

- Blood at the tip of the penis
- The foreign body may be felt at the undersurface of urethra (objects above the urogenital diaphragm may not be palpable)
- Foreign body at the urethral meatus can be seen directly (Fig 13.2)
- Distended urinary bladder in total obstruction of urethra
- Diagnosis is by clinical history (proper history may not be available in mentally disturbed patients).



FIG. 13.2: Foreign body (coconut kernel) in anterior urethra
(Courtesy: Dr N Mohan)

Relevant Investigations

- Urine examination – gross or microscopic hematuria
- X-ray – radiopaque foreign bodies can be identified
- Urethroscopy will reveal foreign bodies in the proximal urethra
- US, IVU and cystogram may reveal radiolucent objects.

Treatment

- Endoscopic manipulation and extraction using forceps, snares, balloon wires and retrieving baskets
- Urethrotomy may be needed when endoscopic procedures are unsuccessful.

SCROTAL LACERATION

Incidence and Etiology

It occurs commonly following:

- Blunt injury (road traffic accident)
- A fall
- Gunshot injuries.



FIG. 13.3: Laceration of penis and scrotum with exposure of left testis

- Intra-abdominal, chest and head injuries may be associated
- The tunica albuginea of testis may be damaged with loss of testicular substance.

Symptom

Profuse bleeding.

Signs

- The laceration may be confined to a small area of the scrotum
- Sometimes, there may be loss of entire scrotal skin with exposure of the testis (Fig. 13.3).

Relevant Investigation

No special investigation is necessary.

Treatment

- Cleaning and primary repair of simple tears
- Placing the exposed testis in the subcutaneous plane of the thigh, and dressings for complex injuries
- Skin grafting and, or scrotal reconstruction may be needed at a later date
- Any tear in the tunica albuginea needs repair to prevent loss of testicular substance.

Female Genital Injuries

COITAL INJURIES

Incidence and Etiology

Injuries commonly occur during forced intercourse and sometimes at the time of first intercourse.

Symptom

Vulval or vaginal bleeding.

Signs

Local examination reveals irregular abrasions in the vulva or vagina.

Relevant Investigations

Local examination is conclusive and no special investigation is necessary.

Treatment

- Application of pressure with gauze pack is effective in controlling most bleeds
- Suturing is done for larger bleeds
- Uncontrolled bleeding should prompt a search for coagulation disorders.

INJURIES DURING CHILDBIRTH

Incidence and Etiology

Injuries of the genital tract occur during childbirth, especially due to:

- Delivery of large babies
- Tight introitus
- Deliveries conducted with the aid of instruments.

Clinical Presentation

- Bleeding from the wound
- Local examination reveals a laceration.

Relevant Investigations

No special investigation is necessary.

Treatment

Primary repair of the tear with correct approximation of layers is mandatory as healing by secondary intention weakens the area resulting in anal or urinary incontinence or uterine prolapse.

Hand Injuries

Incidence and Etiology

- Hand injuries form nearly one-third of all injuries to human body
- They can be:
 - ❖ Open
 - ❖ Closed
- Acute injuries can be classified depending on the nature of force and on contamination as tidy, untidy and indeterminable injuries:
 - ❖ *Tidy injuries*: Clean cuts and incised wounds, usually resulting from sharp force
 - ❖ *Untidy injuries*: Ragged skin wounds, may be with associated fractures, usually resulting from crush or avulsion force
 - ❖ *Indeterminable injuries*: Injuries where it is difficult to determine the extent of injury and the viability of tissues, usually caused by very severe force
- Injuries to the vessels, nerves, bones and joints may be present in any of the above injuries.

Complications: Post-traumatic deformities like contractures, neurovascular deficit.

Clinical Presentation

The patient presents to the surgeon either in the acute stage or late.

- In acute injuries (Figs 15.1A to C), the pain is excruciating, and assessment is difficult, unless pain is alleviated by analgesics. When patients arrive late, infection gets superadded, and result in post-traumatic sequelae.



FIG. 15.1A: Traumatic amputation of tip of middle finger



FIG. 15.1B: Traumatic amputation of entire index finger



FIG. 15.1C: Penetrating injury of three fingers

- ❖ *Nail injury:* Avulsion of nail (complete or partial) (Fig. 15.2A)
- ❖ *Skin damage:* Loss of skin, especially degloving
- ❖ *Nerve damage:* Sensory or motor loss depending on the nerve affected
- ❖ *Damage to arteries:* Suspected by profuse hemorrhage (incomplete tears), gangrene (complete injuries) (Figs 15.2B to D)
- ❖ *Damage to tendon sheaths and tendons:* Lack in active movement
- ❖ *Damage to bones and joints:* Pain, deformity, tenderness and loss of movement.



FIG. 15.2A: Avulsion of nails by crush injury



FIG. 15.2B: Traumatic gangrene of middle finger



FIG. 15.2C: Gangrene of fingertip following crush injury



FIG. 15.2D: Traumatic gangrene of little finger

In acute injuries, for assessment of damage, surgical exploration may be necessary when in doubt and it should be done meticulously. The structures should be considered divided until otherwise proved.

- In late post-traumatic cases detailed examination of movements and sensation can be done with ease, and damage assessed completely.
 - ❖ *Skin injuries:* Necrosis of skin, may manifest as raw area, when very late, can present with contracture due to secondary healing.
 - ❖ *Vascular injuries:* Necrosed skin and deeper tissues



FIG. 15.3A: Boutonnière deformity of little finger

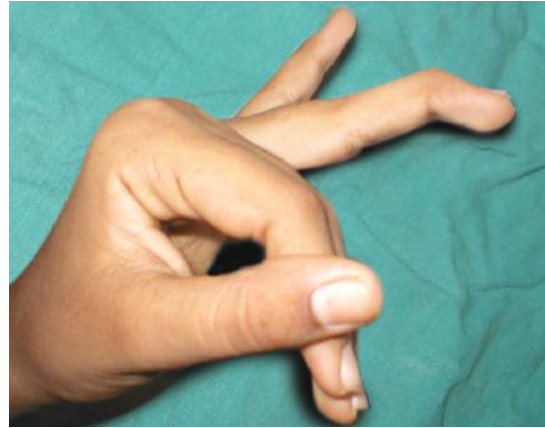


FIG. 15.3B: Mallet finger



FIG. 15.3C: Swan-neck deformity

- ❖ *Extensor tendon injuries:* Dropped finger (if injury is at dorsum or proximal level), Boutonnière deformity (when injury is at proximal finger level) (Fig. 15.3A) and Mallet finger (when injury is at distal finger level) (Fig. 15.3B). Neglected or untreated Mallet fingers may progress to Swan-neck deformity (Fig. 15.3C).

- ❖ *Flexor tendon injuries:* Lack of movement of PIP joint suggest flexor digitorum superficialis injury and DIP joint suggests flexor digitorum profundus injury
- ❖ *Nerve damage:* Wasting of muscle groups, lack of sweating and trophic changes
- ❖ *Bone or joint injuries:* Fractures with or without dislocations of joints.

Relevant Investigations

- X-rays (in various views) are necessary to assess bone injuries
- Doppler studies are required to assess vascular damage.

Treatment

- Bleeding can be controlled by elevating the limb or by applying pressure directly over the site with pads
- In acute injuries:
 - ❖ *Skin injuries:* Primary repair wherever possible
 - ❖ *Vascular injuries:* Primary microvascular repair
 - ❖ *Bone and joint injuries:* Proper debridement and splinting, reduction of dislocations and immobilization
 - ❖ *Nerve injuries:* Primary repair under magnification where possible
 - ❖ *Tendon injuries:* Primary repair in clean wounds.
- In late post-traumatic cases:
 - ❖ *Skin injuries:* Wounds healing by secondary intention cause severe fibrosis and contractures, and may need release and repair and skin grafting in some cases
 - ❖ *Vascular injuries:* Grafts wherever required
 - ❖ *Bone and joint injuries:* Malunion or nonunion should be treated accordingly
 - ❖ *Nerve injuries:* Nerve grafts
 - ❖ *Tendon injuries:* Tendon transfers or grafts.

Note: Dirty wounds should be debrided well and converted into a clean wound and further treatment is carried out.

Section V

Organs and Systems

- 16. Oral Cavity
 - Acute Odontogenic Infections
- 17. Head and Neck
 - Acute Suppurative Sialadenitis
 - Ludwig's Angina
- 18. Thorax
 - Acute Mediastinitis
 - Acute Pleuritis
 - Pleural Effusion
 - Acute Empyema Thoracis
 - Spontaneous Pneumothorax
 - Foreign Bodies in the Respiratory Tract
 - Suppurative or Aspiration Pneumonia
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 - Pulmonary Embolism
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- 20. Spine
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- 21. Gastroenterology
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 - Acute lower abdominal pain
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 - Primary Sclerosing Cholangitis
 - Splenic Abscess
 - Acute Hemorrhagic Pancreatitis
 - Acute Appendicitis
 - Acute Mesenteric Lymphadenitis
 - Acute Colonic Diverticulitis
 - Acute Meckel's Diverticulitis
 - Acute Solitary Cecal Diverticulitis
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 - Acute Intestinal Obstruction
 - Gallstone Ileus
 - Acute Intussusception
 - Swallowed Foreign Bodies
 - Perforated Bowel Pathologies

- Intestinal Strictures
 - Bands and Adhesions
 - Enteroliths/Food Bolus
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 - Sigmoid volvulus
 - Cecal volvulus
 - Midgut volvulus
 - Gastric volvulus
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 - Paralytic Ileus
 - Torsion of Mesenteric Cyst
 - Torsion of Omentum
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 - Hemorrhoids
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 - Furuncle
 - Cellulitis
 - Acute Pyogenic Abscess
 - Carbuncle
 - Burns
 - Necrotizing Fasciitis

Oral Cavity

ACUTE ODONTOGENIC INFECTIONS

Incidence and Etiology

Acute odontogenic infections present in three ways:

- i. Abscess
- ii. Cellulitis
- iii. Fulminating infections.

The odontogenic abscesses present in many ways:

- Acute periapical abscess (cause – Infective necrosis of pulp)
- Acute dentoalveolar abscess
- Acute periodontal abscess
- Acute pericoronal abscess.

Causative organisms

- Staphylococci
- Streptococci
- Bacteroides.

Pathogenesis

The infection follows the path of least resistance, the deciding factor being the fascial planes:

- Mandibular 2nd and 3rd molar infections perforate the lingual cortex and spread to submandibular space, as the roots of these teeth lie below the mylohyoid line

- Mandibular premolars and first molars, involve the sublingual space, as the roots of these teeth lie above the mylohyoid line.

Spread of Infection

- *Acute periapical abscess (cause–Infective necrosis of pulp)*: Organisms from infected pulp invade periapical tissue through apical foramina
- *Acute dentoalveolar abscess*: Continuation of periapical abscess
- *Acute periodontal abscess*: Arises in the periodontal membrane adjacent to a periodontal pocket
- *Acute pericoronal abscess*: Arises around the crown of impacted or erupting teeth.

Symptoms and Signs

- *Acute periapical abscess*: Severe throbbing pain in the affected tooth which may be carious
- *Acute dentoalveolar abscess*: Severe pain, submucosal and or extraoral swelling
- *Acute periodontal abscess*: Dull pain, rarely severe, pus discharge via gingival pocket
- *Acute pericoronal abscess*: Dull continuous pain, swelling of gingiva around the crown with or without pus discharge.

Relevant Investigations

Diagnosis is more clinical. X-rays are not diagnostic.

Treatment

- *Medical*: Hydration, soft diet, analgesics, mouthwashes, broad-spectrum antibiotics
- *Surgery*: Extraction of affected tooth or incision and drainage of abscess when possible
- *Conservative*: Root canal treatment for acute periapical abscess.

Head and Neck

ACUTE SUPPURATIVE SIALADENITIS

Incidence and Etiology

- Occurs generally in:
 - ❖ People with poor oral hygiene
 - ❖ Postoperative patients
 - ❖ Dehydration and reduced or obstructed salivary flow
 - ❖ By ascending infection from oral flora, usually *Streptococcus viridans* or pneumococci
- Parotid gland is usually involved
- Submandibular gland may also be involved.

Clinical Presentation

- Acute parotitis:
 - ❖ Painful unilateral parotid swelling with trismus, fever and tachycardia
 - ❖ On examination, the parotid is diffusely enlarged as a brawny swelling (Fig. 17.1) and extremely tender with purulent discharge through the Stenson's duct.
- Acute submandibular sialadenitis:
 - ❖ Painful swelling of the submandibular gland
 - ❖ On examination, the gland is enlarged and tender (Fig. 17.2) purulent discharge through the Wharton's duct
 - ❖ Obstructing calculus may be seen or felt in the floor of the mouth (Fig. 17.3).



FIG. 17.1: Acute suppurative parotitis

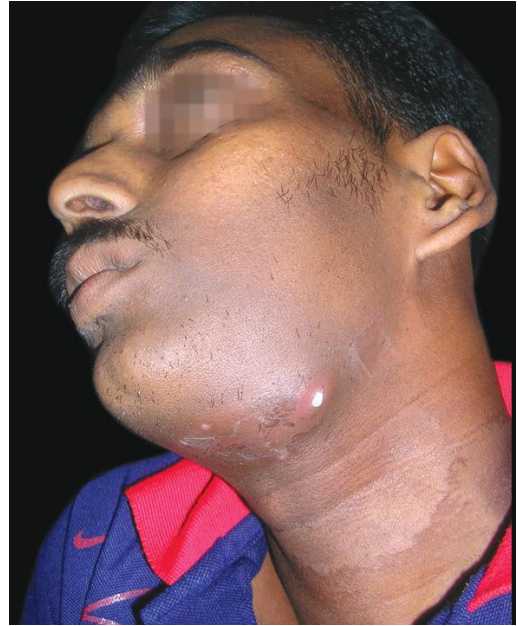


FIG. 17.2: Submandibular salivary gland abscess



FIG. 17.3: Calculus in the Wharton's duct (*Courtesy: Dr Jacinth Cornelius*)

Relevant Investigation

No special investigation is required.

Treatment

- Broad-spectrum antibiotics are necessary
- If abscess is formed, external drainage is necessary without waiting for fluctuation to establish.

LUDWIG'S ANGINA

Incidence and Etiology

- Usually due to dental infections
- The infection is a mixture of aerobic and anaerobic organisms.

Pathogenesis

It is cellulitis involving the sublingual and submandibular spaces beneath the deep cervical fascia.

Complication: Edema of glottis.

Symptoms

- Severe pain and swelling of neck (Fig. 17.4)
- May cause respiratory embarrassment.

Signs

- Severely swollen neck
- Marked tenderness.

Relevant Investigation

Culture of pus and identifying the organism.

Oral cavity examination is mandatory.



FIG. 17.4: Ludwig's angina

Treatment

- Broad-spectrum antibiotics
- Incision and drainage, if abscesses are found in the dental planes, with or without dental extractions.

Thorax

ACUTE MEDIASTINITIS

Incidence and Etiology

Acute suppurative mediastinitis occurs due to varied causes. They are:

- Injury to cervical or thoracic esophagus
- Extension of cervical infections
- Secondary to osteomyelitis of ribs and sternum
- Extension of retroperitoneal infections
- Suppuration of mediastinal lymph nodes.

Clinical Presentation

- Chest pain, fever and chills are commonly present
- Symptoms related to primary cause are usually present.

Relevant Investigations

- X-ray chest (Fig. 18.1) shows mediastinal widening
- CT chest is diagnostic, especially of the primary cause.



FIG. 18.1: X-ray—Superior mediastinitis

Treatment

- Broad-spectrum antibiotics are necessary
- Immediate surgery with adequate mediastinal drainage for esophageal injuries.

ACUTE PLEURITIS

Incidence and Etiology

- A simple term denoting the pleural involvement of any disease giving rise to pleuritic pain or evidence of pleural friction
- Common feature of pleural invasion by pulmonary tuberculosis or bronchogenic carcinoma.

Symptom

Pain characteristically inspirational.

Sign

Chest movement may be restricted locally with an audible pleural rub.

Relevant Investigations

X-ray chest is useful in identifying the underlying cause.

Treatment

Analgesics are required to treat pleuritic pain with treatment focused towards the primary cause.

PLEURAL EFFUSION

Incidence and Etiology

Denotes accumulation of serous fluid in the pleural space, which results due to:

- Increased hydrostatic pressure or decreased osmotic pressure (transudative effusion). The causes are:
 - ❖ Cardiac failure
 - ❖ Hepatic failure
 - ❖ Renal failure
- Increased microvascular permeability due to disease of pleura or adjacent lung (exudative effusion). The causes are:
 - ❖ Infections (e.g. bacterial, tuberculous, fungal, parasitic, viral)
 - ❖ Collagen vascular disease (e.g. rheumatoid, lupus)
 - ❖ Malignancy (e.g. mesothelioma, lung cancer, metastases)
 - ❖ Pulmonary embolism
 - ❖ Abdominal disease (e.g. pancreatitis, subphrenic abscess)
- The effusion can be:
 - ❖ Unilateral (e.g. tuberculosis, malignancy)
 - ❖ Bilateral (e.g. cardiac failure, hypoproteinemia).

Symptom

Breathlessness is the most common symptom.

Signs

- Reduced chest wall movement on the affected side, dullness on percussion and reduced or absent breath sounds and vocal resonance
- Large effusions shift the trachea to the opposite side.



FIG. 18.2A: X-ray chest—Left pleural effusion

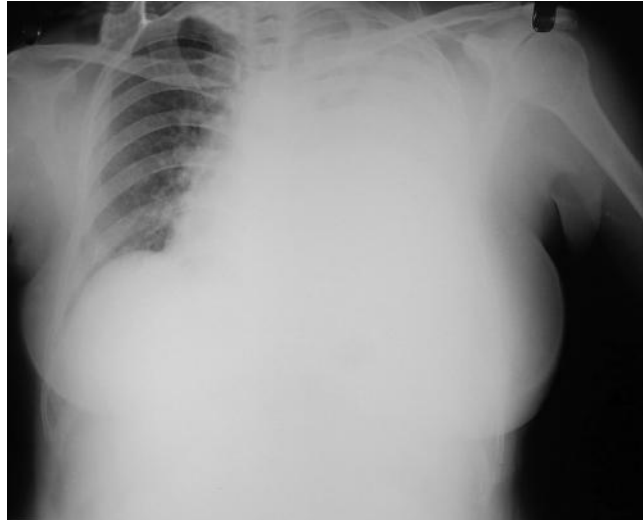


FIG. 18.2B: X-ray chest—Massive pleural effusion left side

Relevant Investigations

- Chest X-ray shows a dense uniform opacity in the lower and lateral parts of hemithorax, shading off above and medially into translucent lung (more than 400 ml is required to blunt the costodiaphragmatic angle) (Figs 18.2A and B)
- US is very useful in differentiating the effusion and tumor
- CT (Fig. 18.3) is diagnostic
- Aspiration of pleural fluid is necessary for:
 - ❖ Nature (blood – malignancy, embolus; milky – chyle; pus – empyema; straw color – tuberculosis and transudates)
 - ❖ Microbiology – culture sensitivity
 - ❖ Cytology
 - ❖ Biochemistry (Total proteins >3 g% - exudate, pleural LDH/serum LDH ratio >0.6 – exudates, high levels of triglycerides >110 mg% - chylothorax, amylase – pancreatitis, malignant effusions)
- Bronchoscopy biopsy, thoracoscopy and biopsy of enlarged lymph nodes may be needed to identify the primary cause
- Pleural biopsy yields good results.



FIG. 18.3: CT—Right sided pleural effusion

Treatment

Aspiration of pleural fluid may be necessary to relieve breathlessness, but treatment of underlying cause is necessary.

ACUTE EMPYEMA THORACIS

Incidence and Etiology

- Empyema indicates pus in the pleural space, which may involve a part (loculated) or the whole of it
- Always secondary to infection in a neighboring structure, usually the lung (bacterial pneumonia and tuberculosis)
- Other causes being, infection of hemothorax or rupture of subphrenic abscess or liver abscess.

Complications: Bronchopleural fistula, empyema necessitans, (empyema burrowing through the chest wall to present as an abscess externally), pleurocutaneous fistula, pericarditis, mediastinal abscess.

Symptom

High intermittent pyrexia, with rigors, sweating, malaise and weight loss.

Signs

- Pleural pain, breathlessness and cough with purulent sputum
- Clinical signs are those of pleural effusion.

Relevant Investigations

- Chest X-ray is diagnostic
- US and CT give invaluable information
- Aspiration of pus is confirmatory
- Isolation of organism from sputum or pleural aspirate is useful in treatment.

Treatment

- General: Supportive respiratory care, physiotherapy
- Drainage of pleural space (thoracentesis or close drainage) and irrigation with normal saline is necessary to clear the pleural space of the pus, under cover of appropriate antibiotics
- Underlying cause like pneumonia and tuberculosis need active treatment.

SPONTANEOUS PNEUMOTHORAX

Incidence and Etiology

- Pneumothorax is presence of air in the pleural cavity
- This is a sudden event
- Spontaneous pneumothorax can be:
 - ❖ Primary—without any obvious evidence of pulmonary pathology (e.g. rupture of small emphysematous bulla)
 - ❖ Secondary—due to underlying pulmonary pathology (e.g. COPD, tuberculosis).

Symptom

Sudden unilateral chest pain or breathlessness.

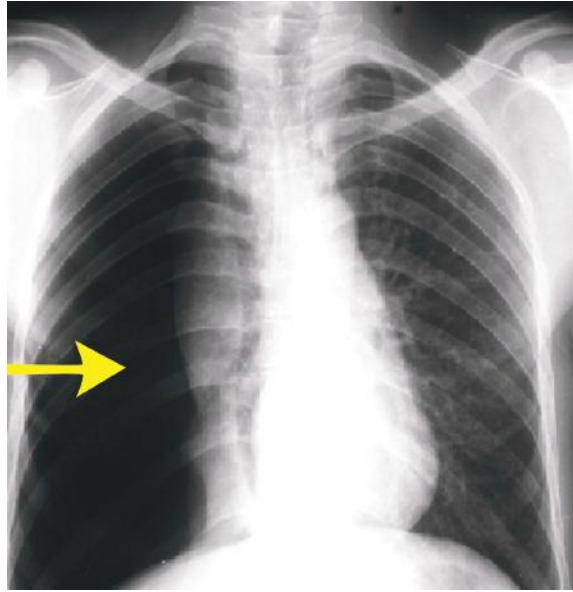


FIG. 18.4: Chest X-ray—Right pneumothorax

Sign

Small pneumothorax shows no clinical signs, whereas, large ones show decreased movement of chest wall, hyper-resonant percussion note and decreased or absent breath sounds.

Relevant Investigations

- Chest X-ray (Fig. 18.4) shows sharply defined edge of the deflated lung with complete translucency between the line and the chest wall with no lung markings
- CT is useful in defining the underlying pathology.

Treatment

- Percutaneous needle aspiration of air is necessary for full lung expansion
- Intercostal tube drainage with underwater seal may be needed [See Chapter 31 (Insertion of Chest Drains)]
- Smoking should be completely avoided and the underlying pathology treated.

FOREIGN BODIES IN THE RESPIRATORY TRACT

Incidence and Etiology

- Foreign bodies get lodged in the trachea and bronchi accidentally, and are more common in:
 - ❖ Children than in adults
 - ❖ The bronchus than the trachea
 - ❖ The right bronchus than the left, as the right bronchus is a direct continuation of trachea and is wider than the left bronchus.
- In children the objects are in the form of seeds, buttons, cells, etc. and in adults, they are dentures, loose teeth, etc.
- In adults, accidental lodging when they are under the influence of alcohol.

Pathogenesis

- Foreign bodies act like a valve and cause symptoms (Table 18.1).

Symptoms

Dyspnea, cough, stridor, cyanosis and fever.

Signs

- Rhonchi
- Reduced breath sounds.

Relevant Investigations

- X-rays reveal the radio-opaque foreign bodies, and collapsed lungs
- Bronchoscopy is diagnostic (Figs 18.5 and 18.6).

Table 18.1: Pathogenesis of foreign body obstruction in lower respiratory tract

<i>Mechanism</i>	<i>Pathophysiology</i>
Stop valve	Foreign body causes total obstruction and does not allow air entry (ingress) or allow air and secretions to escape (egress). The lobe of lung may collapse and consolidate
Bypass valve	When the foreign body is small or has a hole, it allows ingress and egress of air
Ball valve	During inspiration, the bronchi dilate allowing air to enter (ingress), but does not allow air to escape (egress) as the bronchi constrict during expiration. The trapped air caused pneumothorax or emphysema

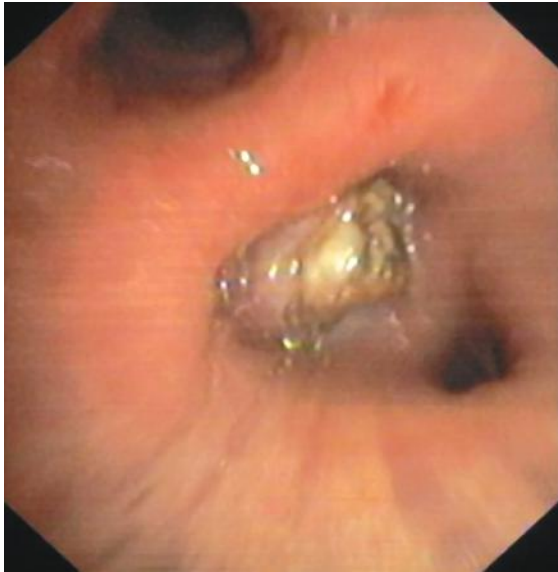


FIG. 18.5: Bronchoscopy—Tooth in the bronchus
(*Courtesy:* Dr R Narasimhan and Dr Gayathri)

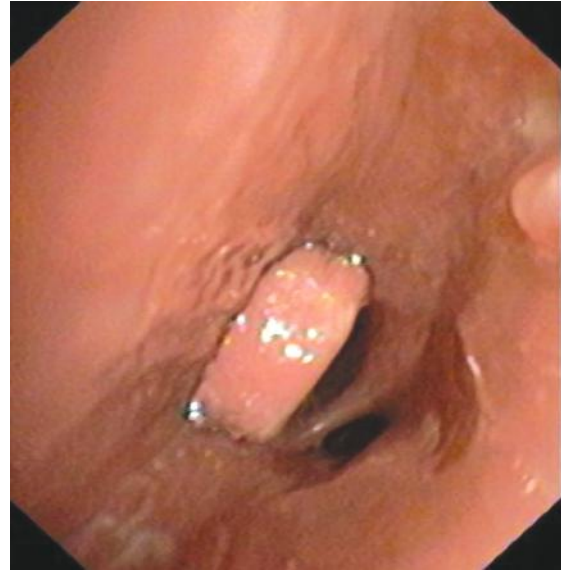


FIG. 18.6: Bronchoscopy—Tablet in the bronchus
(*Courtesy:* Dr R Narasimhan and Dr Gayathri)

Treatment

Bronchoscopic removal (using a rigid bronchoscope) is curative.

SUPPURATIVE OR ASPIRATION PNEUMONIA

Incidence and Etiology

- Suppurative pneumonia or pneumonic consolidation denotes destruction of lung parenchyma by inflammatory process with microabscess formation on histology
- *Staphylococcus aureus* and *Klebsiella pneumoniae* infections cause suppuration
- Suppuration has an insidious onset whereas aspiration is acute.

Pathogenesis

- Inhalation of septic material during endotracheal anesthesia or by aspiration of gastric contents
- Aspiration may lead to severe acute respiratory distress syndrome (ARDS).

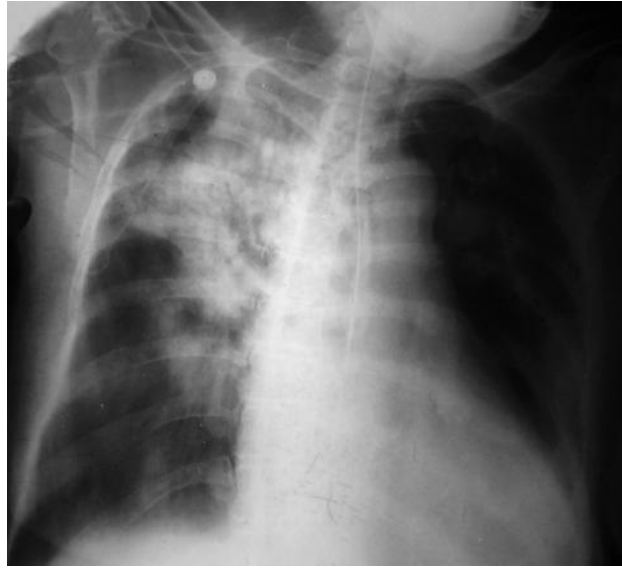


FIG. 18.7: X-ray—Aspiration pneumonia right lung

Symptoms

- Productive cough (fetid or blood stained)
- Pleural pain may be present
- High remittent pyrexia is common.

Sign

Sign of consolidation, with pleural rub.

Relevant Investigation

Chest X-ray (Fig. 18.7) shows homogeneous lobar or segmental opacity. A cavity with fluid level may indicate an abscess.

Treatment

- Broad-spectrum antibiotics form the mainstay of treatment
- Surgical intervention may be required for abscess, which does not respond to medical therapy.

HOSPITAL ACQUIRED PNEUMONIA

Incidence and Etiology

- Refers to a new episode of pneumonia 2 to 3 days after admission to hospital.
- The predisposing factors for the development of pneumonia are:
 - ❖ Immunocompromised state (e.g. corticosteroid treatment, malignancy, AIDS)
 - ❖ Reduced cough reflex (e.g. postoperative)
 - ❖ Aspiration of gastric contents (e.g. vomiting, severe reflux, nasogastric intubation)
 - ❖ Endotracheal intubation (e.g. mechanical ventilation)
 - ❖ Bacteremia (e.g. sepsis).

Symptoms

- Cough with purulent expectoration is the predominant symptom
- Breathlessness will appear soon.

Signs

- Cyanosis
- Crepitations are heard on auscultation.

Relevant Investigations

- Leukocytosis is present
- Chest X-ray will show mottled opacities in both lung fields.

Treatment

- Broad-spectrum intravenous antibiotics are necessary
- Physiotherapy is mandatory in immobile patients.

ACUTE LUNG ABSCESS

Incidence and Etiology

- A suppurative focus within the lung associated with necrosis
- The bacteria responsible for a lung abscess may reach the lung through various routes. They are:
 - ❖ Aspiration down the tracheobronchial tree (e.g. bronchiectasis)
 - ❖ By blood (e.g. septicemia)
 - ❖ Trauma (e.g. penetrating trauma)
 - ❖ Extension of adjacent suppurative focus (e.g. liver abscess).

- Other causes are:
 - ❖ Tumors of bronchus (e.g. bronchogenic carcinoma)
 - ❖ Suppuration of hematoma (e.g. penetrating trauma).

Symptoms

- Appear acutely ill
- Cough with foul smelling purulent expectoration
- Hemoptysis may occur
- Fever
- Breathless
- Chest pain.

Sign

Crepitations may be heard.

Relevant Investigations

X-ray and CT are diagnostic.

Treatment

- Antimicrobial therapy resolves most abscesses
- Surgical intervention (lobectomy or segmentectomy), is reserved for those which do not respond.

PULMONARY EMBOLISM

Incidence and Etiology

Majority of pulmonary emboli result from deep venous thrombosis of lower limbs, and they can be acute (minor and massive) or chronic.

Symptoms and Signs

The symptoms and signs are tabulated in Table 18.2.

Table 18.2: Symptoms and signs of pulmonary embolism

Type	Symptoms	Signs
Acute minor	Shortness of breath and hemoptysis – pleurisy	Pleural rub and signs of pleural effusion
Acute massive	Central chest pain, apprehension, low cardiac output and syncope	Sinus tachycardia, hypotension and peripheral vasoconstriction
Chronic	Exertional dyspnea, syncope and chest pain over months and years	Pulmonary hypertension, loud pulmonary component of second heart sound and a right ventricular heave.

Relevant Investigations

Chest radiographs, ECG, arterial blood gases, ventilation-perfusion lung scanning and pulmonary angiography are useful investigations.

Treatment

- *General:* Opiates to relieve pain and distress, resuscitation by external cardiac massage, with oxygen support
- Anticoagulation is necessary atleast for 5 days
- Thrombolytic therapy is used in acute massive types.

Breast

BREAST HEMATOMA

Incidence and Etiology

Associated with history of trauma such as seatbelt injury during a road traffic accident, or following a violent contraction of pectoralis muscles responding to a blow.

Symptoms

- Pain in the breast
- History of trauma.

Sign

Presence of bruise over the breast may be the contributory finding for diagnosis.

Relevant Investigations

Fine needle aspiration cytology (FNAC) and mammography are needed to rule out malignancy.

Treatment

Exploration and histopathological confirmation.



FIG. 19.1: Acute breast abscess

ACUTE BREAST ABSCESS

Incidence and Etiology

Collection of pus resulting from mastitis.

Symptoms

- Starts with dull ache, which proceeds on to throbbing pain
- Systemic manifestations like fever and malaise may develop.

Signs

- Superficial abscesses may show as fluctuant tender lumps
- Deep abscesses may show as severe cellulitis with edema of breast (Fig. 19.1) without fluctuation.

Relevant Investigations

- Polymorphonuclear leukocytosis is generally present
- Diagnostic aspiration may confirm the presence of pus.

Treatment

- Surgical drainage gives full relief
- Spontaneous or inadequate drainage may result in the formation of antibioma, chronic abscess, mammary fistula with purulent or sero sanguinous discharge.

Spine

DEGENERATIVE DISEASES OF DISK AND FACET JOINTS

Incidence and Etiology

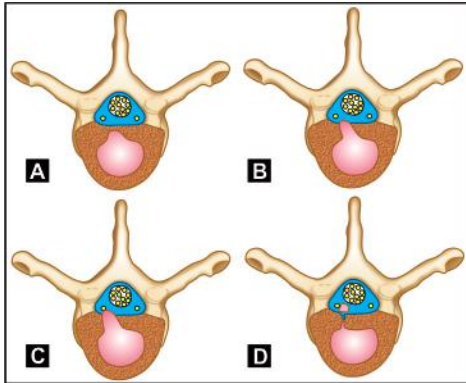
Degenerative diseases of the spine are very common. They affect the lumbar, cervical and thoracic regions in decreasing order of frequency.

- Lumbar degenerative disease:
 - ❖ Prolapse of intervertebral disk: Disks between L5/S1 and L4/L5 are most often affected. Part of the gelatinous nucleus pulposus protrudes through the rent in the annulus fibrosus at its weakest part, which is posterolateral and compress the nerve roots (compressive radiculopathy) (Figs 20.1A to D). Occasionally, a large central disk prolapse at L5/S1 compresses the cauda equina (Cauda equina syndrome).
 - ❖ Facet joint disease: Degenerative changes cause root compression.
 - ❖ Lumbar canal stenosis: Increasing wear and tear, bony overgrowth and ligamentous hypertrophy can cause narrowing of the spinal canal.
- Cervical degenerative disease: Pathogenesis is the same as in lumbar disease.

Cauda equina syndrome consists of back pain, bilateral sciatica and urinary retention.

Symptoms and Signs

- Lumbar degenerative disease: Back pain is the constant feature, but varies according to the compression of the root:



FIGS 20.1A to D: Stages of prolapse of intervertebral disk: (A) Torn annulus fibrosus with disk bulge; (B) Extrusion of nucleus pulposus protrusion not pressing the nerve; (C) Extrusion of nucleus pulposus protrusion pressing the nerve; (D) Sequestration of disk



FIG. 20.2: MRI—Prolapse of L4-L5 intervertebral disk

- ❖ Prolapse of intervertebral disk: Back pain, radiating to
 - Inner thigh (L4 root compression—L3/L4 prolapse)
 - Outer aspect of the leg to the big toe (L5 root compression—L4/L5 prolapse)
 - The sole of foot (S1 root—L5/S1 prolapse), is the presenting symptom.

Acute radiating pain and inability to move, aggravated by coughing and sneezing is the symptom of acute disk prolapse. Straight leg raising test is positive on the affected side.

- ❖ Facet joint disease presents with pain similar to the synovial joint pain (more after a period of rest like early mornings and becomes less after movement)
- ❖ Lumbar canal stenosis causes numbness and weakness of lower limbs on walking.
- Cervical degenerative disease: The presenting symptom is neck pain related to movements with radiculopathy.

Relevant Investigations

CT and MRI (Fig. 20.2) are diagnostic.

Treatment

- Acute attacks require rest and analgesics, followed by physiotherapy
- Chronic, persistent or progressive symptoms require surgery to remove the prolapsed disk or decompressive laminectomy.

SPONDYLOLISTHESIS

Incidence and Etiology

- This term signifies slipping of one vertebra over the lower, which is usually forwards, but may be backwards. In the normal spine, forward displacement of a vertebral body is prevented by engagement of the articular processes with those of the segment next below it, and this check mechanism is weak in spondylolisthesis
- The displacement is most common in the 5th lumbar vertebra, and the displacement may increase slowly over years, and reach a severe degree of spondyloptosis.
- The predisposing factors are:
 - ❖ Spondylolysis (a defect in the pars interarticularis of the neural arch)
 - ❖ Osteoarthritis of the posterior (facet) joints (degenerative)
 - ❖ Congenital malformation of the articular processes (rare).

Symptom

Chronic backache with or without sciatica.

Signs

- A step above the sacral crest
- Lumbar vertebral bodies may be felt per abdomen due to its forward displacement
- Straight leg raising (SLR) test may be positive.

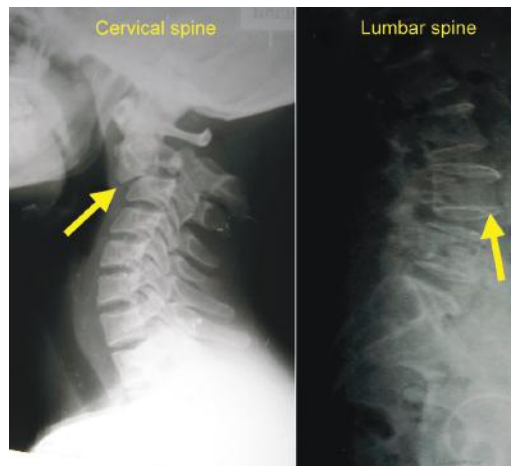


FIG. 20.3: X-rays—Spondylolisthesis

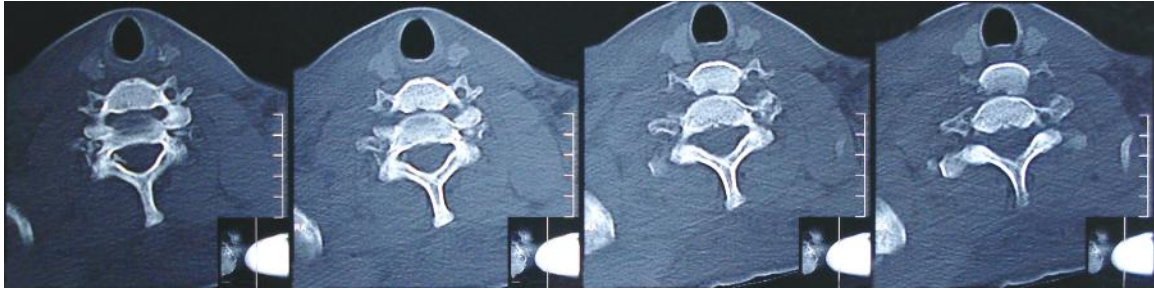


FIG. 20.4: CT—Spondylolisthesis (Courtesy: Dr V Thulasiraman)



FIG. 20.5: MRI spine—C4-C5 spondylolisthesis

Relevant Investigations

- X-rays (Fig. 20.3) and CT (Fig. 20.4) are diagnostic
- MRI (Fig. 20.5) is used to assess root compression.

Treatment

- Asymptomatic patients require no treatment
- Mild cases require surgical corset
- Surgery is justified only when the disability is severe.

Gastroenterology

ACUTE ABDOMEN

Introduction

Acute pain often denotes the presence of a disease process or injury, which needs to be treated with elimination of cause. It is a signal of ongoing or impending tissue damage. Acute abdominal pain denotes similar intra-abdominal organ disease or pathology, which warrants emergent care.

The main visceral pain receptors in the abdomen respond to mechanical and chemical stimuli.

- *Mechanical stimuli*: Stretch, distension, contraction, compression and torsion
- *Chemical stimuli*: Bradykinin, substance P, serotonin and prostaglandins. These receptors are located on the serosal surfaces, within the mesentery and within the walls of hollow viscera
- Gut related visceral pain is usually perceived in the midline because it is a midline structure in an embryo and has bilateral symmetric innervations, except for pains originating from the gallbladder and the ascending and descending colon. Pain from other intra-abdominal organs tends to be unilateral.
 - ❖ *Pain at epigastrium*: Diseases of the foregut (abdominal esophagus, stomach and proximal half of second part of duodenum and their offshoots like liver, gallbladder, pancreas and spleen) (e.g. gastric and duodenal ulcers)
 - ❖ *Pain at the umbilical region*: Diseases of midgut (distal half of second part of duodenum, small bowel, colon up to the proximal 2/3 of transverse colon) (e.g. intestinal tuberculosis)
 - ❖ *Pain at the hypogastrium*: Diseases of hindgut (distal 1/3 of transverse colon to the anorectal junction) (e.g. colorectal and urinary bladder pathologies)

The abdomen is divided into 10 arbitrary regions for convenience of understanding and localizing. The two lateral vertical planes pass from the costal margin close to the tip of the ninth cartilage above to the femoral artery below. The horizontal plane (the subcostal plane) connects the lowest points on the costal margins and the interiliac plane connects the tubercles of the iliac crests (Fig. 21.1). These divisions will help in localizing the diseases.

Pain

Nature of Pain

- *Sudden onset pain*: Pain is sudden in otherwise healthy and asymptomatic persons. It increases in a very short time (e.g. pain due to perforations of duodenal ulcer and appendicitis)
- *Sudden onset with pain-free intervals*: The pain of quick onset can reach a peak making the patient writhe in pain and buckle up, and also quickly recede to absolutely pain-free period, only to recur again (e.g. colics)
- *Dull continuous pain increasing in severity*: A persistent pain may increase in severity over a period of time (e.g. a dull pain of subacute appendicitis in the right iliac fossa may become severe when the appendicitis becomes severe and acute due to superadded infection and inflammation)
- *Burning pain*: The pain may be of burning nature occurring suddenly (e.g. pain in acid peptic disease)
- *Constant or continuous pain*: Persistent pain without variation in intensity (e.g. peritonitis)
- *Agonizing pain*: Very severe pain which upsets the morale of the patient (e.g. pancreatitis, torsion of pedicled organ)

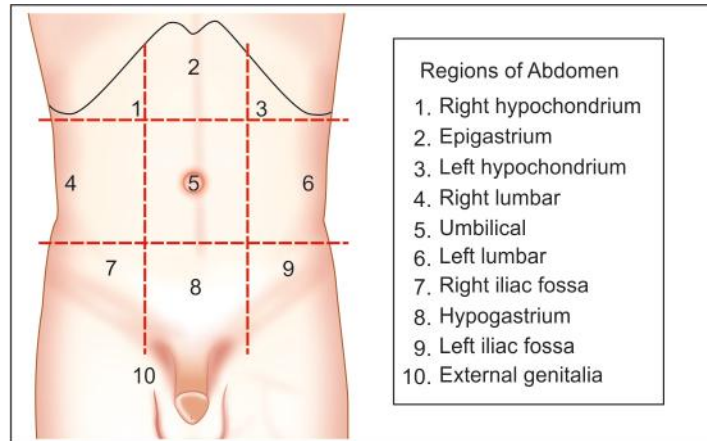


FIG. 21.1: Regions of abdomen

- **Throbbing pain:** Continuous pain throbbing in nature (e.g. acute cholecystitis—due to inflammation being inside closed confines of a structure).

Shift, Migration and Referred Pain

- **Shifting pain:** Shifting pain is defined as the pain, which originates in one region and shifts totally to another region, without any pain at the point of origin (e.g. pain in acute appendicitis originates at the umbilical region and shifts to right iliac fossa later)
- **Migrating pain:** Migrating pain is that pain which originates at one region and shifts to another region, but does continue to stay at a lesser degree at the point of origin (e.g. pain in perforated duodenal ulcer is right hypochondrial to start with, and spreads to the right iliac fossa due to the flow of the gastric contents down the right paracolic gutter)
- **Referred pain:** The pathology and the area of the pain are different, since the organ of pathology and the area of pain share the same nerve supply. The examples are given in Figure 21.2

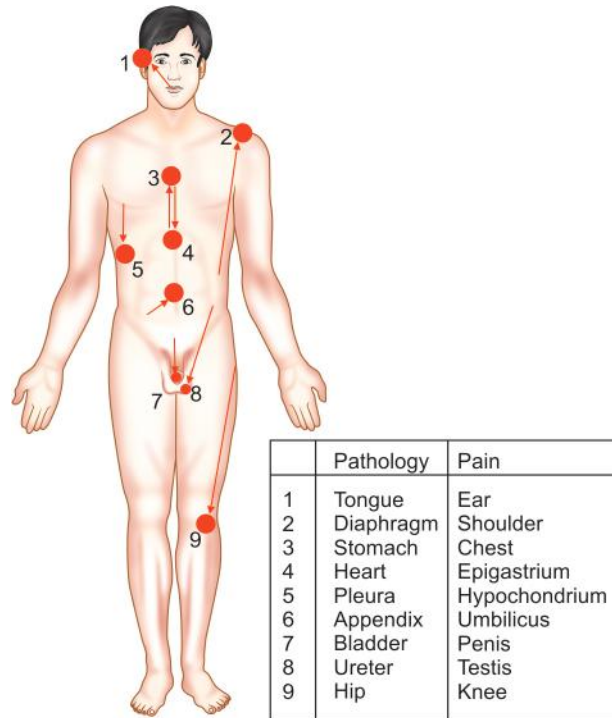


FIG. 21.2: Referred pain

- The perception of visceral pain corresponds to the spinal segments where the visceral afferent nerve fibers enter the spinal cord. Table 21.1 shows some common spinal segments where visceral pain is perceived.

Effects of Movements, Food or Habits

The abdominal pain may be aggravated by certain factors like movements, food or habits. Some examples are given in Table 21.2.

The pain may get relieved by certain factors. Some examples are given in Table 21.3.

Table 21.1: Visceral pain and dermatomal perceptions

<i>Organ of pathology</i>	<i>Site of pain</i>	<i>Dermatome</i>
Stomach	Epigastrium	T5-T10
Small bowel	Umbilicus	T9-T10
Large bowel up to splenic flexure	Umbilicus	T11-L1
Large bowel from splenic flexure	Hypogastrium	L1-L2
Gallbladder	Epigastrium, scapular region	T7-T9
Pancreas	Epigastrium	T6-T10
Ureter	Loin to groin	Genitofemoral nerve (L1-L2)
Testis and ovary	Umbilicus	T10-T11

Note: Segmental nerve supply mentioned here is sympathetic supply of the viscus. Parasympathetic supply is from the vagus nerve, excepting for the hindgut and the urinary bladder, which is from the sacral segments.

Table 21.2: Aggravating factors of abdominal pain

<i>Aggravating factor</i>	<i>Pathology</i>
Physical movements like jolting	Appendicitis, peritonitis
Deep inspiration	Pleurisy
Intake of fatty food	Cholecystitis
Intake of spicy food, alcohol	Acid peptic disease
Lying supine	Pancreatitis
Drugs—Analgesics, NSAIDs	Acid peptic disease

Table 21.3: Relieving factors of abdominal pain

Relieving factor	Pathology
Vomiting	Peptic ulcer pain
Intake of bland food	Peptic ulcer pain
Local pressure	Colicky pain
Leaning forward	Pancreatitis
Drugs—antacids, H ₂ blockers	Acid peptic disease

Vomiting

Vomiting is a very common feature associated with pain in acute abdominal emergencies. The patient should be asked about the following details, as each has its own significance. They are:

- *Character of vomiting*
 - ❖ *Projectile*: Involuntary projectile ejection of large quantities of vomitus (e.g. high intestinal obstruction)
 - ❖ *Regurgitative*: Effortless involuntary regurgitation of intestinal contents (e.g. peritonitis due to perforation)
- *Frequency of vomiting*
 - ❖ *Constant*: Persistent vomiting even in the absence of food intake (e.g. acute intestinal obstruction, acute pancreatitis)
 - ❖ *Periodical*: Vomiting with some periodicity or following food intake indicates bowel obstruction (e.g. acute peptic ulcer, gastric outlet obstruction)
- *Nature of vomitus*
 - ❖ Coffee ground vomitus (Brown to dark brown colour)—gastric contents with altered blood (e.g., bleeding duodenal ulcer)
 - ❖ Bloody (red in colour) (e.g. bleeding oesophageal varices)
 - ❖ Faeculent (yellowish green and foul smelling) (e.g. gastric contents followed by duodenal and intestinal contents in intestinal obstruction)
- *Quantity of vomitus*
 - ❖ Large quantities indicate distal bowel obstruction
 - ❖ Small quantities indicate gastric outlet obstruction
- *Relationship with pain*: The pain may precede, accompany or follow abdominal pain. The examples are given in Table 21.4.

Table 21.4: Relationship of vomiting with abdominal pain

<i>Relationship of vomiting with abdominal pain</i>	<i>Pathology</i>
Pain preceding vomiting	Acute appendicitis, acute peptic ulcer, biliary and renal colic
Pain and vomiting occurring together	High intestinal obstruction
Vomiting occurs much later than the pain	Low intestinal obstruction
Vomiting as a late feature or absent	Large intestinal obstruction

- Abdominal pain due to acute peptic ulcer may get relieved by vomiting, but gives temporary relief in colics
- Vomiting is not a constant feature in acute appendicitis as the stomach gets empty after one bout, but nausea persists
- In peritonitis, vomiting is absent but appears at a later stage.

Alteration in Bowel Habits

- **Obstipation:** Inability to pass feces and flatus (absolute constipation) (e.g. acute intestinal obstruction)
- **Constipation:** Inability to pass stools but able to pass flatus (due to solidified fecal mass impacted at the anal verge) (e.g. acute fissure in ano)
- **Tenesmus:** A desire to evacuate but results in passage of flatus only (e.g. acute appendicitis)
- **Passage of blood stained stools:** Red currant jelly stools in acute intussusception, mesenteric vascular obstruction, mesenteric thrombosis, malignancy
- **Diarrhea:** Passage of loose stools—rarely seen in illnesses requiring emergency surgery (e.g. ulcerative colitis, Crohn’s disease, acute enteritis).

Urinary Symptoms

Patients presenting with abdominal pain may have associated urinary symptoms:

- **Frequency in micturition:** Patients presenting with renal or ureteric colic, may have associated urinary infections along with urolithiasis. Frequency is one of the common symptoms
- **Strangury:** Frequent passing of urine with excruciating pain. (e.g. impacted stones in the urinary tract, pelvic or retrocecal appendicitis). Patient succeeds in passing a small quantity of blood stained urine
- **Hematuria:** Passing blood in the urine (e.g. stones in the urinary tract, rarely retrocecal or pelvic appendix lying close to the ureter).

Causes

Right Hypochondrium

- Acute cholecystitis
- Acute cholangitis
- Acute hepatitis
- Acute hyperacidity
- Perforated duodenal ulcer

Epigastrium

- Acute hyperacidity
- Acute pancreatitis
- Perforated duodenal ulcer
- Acute hepatitis (left lobe)

Left Hypochondrium

- Acute pancreatitis
- Acute hyperacidity
- Splenic infarct

Eliciting History

1. *Nature of pain*

- ❖ Continuous (e.g. acute pancreatitis)
- ❖ Episodic (e.g. acute hyperacidity)
- ❖ Colicky (e.g. biliary colic).

2. *Location of pain*

- ❖ Epigastric pain (e.g. acute hyperacidity, acute pancreatitis, acute colitis)
- ❖ Left hypochondrial pain (e.g. acute hyperacidity, left renal colic)
- ❖ Right hypochondrial pain (e.g. acute cholecystitis, acute hepatitis, right renal colic).

Renal colics can present as upper abdominal pain in their respective sides.

- 3. *Association of vomiting*: Presence of vomiting is not a very reliable symptom to narrow down the diagnosis, as it can be present with any severe painful pathology in the upper abdomen.
- 4. *Association of fever*: Fever indicates infective pathology (e.g. acute cholecystitis, acute pancreatitis, acute colitis, perforated duodenal ulcer).

5. *Association of jaundice*: Jaundice may be present with acute cholangitis, acute hepatitis or acute cholecystitis.
6. *Association of loose stools*: Association of loose stools may indicate colitis or rarely acute pancreatitis.
7. *Radiation*: Radiation to right scapula or shoulder is common with acute cholecystitis, perforated duodenal ulcer due to irritation of diaphragm.
8. *Aggravating factors*
 - ❖ Food—in acute hyperacidity
 - ❖ Lying supine—in acute pancreatitis
 - ❖ Deep breathing—in acute cholecystitis.
9. *Relieving factors*: Leaning forward while sitting—acute pancreatitis.
10. *Referred pain*: In some pathologies, the pathology and the area of the pain are different, since both of them share the same nerve supply.

Past History

- History of pain (e.g. acute on chronic cholecystitis)
- Previous surgery (e.g. cholecystectomy will rule out cholecystitis from consideration).

Family History

Gallstones

Clinical Examination

General

- Breath for fetor (e.g. alcoholic hepatitis, acute pancreatitis)
- Conjunctiva for anemia, jaundice
- Tongue for anemia
- Neck for lymphadenopathy
- Hands for signs of liver failure (e.g. clubbing, palmar erythema, liver flap, etc).

Abdomen

Inspection

Distension:

- Generalized (e.g. perforated duodenal ulcer or gallbladder with peritonitis)
- Right upper abdominal (e.g. hepatomegaly)
- Epigastric (e.g. left lobar hepatomegaly, carcinoma stomach)
- Left upper abdominal (e.g. splenomegaly)
- Scars, swellings and sinuses.

Palpation*Tenderness:*

- All quadrants—generalized peritonitis
- Right upper quadrant (e.g. acute hepatitis, acute cholecystitis, acute hyperacidity)
- Epigastric (e.g. acute gastritis, acute hepatitis)
- Left upper quadrant (e.g. acute gastritis, acute pancreatitis).

Lump:

- Right upper quadrant (e.g. hepatomegaly, distended gallbladder)
- Epigastric (e.g. carcinoma stomach, left lobar hepatomegaly)
- Left upper quadrant (e.g. carcinoma stomach, splenomegaly).

Percussion*Percuss the liver for:*

- Its enlargement (e.g. acute hepatitis)
- Obliteration of liver dullness (e.g. perforated duodenal ulcer).

Auscultation

- Absence of bowel sounds indicates paralytic ileus (e.g. perforated peritonitis)
- Exaggerated bowel sounds may indicate obstruction of small bowel (e.g. intestinal colic)
- Normal bowel sounds indicate that there is no gross infection of the peritoneum.

Examination of

- Groins (e.g. obstructed hernia)
- Genitalia (e.g. obstructed hernia)

Differential Diagnosis by Clinical History and Examination

- *Right hypochondrial pain*
 - ❖ Without fever and but local tenderness (e.g. acute cholecystitis, acute hepatitis, acute hyperacidity)
 - ❖ With fever and local tenderness
 - + Distension (e.g. acute cholecystitis)
 - Obliteration of liver dullness (e.g. perforated duodenal ulcer)
 - + Hepatomegaly (e.g. acute liver abscess)
 - ❖ With vomiting
 - Local tenderness (e.g. acute cholecystitis, acute hyperacidity)
 - Local tenderness with obliteration of liver dullness (e.g. perforated duodenal ulcer)
 - ❖ With diarrhea and local tenderness (e.g. acute colitis)

- ❖ With jaundice
 - Local tenderness + hepatomegaly (e.g. acute hepatitis, acute cholecystitis, choledocholithiasis, cholangitis, acute liver abscess)
 - Local tenderness, fever, +/- abdominal lump (e.g. mucocele gallbladder, choledochal cyst)
- ❖ Colicky in nature +/- local tenderness (e.g. biliary colic, right renal colic)
- *Epigastric pain*
 - ❖ Without fever but with local tenderness (e.g. acute hyperacidity, acute pancreatitis, acute hepatitis—left lobe)
 - ❖ With fever and local tenderness + distension (e.g. acute pancreatitis, perforated duodenal or gastric ulcer)
 - ❖ With vomiting and
 - Local tenderness (e.g. acute hyperacidity, acute pancreatitis)
 - Local tenderness and obliteration of liver dullness (e.g. perforated ulcer)
 - ❖ With diarrhea and local tenderness (e.g. acute colitis)
 - ❖ With jaundice and local tenderness
 - ❖ + tender hepatomegaly (e.g. left lobar hepatitis, left lobar liver abscess)
 - ❖ Nontender hepatomegaly (e.g. metastatic liver)
- *Left hypochondrial pain*
 - ❖ Without fever but with local tenderness (e.g. acute gastritis, acute pancreatitis)
 - ❖ With fever and local tenderness + +/- splenomegaly (e.g. acute pancreatitis, splenic infarct)
 - ❖ With diarrhea and local tenderness (e.g. acute colitis)
 - ❖ Colicky in nature + local tenderness (e.g. left renal colic).

- Perforated bowel can give rise to generalized abdominal distension due to generalized peritonitis
- Obstructed groin hernia is one of the common causes of intestinal obstruction causing generalized distension.

Relevant Investigations

Hematology

- Leukocytosis in infective pathologies (e.g. acute cholecystitis, perforated duodenal ulcer, perforated cholecystitis, acute pancreatitis)
- Raised ESR in all infective pathologies.

Radiology

- *Plain X-ray abdomen:* Gas under the diaphragm (e.g. perforated hollow viscus)
- *Ultrasonography:* Radiopaque shadows in right upper abdomen (e.g. gallstones, renal stones)

- *Nonperforated pathologies:* Medical management
- *Perforated pathologies:* Early surgical management
- Diagnosis not clear and not responding to medical management—exploratory laparotomy.

ACUTE LOWER ABDOMINAL PAIN

Causes

Right Iliac Fossa

- Acute appendicitis
- Perforated appendicitis
- Acute mesenteric adenitis
- Ureteric colic
- Acute Meckel's diverticulitis
- Acute cecal diverticulitis
- Torsion of cyst of right ovary
- Pelvic inflammatory disease
- Incarcerated right inguinal hernia
- Ruptured ectopic gestation
- Right ureteric colic.

Hypogastrium

- Acute cystitis
- Acute congestive dysmenorrhea
- Uterine fibroid.

Left Iliac Fossa

- Acute diverticulitis
- Ureteric colic
- Torsion of cyst of left ovary
- Pelvic inflammatory disease
- Incarcerated left inguinal hernia
- Ruptured ectopic gestation
- Left ureteric colic.

Diabetic ketoacidosis is one of the important metabolic causes of acute lower abdominal pain.

Eliciting History

Nature of pain

- Continuous (e.g. acute appendicitis)
- Episodic (e.g. acute cystitis, ruptured ectopic gestation)
- Colicky (e.g. appendicular colic, ureteric colic, dysmenorrhea).

Association of nausea and vomiting

Presence of vomiting is not a very reliable symptom to narrow down the diagnosis, as it can be present with any severe painful pathology in the lower abdomen. Nausea is a predominant symptom of acute appendicitis, whereas vomiting is a common symptom of ureteric colic.

Association of fever

Fever indicates infective pathology (e.g. acute appendicitis, acute cystitis, acute colitis, perforated appendicitis).

Association of loose stools

Association of loose stools may indicate colitis, acute diverticulitis.

Radiation

Radiation to pain to external genitalia occurs with ureteric colic.

Past History

- History of pain (e.g. acute on chronic appendicitis, ureteric colic)
- Previous surgery (e.g. appendectomy will rule out appendicitis from consideration).

Personal History

Menstrual irregularities

- ❖ Dysmenorrhea (e.g. congestive dysmenorrhea)
- ❖ Irregularities (e.g. pelvic inflammatory diseases)
- ❖ Amenorrhea (e.g. ruptured ectopic gestation).

Family History

- Diverticulosis
- Colonic malignancy.

General

- Conjunctiva for anemia (e.g. ruptured ectopic gestation)
- Tongue for anemia (e.g. ruptured ectopic gestation)
- Neck for lymphadenopathy (e.g. mesenteric adenitis as a part of tuberculosis).

Abdomen■ **Inspection***Distension:*

- ❖ Generalized (e.g. perforated appendicitis with generalized peritonitis)
- ❖ Right lower abdominal (e.g. ruptured appendicitis, torsion of right ovary or its cyst)
- ❖ Hypogastric (e.g., distended urinary bladder in cystitis, enlarged uterus)
- ❖ Left lower abdominal (e.g. torsion of left ovary or its cyst)

Scars, swellings and sinuses■ **Palpation***Tenderness:*

- ❖ All quadrants – generalized peritonitis
- ❖ Right lower quadrant (e.g. acute appendicitis, acute mesenteric adenitis)
- ❖ Hypogastric (e.g. acute cystitis)
- ❖ Left lower quadrant (e.g. acute colitis, acute diverticulitis)

Lump:

- ❖ Right lower quadrant (e.g. appendicular abscess, mesenteric adenitis, right ovarian cyst)
- ❖ Hypogastric (e.g. distended urinary bladder, uterine fibroids)
- ❖ Left lower quadrant (e.g. carcinoma colon, left ovarian cyst)

■ **Percussion**

Percuss the liver for:

- ❖ Its enlargement (e.g. associated metastases liver)
- ❖ Obliteration of liver dullness (e.g. perforated appendicitis and diverticulitis)

■ **Auscultation**

- ❖ Absence of bowel sounds indicates paralytic ileus (e.g. perforation and peritonitis)
- ❖ Exaggerated bowel sounds may indicate obstruction of small bowel (e.g. intestinal colic)
- ❖ Normal bowel sounds indicate that there is no gross infection of the peritoneum

Differential Diagnosis by Clinical History and Examination

■ *Pain in the right iliac fossa with*

- ❖ Nausea/vomiting and fever and local tenderness (e.g. acute catarrhal/perforated appendicitis, acute typhlitis, acute Meckel's diverticulitis, acute mesenteric adenitis)
- ❖ Vomiting and
 - +/- Local tenderness (colicky pain—e.g. right ureteric colic, appendicular colic, radiating pain—right ureteric colic)
 - Menstrual irregularities and palpable lump (e.g. right ovarian cyst torsion)
 - Amenorrhea with local tenderness and anemia (e.g. ruptured ectopic gestation)
 - Abdominal lump (e.g. ileocecal tuberculosis, Crohn's disease, cecal malignancy, right ovarian malignancy, right ovarian cyst)
- ❖ Diarrhea or dysentery and local tenderness (e.g. acute amebic typhlitis, cecal diverticulitis)

■ *Pain in the left iliac fossa with*

- ❖ Vomiting
- ❖ +/- Local tenderness (e.g. left ureteric colic)
 - Menstrual irregularities and palpable lump (e.g. left ovarian cyst torsion)
 - Amenorrhea with local and cervical tenderness and anemia (e.g. ruptured ectopic gestation)
 - Abdominal lump (e.g., colonic malignancy, left ovarian malignancy, left ovarian cyst)
 - Diarrhea or dysentery with or without local tenderness (e.g. acute colitis, acute diverticulitis)

■ *Hypogastric pain with*

- ❖ Urinary symptoms with
 - Local tenderness (e.g. acute cystitis)
 - Distended urinary bladder (e.g. acute distension of bladder—bladder outlet obstruction)
- ❖ Menstrual irregularities
 - +/- With or without local tenderness (e.g. congestive dysmenorrhea, pelvic inflammation)
 - Enlarged uterus (e.g. uterine fibroids, endometrial malignancy)

■ *Acute lower abdominal pain (any or all quadrants of lower abdomen) with*

- ❖ Menstrual irregularities and local tenderness (e.g. pelvic inflammatory disease)
- ❖ Vague symptoms and signs (e.g. metabolic causes—diabetes mellitus).

Relevant Investigations

Hematology

- Reduced hematocrit (e.g. ruptured ectopic gestation, colonic malignancy)
- Leukocytosis in infective pathologies (e.g. acute appendicitis, perforated appendicitis, and diverticulitis, duodenal ulcer)
- Raised ESR in all infective pathologies.

Radiology

- Plain X-ray abdomen
 - ❖ Gas under the diaphragm (e.g. perforated appendicitis and diverticulitis)
 - ❖ Opaque abdomen (e.g. peritonitis)
 - ❖ Radiopaque shadows (e.g. ureteric stone)
- Ultrasonography
 - ❖ Cystic swelling (e.g. ovarian cyst torsion)
 - ❖ Collection of pus (e.g. appendicular abscess, paracolic abscess)
 - ❖ Radiopaque shadows in the lines of ureters (e.g. ureteric calculus) and bladder region (e.g. vesical calculus)
 - ❖ Dilated pelvicalyceal system or ureters (e.g. obstructed ureteric stone).

Treatment Plan

- Nonperforated pathologies—medical management (except acute appendicitis)
- Perforated pathologies—early surgical management
- Diagnosis not clear and not responding to medical management—exploratory laparotomy or diagnostic laparoscopy.

ACUTE LIVER ABSCESS

Incidence and Etiology

- *Amebic liver abscess*: Amebic infections causing hepatitis and dysentery may resolve into a liver abscess (usually in alcoholics)
- *Pyogenic liver abscess*: Septicemic patients may present with pyemic abscesses in the liver.

Pathogenesis

- *Amebic liver abscess*: Amebic infections originating in the colon as amebic colitis, travel through the portal blood to reach the liver, forming an abscess more commonly in the right lobe of the liver
- *Pyogenic liver abscess*: Septicemic patients' infected blood reach the liver through the systemic and portal circulation, forming multiple abscesses in both lobes of the liver.

Symptoms

- Constitutional symptoms with high-grade fever, tachycardia and sometime shock.
- Patients with amebic etiology may give history of preceding diarrhea or dysentery.

Signs

- Intercostal tenderness is pathognomonic of a right lobar liver abscess.

Differential Diagnosis

- Lower thoracic lesions (basal pleurisy, pneumonia, and lung abscess), which irritate the diaphragm
- Acute cholecystitis
- Acute hepatitis.

Relevant Investigations

- Plain X-ray of abdomen or chest X-ray will reveal elevation of the right dome of diaphragm (Fig. 21.3). Right pleural effusion is common
- Fluoroscopy will show reduced mobility of the right dome
- US and CT (Fig. 21.4) are diagnostic
- Aspiration is confirmatory
- Stool examination is routine
- Isolation of organism in culture is required
- Serologic test (for amebiasis) is positive in majority of cases.



FIG. 21.3: Elevated right dome of diaphragm in amebic liver abscess

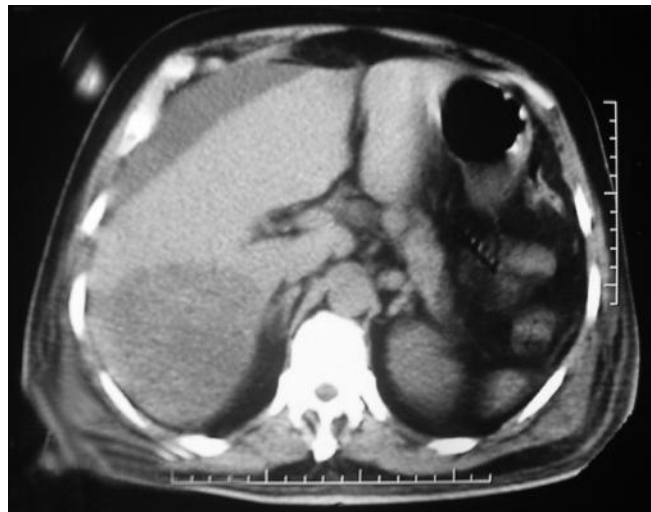


FIG. 21.4: CT—Amebic liver abscess right lobe with subphrenic collection

- Antiamebic treatment is administered for small abscess and hepatitis of amebic etiology
- Drainage under US guidance with broad-spectrum antibiotics and antiamebic drugs is required for large abscess
- Growth of pyogenic organisms in culture of pus requires appropriate treatment.

ACUTE CHOLECYSTITIS

Incidence and Etiology

- Common in fat, flatulent, fertile, female of fifty (Five Fs)
- Ninety-five percent people with acute cholecystitis have gallstones.

Pathogenesis

- Inflammation of gallbladder occurs due to detergent action of bile (chemical cholecystitis), and infection by bacteria supervenes (commonly enteric organisms)
- Obstruction caused by a calculus in the cystic duct.

Complications

- Acute obstruction of the cystic duct causes distension and the bile can be replaced by mucus (mucocele) or pus (empyema)
- Ongoing inflammation causes gangrene and perforation of gallbladder (common in diabetics).

Symptoms

- Severe colicky pain (biliary colic) in the right hypochondrium, radiating to the inferior angle of the right scapula and the right shoulder
- Pain may be associated with vomiting.
- Fever and jaundice* are associated when there is associated cholangitis
- Symptom complex of pain, jaundice and fever with chills is called “Charcot’s triad”.

*Jaundice may occur due to the obstruction of the common bile duct caused by an impacted stone in the cystic duct (Mirizzi’s syndrome type 1)

Signs

- Tenderness at the tip of the right ninth costal cartilage (Murphy's sign)
- Hyperesthesia between the right 9th and 11th ribs posteriorly (Boas's sign)
- There may be associated guarding and rigidity in the right hypochondrium
- Palpable mass in the RUQ (inflamed and distended gallbladder).

Differential Diagnosis

- Acute appendicitis
- Acute right pyelonephritis
- Duodenal ulcer perforation
- Right basal pleurisy
- Myocardial infarction.

Relevant Investigations

- Elevated leukocyte count
- Deranged LFT (due to inflammation and edema of biliary tract)
- Plain X-ray abdomen (About 10% of gallstones are visible)
- US (Figs 21.5A and B) is diagnostic
- Rarely CT scan (Figs 21.6A to C) may be required
- Isotope scans are contributory.

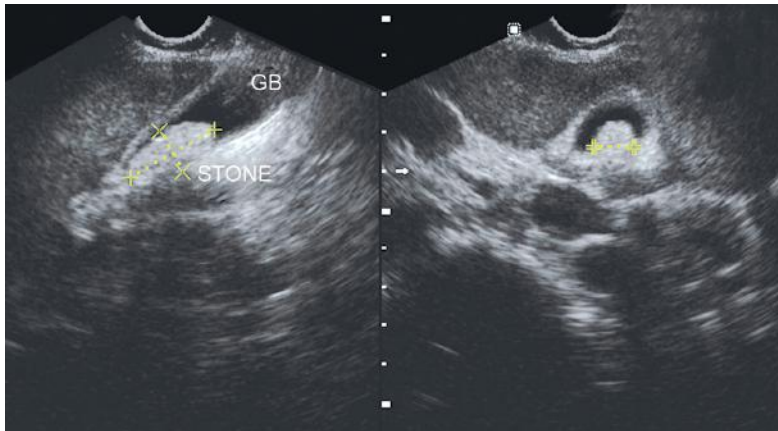


FIG. 21.5A: US—Stone in the cystic duct of gallbladder
(Courtesy: Dr V Ganesan)

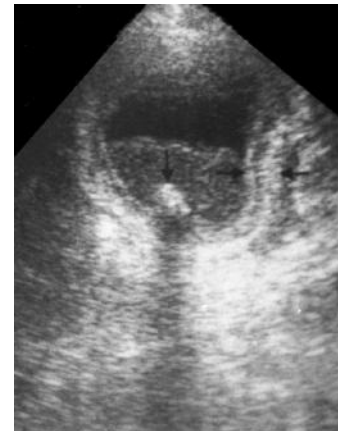


FIG. 21.5B: US—Acute edematous cholecystitis



FIG. 21.6A: CT—Stone in the gallbladder

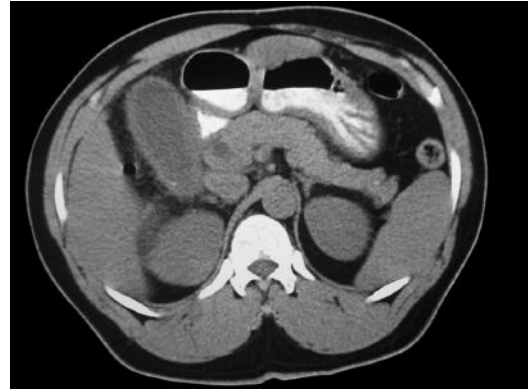


FIG. 21.6B: CT—Edematous gallbladder of acute cholecystitis

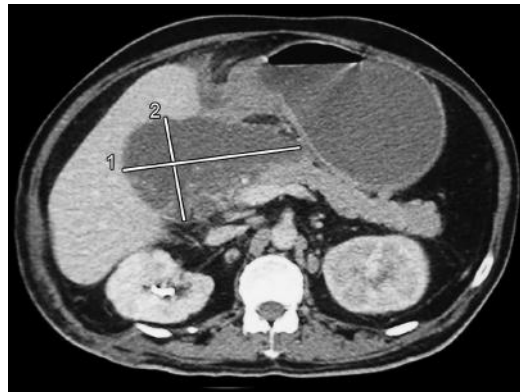


FIG. 21.6C: CT—Ruptured empyema of gallbladder

Treatment

- Acute cholecystitis is treated conservatively with antibiotics followed by elective cholecystectomy (Laparoscopic or open) **
- Common duct stones need to be removed by ERCP, sphincterotomy and basketing/stenting or by open choledochotomy, to relieve cholangitis and jaundice followed by cholecystectomy later.

** Laparoscopic cholecystectomy can be performed within 72 hours of onset of symptoms, with no higher conversion or complication rate, than a delayed operation.

ACUTE CHOLANGITIS

Incidence and Etiology

- Approximately 10 percent of patients with gallstones have associated common bile duct stones (choledocholithiasis)
- Obstructive lesions in the common bile duct can cause cholangitis (Fig. 21.7). They are:
 - ❖ Stricture
 - ❖ Tumor
 - ❖ Foreign body (e.g. stent)
 - ❖ Acute pancreatitis
 - ❖ ERCP

Pathogenesis

Obstructing lesion causes bile stasis, and this obstructed flow of bile encourages multiplication of bacteria, which have entered the biliary tract from the intestines through the sphincter of Oddi, causing ascending cholangitis.

Symptoms

RUQ pain, fever with rigors and jaundice (Charcot's triad).

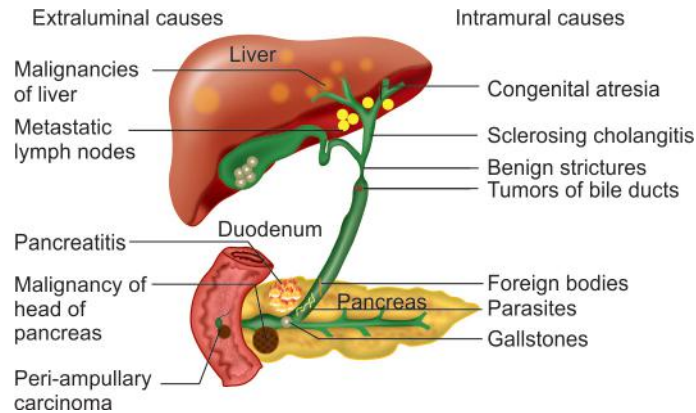


FIG. 21.7: Causes of mechanical obstruction of biliary system

- Pyrexia, tachycardia, tachypnea
- Hypotension in shocked in individuals
- Jaundice
- Tenderness and guarding in RUQ.

Relevant Investigations

- Elevated leukocyte count
- Conjugated bilirubinemia
- Deranged LFT (due to inflammation and edema of biliary tract)
- Plain X-ray abdomen (About 10% of gallstones are visible)
- US and CT may reveal the dilated intra-hepatic radicals
- ERCP (Fig. 21.8) and MRCP (Fig. 21.9) will reveal the level and severity of stricture
- PTC (Fig. 21.10) is useful in impassable strictures
- MDCT with MPR technique (Fig. 21.11) has sensitivity of about 90 to 95 percent in localizing the causes of biliary obstruction.

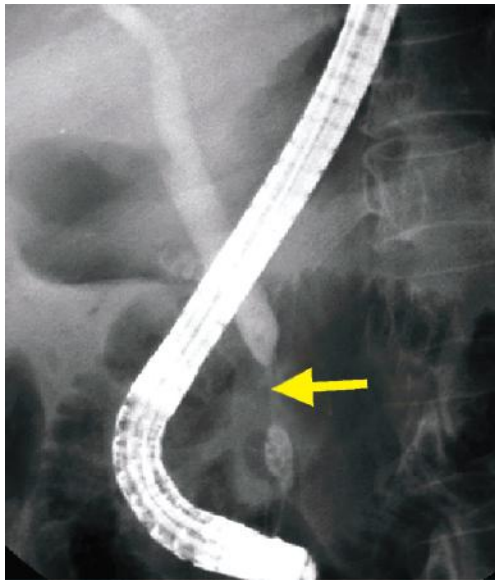


FIG. 21.8: ERCP—Benign stricture of CBD



FIG. 21.9: MRCP—Benign stricture of CBD



FIG. 21.10: PTC—Grossly dilated intrahepatic bile ducts due to CBD obstruction

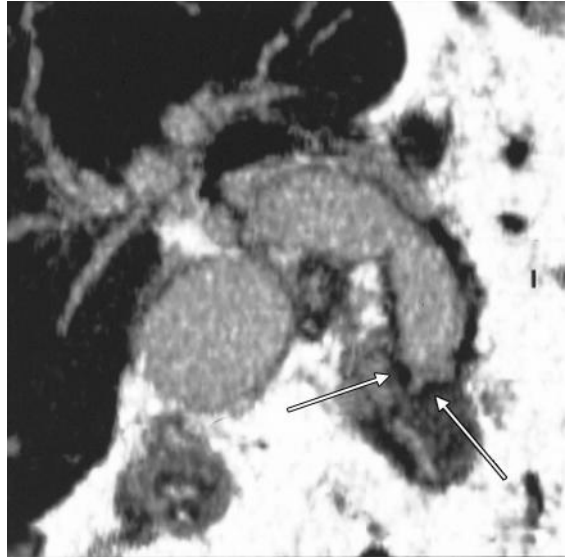


FIG. 21.11: MDCT—Cholangiocarcinoma

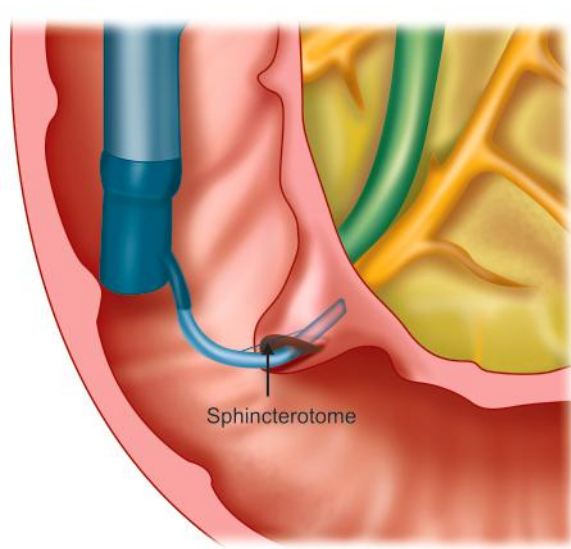


FIG. 21.12: Sphincterotomy

- Inpatient care/Intensive care
- Analgesics
- Broad spectrum antibiotics
- Fluid resuscitation
- Emergency ERCP, sphincterotomy and basketing/stenting (Fig. 21.12) to relieve cholangitis and jaundice followed by cholecystectomy.
- Surgical treatment is required for impassable strictures
 - ❖ Resectable strictures can be resected with primary anastomosis
 - ❖ Strictures of the retropancreatic region require, choledocho-duodenostomy
 - ❖ Strictures of the common hepatic duct, require hepatico-jejunostomy.

Acute suppurative cholangitis is uncommon, but serious enough and if left untreated, has 100 percent mortality.

PRIMARY SCLEROSING CHOLANGITIS

Incidence and Etiology

- Rare condition of autoimmune origin
- Results in progressive fibrosis of the biliary system
- Causes luminal narrowing and progressive obstructive jaundice and secondary cirrhosis
- Strongly associated with inflammatory bowel disease, ulcerative colitis (75% of patients with PSC have inflammatory bowel disease and 7.5% with ulcerative colitis have PSC)
- Risk of malignant transformation (cholangiocarcinoma) is 15 percent.

Clinical Presentation

- Progressive obstructive jaundice
- Low-grade fever with chills, sweats.

Relevant Investigations

- CT (Fig. 21.13) and ERCP (Fig. 21.14) are diagnostic, classically shows diffuse stricturing and beading involving both intra and extrahepatic bile ducts, but indistinguishable from cholangiocarcinoma
- Liver biopsy may show the characteristic lesion of concentric fibrosis around small bile ducts, termed 'onion skin' fibrosis
- Peripheral antineutrophil cytoplasmic antibody (pANCA) is detected.

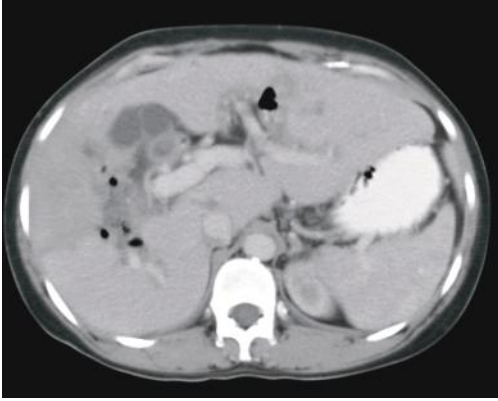


FIG. 21.13: CT—Sclerosing cholangitis



FIG. 21.14: ERCP—Sclerosing cholangitis

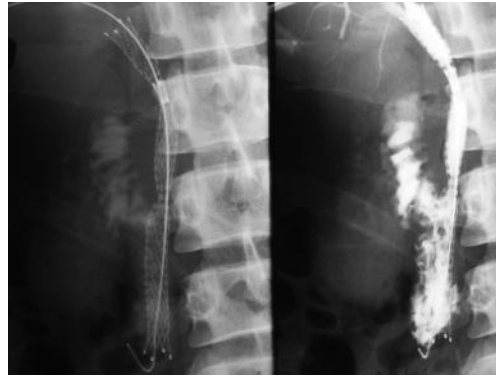


FIG. 21.15: Biliary stenting with endoprosthesis for cholangiocarcinoma

Treatment

- May settle spontaneously
- May respond to antibiotics, and UDCA
- Stenting of the biliary tree (Fig. 21.15) is very useful
- Liver transplantation is used widely for advanced disease.

SPLENIC ABSCESS

Incidence and Etiology

- Cyst of the spleen is a rare condition
- They can be:
 - ❖ Congenital
 - ❖ Parasitic (hydatid).

Pathogenesis

Severe systemic infections cause splenic abscesses which are usually multiple.

Symptoms

- Constitutional symptoms (e.g. high-grade fever)
- Previous history of severe intra-abdominal sepsis may be present.

Sign

Tender splenomegaly may be present.

Relevant Investigation

US and CT (Fig. 21.16) are diagnostic.

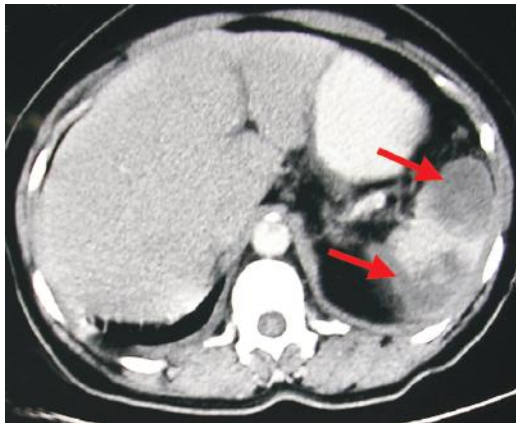


FIG. 21.16: CT abdomen—Splenic abscesses

Treatment

- Broad spectrum antibiotics
- Splenectomy is done for large spleen with abscesses.

ACUTE HEMORRHAGIC PANCREATITIS

Incidence and Etiology

- Common in the third decade
- Classification:
 - ❖ *Mild type*: Pancreatitis with minimal or no organ dysfunction and has a self-limiting course with uneventful recovery
 - ❖ *Severe type*: Pancreatitis with multiorgan failure and/or local complications like pseudocyst, necrosis or abscess
- Common causes are (nemonic: I get smashed)
 - ❖ I - Idiopathic
 - ❖ G - Gallstone (in the ampulla of Vater)
 - ❖ E - Ethanol
 - ❖ T - Trauma
 - ❖ S - Steroids
 - ❖ M - Mumps (paramyxovirus, Epstein-Barr virus, cytomegalovirus)
 - ❖ A - Autoimmune (Polyarteritis nodosa, Systemic lupus erythematosus)
 - ❖ S - Scorpion sting (Tityus Trinitatis), Snake bite
 - ❖ H - (Hypercalcemia, hyperlipidemia hypertriglyceridemia and hypothermia)
 - ❖ E - (Endoscopic Retrograde Cholangiopancreatography—ERCP)
 - ❖ D - Drugs (Steroids and Sulfonamides, Azathioprine, NSAIDs, Diuretics—SAND)
- Less common causes are:
 - ❖ Pancreas divisum
 - ❖ Long common duct
 - ❖ Carcinoma of the head of pancreas, and other cancer
 - ❖ Ascaris blocking pancreatic outflow
 - ❖ Chinese liver fluke
 - ❖ Ischemia from bypass surgery
 - ❖ Fatty necrosis
 - ❖ Pregnancy

- ❖ Infections other than mumps, including varicella zoster
- ❖ Repeated marathon running
- ❖ Cystic fibrosis.
- Most common causes of pancreatitis, by demography are as follows:
 - ❖ *Western countries:* Chronic alcoholism and gallstones
 - ❖ *Eastern countries:* Gallstones.

Pathogenesis

- In acute pancreatitis, there is edema, hemorrhage and necrosis of the organ partly due to autodigestion. The inflamed pancreas may return to normal, but may recur, and likely to occur under two circumstances:
 - ❖ The initiating cause has not been removed (gallstones, alcohol consumption)
 - ❖ Major pancreatic necrosis, resulting in chronic pancreatitis or stricture of main pancreatic duct.

Complications

- Systemic complications:
 - ❖ Respiratory failure
 - ❖ Renal failure
 - ❖ Metabolic abnormalities
 - ❖ Coagulation disorders
 - ❖ Multiple organ failure.
- Local complications:
 - ❖ Pancreatic necrosis
 - ❖ Infection of pancreatic necrosis
 - ❖ Fungal infections
 - ❖ Hemorrhage
 - ❖ Pancreatic pseudocyst, pancreatic fistula, pancreatic abscess.

Symptoms

- Very severe, unbearable constant epigastric pain radiating to the back, relieved by sitting and bending forwards
- Nausea and vomiting are marked, frequent and persistent.

Signs

- Shock and cyanosis are marked
- Elevated temperature, tachycardia, tachypnea

- Epigastric tenderness but guarding and rigidity are not marked
- Retroperitoneal hemorrhage may lead to characteristic discoloration:
 - ❖ Around the umbilicus (Cullen's sign) (Fig. 21.17)
 - ❖ Of the flanks (Grey Turner's sign) (Fig. 21.18)(These signs are not pathognomonic of pancreatitis, as they can be seen with ruptured ectopic pregnancy as well).

Differential Diagnosis

- Perforated peptic ulcer
- Leaking aortic aneurysm
- Myocardial infarction
- Acute cholecystitis
- Acute hyperacidity

Relevant Investigations

- Elevation of serum amylase over 400 Somogyi units is indicative and more than 1000 Somogyi units is diagnostic (It usually rises 2 to 12 hours from the onset of symptoms, and normalizes within 48 to 72 hours)
- Serum lipase levels are elevated (It rises 4 to 8 hours from the onset of symptoms and normalizes within 7 to 14 days).

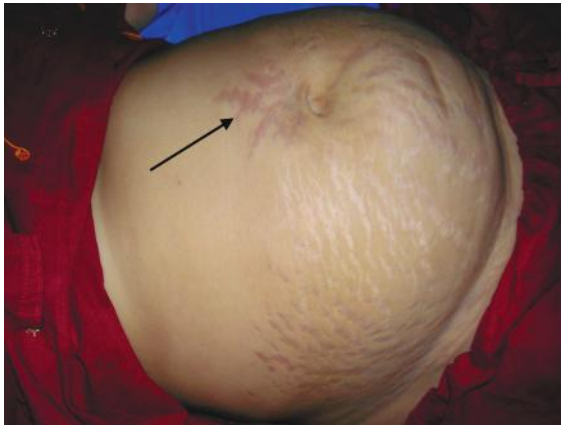


FIG. 21.17: Cullen's sign



FIG. 21.18: Grey Turner's sign

Serum amylase may be normal (in 10% of cases) for cases of acute or chronic pancreatitis (depleted acinar cell mass) and hypertriglyceridemia. Reasons for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase) and macroamylasemia. If the lipase level is about 2.5 to 3 times that of Amylase, it is an indication of pancreatitis due to alcohol

- Plain X-rays of abdomen show characteristic features:
 - ❖ ‘Sentinel loop sign’ – duodenum – which represents a focal dilated jejunal loop in the left upper quadrant
 - ❖ ‘Cut off sign’ – Transverse colon (Fig. 21.19) - Inflammatory exudate of acute pancreatitis extends into the phrenicocolic ligament directly spreading through the lateral attachment of the transverse mesocolon causing functional spasm and/or mechanical narrowing of the splenic flexure at the level where the colon returns to the retroperitoneum.
- (Absence of gas under the diaphragm eliminates the diagnosis of perforated duodenal ulcer)

■ US may not be very useful at all times (Fig. 21.20)

■ Contrast enhanced computed tomography (CECT) is very useful in assessing the size of the pancreas (Fig. 21.21), and also in determining the causes like the biliary or pancreatic duct calculi (Fig. 21.22).

Predicting the severity of an attack of acute pancreatitis is made on Glasgow or Imrie criteria (simplified Ranson criteria) Box 21.1.

Presence of three or more criteria reached before or at 48 hours of an attack predicts a severe attack and two or less predicts a mild attack.

APACHE II score (Acute Physiology and Chronic Health Evaluation) can be applied at anytime but it is cumbersome as it requires 15 different or biochemical criteria.

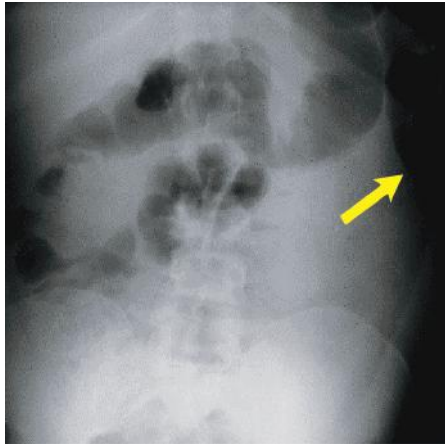


FIG. 21.19: X-ray—Colon cut-off sign

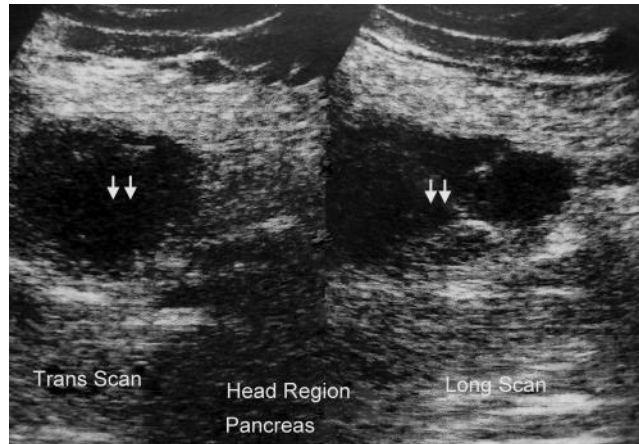


FIG. 21.20: US—Acute pancreatitis

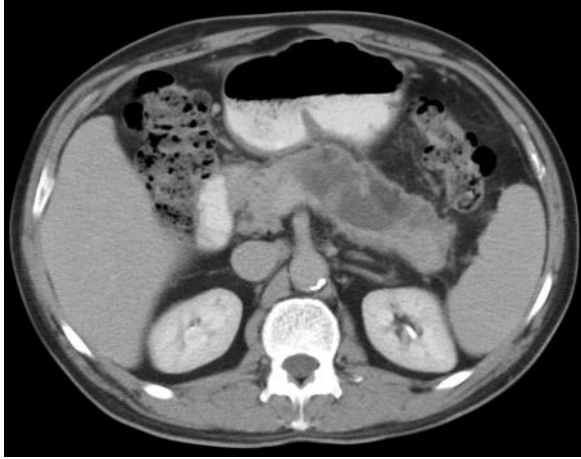


FIG. 21.21: CT—Enlarged and edematous pancreas - Acute pancreas



FIG. 21.22: CT—Pancreatic calculi

Box 21.1: Criteria to assess the severity of acute pancreatitis

P	Arterial PaO ₂ <9 kpa
A	Albumin <32 g/L
N	Urea Nitrogen >10 mmol/L
C	Calcium <2 mmol/L
R	Raised white cell count >16 mmol/L
E	Enzyme – lactate dehydrogenase >600 mmol/L
A	Age >55 years
S	Sugar glucose >10 mmol/L

Treatment

- Initial management is conservative in intensive care unit:
 - ❖ Continuous arterial and CVP monitoring
 - ❖ Assisted ventilation if required
 - ❖ Inotropic support

- ❖ Enteral feeding
- ❖ Parenteral feeding if required
- ❖ Hemodialysis of renal failure if warranted
- Endoscopic treatment:
 - ❖ ERCP sphincterotomy and extraction of stones followed by laparoscopic cholecystectomy
- Pancreatic necrosectomy is the treatment of choice with questionable outcome in a grave situation.

ACUTE APPENDICITIS

◆ Incidence and Etiology

- Most common acute abdominal condition, occurring at any age
- Uncommon only below the age of two
- Most commonly seen between the age of 18 and 35.
- The two varieties of appendicitis are:
 1. Catarrhal appendicitis
 2. Obstructive appendicitis.

Pathogenesis

- *Catarrhal appendicitis*: Occurs due to acute inflammation of the appendix, which produces edema and even gangrene due to vascular involvement in inflammatory process
- *Obstructive appendicitis*: Caused by obstruction of its lumen by worms, fecoliths (Fig. 21.23) or hypertrophied lymphoid follicles. The appendix itself may be filled with pus (Fig. 21.24).

Complications

- When the adjacent tissues and omentum wall off the appendix or its perforation, it forms a mass called 'Appendicular mass'.
- When there is suppuration, it forms an abscess named 'Appendicular abscess', which may burst into peritoneal cavity to produce severe peritonitis and even death.

Symptoms

- *Pain*: A dull continuous ache starting at the umbilical region (visceral pain) and then localizing at the right iliac fossa (parietal pain) - catarrhal variety. Obstructive appendicitis presents with colicky pain (appendicular colic) in the right lower abdomen
- Nausea, vomiting and anorexia are usually present and are diagnostic



FIG. 21.23: Appendix with fecoliths



FIG. 21.24: Acute suppurative appendicitis (note the pus in the cup)

- Fever is the last to develop
(Pain, vomiting and fever in appendicitis is called Murphy's syndrome).

Signs

- Hyperesthesia over Sherrren's triangle
- Tenderness over McBurney's point
- Guarding and rigidity in the right iliac fossa
- Rovsing's sign (pain in the right iliac fossa on application of pressure in the left iliac fossa) may be elicited
- Tender mass may be felt (appendicular mass/abscess)
- Dullness on percussion (if mass already formed).

The signs and symptoms of appendicitis vary according to the position of the appendix (Fig. 21.25). They are given in Table 21.5.

Special Situations

- In children:
 - ❖ Constitutional symptoms like fever and tachycardia are more predominant
 - ❖ Use the child's hand itself for palpation, and if there is tenderness in the McBurney's point, the child will withdraw its hand
 - ❖ Appendicular mass is rare as the omentum is small in size and does not reach the appendix.

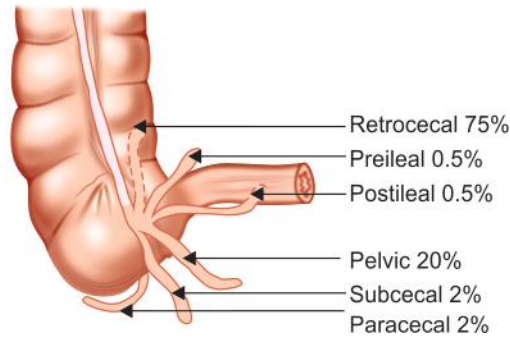


FIG. 21.25: Various positions of appendix

Table 21.5: Signs and symptoms of acute appendicitis related to the position of the appendix

<i>Symptoms and signs</i>	<i>Retrocecal and paracecal appendicitis</i>	<i>Pelvic and subcecal appendicitis</i>	<i>Pre and post-ileal appendicitis</i>
Pain	Right flank and back lateral to sacrospinalis muscle	Right iliac fossa	Right iliac fossa
Diarrhea	Absent	Absent	May be present
Tenderness and guarding	Not marked	Absent	Present
Positive test	Cope's psoas test Baldwing's test	Obturator test	Nil specific
Tenderness in rectal examination	Absent	Present	May be present

- In the elderly:
 - ❖ Guarding and rigidity are not pronounced as the abdominal musculature is weak
 - ❖ Incidence of gangrene is more as there is associated atherosclerosis
 - ❖ Peritonitis supervenes early.
- In pregnant women:
 - ❖ The point of tenderness is shifted up, as the appendix itself is pushed up by the enlarged gravid uterus
 - ❖ Pyelitis and cystitis of pregnancy adds to the difficulties in diagnosis of appendicitis
 - ❖ Accidental hemorrhage mimics acute appendicitis.

Cardinal signs and symptoms of acute appendicitis are:

- Periumbilical pain shifting to the right iliac fossa (very important)
- Association of nausea
- History of similar episodes in the past
- Tenderness at McBurney's point
- Guarding and rigidity in the right iliac fossa.

Differential diagnosis

- Right ureteric colic (most common)
- Right ovarian pathology
- Acute mesenteric adenitis
- Acute cholecystitis.

The differentiating features of acute appendicitis and right ureteric colic are given in Table 21.6.

Relevant Investigations

- US (Figs 21.26A and B) may be contributory. The immobile swollen appendix with free fluid in the right iliac fossa may be imaged. Mildly swollen appendix is generally not seen in an ultrasound scan. The scan helps to eliminate other lesions like the ureteric calculus, ovarian pathology, which can be imaged by US
- CT is useful in identifying inflamed appendix and (Fig. 21.27) appendicular mass. The signs of appendicitis are tabulated in Table 21.7.

Table 21.6: Differentiating features of acute appendicitis and right ureteric colic

<i>Sign and symptom</i>	<i>Pathology</i>	
	<i>Acute appendicitis</i>	<i>Right ureteric colic</i>
Pattern of pain	Periumbilical pain shifting to right iliac fossa	Loin to groin radiation on the right side
Nature of pain	Dull and continuous	Very severe and colicky with painfree intervals
Onset of pain	Slow and continuous	Sudden onset
Relationship to body movements	Aggravated	Not related to movement
Urinary symptoms	Absent	May be present
Rebound tenderness	May be present	Absent
Plain X-ray of abdomen	Nonspecific	Ureteric calculus

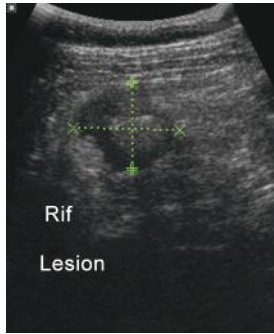


FIG. 21.26A: US—Inflamed appendix (*Courtesy: Dr V Ganesan*)

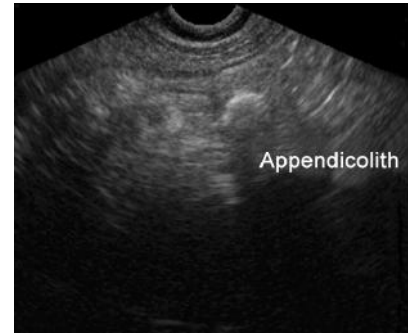


FIG. 21.26B: US—Appendicolith (*Courtesy: Dr V Ganesan*)

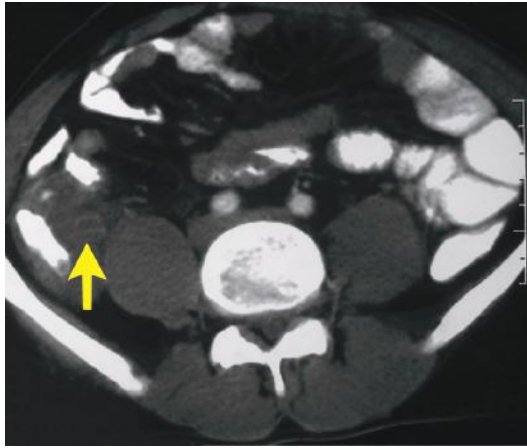


FIG. 21.27: CT—Appendicular mass

Table 21.7: CT findings associated with acute appendicitis

<i>Appendiceal signs</i>	<i>Periappendiceal signs</i>
Appendix >6 mm in anteroposterior diameter	Increased fat attenuation (stranding) in the right lower quadrant
Failure of appendix to fill with oral contrast or gas to its tip	Cecal wall thickening
Enhancement of appendix with IV contrast	Phlegmon in the right lower quadrant
Appendicolith	Abscess or extraluminal gas
	Fluid in the right lower quadrant or pelvis

Treatment

- Emergency appendicectomy (Laparoscopic or open) is the treatment of choice
- Conservative management is adopted for appendicular mass, subacute or chronic appendicitis (Oschner Scherren regime), followed by appendicectomy at a later date (interval appendicectomy)
- Appendicular abscess requires drainage, followed by interval appendicectomy.

ACUTE MESENTERIC LYMPHADENITIS

Incidence and Etiology

- Seen commonly in the pediatric age group
- Acute infection and inflammation of the mesenteric lymph nodes mostly of the ileocolic group (Fig. 21.28).

Pathogenesis

Commonly caused by viral infections.

Symptoms

- Periumbilical pain associated with high grade fever
- Vomiting is rare.

Signs

- The point of tenderness is usually in the right iliac fossa in the supine position, and shifts to the left side, if the patient is made to lie on the left side and vice versa (“Klein’s sign”)
- Guarding is not predominantly present
- In thin children, enlarged lymph nodes may be felt.

Differential Diagnosis

- Tubercular infection
- Acute appendicitis.

Relevant Investigations

- Clinical suspicion is important
- US (Fig. 21.29) and CT may show large lymph node swellings.

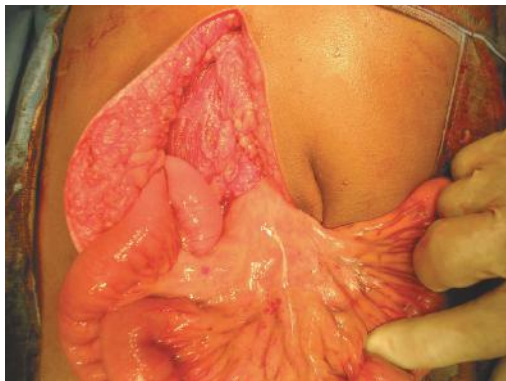


FIG. 21.28: Mesenteric lymphadenitis

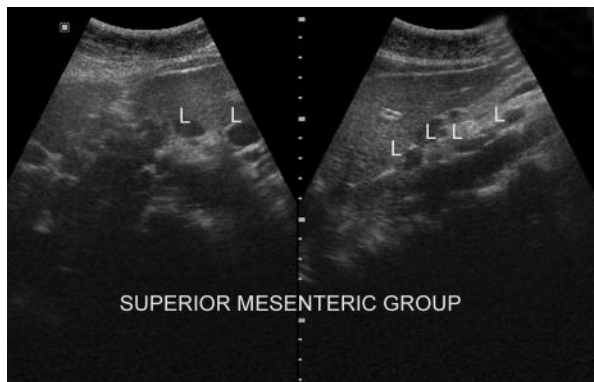


FIG. 21.29: US—Mesenteric lymphadenitis
(Courtesy: Dr V Ganesan)

Treatment

Medical management with antibiotics and supportive measures.

ACUTE COLONIC DIVERTICULITIS

Incidence and Etiology

- Common in the West
- Disease of the elderly
- Sigmoid colon is most commonly affected.

Pathogenesis

- Acute inflammation of the diverticulum of large bowel (colonic diverticulitis)
- They are thought to arise from increased pressure in the colonic lumen, occurs at weak areas between the taeniae where vessels perforate through the submucosal layer.

Complications

- Bleeding
- Perforation
- Peritonitis
- Abscess.



FIG. 21.30: Barium enema— Diverticulitis of colon

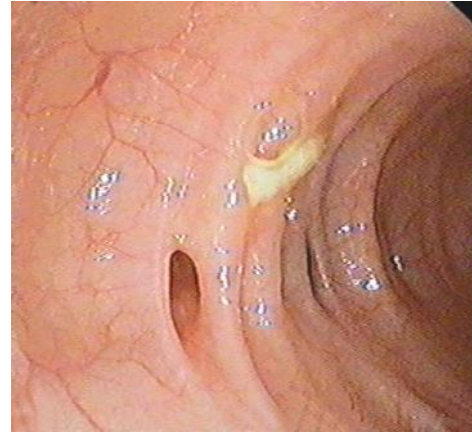


FIG. 21.31: Colonoscopy—Acute diverticulitis
(Courtesy: Dr Mani Veeraghavan)

The severity of diverticulitis is classified by Modified Hinchey classification

- Stage 1 – pericolic abscess
- Stage 2a – distant abscess amenable to percutaneous drainage
- Stage 2b – complex abscess associated with/without fistula
- Stage 3 – generalized purulent peritonitis
- Stage 4 – fecal peritonitis

Symptoms

- Colonic diverticulitis presents with colicky pain in the left flank
- When perforated, it may form an abscess in the paracolic region and present with high grade fever and a palpable tender lump.

Signs

- Left iliac fossa tenderness
- Tender mass (abscess) may be palpable.

Relevant Investigations

- Double contrast barium enema (Fig. 21.30) is informative
- Colonoscopy (Fig. 21.31) is diagnostic
- US and CT (Fig. 21.32) are useful in diagnosing abscesses.

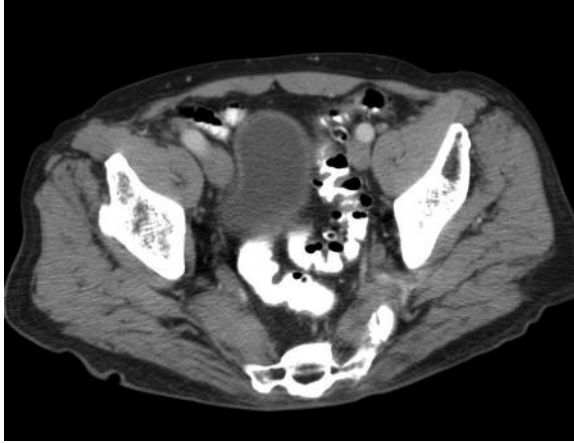


FIG. 21.32: CT—Diverticulitis of sigmoid colon

Treatment

- Uncomplicated diverticulitis needs to be treated with antibiotics
- Perforated diverticulitis with or without abscess formation needs surgical intervention.

ACUTE MECKEL'S DIVERTICULITIS

Incidence and Etiology

- Meckel's diverticulum* - An embryological remnant (Fig. 21.33) present as a free diverticulum with a wide mouth, about 25 to 30 cm from the ileocecal junction; may contain ectopic gastric mucosa (reason not known)
- Occurrence follows rough rule of 2s:
 - ❖ 2 percent of the population
 - ❖ 2 feet from the ileo-cecal junction
 - ❖ 2 inches in length
 - ❖ 2 times more common in males than in females.

Complications: Perforation and peritonitis.

* Meckel's diverticulum is often found incidentally during laparotomy and remains asymptomatic in majority of individuals.



FIG. 21.33: Meckel's diverticulum

Symptom

Right iliac fossa pain.

Sign

Tenderness right iliac fossa (McBurney's point).

Meckel's diverticulitis may present with peptic ulceration, lower GI hemorrhage, perforation, intussusception and intestinal obstruction.

Differential Diagnosis

- Acute appendicitis
- Right ureteric colic
- Right ovarian pathology in women.

Relevant Investigations

No investigation is useful.

- Acute diverticulitis warrants diverticulectomy
- Perforation and peritonitis need appropriate management.

ACUTE SOLITARY CECAL DIVERTICULITIS

Incidence and Etiology

- Solitary diverticulum is common in the cecum
- Inflammation of the diverticulum presents like acute appendicitis and mislead the examiner.

Symptom

Pain in the right iliac fossa (similar to acute appendicitis).

Sign

A lump may be felt in the right iliac fossa.

Clinically it mimics acute appendicitis, and many times, cecal diverticulitis is identified in second surgery, after appendectomy.

Relevant Investigations

US and CT may be useful.

Treatment

Diverticulectomy or right hemicolectomy is the treatment of choice.

ACUTE ULCERATIVE COLITIS

Incidence and Etiology

- A chronic inflammatory bowel disease with relapses and remissions
- Acute inflammation is one of its clinical presentations.

Complications: Perforation and peritonitis.



FIG. 21.34: X-ray—Toxic megacolon

Symptoms

- Incessant diarrhea, mixed with blood, mucus and pus with constitutional symptoms
- They present an emaciated appearance.

Signs

Deep ulcers involving the entire colon is called *Toxic megacolon*, presenting as grossly distended abdomen.

Relevant Investigations

- Plain X-ray abdomen (Fig. 21.34) is diagnostic
- Colonoscopy is essential for diagnosis.

Treatment

- Uncomplicated cases are treated medically
- Perforation and peritonitis need appropriate treatment.

ACUTE INTESTINAL OBSTRUCTION

- Intestinal obstructions may be dynamic or adynamic.
 - ❖ Dynamic obstruction is a physical or mechanical obstruction of the intestinal lumen due to various causes. They are:
 - Intramural causes (e.g. fecal impaction, worms)
 - Mural causes (e.g. strictures or stenosis due to tuberculosis, malignancies)
 - Extramural causes (e.g. adhesions, obstructed hernia)
 - ❖ Adynamic obstruction of the bowel is due to its neural (autonomic) paralysis, commonly seen after abdominal surgery in the immediate postoperative period, peritonitis or any other cause like fractures, tight bandages, etc.
- Intestinal obstruction may be at different levels. They are:
 - ❖ Small intestinal (high or low)
 - ❖ Large intestinal (colonic or sigmoid)
- Causes of intestinal obstruction are different for different age groups (Table 21.8).

Symptoms

1. Sudden episodic colicky abdominal pain
2. Vomiting
3. Constipation
4. Abdominal distension

(The symptoms vary according to the level of obstruction)

- *Abdominal pain:* It is sudden and squeezing, and the patient doubles up. It is felt in the umbilical region, sometimes accompanied by the appearance of a contracting loop. There may be pain free intervals. Colonic pain presents in the hypogastrium

Table 21.8: Causes of intestinal obstruction in different age groups

<i>Newborn</i>	<i>Infants</i>	<i>Adolescents</i>	<i>Adults</i>	<i>Elderly</i>	<i>Rare causes</i>
Duodenal atresia	Helminths	Bands	Postoperative adhesions	Growth	Enteroliths
Pyloric stenosis	Intussusception	Intussusception	Intussusception	Intussusception	Foreign bodies
Meconium ileus		Meckel's diverticulum	Volvulus	Obstructed or strangulated hernia	Gallstones
Hirschsprung's disease		Obstructed or strangulated hernia	Obstructed or strangulated hernia		Trichobezoar
			Growth		Phytobezoar

- **Vomiting:** Vomiting is predominant in high obstructions. The vomitus consists of gastric contents, followed by the duodenal and lastly the intestinal, depending on the level of obstruction. In the late stages, the vomitus becomes feculent – ominous sign. Vomiting by itself is a late sign of chronic intestinal obstruction
- **Constipation:** The patient evacuates his bowel (contents distal to obstruction) once or twice, and constipation becomes a noticeable feature after 24 hours

Diarrhea can be a feature in certain situations like intussusception (red currant jelly stools), Richter's hernia, adynamic obstruction caused by mesenteric vascular occlusion, pelvic abscess, etc.

- **Abdominal distension:** Common feature of intestinal obstruction. Distension is
 - ❖ centrally located in small bowel obstruction (ladder pattern),
 - ❖ more on the flanks when distal colon is obstructed (asymmetrical)
 - ❖ more on the left flank in sigmoid volvulus
- **Dehydration:** When vomiting is pronounced as in high level obstructions, dehydration is a presenting feature.

Early dehydration and less abdominal distension suggests duodenal or jejunal obstruction whereas, late dehydration and more abdominal distension suggests distal ileal obstruction. Vomiting and dehydration are usually not present in isolated acute colonic obstruction.

Signs

- **General:** Pulse rate and blood pressure are maintained at normal levels in the initial stages. As dehydration becomes prominent, tachycardia and hypotension result
 - **Abdomen:** Bowel sounds are not heard as obstruction worsens.
- The summary of signs and symptoms related to intestinal obstruction are given in Table 21.9.

Relevant Investigations

- Plain X-rays of abdomen in the erect posture will reveal multiple air fluid levels (Fig. 21.35) and colonic obstruction may show distended colon also (Fig. 21.36).

Table 21.9: Signs and symptoms related to intestinal obstruction

Level of obstruction	Signs and symptoms					
	Duration of colic	Pain free interval	Vomiting	Distension of abdomen	Constipation	Dehydration
High	Short	Short	More	Minimal	Not constant	Severe
Low	Long	Long	Less	More	Late feature	Mild-to-moderate

(Findings of clinical examination and treatment of individual diseases are discussed at appropriate headings in the following pages)



FIG. 21.35: X-ray—Multiple air fluid levels of small bowel obstruction



FIG. 21.36: X-ray—Distention of large bowel

Treatment

- Inpatient/intensive care
- Nil by mouth
- Intravenous fluids
- Correction of electrolytes
- Nasogastric decompression
- Urinary catheterization of better monitoring
- Identifying and treating the cause.*

* If adhesions are the likely cause, a trial of conservative management is justified.

GALLSTONE ILEUS

Incidence and Etiology

Pathogenesis

- Gallstone enters the bowel through a perforated gallbladder (postcholecystitis) adherent to the small bowel (cholecysto-enteric fistula)
- When the stone is big and reaches the ileocecal junction, it causes small bowel obstruction.

Symptoms

Previous history of vague attacks of right upper quadrant pain, suggesting frequent cholecystitis.

Signs

Features of intestinal obstruction.

Relevant Investigations

- Plain X-ray abdomen (Fig. 21.37) almost always shows air in the biliary tree as bowel gas passes through the cholecystoenteric fistula. A gallstone may also be seen in the right lower quadrant, if it is radiopaque
- CT (Fig. 21.38) is more informative. Rarely, a gallstone may also be seen in the small bowel, and also in the gallbladder if there are many.

Treatment

- During laparotomy:
 - ❖ Simple crushing of stone with finger from outside the bowel may be enough if it is soft
 - ❖ Simple surgical removal through an enterotomy is required if the stone is hard and big
 - ❖ Cholecystectomy must be performed with closure of fistula.

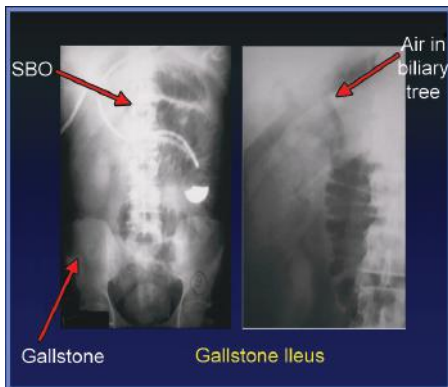


FIG. 21.37: X-ray—Gallstone ileus



FIG. 21.38: Air in the biliary system

ACUTE INTUSSUSCEPTION

Incidence and Etiology

Intussusception is the invagination of a segment of bowel into the distal adjacent loop (proximal into the distal) (Fig. 21.39).

In Children

- Two per thousand infants are affected with male preponderance, commonly affecting the age group of 3 months to 1 year
- Commonly, it is secondary to an enlarged Peyer's patch due to viral or bacterial infections
- The other less common causes are:
 - ❖ Meckel's diverticulum
 - ❖ Duplication cyst in the bowel wall
 - ❖ Polyp.

In Adults

- Intussusception of small bowel is always secondary to a polypoid lesion, a lipoma (Figs 21.40A and B)
- In large bowel, it is due to a malignant polypoid lesion (Figs 21.41A and B).



FIG. 21.39: Ileoileal intussusception



FIG. 21.40A: Ileoileal intussusception



FIG. 21.40B: Lipoma the cause of ileoileal intussusception

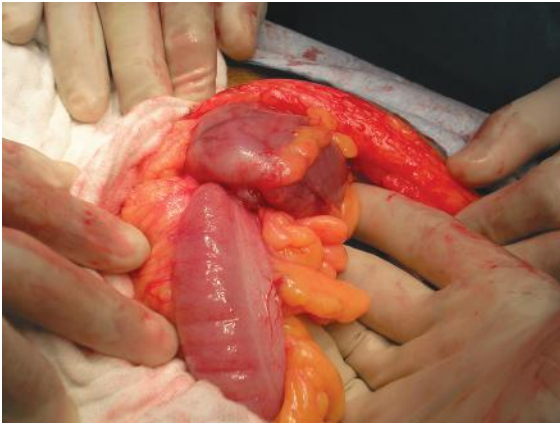


FIG. 21.41A: Colocolic intussusception



FIG. 21.41B: Malignant growth of colon cause for intussusception

Complications: When the mesentery is drawn between the loops, it may result in vascular compromise, which may lead to strangulation, gangrene and perforation.

Symptoms

- In children, there may a history of preceding gastroenteritis following a change in diet (weaning from milk to solid food)

- Severe acute colicky pain, with abdominal distension
- Passing of frequent semisolid stools with bright red blood may be predominant (red currant jelly).

Signs

- During the attacks of pain, a sausage-shaped mass may be felt, which appears during the time of colic and disappears after the colic disappears. The right iliac fossa is empty – Sign de Dance
- Rectal examination may reveal bloodstain on the examining finger (red-currant jelly)
- Colo-rectal intussusception may be felt by the examining finger on rectal examination, or it may even present through anus, resembling a rectal prolapse.

Relevant Investigations

- Plain X-ray abdomen – soft tissue shadow in the region of transverse colon with empty distal colon. Multiple air fluid levels may be seen when obstruction predominates
- Barium enema may show a filling defect called pincer-shaped filling defect (caused by the intussusceptum with the intussusciptiens)
- Colonoscopy can identify, colonic intussusceptions (Fig. 21.42A)
- US and CT (Figs 21.42B and C) will reveal the intussuscepting mass (pseudokidney appearance).

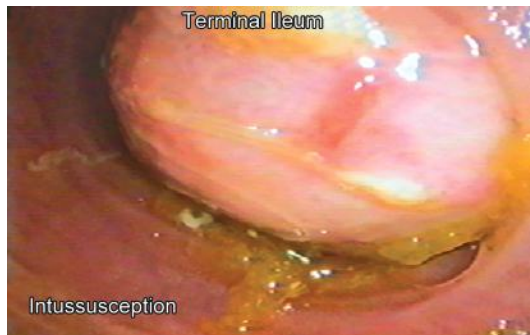
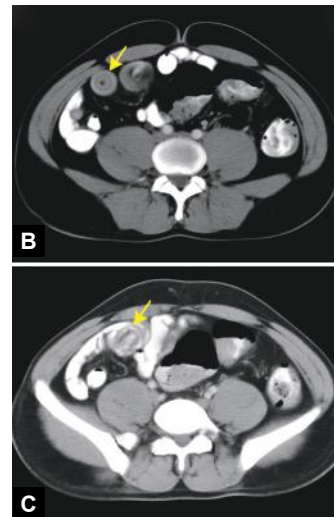


FIG. 21.42A: Intussusception of small bowel
(Courtesy: Dr Mani Veeraraghavan)



FIGS 21.42B and C: CT—Intussusception

Treatment

- Barium enema and colonoscopy, by themselves may reduce the colonic intussusception
- Laparotomy is required to reduce the small bowel intussusception, and treat the cause appropriately
- Bowel resections may be needed if the bowel segment is strangulated, and nonviable
- Perforation and peritonitis need appropriate treatment.

SWALLOWED FOREIGN BODIES

Incidence and Etiology

- Common in children and psychotic individuals
- Most foreign bodies pass through GIT without any difficulty.

Symptoms

- Abdominal pain may be present
- Vomiting may supervene.

Signs

- Signs of intestinal obstruction may appear
- Chest signs may appear.

Examine the oral cavity, oropharynx or nasopharynx for impacted foreign body, before asking for radiographs.

Relevant Investigations

- Plain X-ray of throat (Fig. 21.43), chest and abdomen may identify the foreign body
- Bronchoscopy/Gastroscopy may be required.

- By the time the patient is seen by the clinician, the foreign body might be expelled through feces, if it is small and blunt
- Since button cells have the tendency to erode through the bowel wall, it is better to monitor its position by serial radiography, and removed if stuck in a place.

Treatment

- Blunt objects reaching the stomach will pass without difficulty
- Sharp foreign bodies impacted in the GIT should be removed (endoscopically or by open surgery)
- Catastrophic bleed, obstruction and perforation peritonitis need laparotomy.



FIG. 21.43: Foreign body larynx (Courtesy: Bharat scans)

PERFORATED BOWEL PATHOLOGIES

There are a variety of intra-abdominal pathologies, which are serious emergencies, and also threaten life, if not recognized early and treated. Such conditions are called “acute abdominal catastrophes”. Most of these conditions are a result of infection of peritoneum called ‘peritonitis’, caused by the leak of infected intestinal contents into the peritoneal cavity. Since the peritoneum is a semi-permeable membrane, it allows the spread of infection systemically into the blood to result in septicemia, a fatal condition.

Peritonitis consists of three stages. They are:

1. *Stage of peritonism:* Occurs due to the chemical irritation of the peritoneum caused by the leaked contents of the bowel, and the patient feels sudden excruciating pain. Constitutional symptoms are not prominent, though tenderness and guarding can be elicited
2. *Stage of reaction:* The contents of the bowel are neutralized by the peritoneal exudates, and pain becomes less. Muscle guarding may be present, and shifting dullness may be elicited indicating the presence of free fluid in the abdomen
3. *Stage of spreading peritonitis:* This is an ominous stage, as there are signs of severe infection. They exhibit classic facies, called “Facies Hippocratica” – sunken eyes, hollow cheeks and anxious face. Severe tachycardia (fast thready pulse) is present. Abdomen shows board like rigidity, indicating generalized peritonitis. Death becomes imminent if not attended to, surgically.

The signs and symptoms of perforations caused by various parts of the bowel have certain indicative findings. They are given in the Table 21.10.

Table 21.10: Signs and symptoms of perforations caused by various diseases of the bowel

<i>Signs and symptoms</i>	<i>Perforation of</i>						
	<i>Duodenal or gastric ulcer (benign/malignant)*</i>	<i>Appendicitis**</i>	<i>Cholecystitis</i>	<i>Typhoid/Tubercular ulcer</i>	<i>Ulcerative colitis</i>	<i>Diverticular disease</i>	<i>Colonic malignancy</i>
Previous history	Frequent use of antacids	Nil or pain in the right iliac fossa in the past (treated or untreated)	Gallstones or chronic cholecystitis	Prolonged fever (high grade in typhoid and low grade in tuberculosis)	Repeated attacks of diarrhea and abdominal pain	Repeated attacks of constipation and abdominal pain	Constipation
History of drug intake	NSAIDs	Nil	Nil	Treatment for typhoid fever	Medical management	Medical management	Laxatives
Area of abdominal pain	Upper abdominal	Umbilical to start and then in the right iliac fossa	Right hypochondrial	Umbilical or lower abdominal	Flanks and hypogastric	Umbilical or flanks	Flank on the side of lesion
Gastro-intestinal bleeds	Upper	Nil	Nil	Lower	Lower	Lower	Lower
Treatment	Surgical						

* Leaks of gastric contents due to perforated gastric ulcer (posteriorly placed), into the lesser sac may mask abdominal symptoms
 ** Clinical presentation of perforated Meckel's diverticulitis is the same as that of perforated acute appendicitis.

Differential Diagnosis

- Myocardial infarction
- Diaphragmatic irritation caused by lower lobar lung lesions
- Acute pancreatitis
- Ruptured or dissecting aortic aneurysm.

Relevant Investigations

- Plain X-rays of abdomen in the erect posture is confirmatory (gas under the diaphragm) in most cases (Figs 21.44 and 21.45) and peritonitis gives the classic 'ground glass appearance' (Fig. 21.46)
- CT (Fig. 21.47) is useful.

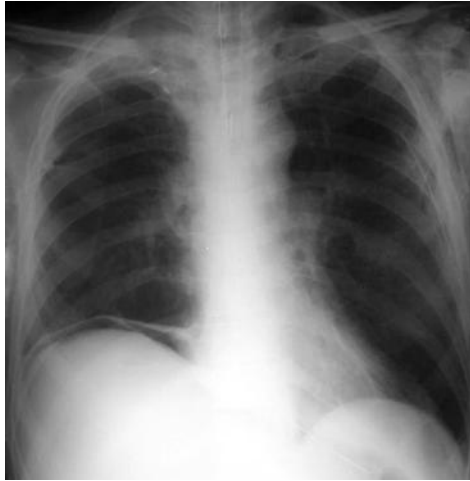


FIG. 21.44: X-ray—Air under the right dome of diaphragm—duodenal ulcer perforation



FIG. 21.45: Minimal air under right dome of diaphragm - appendicular perforation



FIG. 21.46: X-ray abdomen - Ground glass appearance of peritonitis

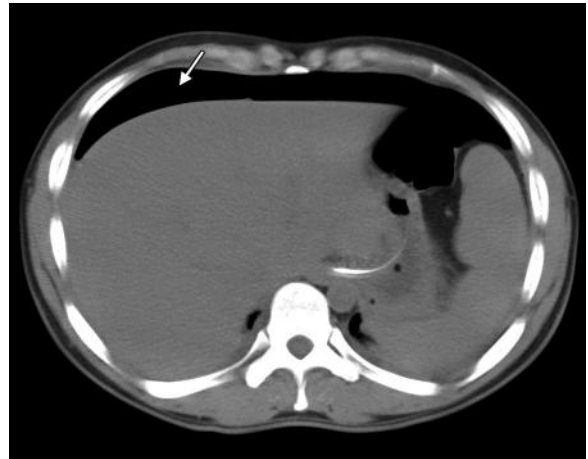


FIG. 21.47: CT—Pneumoperitoneum

Treatment

Treatment is always surgical:

- ❖ Closure of bowel perforation if it is simple and of benign cause
- ❖ Cholecystectomy (gallbladder perforations)/ appendicectomy (appendicular perforations)/appropriate bowel resections (benign or malignant bowel perforations)
- ❖ Peritoneal toileting is mandatory and should be complete.

INTESTINAL STRICTURES

Incidence and Etiology

- Obstructions can be caused by strictures due to:
 - ❖ Tuberculosis (healing lesions)
 - ❖ Malignant lesions
- Postoperative strictures are not uncommon.

Symptoms

- Symptoms of subacute or acute intestinal obstruction
- History of weight loss, low-grade pyrexia, anemia and vague abdominal pain may be present.

Signs

- Clinical examination may show a mass in the right iliac fossa (differential diagnosis—Crohn's disease)
- Ascites may be present.

Relevant Investigations

- Plain X-ray abdomen (erect) shows air fluid levels
- X-ray chest may reveal a primary tubercular lesion.

Treatment

- Laparotomy is needed for acute obstructions
- Strictureplasty or bypass procedures or resections are done for tubercular strictures
- Radical resections or bypass procedures are done for malignant strictures.

BANDS AND ADHESIONS

Incidence and Etiology

- Bands may be congenital, but adhesions are post surgical
- The adhesions may be like a small band or generalized, flimsy or dense, single (Figs 21.48 and 21.49) or multiple.

Symptoms and Signs

Features of subacute or acute intestinal obstruction, months or years after surgery.

Relevant Investigations

- Plain radiographs of abdomen are useful in diagnosis
- Diagnostic laparoscopy is conclusive.

Treatment

- Release of adhesions by laparoscopy or by open surgery
- Open surgery and release of adherent bowel loops for dense adhesions obstructing the bowel
- It is better to manage the acute obstructions by trial medical management with gastric suction and intravenous fluids

Note: Surgery has the disadvantage of recurrence, especially in generalized adhesions.



FIG. 21.48: Band in the pelvis causing internal rotation of small bowel

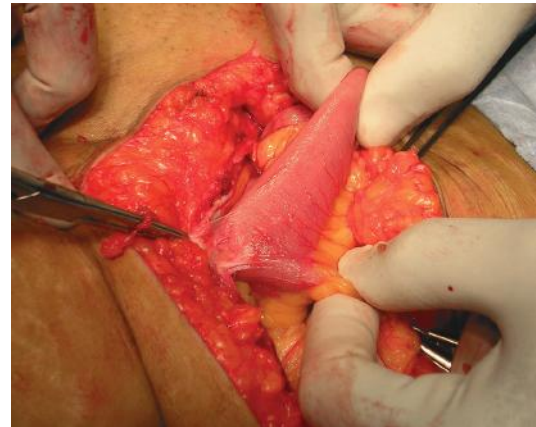


FIG. 21.49: Adhesions of small bowel to previous surgery scar

ENTEROLITHS/FOOD BOLUS

Incidence and Etiology

Obstructions may be caused by enteroliths or food bolus, resulting from poor chewing in an edentulous patient, high consumption of high fiber (e.g. orange pith), usually at a pre-existing narrowing due to tuberculosis, Crohn's disease and surgery.

Symptoms and Signs

Symptoms of subacute or acute intestinal obstruction.

Relevant Investigations

Plain X-ray abdomen (erect) shows air fluid levels. Diagnosis is more clinical and is difficult.

Treatment

Surgery is indicated for acute obstructions, and the bolus or enterolith can be milked into the large intestine and rarely it is necessary to open the bowel to remove it.

VOLVULUS

- Volvulus is defined as a twist of the bowel around its mesenteric axis
- It is more common in the large bowel (commonly the sigmoid colon) than in the small bowel
- Rotation of more than 180 degrees may result in strangulation.

SIGMOID VOLVULUS

Incidence and Etiology

Disease of the middle aged and elderly.

Pathogenesis

Rotation of the sigmoid around its axis occurs when its mesentery is unusually long.

Symptoms

- Sudden severe pain, frequently when straining to pass stool. The patient retches and develops hiccoughs
- The patient may give history of attacks of abdominal pain with constipation, relieved by passing watery stools and large volumes of flatus.



FIG. 21.50: X-ray – Sigmoid volvulus

Signs

- Abdomen rapidly distends, disproportionate to the duration of pain, and the distension is confined more to the left flank
- Rectum is empty on examination.

Relevant Investigations

Plain radiograph of the abdomen will reveal a distended sigmoid, coffee bean appearance – Freeman Dahl sign (Fig. 21.50) (Convergence of three white lines towards the base of the pedicle).

Treatment

- Untwisting of the volvulus, and fixing the colon to parietal peritoneum to prevent recurrence
- Sigmoidectomy is the treatment of choice in long redundant sigmoid colon.

CECAL VOLVULUS

Incidence and Etiology

- This occurs in those whose entire right colon has a mesentery continuous with that of the small bowel, and the cecum does not lie in the right iliac fossa
- More common in women and during pregnancy.

Clinical Presentation

- A distended, tense palpable resonant mass in the umbilical region, with an empty right iliac fossa
- Features of distal small bowel obstruction.

Relevant Investigations

Plain radiograph of the abdomen is diagnostic (Cecal bubble is seen).

Treatment

Untwisting of the volvulus with or without resection of the segment of the bowel and cecopexy is required.

MIDGUT VOLVULUS

Incidence and Etiology

- In children, this occurs due to malrotation and failure of fixation of midgut, usually before one year, rarely in neonates
- In adults, a loop of bowel rotates around a point of adhesion (to the abdominal wall or to an adjacent viscera).

Symptom

Severe abdominal pain.

Signs

Signs of intestinal obstruction.

Relevant Investigation

Contrast study shows 'Cork-screw effect'.

Treatment

Emergency laparotomy and correction of the defect with fixing of the bowel.

Incidence and Etiology

- Rare, potentially life threatening
- Stomach twists by more than 180 degrees causing in the long axis (organo-axial rotation) causing closed loop obstruction
- Mesentero-axial rotation occurs in one-third of cases.

Symptoms*

- Severe upper abdominal pain
- Vomiting
- Dyspnea.

* Subdiaphragmatic gastric volvulus causes chest pain.

Sign

Upper abdominal distension.

Relevant Investigation

Plain X-ray shows gas filled bowel in lower chest or epigastrium.

Treatment

Emergency laparotomy to reduce the volvulus and gastropexy.

INTESTINAL OBSTRUCTION DUE TO HERNIAE (INTERNAL AND EXTERNAL)

Incidence and Etiology

Internal hernias may occur due to congenital and acquired reasons.

- *Congenital*: Herniation through the pockets under the superior mesenteric and inferior mesenteric vessels
- *Acquired*: External hernia like strangulated inguinal or femoral hernia can cause intestinal obstruction.



FIG. 21.51: Internal herniation of small intestines



FIG. 21.52: Intestinal obstruction due to obstructed inguinal hernia

Symptoms

- Internal hernia may present with features of intestinal obstruction (Fig. 21.51). Symptoms depend on the level of obstruction.

Sign

External hernia is clinically evident and may show signs of obstruction or strangulation (Fig. 21.52).

Relevant Investigation

Plain radiographs and CT are useful in diagnosis.

Treatment

- Reduction of herniating bowel and closure of defects
- Strangulated bowel may need resection.

PARALYTIC ILEUS

Incidence and Etiology

- Prolongation of ileus after abdominal operations beyond the third postoperative day and last for a week or more

Table 21.11: Causes of paralytic ileus

<i>Causes</i>	<i>Pathology</i>	<i>Mechanism</i>
Sympathetic dysfunction	Postoperative ileus	Reflex inhibition
	Spinal injury	
	Acute renal colic	Retroperitoneal hemorrhage
	Trauma	
	Acute pancreatitis	
Local causes	Retroperitoneal malignancy	Malignant infiltration
	Peritonitis	Bacterial infection
	Advanced mechanical obstruction	Excessive distension of bowel
Pharmacological	Anticholinergics	Interference with smooth muscle contractility
	Antidiarrheals	
	Ganglion blockers	
Biochemical	Hypokalemia	
	Uremia	
	Diabetic crisis	
	Hypoxia	

- Usual cause being electrolyte imbalance in the postoperative period
- Retroperitoneal or intraperitoneal hemorrhage and sepsis are the other causes
- Trivial causes like an injection, application of Plaster-of-Paris bandage and any injury anywhere, fractures, etc. (Table 21.11).

Symptoms

- Abdominal distention without pain
- Vomiting is a predominant symptom.

Sign

The abdomen is resonant with the characteristic absence of bowel sounds.

Relevant Investigations

- Serum electrolyte levels show abnormalities
- Plain radiographs of the abdomen will show ‘step-ladder pattern’ of small bowel (Fig. 21.53) with distension of both small and large bowels.



FIG. 21.53: Step-ladder pattern of paralytic ileus

Treatment

- Correction of electrolyte imbalances
- Treatment of retroperitoneal or intraperitoneal causes like hemorrhage and sepsis.

TORSION OF MESENTERIC CYST

Incidence and Etiology

- Cystic lesions of mesentery are more common than solid tumors (2:1)
- Cystic tumors of mesentery are:
 - ❖ Chylous cyst, serous cyst (developmental) – more common
 - ❖ Lymphangioma (lymphatic tissue) – more common
 - ❖ Traumatic cyst (trauma)
 - ❖ Enteric cyst, dermoid (embryonic rests)
- Majority of cysts are benign (except lymphangiosarcoma, malignant teratoma).

Torsion occurs more by its weight, and mobile nature of mesentery encourages this torsion.

Symptom

Sudden acute abdominal pain.

Signs

- Central abdominal tenderness with guarding and rigidity
- A cystic lump may be felt in the central abdomen.

Relevant Investigation

US and CT of abdomen are diagnostic.

Treatment

Emergency laparotomy is needed. The cyst should be removed.

TORSION OF OMENTUM

Incidence and Etiology

- More common in 4th to 5th decades
- Equal sex incidence
- Causes of omental torsion are:
 - ❖ *Primary*: Always unipolar, cause is unknown
 - ❖ *Secondary*: Usually bipolar, associated with adhesions (intra-abdominal inflammation—tuberculosis, postsurgical adhesions, internal and external herniae).

Pathogenesis

- The omentum twists on its long axis to an extent causing vascular obstruction
- May vary from mild vascular obstruction producing edema to complete strangulation leading to infarction and gangrene
- The situations to cause torsion are:
 - ❖ Redundant and mobile segment
 - ❖ Fixed point around which the segment can twist

- The precipitating factors for torsion are:
 - ❖ Anatomic variations (bifid omentum, accessory omentum, large and bulky omentum)
 - ❖ Venous redundancy
 - ❖ More common in right side (due to increased bulk and mobility).

Symptoms

- Acute abdominal pain usually is localized to right lower quadrant
- Movement increases the pain
- Nausea and vomiting are common.

Signs

- Tenderness, rigidity and guarding of abdomen
- A vague tender mass may be felt in the upper abdomen.

Differential Diagnosis

- Acute cholecystitis
- Acute appendicitis
- Torsion of right ovary.

Relevant Investigation

US and CT of abdomen are diagnostic.

Treatment

- Emergency laparotomy is needed.
 - ❖ Twisted omentum needs to be excised with release of adhesions
 - ❖ Associated hernia needs repair.

COLICS

Incidence and Etiology

Colic is defined as a sudden squeezing or griping pain lasting for about 3 to 5 minutes with pain free intervals.

- Nausea, vomiting and retching are common accompaniments
- The cause of a colic is partial obstruction of a tubular structure due to varied causes.

Table 21.12: Clinical features of various colics

<i>Clinical feature</i>	<i>Biliary colic</i>	<i>Renal colic</i>	<i>Colics</i>		
			<i>Ureteric colic</i>	<i>Intestinal colic</i>	<i>Appendicular colic</i>
Incidence	Fat, fertile, flatulent, female of fifty	Young age	Younger age	Any age	Any age
Etiology	Gallstones	Renal calculus	Ureteric calculus, papilloma, clot, stricture	Parasitic infestations (younger age), strictures (middle age), malignancy (old age)	Fecoliths, Worms
Nature of pain	Right hypochondrial pain, referred to right scapula or shoulder	Severe pain in the lumbar region radiating towards umbilicus	Severe colicky pain radiating from loin to groin, testis or thigh	Colicky pain in the umbilical region	Colicky pain in the right iliac fossa
Associated symptom	Dyspepsia	Urinary symptoms	Urinary symptoms	Constipation or diarrhea	Repeated attacks of dull pain in right iliac fossa

Symptoms

- Severe griping pain with pain free intervals
- Location of pain is definite (Table 21.13)
- Radiation (Fig. 21.54) may be present:
 - ❖ Radiation to right scapula or shoulder is common with biliary colic
 - ❖ Radiation to external genitalia and groins (e.g. ureteric colic)
- Vomiting is usually present

Table 21.13: Location of pain related to regions of abdomen

<i>Right hypochondrium</i>		<i>Left hypochondrium</i>
Biliary colic		Left renal colic
Right renal colic		
<i>Right lumbar region</i>	<i>Umbilical region</i>	<i>Left lumbar region</i>
Right renal colic	Intestinal colic	Left renal colic
<i>Right iliac fossa</i>	<i>Hypogastrium</i>	<i>Left iliac fossa</i>
Appendicular colic	Uterine colic	Left ureteric colic
Right ureteric colic	Urethral colic	

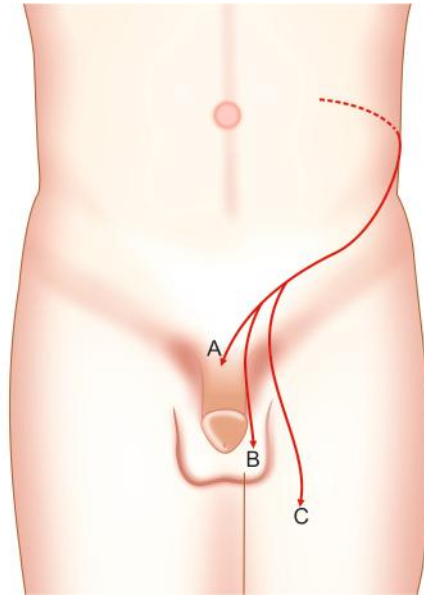


FIG. 21.54: Ureteric colic: (A) Base of penis; (B) Genitalia; (C) Thigh

- Fever may be present if infection is associated
- Jaundice may be present if cholangitis is associated
- Loose stools may be present if colitis is associated
- History of similar episodes in the past
- Family history may present regarding biliary and urinary calculi.

Signs

- Tenderness in the region of pathology
- Mass may be felt proximal to the level obstruction:
 - ❖ Right upper quadrant (e.g. distended gallbladder)
 - ❖ Right lumbar (e.g. obstructed and enlarged kidney as in PUJ obstruction)
 - ❖ Umbilical (e.g. distended bowel)
 - ❖ Hypogastric (e.g. distended uterus in dysmenorrhea).

- Biliary colic, jaundice, palpable gallbladder (e.g. cholangitis with mucocele, gallbladder choledochal cyst)
- Renal colic, urinary symptoms, palpable kidney (e.g. obstruction at PUJ)
- Ureteric colic, urinary symptoms, tenderness in iliac fossa (e.g. obstruction of ureter)
- Intestinal colic, exaggerated bowel sounds (e.g. obstruction of small bowel)
- Appendicular colic, tenderness right iliac fossa (e.g. obstructive appendicitis)
- Uterine colic, dysmenorrhea, palpable uterus (e.g. congestive dysmenorrhea).

Relevant Investigations

Hematology

- Leukocytosis in infective pathologies (e.g. acute cholecystitis)
- Raised ESR in all infective pathologies.

Radiology

- *Plain X-ray abdomen:* Gas filled loops of bowel (e.g. acute intussusceptions, acute intestinal obstruction)
- *Ultrasonography:* Radiopaque shadows in the abdomen (e.g. renal stones, ureteric stones).

Treatment Plan

- Medical management will suffice in most instances
- Obstructive pathologies may require surgery or removal to relieve the cause of obstruction
- Repeated attacks of colic will require evaluation and management.

GASTROINTESTINAL HEMORRHAGE

Definitions

- Hematemesis—vomiting blood, which may be fresh or partly altered.
- Melena—passing altered blood (meaning digested blood) per rectum.
- Hematochezia—passing blood per rectum bright in color
- Occult bleeding—bleeding not visible to naked eye, but microscopic.

Types of Gastrointestinal Hemorrhages

The blood loss due to gastrointestinal hemorrhage is divided into three types:

1. Mild—less than 500 ml.
2. Moderate—500 to 1500 ml.
3. Severe—more than 1500 ml.

- Gastrointestinal hemorrhages, when they are in large quantities, called major hemorrhages present either as vomiting of blood or passage of blood per rectum, but usually they coexist
- Many times, melena is the sole clinical presentation of upper gastrointestinal bleed.

While evaluating a GI hemorrhage, the following pathologies are to be kept in mind (Fig. 21.55)

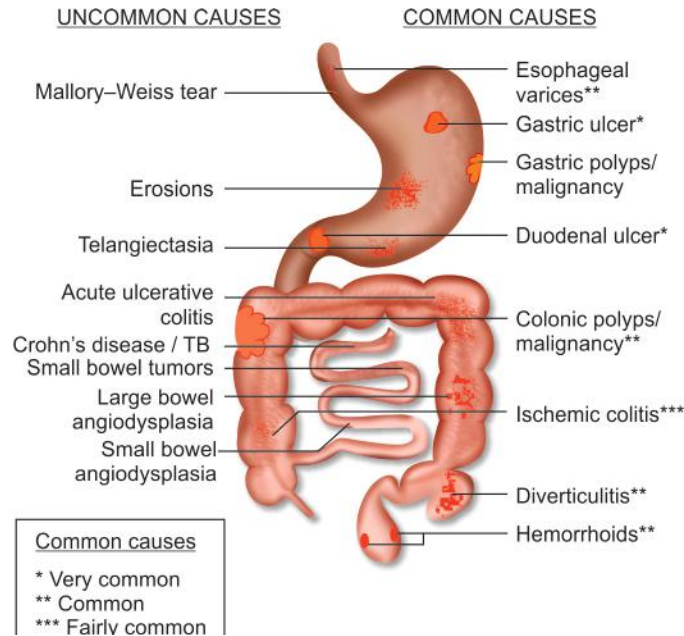


FIG. 21.55: Causes of gastrointestinal bleeding

Esophagus

- Reflux esophagitis
- Esophageal varices
- Mallory–Weiss tears
- Corrosive poisoning.

Stomach and Duodenum

- Acid peptic disease
- Gastric polyps
- Gastric lymphoma
- Carcinoma stomach
- Corrosive poisoning.

Melena*Small Bowel*

- Angiodysplasia
- Diverticulitis
- Radiation enteritis
- Infections and inflammations
- Ischemic disease
- Intussusception
- Richter’s hernia
- Benign tumors
- Malignant tumors.

Hematochezia*Large Bowel*

- Angiodysplasia
- Diverticulitis
- Radiation colitis
- Infections and inflammations

- Ischemic disease
- Inflammatory bowel disease
- Benign polyps
- Malignant tumors.

Rectum and Anus

- Polyps
- Malignant tumors
- Hemorrhoids, fissures.

Lower gastrointestinal hemorrhages do not present with hematemesis.

Eliciting History

Hematemesis

1. *Nature of bleed*
 - ❖ Frank blood (e.g. esophageal varices)
 - ❖ Altered blood (e.g. peptic ulcer, gastric malignancy).
2. *Duration of hematemesis*
 - ❖ Short duration (e.g. acute hyperacidity)
 - ❖ Recurrent attacks (e.g. chronic duodenal ulcer, gastric malignancy).
3. *Association of rectal bleed*
 - ❖ Melena (e.g. upper GI bleed)
 - ❖ Hematochezia (e.g. lower GI bleed).
4. *Association of abdominal pain*
 - ❖ Absence of abdominal pain (e.g. esophageal varices)
 - ❖ Presence of abdominal pain (e.g. chronic duodenal ulcer, gastric malignancy).
5. *Association of fever*: Fever – usually low grade (e.g. GI malignancy).
6. *Association of jaundice*: Jaundice may be present (e.g. periampullary carcinoma, hepatic failure).

Hematochezia

1. *Duration of bleed*
 - ❖ Short duration (e.g. hemorrhoids, inflammatory bowel disease, colonic malignancy)
 - ❖ Recurrent attacks and long duration (e.g. hemorrhoids, inflammatory bowel disease).

2. *Association of abdominal pain*
 - ❖ Absence of abdominal pain (e.g. hemorrhoids, malignancy)
 - ❖ Presence of abdominal pain (e.g. inflammatory bowel disease).
3. *Association of fever: Fever* (e.g. inflammatory bowel disease)
4. *Association of jaundice*
Jaundice may be present (e.g. portal hypertension, metastatic colonic malignancy)
5. *Association of bowel disturbances*
 - ❖ Constipation (e.g. colorectal malignancies)
 - ❖ Diarrhea (e.g. inflammatory bowel disease).

Past History

- History of pain (e.g. chronic duodenal ulcer, diverticulitis)
- History of bleeds (e.g. chronic duodenal ulcer, diverticulitis, hemorrhoids)
- Previous surgery (e.g. recurrent malignancy of GIT).

Family History

- Familial polyposis
- Colonic malignancy.

Clinical Examination

General

- Vital signs (e.g. shock)
- Breath for fetor (e.g. hepatic failure)
- Conjunctiva for anemia (e.g. GI malignancy), jaundice (e.g. hepatic failure)
- Tongue for anemia (e.g. GI malignancy)
- Neck for lymphadenopathy (e.g. GI malignancy)
- Hands for clubbing, palmar erythema, liver flap, etc. (e.g. liver failure).

- Mild hemorrhages are rarely associated with systemic signs. When the blood loss approaches about 40 percent of the blood volume, shock ensues
- The volume of blood loss either by vomiting or through rectum, is not a very reliable measure, as large amounts stay in the bowel.

Abdomen

■ Inspection

Distension

- ❖ Generalized (e.g. ascites of liver failure, malignant ascites)
- ❖ Right upper abdominal (e.g. hepatomegaly)
- ❖ Epigastric (e.g. left lobar hepatomegaly, carcinoma stomach)
- ❖ Left upper abdominal (e.g. splenomegaly).

Scars, swellings and sinuses

■ Palpation

Tenderness

- ❖ Right upper quadrant (e.g. hepatic failure)
- ❖ Epigastric (e.g. chronic duodenal ulcer)
- ❖ Left upper quadrant (e.g. chronic duodenal ulcer).

Lump

- ❖ Right upper quadrant (e.g. hepatomegaly, distended gallbladder)
- ❖ Epigastric (e.g. carcinoma stomach, left lobar hepatomegaly)
- ❖ Left upper quadrant (e.g. carcinoma stomach, splenomegaly).

■ Percussion

Percuss the liver for

- ❖ Its enlargement (e.g. chronic hepatitis, metastatic liver).

■ Auscultation

- ❖ Bowel sounds are usually normal
- ❖ Exaggerated bowel sounds may indicate obstruction of small bowel (e.g. intestinal colic).

Differential Diagnosis by Clinical History and Examination

- Hematemesis and melena (e.g. pathologies of upper GI tract proximal to ligament of Treitz)
 - ❖ With upper abdominal pain, epigastric tenderness (e.g. bleeding gastric/duodenal ulcer)
 - ❖ Without abdominal pain and
 - No clinical findings (e.g. esophageal varices, vascular malformations, upper GI malignancy)
 - Abdominal lump (e.g. upper GI malignancy).
 - ❖ Without abdominal pain and
 - Increasing jaundice, abdominal lump, + / – hepatomegaly (e.g. upper GI malignancy + / – liver metastases)
 - Fluctuating jaundice, palpable GB, + / – hepatomegaly (e.g. periampullary carcinoma + / – liver metastases).

- Melena (e.g. pathologies of lower GI tract distal to ligament of Treitz)
 - ❖ With abdominal pain
 - Epigastric tenderness (e.g. bleeding duodenal/gastric ulcer)
 - Umbilical tenderness (e.g. inflammatory bowel disease)
 - Flank tenderness (e.g. ulcerative colitis)
 - Palpable lump (e.g. proximal colonic malignancy).
 - ❖ Without abdominal pain
 - No clinical findings (e.g. esophageal varices, vascular malformations)
 - Abdominal lump (e.g. upper GI malignancy).
- Hematochezia (e.g. pathologies of lower GI tract in the colorectum)
 - ❖ With pain during defecation, positive inspection findings (e.g. acute fissure in ano)
 - ❖ Without perianal pain
 - Positive proctoscopy findings (e.g. hemorrhoids, rectal pathologies)
 - Negative proctoscopy findings (e.g. colonic pathologies)
 - ❖ With abdominal pain + / – fever, local tenderness (e.g. inflammatory bowel disease, diverticulitis)
 - ❖ With colicky pain, abdominal lump (e.g. obstructing colonic malignancy)
 - ❖ With jaundice + / – abdominal pain, + / – abdominal lump, + / – hepatomegaly (e.g. colonic malignancy with liver metastases).

Whatever be the external visible loss, signs of hypovolemia should be watched for.

Relevant Investigations

Hematology

- Hemoglobin and PCV for anemia
- Total and differential leukocyte count (e.g. tuberculosis, ulcerative colitis)
- Thrombocytopenia (e.g. hypersplenism)
- ESR may be raised in infections and malignancies
- Coagulation profile (e.g. bleeding disorders).

Coagulation profile should be assessed in all cases of GI bleed, as bleed by itself the sole presentation of coagulation disorders.

Liver Function Tests

- Serum bilirubin (e.g. raised levels indicate jaundice)
- Serum transaminases (e.g. raised levels indicate liver cell disease)
- Serum alkaline phosphatase (e.g. raised levels may indicate biliary obstruction due to hepatic metastases).

- Serum proteins (e.g. reduced levels indicate undernutrition)
- Prothrombin time is prolonged in liver diseases and needs to be corrected by administration of vitamin K, in the management of gastrointestinal bleeds.

Stool Examination

Examination of stool for occult blood is required in cases of occult bleeding from GIT.

Occult bleeding from GIT is common, but the bleed should be at least 10 ml to identify by examination.

Radiology

- Chest X-ray (e.g. aspiration pneumonitis, mediastinal widening and hilar lymphadenopathy of esophageal malignancy)
- Contrast studies of bowel
 - ❖ Barium swallow (e.g. esophageal malignancy)
 - ❖ Barium meal (e.g. gastric malignancy, periampullary carcinoma)
 - ❖ Barium enema (e.g. colonic malignancy)
- CT/MRI scan
 - ❖ Chest (e.g. esophageal malignancy, and paraesophageal pathology like lymph nodes, pulmonary secondaries)
 - ❖ Abdomen (e.g. cirrhosis of liver, malignant deposits of liver, lymph node metastases of GI malignancies, intestinal tuberculosis)
- Ultrasonography of abdomen (e.g. malignant deposits, dilated portal vein and biliary radicals)
- Magnetic resonance cholangiopancreatography (MRCP) (e.g. periampullary carcinoma).

Endoscopy

- Upper gastrointestinal endoscopy (e.g. gastroesophageal reflux disease, esophageal tears, gastric malignancies, periampullary carcinoma)
- Lower gastrointestinal endoscopy (e.g. polyps, tumors of rectum and colon)

- Endoscopy has the advantage of obtaining tissue for histopathology
- Endoscopy may be used as a therapeutic tool at the same sitting (e.g. sclerotherapy in bleeding esophageal varices and endoclipping of bleeding vessel)
- Endoscopy should be performed at the earliest opportunity and after adequate resuscitation.

Radioisotope Studies

Radioisotope scanning using the patient's own labeled red blood cells can be useful in small bowel bleeds, especially those from angiodysplasia.

Selective arteriography (superior mesenteric arteriography) may be useful in determining the small bowel bleeds, particularly those from angiodysplasia.

Capsule Endoscopy

Swallowing a small capsule with a video camera, and recording the images of the lumen of small bowel gives tremendous information of bleeding from small bowel. Histopathology cannot be obtained by capsule endoscopy.

Histopathology

Biopsy through endoscopy (upper and lower) is confirmative.

Treatment

Medical Management

- Large bore vascular access and correction of hypovolemia and hematocrit
- Correction of coagulopathy
- Emergency endoscopy
 - ❖ For nonvariceal bleeding
 - IV PPI (80 mg 6 hrly)
 - Nasogastric suction and gastric lavage
 - ❖ For variceal bleeding
 - Insertion of Sengstaken-Blakemore tube for compression of varices
 - Administration of vasopressin (upto 20 units SC or slow IV).
 - Variceal injection or banding
 - Transhepatic intravenous portosystemic shunt (TIPSS) for uncontrolled bleeding.

Surgery is needed for persistent uncontrollable GI bleeding.

Patient requiring more than six units of blood for resuscitation for acute bleed, will require surgical management.

Surgical Treatment

Surgery is indicated in recurrent esophageal variceal bleeding, complicated inflammatory bowel disease, polyps, benign and malignant tumors of GIT and hemorrhoids.

Anorectum

ACUTE ANAL FISSURE

Incidence and Etiology

- Anal fissures are of two types. They are:
 - i. *Acute fissures*: Tear in the anal skin due to forceful expulsion of hard fecal matter
 - ii. *Chronic fissures*: Nonhealing of acute fissure due to repeated trauma caused by hard fecal matter
- Anal fissures can occur secondary to Crohn's disease, ulcerative colitis, syphilis and tuberculosis (secondary fissures).

Pathogenesis

It is a tear in the anal skin, usually found in the 6 o'clock or 12 o'clock positions, following a bout of constipation and passage of a large hard stool.

- Posterior fissures are common than the anterior due to following reasons:
 - ❖ Anal canal is posteriorly angulated
 - ❖ Anal orifice is elliptical in shape
 - ❖ Posterior part of the anus is not supported by the muscles
 - ❖ Local ischemia.

Symptoms

- Acute fissure is a very painful condition associated with fresh bleeding (streak of blood on the hard fecal matter)
- Chronic fissure is moderately painful with blood stained fecal matter.

Signs

- Acute fissure presents with a linear tear in the anal skin (Fig. 22.1)
- Chronic fissure has a swollen skin at its lowest part called ‘sentinel pile’ (Figs 22.2A and B).



FIG. 22.1: Acute fissure in ano — 6 o'clock position



FIG. 22.2A: Chronic fissure in ano — 6 o'clock position



FIG. 22.2B: Chronic fissure in ano with sentinel pile and hemorrhoids

Relevant Investigation

No specific investigation is necessary, excepting a proctoscopy.

Treatment

- Acute fissure heals when constipation is taken care of, with probable anal dilatation
- Chronic fissure requires excision
- Secondary fissures require appropriate management.

ANORECTAL ABSCESS

Incidence and Etiology

- They are of four types (Fig. 22.3):
 - i. Pelvirectal abscess
 - ii. Submucous abscess
 - iii. Ischiorectal abscess
 - iv. Perianal abscess.

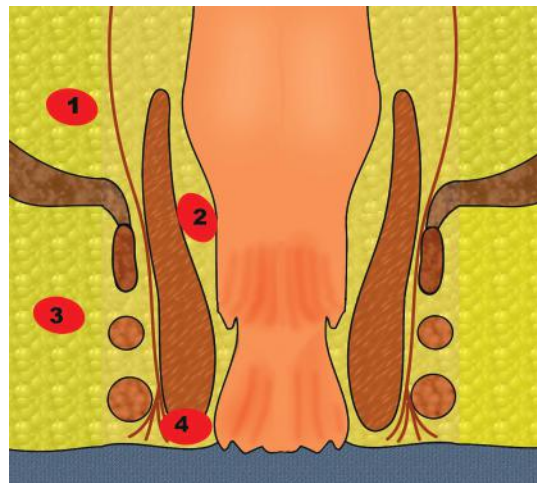


FIG. 22.3: Anorectal abscesses: (1) Pelvirectal abscess (2) Submucous abscess (3) Ischiorectal abscess (4) Perianal abscess

- Acute infections of the anal intersphincteric glands caused by aerobic and anaerobic organisms
- The infection originating in the intersphincteric space may spread in three directions:
 - i. Upwards
 - ii. Downwards
 - iii. Horizontally and circumferentially.
- When the infection spreads in the vertical direction, that is upwards and downwards, and opens at two places, forming an internal opening in the rectum and an external opening on the perianal skin, resulting in a fistula.

Symptoms

- A painful lump in the perianal region (Fig. 22.4), associated with fever
- Signs of acute inflammation in the perianal region.

Signs

- Tender mass with surrounding cellulitis
- Fluctuation is difficult to demonstrate.



FIG. 22.4: Recurrent perianal abscess — 5 o'clock position

Relevant Investigation

No special investigation is necessary.

Treatment

- Incision and drainage of painful abscess under general anesthesia, under cover of antibiotics
- Appropriate antibiotics are necessary based on culture examination of pus, after drainage.

HEMORRHOIDS

Incidence and Etiology

- Hemorrhoids (piles) are the varicosities of the hemorrhoidal plexus of veins
- Hemorrhoids are caused by:
 - ❖ Chronic constipation
 - ❖ Purgation
 - ❖ Malignancies
- Hemorrhoids may occur in the late middle age or elderly, secondary to rectal growths infiltrating or compressing the hemorrhoidal veins, called 'symptomatic piles' since it is a symptom of a condition more proximally
- External piles are covered by skin and the internal piles are covered by mucosa
- Hemorrhoids are of four degrees:
 - ❖ *First degree*: Only bleeding (splash in the pan) and no mass
 - ❖ *Second degree*: Masses prolapse on straining and reduce spontaneously (Fig. 22.5A)
 - ❖ *Third degree*: Masses prolapse on straining and need manual reduction
 - ❖ *Fourth degree*: Masses stay prolapsed at all times (Fig. 22.5B).

Complications: Profuse hemorrhage (Fig. 22.6A), prolapse (Fig. 22.6B), strangulation, infection (Fig. 22.6C), thrombosis (Fig. 22.6D), gangrene, ulceration (Fig. 22.6D) and fibrosis.

Symptoms

- Majority of patients present with painless rectal bleed
- Constipation is a common accompaniment
- Mucus discharges and pruritus ani are commonly present
- Pain is felt when the pile masses are thrombosed and strangulated.



FIG. 22.5A: Second degree hemorrhoids



FIG. 22.5B: Third degree hemorrhoids



FIG. 22.6A: Bleeding hemorrhoids

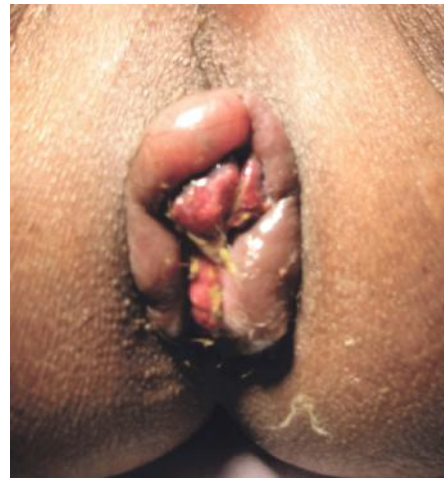


FIG. 22.6B: Prolapsed hemorrhoids

Signs

- Lumps (3,7 and 11 o'clock positions — primary piles) at the anal orifice
- Digital examination to rule out associated sphincter spasm and tumors is important
- Proctoscopy is diagnostic.



FIG. 22.6C: Inflamed hemorrhoids



FIG. 22.6D: Thrombosed and ulcerated pile masses (*Courtesy: Dr Chandrasekar Rao*)

- Uncomplicated hemorrhoids are not felt by the examining finger
- Complicated hemorrhoids warrant emergency surgical consultation
- Thrombosed pile masses are firm to feel and visible on inspection, and present with gross edema and ulcerations.

Relevant Investigation

No special investigation is required unless other pathologies like malignancies are suspected.

Treatment

- Hemorrhoidal bleeds are usually self-limiting
- Banding or infrared coagulation controls acute bleeds
- Venotonics control chronic recurrent rectal bleeds
- Laxatives to manage constipation
- Hemorrhoidectomy is curative.

PERIANAL HEMATOMA

Incidence and Etiology

- It is sometimes called as ‘thrombosed piles’, but it is not related to hemorrhoids
- The cause is not exactly known.

Pathogenesis

Occurs due to thrombosis of a subcutaneous vein below the transitional zone.

Symptoms*

- A discrete painful swelling (Fig. 22.7)
- Ulceration (Fig. 22.8) can occur with greater pain.

* Disturbing pain brings the patient to the doctor for an emergency consultation.

Sign

Tender mass external to the anal canal.



FIG. 22.7: Perianal hematoma



FIG. 22.8: Ulcerated perianal hematoma

Relevant Investigation

- No special investigation is necessary
- Proctoscopy to rule out associated hemorrhoids.

Treatment

- No treatment as most of them resolve
- Incision and curettage gives quick relief from painful swellings
- Since it opens up a vein, bleeding may be troublesome.

PROLAPSE OF RECTUM

Incidence and Etiology

- Generally caused by chronic constipation and straining at stool and when the pelvic floor is weak
- Occurs at extremes of age.

Pathogenesis

Pelvic floor weakness and the lack of mechanical support allows the rectum to prolapse.

Symptoms

- The rectum presents as a prolapsed mucosa, varying from partial to full thickness (Fig. 22.9), appearing more during the act of defecation
- It can reduce spontaneously or may need manual reduction
- There may be associated mucous discharge, bleeding, pain and incontinence
- Patient may have to strain to make it prolapse for clinical examination, in most cases.

Signs

- Prolapse up to 5 cm is considered to be partial and more than that is considered complete
- They can be differentiated by feeling the wall with the thumb and the index finger.

Differential Diagnosis

- Hemorrhoids
- Intussusception.



FIG. 22.9: Prolapse rectum (*Courtesy: Dr V Srinivasan*)

Relevant Investigation

Proctoscopy and colonoscopy may be needed to rule out the causes of constipation, and associated malignancy.

Treatment

Medical

- Manual reduction
- Dietary modification and laxatives.

Surgical

Various operative procedures are available for the treatment of prolapse.

Vascular System

ACUTE LIMB ISCHEMIA

Incidence and Etiology

- Causes of acute ischemia are:
 - ❖ Acute thrombosis
 - Occurs in a vessel with pre-existing atherosclerosis due to plaque rupture or due to hypercoagulable condition
 - Can occur *de novo* in a normal artery due to thrombophilia
 - ❖ Embolism - Emboli originate from:
 - Heart (e.g. myocardial infarction and rheumatic heart disease with atrial fibrillation – commonest, atrial myxoma – rare)
 - Proximal artery (e.g. arterial emboli arise from an ulcerated atheromatous plaque or from an aneurysm - Popliteal aneurysm and subclavian aneurysm following thoracic outlet compression). The common sites for emboli are the brachial bifurcation and common femoral artery bifurcation. Multiple shower of emboli in the foot produces the characteristic trash foot.
 - ❖ *Dissection*: Seen in patients with hypertension or connective tissue disorders like Marfan's syndrome. There is a tear in the intima which allows the blood to enter between the intima and the media (false lumen) causing the true lumen to occlude. It usually starts in the arch of aorta and extends all the way upto the iliac arteries
 - ❖ *Arterial trauma*: Can be homicidal or iatrogenic (e.g. surgical intervention, accidents). The arterial injury may be laceration, transection, contusion, thrombosis or spasm. There is usually associated bony and or soft tissue injury

- Rapid changes occur in the tissues distal to the occlusion and the limb survival depends on the presence of collaterals
- Skeletal muscle and nerve tissue are the most sensitive to hypoxia and suffer damage early.

Complications: Compartment syndrome, acute renal failure (acidosis), muscle contracture (Volkmann's ischemic contracture).

Symptoms

- Acute ischemia produces classical symptoms (6 Ps)
 - ❖ *Pain:* Usually excruciating, sudden in onset and continuous
 - ❖ *Pallor:* Affected limb appears pale due to lack of blood
 - ❖ *Pulselessness:* Pulses distal to the block are absent
 - ❖ *Paresthesia:* Due to nerve damage
 - ❖ *Paralysis:* Sensory motor deficit due to ischemic nerve damage secondary to severe ischemia (a late sign)
 - ❖ *Poikilothermia:* The affected limb appears cold.

Signs

- Affected muscle gets swollen, tense and is very tender, due to persistent ischemia
- On palpation, muscle gives a characteristic rubbery feel (can be mistaken for DVT)
- Later skin changes like mottling (Fig. 23.1) and blisters start and ultimately the limb becomes gangrenous (Fig. 23.2).



FIG. 23.1: Mottling of ischemic limb (Courtesy: Dr N Sekar)



FIG. 23.2: Gangrene of acute ischemia of left lower limb (Courtesy: Dr N Sekar)

Relevant Investigations

All investigations should be done without delay:

- Duplex scan is done to confirm diagnosis in early cases
- Angiogram for treatment planning
- Tests for thrombophilia
- Hyperkalemia when renal shut down ensues.

Treatment

- Acute arterial occlusion is a surgical emergency. Usually irreversible damage occurs by 4 to 6 hours. Hence, revascularization should be done within 6 to 8 hours for limb salvage (Golden hour).
 - ❖ Intravenous heparin should be administered as soon as the diagnosis of acute ischemia of the limb is made. Best results are seen in those who undergo revascularization within 6 to 8 hours after the onset of ischemia
 - ❖ Embolism is best managed by embolectomy done through the brachial artery at the elbow or the femoral artery at the groin
 - ❖ Acute arterial thrombosis can be managed by surgical bypass or by catheter directed thrombolytic therapy. Frequently both modalities may be required to achieve revascularization
 - ❖ Fasciotomy will be required to avoid muscle damage in compartment syndrome caused by delayed revascularization
 - ❖ Early amputation for limbs with irreversible ischemia.

Note: Those who present late should be taken up for surgery without imaging and intraoperative angiogram should be done to reduce time delay.

ACUTE INTESTINAL ISCHEMIA

Incidence and Etiology

Intestinal ischemia occurs usually due to occlusion of mesenteric artery caused by:

- Thrombosis on an atheromatous plaque (e.g. origin of superior mesenteric artery)
- Embolus following atrial fibrillation/myocardial infarction/detached atheromatous plaque.

Clinical Presentation

- Severe acute abdominal pain with copious vomiting
- Very fast deterioration of health

- Hematemesis and melena also occur in some cases
- The clinical signs are disproportionate to the symptoms, and rarely an area of tenderness may be felt near the infarcted bowel.

Relevant Investigations

- ECG, plain X-ray and US abdomen are useful
- Selective angiography is informative.
 - Systemic inflammatory response, oliguria, persistent acidosis, raised serum amylase and bloody diarrhea may all occur but none is specific.

Treatment

- Conservative management to stabilize the patient
- Laparotomy and resection of nonviable bowel will be necessary, if the patient is stable
- Balloon angioplasty or bypass grafts may be feasible in select cases.

Note: Outcome is guarded in most cases.

- Intestinal ischemia should be considered as a possible cause of deterioration, particularly in the elderly with pre-existing vascular disease or vasculitis.

LEAKING OR DISSECTING AORTIC ANEURYSM

Incidence and Etiology

- Aneurysm of aorta (> 4 cm diameter – normal 1.5 to 2.5 cm) is the disease of elderly, and rupture is the deadly complication, with a mortality of more than 80 percent
- The rupture can occur into the retroperitoneum or into the peritoneal cavity.

Clinical Presentation

- Acute severe upper abdominal pain or in the lower chest
- Radiation to the back mimics acute pancreatitis
- Aneurysm may be felt per abdomen with guarding and rigidity
- Lower limb pulses are feeble or absent
- Dissecting aneurysms present with retrosternal pain, as the pathology starts at the aortic arch.

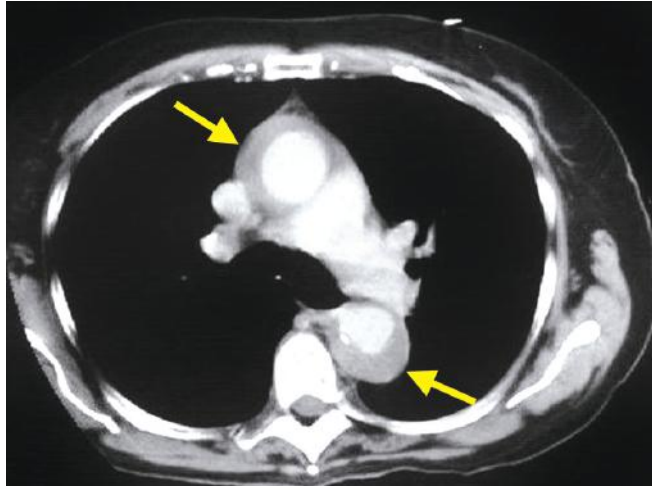


FIG. 23.3: CT chest—Dissecting aneurysm of thoracic aorta

Relevant Investigation

US and CT of the abdomen are conclusive (Fig. 23.3).

Treatment

Emergency surgical intervention is necessary, along with correction of shock.

- Only a third with rupture of aneurysm live to reach the hospital.

Urology

ACUTE RETENTION OF URINE

Definition

Defined as inability to micturate even when the bladder is full.

Incidence and Etiology

- This occurs when the sphincter is unable to relax or with proximal urethral obstruction like in prostatic enlargement
- Acute retention is of short duration and the bladder distension is painful
The causes of acute retention of urine are given in Table 24.1.

Symptoms

- Severe lower abdominal pain
- Inability to void urine
- Symptoms relating to underlying cause (e.g. LUTS).

Signs

- Palpable distended tender urinary bladder
- Rectal examination: Anal sphincter spasm, enlarged prostate.

Table 24.1: Causes of acute retention of urine

<i>Males</i>	<i>Females</i>	<i>Both sexes</i>
Prostatic enlargement	Multiple sclerosis	Clot retention
Urethral stricture	Urethral stenosis	Calculus at bladder neck or urethra
Tight Phimosis / Meatal stenosis	Retroverted gravid uterus	Rupture of urethra
Acute urethritis	Cervical fibroid	Spinal anesthesia
Postoperative	Bladder neck hypertrophy	Neurogenic
		Fecal impaction
		Anal pain (post hemorrhoidectomy)
		Drugs (antihistamines, antihypertensives, anticholinergics, antidepressants)

Relevant Investigations

- Hemogram
- Urea/creatinine
- PSA levels may be high
- US may confirm the cause of obstruction.

Treatment

- Immediate urethral catheterization
- Urine examination for sugar and infective organisms
- Treatment of the cause.

HEMATURIA

Definition

Passing of blood in the urine is called hematuria.

- Frank hematuria: Presence of frank blood or blood clots in the urine
- Microscopic hematuria: Presence of blood discovered by microscopy or dipstick.

Incidence and Etiology

- Whatever the cause, hematuria is usually episodic:
 - ❖ Hematuria (painless) (e.g. tumors of urological system)
 - ❖ Hematuria with dysuria (e.g. urological infections)
 - ❖ The causes of hematuria are given in Table 24.2.

Table 24.2: Causes of hematuria

<i>Etiology</i>	<i>Diseases</i>				
	<i>Kidneys</i>	<i>Ureters</i>	<i>Bladder</i>	<i>Prostate</i>	<i>Urethra</i>
Congenital	Polycystic kidney AV malformation				
Traumatic	Trauma		Trauma		Trauma
Inflammatory	Glomerulonephritis Tuberculosis		Cystitis Schistosomiasis	Prostatitis	Acute urethritis
Neoplastic	Malignancy	Urothelial tumors	Malignancy	Malignancy	Malignancy
Others	Calculi	Calculi	Calculi	Benign enlargement	Calculi
Nonurological	Coagulation and hematological disorders				

Note: Microscopic hematuria may represent a significant lesion in the urinary tract and should be taken seriously, though in about 5 percent of the cases, no cause can be found.

Symptoms

- Passing dark colored/blood stained urine
- Pain may or may not be present.

Signs

- Clinical signs may vary (palpable kidney in large tumors, palpable bladder in bladder obstruction)
- Anemia (in chronic hematuria or large hematuria)
- Enlarged prostate.

Relevant Investigations

- Hematocrit may be low
- Renal function may be impaired
- Coagulation profile may be altered
- Urine examination for infection (routine and C/S)
- Ultrasonography
- Intravenous urography (IVU)
- Cystoscopy
- CT/MRI may be needed while evaluating malignancies.

Treatment

- Primary treatment requires immediate urethral catheterization
- When catheterization is not possible, suprapubic cystostomy is needed
- Definitive treatment depends on the underlying cause.

RENAL COLIC

Caused by distension of renal capsule and pelvis or stretch of the peritoneum by large renal swellings:

- Nature of pain
 - ❖ Pain localized to renal angle (posterior space between the 12th rib and the sacrospinalis muscle) (Fig. 24.1) (e.g. acute pyelonephritis, renal tuberculosis)—described by putting the hand on the waist with his fingers on the renal angle and thumb pointing the umbilicus (Fig. 24.2)
 - ❖ Pain radiating to umbilicus (e.g. renal tuberculosis)
- Intensity of pain
 - ❖ Severe (e.g. acute pyelonephritis renal stones)
 - ❖ Dull and persistent (e.g. polycystic disease and malignancy)
 - ❖ Acute obstruction of the upper ureter can give a severe colicky pain in the loin called “Renal colic”.

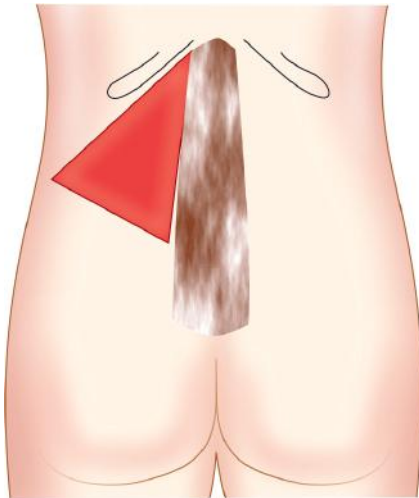


FIG. 24.1: Renal angle



FIG. 24.2: Description of renal pain

- Hematocrit may be low
- Renal function may be impaired
- Urine examination for infection (routine and C/S)
- Ultrasonography will show stones or proximal dilatation
- IVU will show stones, or filling defects (stones, tumors) or proximal dilatations
- Cystoscopy is routine
- CT/MRI may be needed while evaluating malignancies.

◆ Treatment

- Emergency treatment requires antispasmodics to relieve pain, antiemetics for vomiting
- Definitive treatment depends on the underlying cause.

URETERIC COLIC

Caused by obstruction of the ureter commonly by a stone or a clot, and may correspond to the level of obstruction:

- Nature of pain
 - ❖ When the stone is lodged in the upper ureter, the pain radiates to the testicle (T11-12)
 - ❖ When the stone is lodged in the mid ureter, the pain may be in the McBurney's point on the right side and simulate diverticulitis on the left side (T12-L1)
 - ❖ When the stone is lodged in the distal ureter, the pain resembles vesicular pain or may radiate to the genitalia or inner side of thigh (L1-L2)
- Intensity of pain
 - ❖ Dull pain in the side of abdomen (e.g. chronic obstruction of ureter with calculi)
 - ❖ Severe colicky pain—(loin pain radiating to the groin, genitalia or inner thigh—genitofemoral nerve) in acute obstructions of ureter called “Ureteric colic” (Fig. 24.3).

Symptom

Severe pain as described above.

Relevant Investigations

- Hematocrit may be low
- Renal function may be impaired

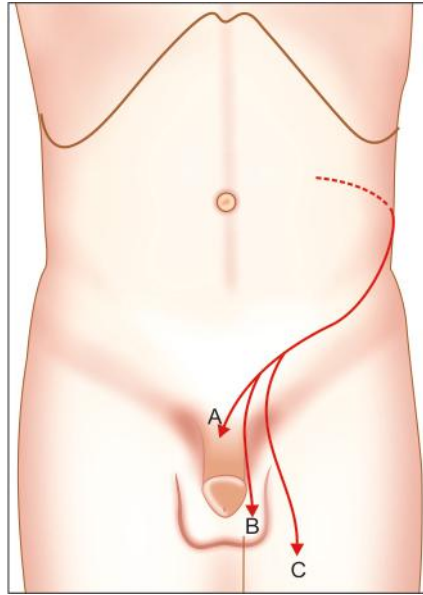


FIG. 24.3: Ureteric colic: (A) Base of penis; (B) Genitalia; (C) Inner aspect of thigh

- Coagulation profile may be altered
- Urine examination for infection (routine and C/S)
- Ultrasonography may show stones or proximal dilations
- IVU will show filling defects (stone, tumors) and proximal dilations
- Cystoscopy may show stones impacted at the ureterovesical junction.
- CT/MRI may be needed while evaluating malignancies.

Treatment

Treatment depends on the underlying cause. Stones need to be removed by crushing or as such, by basketing or ureterolithotomy.

ACUTE URETHRITIS

Incidence and Etiology

- Acute inflammation of paraurethral glands caused usually by gonococcal infections, and rarely by Chlamydia.
- Urethritis can lead to periurethral abscesses, and healing urethritis can lead to stricture, due to scarring.

Symptoms

- White urethral discharge (Fig. 24.4) with severe pain
- History of extra or premarital contact is available.

Sign

Tender swelling in the periurethral area (e.g. abscess).

Relevant Investigations

- Isolation of organism in the discharge or urine
- Urethroscopy (Fig. 24.5) is diagnostic.

Treatment

Appropriate antibiotics.



FIG. 24.4: Urethral discharge of gonococcal urethritis

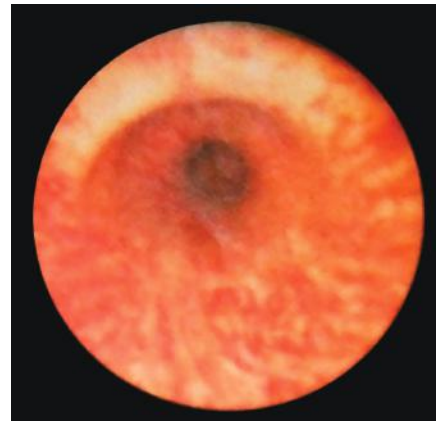


FIG. 24.5: Urethroscopy—Acute urethritis

ACUTE PROSTATITIS

Incidence and Etiology

- Usually caused by infection of urinary tract by *E. coli*, *Klebsiella* or *Pseudomonas*
- Dental and upper respiratory infections are known causes of acute prostatitis
- Epididymo-orchitis may follow due to retrograde spread of infection
- Untreated prostatitis may form prostatic abscess, which may rupture into rectum to form a fistula
- Recurrent acute prostatitis is not uncommon

Symptom

Malaise, fever sometimes with rigor and pain in perineum, sometimes with urinary retention.

Sign

Rectal examination will show tender and swollen prostate.

Relevant Investigation

Leukocytosis and pyuria may be present.

Treatment

- Empirical antibiotics can be curative
- Acute retention of urine due to abscess needs suprapubic cystostomy with antibiotics
- Abscess requires drainage per urethra to avoid iatrogenic fistula formation.

ACUTE PROSTATIC ABSCESS

Incidence and Etiology

- Prostatic abscess results due to untreated prostatitis
- Abscess may rupture into the rectum to form a fistula.

Symptom

Malaise, fever sometimes with rigor and pain in perineum, sometimes with urinary retention.

Rectal examination will show tender and swollen prostate.

Relevant Investigations

- Leukocytosis and pyuria may be present
- US may be conclusive.

Treatment

- Empirical antibiotics
- Acute retention of urine due to abscess needs suprapubic cystostomy with antibiotics
- Abscess requires drainage per urethra to avoid iatrogenic fistula formation.

Male Genitalia

ACUTE SCROTAL PAIN

Definition

Acute severe pain in the scrotum.

Causes of Acute Scrotal Pain

- Torsion of testis
- Torsion of hydatid of Morgagni and other appendages
- Epididymo-orchitis
- Trauma
- Inguinoscrotal hernia
- Testicular tumor
- Scrotal abscess
- Fournier's gangrene

Pain may also be referred to the scrotum from pathologies away from the scrotum:

- Ureteric colic
- Leaking aortic aneurysm
- Hip pathology
- Intra-abdominal sepsis or free fluid (in neonates).

Nature of Pain

- Sudden and severe (e.g. testicular torsion)
- Sudden and gradual increase in intensity (e.g. obstructed inguinoscrotal hernia)
- Insidious (e.g. torsion of appendages of testis/testis, epididymo-orchitis)
- Constant and throbbing (e.g. epididymo-orchitis).

Location of Pain

- Testis and cord (e.g. epididymo-orchitis, testicular torsion)
- Scrotum (e.g. scrotal abscess, Fournier's gangrene).

Association of Vomiting

Vomiting is usually present in testicular torsion, and ureteric colic.

Association of Fever

Fever indicates infective pathology (e.g. acute epididymo-orchitis, scrotal abscess, Fournier's gangrene).

Radiation

Radiation from loin to genitalia is characteristic of ureteric colic, whereas pain of epididymo-orchitis may spread along the cord structures in the inguinal canal.

Past History

- History of pain (e.g. epididymo-orchitis, recurrent torsion, inguinoscrotal hernia)
- Previous surgery (e.g. surgery for torsion on one side, recurrent inguinoscrotal hernia).

Signs

- Swelling of scrotum with:
 - ❖ Erythema and edema (e.g. torsion of testis, acute epididymo-orchitis, scrotal abscess, strangulated inguinoscrotal hernia)
 - ❖ Bluish tinge of skin (e.g. ischemia of testicular torsion)
 - ❖ Dark spot on the upper half of hemiscrotum (e.g. blue dot sign of torsion of testicular appendages).

- Testis
 - ❖ Horizontal lie (e.g. bell-clapper testis of torsion)
 - ❖ Normal lie (e.g. epididymo-orchitis).

On Palpation

Scrotum

- Swelling separate from testis (e.g. obstructed inguinoscrotal hernia)
- Tender skin (e.g. scrotal abscess, torsion of testis and its appendages).

Testis

- Generalized swelling (e.g. epididymo-orchitis, torsion of testis, testicular tumor)
- Tenderness (e.g. epididymo-orchitis, torsion of testis).

Differential Diagnosis by Clinical History and Examination

- Acute scrotal pain, fever
 - ❖ Mild skin changes and
 - Normal lying nontender testis (e.g. scrotal cellulitis, abscess)
 - Normal lying and tender testis (e.g. acute epididymo-orchitis)
 - ❖ Gross skin changes and
 - Normal lying and nontender testis (e.g. Fournier's gangrene)
- Acute scrotal pain, nausea and vomiting
 - ❖ Erythema and edema of tender skin, bell-clapper lie, enlarged and tender testis (e.g. acute torsion of testis)
 - ❖ Swelling separate from normal testis (e.g. obstructed or strangulated inguinoscrotal hernia)
- Colicky pain (loin to genitalia) with normal testis (e.g. ureteric colic)
- Acute scrotal pain after trauma with tender +/- enlarged testis (e.g. traumatic orchitis).

Relevant Investigations

Hematology

- Leukocytosis in infective pathologies (e.g. acute epididymo-orchitis, Fournier's gangrene)
- Raised ESR in all infective pathologies (e.g. acute epididymo-orchitis, Fournier's gangrene).

- Doppler ultrasonography – to assess blood flow to the testis in testicular torsion.

Treatment

- Infective pathologies – antibiotics
- Scrotal abscess – incision and drainage
- Torsion of testis
 - ❖ *Viable testis*: Surgery (Detorsion and fixation of testis when it is viable, with orchidopexy for the contralateral testis)
 - ❖ *Nonviable testis*: Orchidectomy
- Torsion of testicular appendages – medical or surgical (excision)
- Obstructed/strangulated hernia – surgery
- Fournier’s gangrene – surgery (desloughing) with broad-spectrum antibiotics
- Testicular tumor – high orchidectomy with chemoradiation as per stage.

TORSION OF TESTIS

Incidence and Etiology

Common in children between 10 to 15 years, but can also occur in adults.

Pathogenesis

- The testis lies in the scrotum suspended by the cord structures, and the testicular artery is a part of it. When the testis is twisted on the axis of the cord, the blood supply can get impaired, which may lead to gangrene. This twist is usually away from midline corresponding to contraction of cremaster muscle (Torsion)
- Can be precipitated by horizontal lie of the testis or by the presence of a long mesentery between epididymis and body of the testis.

Symptoms

- Sudden onset of severe scrotal pain, usually following an act of physical strain
- Pain in the lower abdomen and suprapubic area, may be present
- There may be mild fever.

Signs

- Swollen cord structures, and in the early stages, a knot can be felt in the cord structure. There may be minimal hydrocele
- If not detected early, there will be swelling of testis and it cannot be separately felt from epididymis.

Relevant Investigation

Doppler studies will show decreased blood flow to the affected side (in epididymo-orchitis, blood flow is increased).

Treatment

It is a surgical emergency and no time should be wasted in treating this condition. Delayed treatment will lead to loss of function of testis.

- Surgery involves untwisting of the torsion to restore blood flow and fixing it
- The opposite testis also needs fixation, to prevent similar problem occurring later
- Gangrenous testis needs orchidectomy (Fig. 25.1).

Note: Even if the diagnosis is in doubt surgical exploration should be done.



FIG. 25.1: Torsion of testis (*Courtesy:* Dr MG Rajamanickam)

TORSION OF APPENDAGES OF TESTIS

Incidence and Etiology

- Defined as torsion of hydatid of Morgagni, which is attached to the anterior aspect of upper pole of testis as a pedunculated structure
- Accounts for 90 percent of cases.

Pathogenesis

- Thought to be due to increased gonadotrophins during puberty which increase the size of hydatid, hence more common in preadolescent age group
- Since it is a pedunculated structure, rotation of this pedicle may compromise the blood supply and lead to infarction.

Symptom

Pain – insidious onset.

Signs

- Distressed patient
- Tender scrotum
- Dark spot may be visible over the testis (Blue dot sign)
- Normal lie of testis in the scrotum
- Hydrocele may be present.

Relevant Investigation

No specific investigation is required.

Treatment

Surgical exploration is required to diagnose, if confirmed the hydatid needs to be excised.

ACUTE EPIDIDYMO-ORCHITIS

Incidence and Etiology

Acute inflammation of testis and epididymis can be caused by:

- Viral infections (e.g. mumps)
- Bacterial infections (e.g. *Neisseria gonorrhoeae*, *E. coli* and *Klebsiella*, and *Chlamydia*)

Pathogenesis

- Retrograde infection from prostate and seminal vesicle
- Blood-borne infection from other focus
- Surgery of urinary tract (TUR)

Symptom

Pain and swelling of testis.

Signs

- Edema of scrotal skin, cord structures very bulky and thickened
- Epididymis is very much enlarged
- Testis is also enlarged and tender.

However, it is possible to make out the testis separate from epididymis whereas this is not possible in torsion testis.

Relevant Investigations

- Blood count – leukocytosis
- Blood culture – positive culture
- Urine culture is routinely requested
- US may be useful in assessing the increased blood flow (reduced in torsion).

Treatment

- Vigorous treatment with antibiotics is necessary
- If the epididymo-orchitis is not settling with one week of antibiotics then tuberculosis or even tumor must be excluded.

TRAUMATIC ORCHITIS

Incidence and Etiology

- It is a common condition, an aseptic inflammation caused by direct trauma, seen in sportsmen involved in contact sports like football
- Any direct trauma to the testis causes this condition.

Clinical Presentation

- Acute pain in the injured testis
- Supporting or lifting the testis may give comfort (pain gets aggravated when it is lifted if it is due to torsion)
- Mild hydrocele may be present.

Relevant Investigation

No special investigation is necessary.

Treatment

Scrotal support and analgesics would suffice.

HEMATOCELE

Definition

Collection of blood in tunica vaginalis sac.

Incidence and Etiology

Occurs as a complication following:

- Any direct blunt trauma to scrotum
- After needle aspiration of pre-existing hydrocele.

Symptoms

- May be asymptomatic and can mimic a testicular tumor
- Scrotal swelling becomes heavy and causes discomfort (Fig. 25.2).



FIG. 25.2: Right hematocele

Signs

- Hard swelling
- The testis may become atrophic following long-standing pressure effect by the hematoma.

Relevant Investigation

Ultrasonography (US) is useful in diagnosing hematocele and determining the size of testis.

Treatment

- Drainage of hematoma and excision of hydrocele sac
- Analgesics and anti-inflammatory drugs.

PYOCOLE

Definition

Collection of pus in tunica vaginalis sac.

Incidence and Etiology

Can result following infection or after aspiration of hydrocele.

Symptom

Severe pain and high-grade fever.

Signs

- Tenderness on pressing the swollen scrotum
- Transillumination is characteristically absent.

Relevant Investigations

- Leukocytosis may be pronounced
- Diagnostic aspiration may reveal the presence of pus.

Treatment

- Needle drainage of pyocele with antibiotic may be sufficient in early stages
- Open drainage and or orchidectomy may be required in delayed cases.

IDIOPATHIC SCROTAL EDEMA

Incidence and Etiology

More common in young and preadolescent children.

Pathogenesis

- Uncertain
- May be a hypersensitivity reaction resulting in angio edema of skin
- May be caused by beta-hemolytic streptococci.

Symptom

Gradual or sudden onset of scrotal swelling (usually unilateral).

Signs

- Swollen and mildly tender scrotum
- Erythema may extend to involve the perineum and inguinal regions
- The lie of the testis is normal.

Relevant Investigations

Ultrasonography (US) may be useful in identifying other scrotal pathologies.

Treatment

- Scrotal support
- Anti-inflammatory drugs
- Antihistamines.

ACUTE SCROTAL ABSCESS

Incidence and Etiology

- Usually, a superadded infection of fungal infections
- The incriminating organisms being staphylococci
- Minor trauma like bruises and scratches are known to initiate this pathology
- More common in diabetics.

Symptoms

- Sudden pain in the scrotum
- Pyrexia.

Sign

Tender swelling of scrotum (Fig. 25.3).

Relevant Investigations

- Screening for diabetes mellitus is useful
- Diagnostic aspiration of pus and isolation of organism is necessary.



FIG. 25.3: Scrotal abscess

Treatment

- Broad-spectrum antibiotics
- Incision and drainage of abscess.

FOURNIER'S GANGRENE

Incidence and Etiology

- It is called a vascular disaster of infective origin
- The incriminating organisms being hemolytic streptococci, staphylococci, *E. coli* and *Cl. Welchii*.
- The three cardinal characteristics of Fournier's gangrene are:
 - i. Sudden appearance of scrotal inflammation
 - ii. Rapid onset of gangrene
 - iii. Predisposing factors
 - ❖ Diabetes
 - ❖ Local trauma
 - ❖ Paraphimosis
 - ❖ Periurethral extravasation of urine

- ❖ Perianal infection
- ❖ Surgery (circumcision/herniorrhaphy)
- Minor trauma like bruises and scratches are known to initiate this pathology.

Symptoms

- Sudden pain in the scrotum
- Prostration
- Pallor
- Pyrexia.

Signs

- Swollen scrotum with necrotic skin
- When cellulitis spreads and scrotal coverings slough, the testes are exposed (Fig. 25.4).

Relevant Investigation

Culture of sloughed tissues or the overlying secretions is diagnostic.

Treatment

- Broad-spectrum antibiotics and wide excision of slough, to stop the spread of gangrene
- Raw area needs split skin grafting.



FIG. 25.4: Fournier's gangrene
(Courtesy: Dr A Chandrasekar Rao)

ACUTE FILARIAL SCROTUM

Incidence and Etiology

In the tropics, the main cause of acute swelling of the scrotum is filariasis.

Clinical Presentation

- The scrotum may be enormously enlarged (Fig. 25.5)
- The skin is red and erythematous, and tender.

◆ Lymphorrhea may be present:

- Penis may also be swollen
- If untreated, may enlarge to massive proportions, a condition called ‘elephantiasis’
- The skin becomes hyperkeratotic, nodular and fissured giving a pachydermous appearance
- There may be associated lower limb edema.

Relevant Investigation

Peripheral smear for microfilaria.

Treatment

- **Medical:** Antifilarial treatment
- **Surgical:** Various plastic surgical procedures are available for chronic swelling of genitalia.



FIG. 25.5: Acute filarial scrotum

FRACTURE PENIS

Incidence and Etiology

- Occurs due to direct injury to tunica albuginea or corpora cavernosa, when the penis is in the erect state
- The tear in the tunica albuginea leads to accumulation of blood outside the corpora cavernosa
- Fracture can occur during:
 - ❖ A fall
 - ❖ Assault by the sexual partner
 - ❖ Forceful manipulation by the patient himself
- Common in the young and middle age.

In about 10 percent of cases, urethral injury may be associated.

Symptoms and Signs

- Erect penis becomes flaccid suddenly, followed by swelling of the penis and scrotum with bluish discoloration (Fig. 25.6)
- The patient is able to hear a click at the time of injury and hence the name ‘fracture penis’
- There may be blood at the urethral meatus with difficulty in passing urine due to associated urethral injury.



FIG. 25.6: Fracture penis (*Courtesy: Dr MG Rajamanickam*)

Relevant Investigations

No specific investigation is necessary, however when in doubt, cavernosogram can be done. This is done by injecting radiopaque contrast into the corpora cavernosa, to visualize the corporal anatomy.

Treatment

- Immediate surgical repair to torn tunica albuginea is required to maintain erectile function
- Urethral injury if present can be repaired at the same time.

PARAPHIMOSIS

Incidence and Etiology

- Occurs due to forceful retraction of the foreskin of pre-existing phimosis, either by manipulation or during sexual intercourse
- The fibrous band resulting from repeated infections, encircles the penis in the subcoronal area to cause congestion of the glans penis
- The patients unaware of phimosis, may develop this condition after the first sexual intercourse, usually after marriage.

Symptoms

- Mild-to-moderate pain in the glans penis
- Swollen prepuce.

Sign

Retracted foreskin just above coronal sulcus is swollen and edematous (Fig. 25.7).

Relevant Investigation

No specific investigation is required.

Treatment

- Immediate reduction of paraphimosis
- Constricting band needs to be incised, when reduction is not possible
- Circumcision should be done as an elective procedure after a couple of weeks, to prevent a recurrence.



FIG. 25.7: Paraphimosis

PRIAPISM

Incidence and Etiology

- In this condition, there is persistent painful erection without any sexual desire
- The causes include:
 - ❖ Hematological disorders
 - Leukemia
 - Sickle cell disease
 - Thalassemia
 - ❖ Neurological disorders
 - Spinal cord lesions and trauma
 - ❖ Secondary malignant deposits
 - ❖ Drugs
 - Intracavernosal injection treatment with drugs (papaverine, alprostadil) for erectile dysfunction (impotence)
 - Antihypertensives
 - Antipsychotics
 - Antidepressants



FIG. 25.8: Priapism

- Anticoagulants
- Recreational drugs (e.g. alcohol, cocaine)

Complications: Ischemia, thrombosis, gangrene, and impotence.

Clinical Presentation

Persistent erection of penis for more than six hours accompanied by pain (Fig. 25.8).

If detumescence does not result in about 6 hours, permanent erectile failure may result due to arterial and venous thrombosis and fibrosis of corpora.

Relevant Investigation

Investigations towards the probable etiology.

Treatment

- It should be treated as an emergency
- The blood from the corpora cavernosa should be drained using a 18 F butterfly needle
- If this fails, corporospongial shunt (anastomosing corpora cavernosa to corpus spongiosum) or cavernosa-saphenous venous shunt may be required
- The underlying pathology needs to be treated.

Hernias

COMPLICATED HERNIAS

Definition

Hernia is a protrusion of an internal part of an organ through an aperture with the enclosing membrane.

Whatever be the cause, the hernia is named conveniently based on its anatomical location (Fig. 26.1).

- Groin hernia
 - ❖ Inguinal
 - ❖ Femoral
- Ventral hernia
 - ❖ Epigastric hernia
 - ❖ Umbilical hernia
 - ❖ Paraumbilical hernia
 - ❖ Spigelian hernia
 - ❖ Incisional hernia (postoperative)
 - ❖ Divarication of recti
- Others
 - ❖ Obturator hernia
 - ❖ Sciatic hernia
 - ❖ Lumbar hernia
 - ❖ Perineal hernia.

To understand the pathology of each hernia, it is necessary to understand the anatomy of that region.

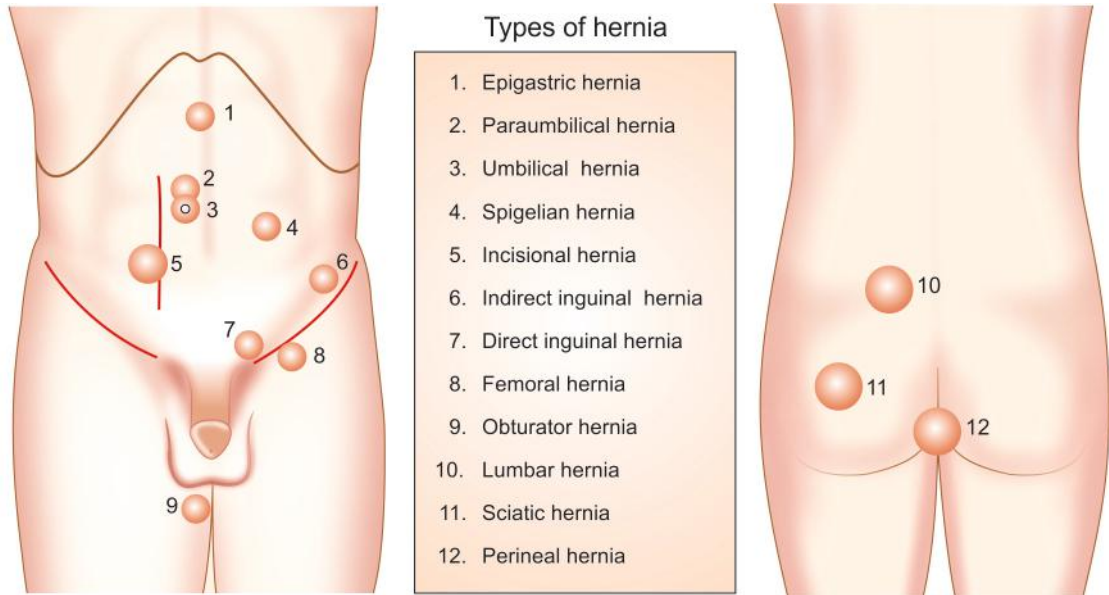


FIG. 26.1: Various types of hernia

Anatomy of the Hernial Sac

- The hernia sac consists of mouth, neck, body and fundus (Fig. 26.2).
- The coverings of a hernia will vary according to its site, but commonly, will be skin, subcutaneous fat, aponeurosis, muscle, endocavity fascia and endothelial lining (pleura or peritoneum)
- The contents will vary depending on its location (e.g. cecum, appendix, small bowel, omentum and right ovary in the right groin hernia, and sigmoid colon, left ovary in the left groin hernia). The contents of the hernia are returnable into the cavity when it is uncomplicated, a property called 'Reducibility'.

Complications

- *Irreducibility*: When the contents cannot be returned into the cavity, it is called irreducible. This may be due to the narrow neck of the sac (e.g. femoral, umbilical), or adhesions between the contents of the sac (e.g. longstanding herniae)
- *Obstruction*: When the neck of the sac is totally obstructed due to the contents of the hernia sac (e.g. distended bowel which forms a closed loop), without vascular impedece, it is said to be 'obstructed'

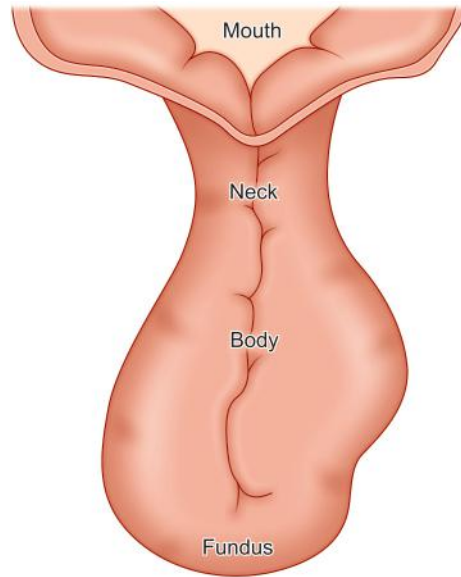


FIG. 26.2: Anatomy of hernia

- *Strangulation:* The venous and lymphatic occlusion distends the bowel further secreting more fluid in the bowel, causing further increase in the swelling. The tissues undergo ischemic necrosis called 'strangulation'. The tissues may slough and become permeable to bacteria and bacteremia results. It may even perforate to cause septicemia and septic shock.
- *Inflammation:* The sac may get inflamed if the contents of the sac like the appendix or Meckel's diverticulum is inflamed.

Symptoms

- Uncomplicated hernia are asymptomatic
- Pain is a symptom indicating a complication (e.g. inflammation, obstruction and strangulation)
- Fever may be a symptom of inflammation
- Vomiting, abdominal distension and constipation are signs of obstruction and strangulation in abdominal hernia.

Signs

- Irreducibility is a sign of complication
- Tenderness indicates complication (e.g. obstruction, strangulation)
- Signs of intestinal obstruction may indicate complications (e.g. obstruction, strangulation).

Relevant Investigations

No specific investigation is required, excepting an X-ray abdomen in intestinal obstruction.

Treatment

- Emergency surgery is mandatory (reduction of hernia, relieving of obstruction, and repair)
- If nonviable bowel is found in the hernia sac, resection is necessary.

Gynecology

ACUTE TORSION OF OVARIAN CYST

Incidence and Etiology

It can occur at any age, may follow major physical exertion, which can initiate the torsion.

Pathogenesis

Due to the large size and vulnerability to rotate over a narrow pedicle, torsion occurs.

Symptom

Severe pain in the lower abdomen, with signs of shock.

Signs

- Guarding and rigidity may be present.
- A tender lump may be palpable, whose lower border cannot be made out.

Relevant Investigations

Ultrasonography (US) and Computed tomography (CT) or Magnetic resonance imaging (MRI) of abdomen (Fig. 27.1) are diagnostic.

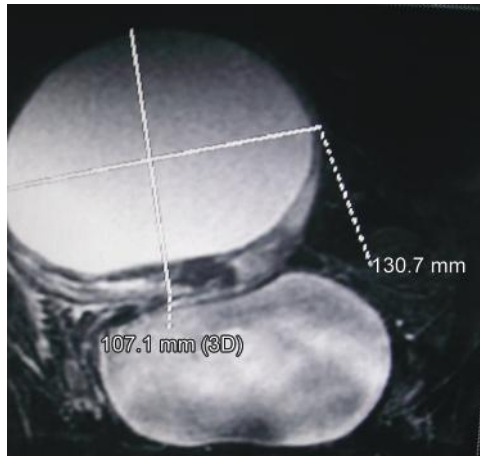


FIG. 27.1: MRI—Torsion of ovarian cyst



FIG. 27.2: Torsion of ovarian cyst

Treatment

Emergency laparotomy (Fig. 27.2) is needed. The cyst should be removed.

ACUTE SALPINGITIS

Incidence and Etiology

- A disease of the sexually active female, sometimes after sexually transmitted diseases, can confuse with acute appendicitis
- Usually associated with menstrual period, abortion or puerperium
- Common infective agents are *N. gonorrhoeae*, coliforms and chlamydiae.

Pathogenesis

Infective organism gains entry through the vagina to infect the salpinx.

Symptoms

Suprapubic pain, with or without white discharge per vagina (leukorrhea).

Signs

- Localized hypogastric tenderness
- Vaginal examination may reveal tender cervix and a bulky uterus.

Differential Diagnosis

Acute appendicitis is to be thought of in right sided salpingitis.

Relevant Investigations

Ultrasonography (US) is useful in diagnosing mass of the right ovary and the bulky uterus.

Treatment

Treatment with antibiotics.

RUPTURE OF LUTEIN CYST

Incidence and Etiology

A common condition in young females.

Clinical Presentation

- Acute abdominal pain associated with vomiting (right sided pathology may be confused with acute appendicitis)
- On examination:
 - ❖ A firm mass may be felt adjacent to the midline or in one of the iliac fossae
 - ❖ Guarding and rigidity may be present
- Vaginal examination may reveal a tender mass.

Relevant Investigations

Ultrasonography (US) of the abdomen is diagnostic.

Treatment

Emergency surgical intervention is mandatory.

Pediatrics

ACUTE INTUSSUSCEPTION

Incidence and Etiology

- Two per 1000 infants are affected with male preponderance, commonly affecting the age group of 2 months to 2 years
- Commonly, it is secondary to an enlarged Peyer's patch due to viral or bacterial infections (Fig. 21.39)
- The other less common causes are:
 - ❖ Meckel's diverticulum
 - ❖ Duplication cyst in the bowel wall
 - ❖ Polyp.

Pathogenesis

- Intussusception is the invagination of a segment of bowel into the distal adjacent loop (proximal into the distal)
- When the mesentery is drawn between the loops, it may result in vascular compromise, which may lead to strangulation, gangrene and perforation.

Symptoms

- In children, there may be a history of preceding gastroenteritis following a change in diet (weaning from milk to solid food)

- Severe acute colicky pain, with abdominal distension
- Passing of frequent semisolid stools with bright red blood may be predominant (red currant jelly).

Signs

- During the attacks of pain, a sausage shaped mass may be felt, which appears during the time of colic and disappears after the colic disappears. The right iliac fossa is empty—Sign de Dance
- Rectal examination may reveal bloodstain on the examining finger (red-currant jelly)
- Colorectal intussusception may be felt by the examining finger on rectal examination, or it may even present through anus, resembling a rectal prolapse.

Relevant Investigations

- Plain X-ray abdomen: Soft tissue shadow in the region of transverse colon with empty distal colon. Multiple air fluid levels may be seen when obstruction predominates
- Barium enema may show a filling defect called pincer shaped filling defect (caused by the intussusceptum with the intussusciptent)
- Colonoscopy can identify, colonic intussusceptions
- US and CT (Fig. 28.1) will reveal the intussuscepting mass (pseudokidney appearance).

Treatment

- Resuscitation
- Hydrostatic or pneumatic radiological reduction

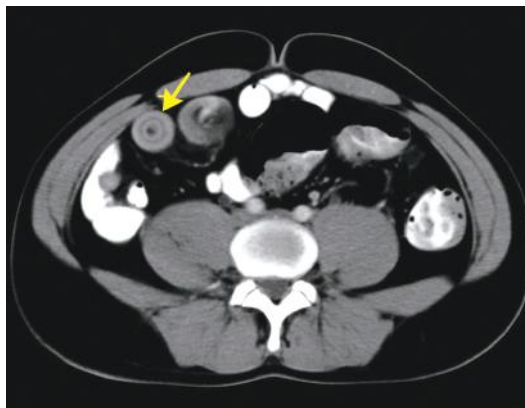


FIG. 28.1: CT—Intussusception

- Barium enema and colonoscopy, by themselves may reduce the colonic intussusception
- Laparotomy is required to reduce the small bowel intussusception, and treat the cause appropriately
- Bowel resections may be needed if the bowel segment is strangulated, and nonviable
- Perforation and peritonitis need appropriate treatment.

CONGENITAL PYLORIC STENOSIS

Incidence and Etiology

Four in 4000, with male predominance, most common in first born child.

Pathogenesis

Congenital inherited condition resulting in hypertrophy of the circular muscle fibers of pylorus.

Symptoms

- Nonbilious projectile vomiting
- Failure to thrive despite hunger.

Signs

- Visible gastric peristalsis
- Palpable mass in abdomen deep to right rectus in the transpyloric plane.

Relevant Investigations

- Serum electrolytes to be checked
- US is diagnostic.

Treatment

- Resuscitation
- Pyloromyotomy when the child is stable.

NECROTIZING ENTEROCOLITIS

Incidence and Etiology

Common in newborns.

Pathogenesis

Thought to be due to ischemia of large bowel wall with translocation of luminal bacteria resulting in systemic sepsis.

Symptoms

- Fever
- Bilious vomiting
- Bloody diarrhea
- Abdominal distension.

Signs

- Abdominal distension
- Absence of bowel sounds (may indicate perforation and peritonitis).

Relevant Investigations

- Leukocytosis
- Abdominal X-ray may show thickened dilated bowel wall containing intramural gas.

Treatment

- Fluid resuscitation
- Broad spectrum antibiotics
- Surgical resection of necrotic bowel may be needed.

TRACHEOESOPHAGEAL FISTULA

Incidence and Etiology

This fistulous communication can occur in newborn infants.

Pathogenesis

It is a developmental abnormality.



FIG. 28.2: Barium swallow—Tracheoesophageal fistula

Symptoms

Cough and fever - due to the entry of swallowed food into the respiratory tract.

Signs

Rales and rhonchi.

Relevant Investigations

Barium swallow (Fig. 28.2) allows the contrast to enter the trachea and bronchi, and is diagnostic.

Treatment

- Surgical closure of a congenital fistula is usually successful
- Malignant fistulae are difficult to handle and death becomes inevitable due to pulmonary infection.

Lymphatic System

ACUTE LYMPHANGITIS

Incidence and Etiology

In the tropics, the main causes are:

- Filariasis
- Tuberculosis.

Clinical Presentation

- Swelling of the limbs and genitalia associated with fever and rigor
- On examination, the limbs are swollen and erythematous with weeping eruptions (Fig. 29.1)
- Secondary infections can lead to localized patchy gangrene (Fig. 29.2).



FIG. 29.1: Acute lymphangitis



FIG. 29.2: Gangrenous patches in acute lymphangitis

Relevant Investigations

Peripheral smear for microfilaria.

Treatment

Antifilarial treatment and compression bandages.

ACUTE VIRAL AND BACTERIAL LYMPHADENITIS

Incidence and Etiology

- Caused by viruses (commonly rhinovirus), bacteria
- Bacterial infections are secondary to oral cavity infections
- Infections may result into abscesses
- Common in the neck.

Symptoms

- Painful swelling (commonly in the neck, rarely in the axilla and inguinal region)
- Fever may be present.

Signs

- Single or multiple tender soft to firm swellings with signs of inflammation on the skin (Figs 29.3 and 29.4)
- Examination of the drainage area is essential to assess the primary cause e.g. acute pharyngitis or dental infections causing secondary enlargements of neck glands.



FIG. 29.3: Acute viral lymphadenitis of neck



FIG. 29.4: Acute bacterial lymphadenitis of neck

Relevant Investigations

- No specific investigation is required in short lived cases
- Biopsy may be needed to differentiate persistent swellings from other causes
- Diagnostic aspiration is useful in abscesses.

Treatment

- No treatment is required for short lived cases (viral) as they resolve spontaneously
- Bacterial lymphadenitis requires broad spectrum antibiotics
- Incision and drainage for abscesses.

ACUTE FILARIAL LYMPHANGITIS AND LYMPHADENITIS

Incidence and Etiology

- Caused by thread like, parasitic filarial worms *Wuchereria bancrofti* or *Brugia malayi*
- Worms lodge in the lymphatic system, and live for 4 to 6 years producing millions of immature microfilariae that circulate in the blood
- Transmitted by mosquitoes *Culex fatigans* (develop and reach the infective stage in 7 to 21 days and migrate to biting mouth parts)
- Causes severe inflammation of the lymphatics and they settle down in the lymph nodes, commonly in the inguinal group.

Symptoms

- High-grade fever with rigor
- Swelling of the lower limbs and groin swellings.

Signs

- Inguinal lymphadenopathy (Fig. 29.5)
- Presentation with swelling of scrotum (hydrocele) (Fig. 29.6) is common.

Relevant Investigation

Demonstration of microfilaria in the peripheral blood smear (Fig. 29.7) is diagnostic.



FIG. 29.5: Acute right inguinal lymphadenitis



FIG. 29.6: Filarial hydrocele left side



FIG. 29.7: Microscopy - *Wuchereria bancrofti*

Treatment

■ Medical

- ❖ Antifilarial drugs like Diethyl carbamazine citrate (DEC) for longer periods of time is useful to control the attacks of fever
- ❖ Antibiotics are useful in controlling secondary infection in acute lymphadenitis
- ❖ Compression bandages are useful in the management of pitting edema.

■ Surgical

- ❖ Surgery is required for conditions like hydrocele
- ❖ Scrotoplasty is performed for filarial scrotums which are grossly swollen
- ❖ Procedures to divert lymph into the circulation (e.g. nodo-venous shunt) are performed for chronic non-pitting lymphedema of lower limbs.

Skin and Subcutaneous Tissues

HEMATOMA

Incidence and Etiology

Injury to the skin and subcutaneous tissue can damage the capillaries in that region, and blood can collect in the intradermal or subcutaneous plane.

Symptom

Painful swelling.

Sign

Irregular bluish patch, rarely raised above the surface of skin (Fig. 30.1), with history of trauma.

Relevant Investigations

No special investigation is necessary.

Coagulation profile may be needed for recurrent attacks.

Treatment

- Many of them resolve on their own
- Thrombolytic creams are helpful.



FIG. 30.1: Hematoma of medial aspect of thigh

ERYSIPELAS

Incidence and Etiology

This is spreading cuticular lymphangitis of the skin following trauma and infection by *Streptococcus pyogenes*.

Symptoms

- Starts as a rose rash, followed by vesicular eruptions (Fig. 30.2)
- When this occurs in regions where loose areolar tissue is found, it resembles cellulitis
- The differentiating features of erysipelas and cellulitis are given in Table 30.1.

Table 30.1: Differentiating features of erysipelas and cellulitis

Feature	Erysipelas	Cellulitis
Rose rash	Disappears on pressure Better felt than seen	Does not disappear on pressure Better seen than felt
Vesicles	Contain serous fluid	Contain pus

Note: In the face, erysipelas does not involve the pinna whereas cellulitis involves the pinna of the ear.



FIG. 30.2: Erysipelas

Investigations

Isolation of the organism in culture.

Treatment

Appropriate antibiotics cure the lesion.

FURUNCLE

Incidence and Etiology

- Infection of the hair follicle by *Staphylococcus aureus*
- Furuncle may be a source of systemic sepsis, in diabetics
- Cavernous sinus thrombosis is rare but a serious complication of furuncle on the face above the line drawn from the angle of the mouth to the tragus of the ear.

Symptom

A painful swelling at the hair root (Fig. 30.3).



FIG. 30.3: Furuncle

Signs

- Indurated swelling containing pus in due course
- Draining lymph nodes may be involved
- Cellulitis is seen in immunocompromised individuals and diabetics.

Relevant Investigations

Culture of the pus and identifying the incriminating organism is necessary.

Treatment

- The abscess may burst spontaneously
- Some may need surgical drainage.

CELLULITIS

Incidence and Etiology

- Spreading inflammation of the subcutaneous and fascial tissues
- Commonly due to *Streptococcus pyogenes*
- Diabetics are mostly affected.

Pathogenesis

Organism gains entry through the broken skin due to trauma, and infection spreads along the subcutaneous tissue planes.



FIG. 30.4: Cellulitis foot

Symptoms

- Affected part appears grossly swollen, painful and red (Fig. 30.4)
- Constitutional symptoms like fever and toxemia are common.

Signs

- It is very tender
- The lymphatics may appear inflamed and appear as red streaks and lymph nodes may be enlarged and tender
- Abscesses may form in the subcutaneous plane and skin may undergo avascular necrosis and become gangrenous.

Relevant Investigations

Organisms should be isolated for culture from discharge if any.

Treatment

Appropriate antibiotics are necessary to control infection.

ACUTE PYOGENIC ABSCESS

Incidence and Etiology

- Abscess is defined as collection of pus
- Incomplete resolution of cellulitis and lymphadenitis are the causes
- When this occurs as a part of pyemia, they are multiple in number.



FIG. 30.5: Acute abscess leg

Symptoms

- A painful swelling (Fig. 30.5)
- Constitutional symptoms like fever, rigor and toxemia will be predominant.

Sign

Tender swelling, may show fluctuation, if the pus is liquid.

Relevant Investigations

- No special investigation is required, if it is solitary
- Isolation of organism in blood by culture if suspected to be part of pyemia
- Diagnostic aspiration may be helpful in localizing deep seated abscess.

Treatment

Incision and drainage of pus will be curative under cover of appropriate antibiotics.

CARBUNCLE

Incidence and Etiology

- An infective gangrene of subcutaneous tissue
- Caused by *Staphylococcus aureus*
- Commonly seen in diabetics.



FIG. 30.6: Carbuncle nape of neck

Symptom

Commences as a painful swelling with marked induration.

Signs

- The skin becomes red and edematous, with the appearance of pustules on it. They burst to form multiple discharging sinuses like a 'sieve' (Fig. 30.6), a characteristic feature of carbuncle
- The slough may involve the deeper structures.

Relevant Investigations

- Isolation of organism in pus by culture
- Determination of blood sugar levels is mandatory.

Treatment

- Control of diabetes is necessary
- Drainage of pus and excision of slough under cover of appropriate antibiotics is curative.

BURNS

Definition

Burns is defined as the damage to the skin by coagulation necrosis caused by heat, cold, electricity, radiation and chemicals.

- Thermal burns: Heat denatures cellular proteins by coagulation necrosis. The damage is directly related to the intensity of heat and the duration of contact of the incriminating agent. Usually the damage is partial thickness or full thickness
- Electrical burns: Electricity (high and low voltage) causes deep tissue destruction both at the point of entry and at the point of exit. Muscle tissue destruction is an integral part but it cannot be assessed accurately in the initial stages
- Radiation burns: Radiation causes full thickness dermal injury due to the deep penetration of ionizing radiation
- Chemical burns: Chemicals cause cell necrosis and the damage depends on the concentration of the chemical and the duration of contact with the skin. The damage ceases only when the agent is chemically expended, and majority of the times the damage is full thickness.

Clinical Features

The burn wounds are classified into four degrees based on the clinical features (Table 30.2). The clinical photographs are shown in Figures 30.7 to 30.17.

Table 30.2: Classification of burn wounds and their clinical features

<i>Degree of burn</i>	<i>Layer of skin involved</i>	<i>Clinical presentation</i>				<i>Tests</i>	
		<i>Pain</i>	<i>Color</i>	<i>Blisters</i>	<i>Touch</i>	<i>Pressure</i>	<i>Capillary filling</i>
First degree (superficial)	Epidermis	Severe	Erythema	Absent	Present	Present	Present
Second degree (partial thickness)	Superficial (superficial dermis)	Severe	Erythema/pallor	Present	Present	Present	Present
	Deep (deep reticular dermis)	Moderate	Pallor	Usually absent	Absent	Present	Present
Third degree burn (full thickness)	Epidermis and dermis	Absent	Pallor	Absent	Absent	Absent	Absent
Fourth degree burns	Skin and subcutaneous structures	Absent	Pallor	Absent	Absent	Absent	Absent

Note: In any patient of burn injury, a combination of degrees occurs. This is important while treating a patient of burns, and repeated assessment and scrutiny of the wounds is necessary.



FIG. 30.7: Superficial burns



FIG. 30.8: Superficial partial thickness burns



FIG. 30.9: Superficial partial thickness burns



FIG. 30.10: Second degree burns with blister formation



FIG. 30.11: Mixed second and third degree burns



FIG. 30.12: Deep partial thickness burns (third degree)



FIG. 30.13: Deep burns of the forearm



FIG. 30.14: Acid burns of the face



FIG. 30.15: Chemical (tar) burns—
Thermal contact



FIG. 30.16: Electrical burns



FIG. 30.17: Thermal burns caused by hyperthermic
reaction of plaster of Paris

The depth of burns varies depending on the incriminating agent and its duration of contact with the skin (Table 30.3).

Table 30.3: Burns based on its etiology

<i>Agent</i>	<i>Exposure time</i>	<i>Depth of burns</i>	<i>Appearance</i>	<i>Pain</i>
Hot liquids	Short exposure	Superficial dermal	Wet, pink, blisters	Severe
	Long exposure	Deep dermal	Wet, red, dark	Minimal
Flame	Flash exposure	Partial thickness	Wet pink blisters	Severe
	Direct contact	Full thickness	Dry, white, waxy or brown, black leathery	Minimal
Chemicals	Direct contact	Full thickness	Light brown to light gray	Severe

Metabolic Effects of Burns

Burn injuries cause severe damage to the body structures and its metabolism. They are shown in Tables 30.4 and 30.5.

Table 30.4: Metabolic disturbances associated with burns (general effects)

<i>Category</i>	<i>Period</i>	<i>Clinical condition</i>	<i>Reason</i>	<i>Clinical presentation</i>
General effects	Immediate	Hypovolemic shock	Extracellular accumulation of water and excessive evaporation from burnt area	Tachycardia, tachypnea, hypotension, prerenal uremia, oliguria
		Hemoconcentration	Loss of water and plasma from burnt area	
		Dehydration	Excessive evaporation of water from burnt area	Tachycardia, tachypnea, hypotension, prerenal uremia oliguria
		Electrolyte imbalance	Retention of sodium and excessive excretion of potassium	Altered sensorium, uremia, oliguria
		Asphyxia and cyanosis	Due to burn of respiratory passages	Difficulty in breathing
	Late	Septicemia (multiorgan failure)	Systemic spread of infection	High grade fever, tachycardia, tachypnea, hypotension oliguria, death
		Acute renal failure	Fluid loss and electrolyte imbalance	Altered sensorium, uremia, oliguria or anuria
		Stress ulcers	Reason not known	Hematemesis and melena

Table 30.5: Metabolic disturbances associated with burns (late effects)

Category	Period	Clinical condition	Reason	Clinical presentation
Local effects	Immediate	Tissue damage	Direct injury of cells	Extensive wounds
		Edema	Excessive accumulation of fluid in the extracellular spaces	Swollen areas surrounding or below the burnt areas
		Water and electrolyte loss	Excessive loss from the burnt area from large wounds	Large dry wounds
		Local sepsis	Bacterial growth on the burnt area	Purulent discharge or slough formation, fever with rigors
	Late	Delayed healing	Inadequate and improper treatment, diabetes mellitus	Chronic wound with unhealthy granulation (Fig. 30.18A)
		Keloid formation	Severe scarring	Elevated thick irregular skin lesion (Fig. 30.18B)
		Marjolin's ulcer	Low grade epidermoid carcinoma arising from the epithelium covering the scar tissue after burns	A very slow growing painless ulcer, on the scar without lymphatic spread, resembling squamous cell carcinoma, but the edge is not raised and floor does not contain florid granulation tissue (Fig. 30.19)
		Contractures	Fibrosis of burnt areas around neck, joints	Contracted areas with restricted mobility (Fig. 30.20)
		Disability	Pain and restricted mobility due to contractures	Extensive fibrotic tissues resulting in debility (Figs 30.21A to D)
		Strictures of tubular organs	Annular healing by fibrosis of tubular structures	Dysphagia, if esophagus is involved and dyspnea, if trachea is involved

PHYSICAL EXAMINATION

Clinical examination of a patient of burns should be done quickly but in detail.

Preliminaries Before Examination

- The patient should be weighed using a trolley weighing machine (determination of weight is necessary to calculate the fluid requirement)
- The patient should be sedated well after ensuring the airway patency, and if required intubated
- The patient should be completely undressed.
- Two intravenous portals should be established for fluid resuscitation and medication.



FIG. 30.18A: Infected burns wound



FIG. 30.18B: Keloid formation after burns
(Courtesy: Dr K Sridhar)



FIG. 30.19: Marjolin's ulcer chest wall
(Courtesy: Dr R Rajaraman)



FIG. 30.20: Postburn contracture of neck
(Courtesy: Dr K Sridhar)



FIG. 30.21A: Postburn contractures
(*Courtesy: Dr K Sridhar*)



FIG. 30.21B: Postburn scarring resulting in syndactyly
(*Courtesy: Dr K Sridhar*)



FIG. 30.21C: Postburns scarring resulting in disability
(*Courtesy: Dr K Sridhar*)



FIG. 30.21D: Postburns contracture of axillary fold
(*Courtesy: Dr K Sridhar*)

Circumferential burn injuries (around the chest and limbs) especially for the third degree burns, escharotomy will have to be performed or otherwise, respiratory embarrassment and ischemic necrosis of limbs may occur.

INSPECTION

The patient should be examined head to foot, with special attention to the areas like the axillae, groins and the perineum. The extent of damage both by area and depth should be assessed quickly and precisely, as this helps in the determination of fluid replacement.

- I. *Extent of area of burns:* This is calculated in two ways:
- For small, multiple and scattered areas: 'Rule of hand' is used; determination of surface area of burns, using the patient's own hand (with adducted fingers), which is equal to 1 percent
 - For large areas: 'Wallace's rule of nine is used (Fig. 30.22A). For children, the rule is modified (Fig. 30.22B), as the surface of area of the head is bigger relative to the other parts of the body.

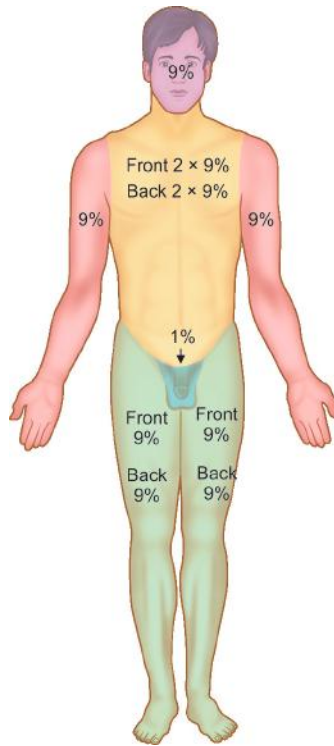


FIG. 30.22A: Wallace's rule of nine for adults

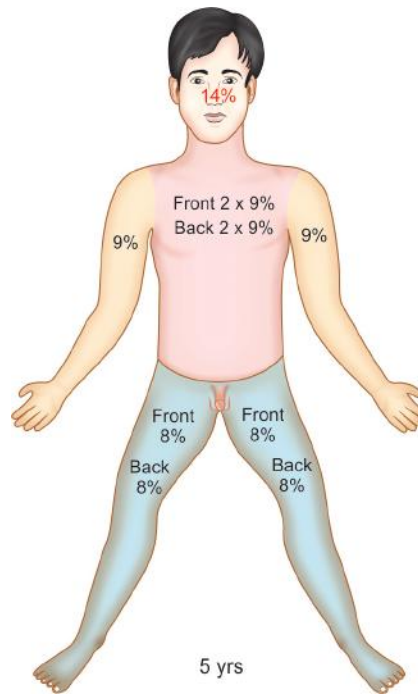
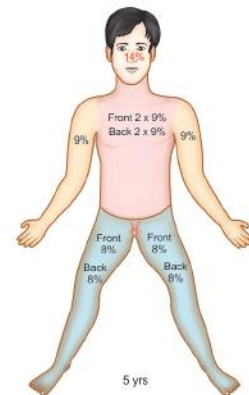


FIG. 30.22B: Modified rule of nine for children



II. *Depth of tissue damage:* This can be done by inspecting the burnt area, and reasonable conclusions may be reached.

It should be remembered that various degrees of tissue damage coexist in the same patient.

- Cutaneous sensation is tested by pricking the burnt area (sterile needle test) with a sterile needle. Presence of sensation is diagnostic of partial thickness burns, but it is not pathognomonic. Plucking the hair from the burnt area can show the depth. In full thickness burns, the hair can be plucked easily, whereas in partial thickness burns it is not.

General Examination

- *Examination of sensorium:* Usually the sensorium remains normal and not willing to answer may indicate a psychiatric problem (e.g. depression or withdrawal). Inability to answer may indicate state of unconsciousness or altered sensorium (e.g. shock, hyponatremia, uremia)
- *Examination of face:*
 - ❖ *Eyes:* Sunken eyes indicate dehydration and undernutrition. Examination of conjunctivae will reveal anemia.
 - ❖ *Hairy areas:* Surging of hair in eyebrow, moustache or over forehead will indicate respiratory burns
- *Examination of skin:* The skin should be examined for dehydration and undernutrition. Generalized edema may be seen in severely burnt patients.
- *Examination of tongue:* Dry tongue indicates dehydration
- *Recording of vital signs:* Tachycardia may indicate infection or hypovolemia. Elevated temperature may indicate associated infections and toxemia (usually seen in the second week). Hyperventilation may indicate hydration, hypovolemia and is predominant in respiratory burns. Hypotension may indicate hypovolemia.

Systemic Examination

- *Examination of oral cavity:* A systematic examination of oral cavity is essential (e.g. chemical burns, burns due to inhalation of gases)
- *Examination of chest:* Auscultation—Adventitious sounds may indicate aspiration pneumonitis or respiratory infections.

Relevant Investigations

- Hematology
 - ❖ Hemoglobin for anemia
 - ❖ Total and differential leukocyte count, e.g. infections

- ❖ ESR may be raised in infections and malignancies
Note: Hemoglobin levels initially remain normal due to hemoconcentration and serial determinations are necessary on subsequent days.
- Biochemistry
 - ❖ *Blood sugar:* Required especially in diabetics. Repeated determination is required in patients on hyperalimentation
 - ❖ *Liver function tests:* Reduced levels of serum proteins indicate loss of proteins through burn wound and will help in the replacement with blood or plasma
 - ❖ *Renal function tests:* Uremia and creatinemia indicate deranged renal status due to dehydration and hypovolemia
 - ❖ *Serum electrolytes:* Determination of serum levels of electrolytes is important in the management of burns, as electrolyte disturbances are commonly found in burns, due to evaporation during injury and later through wound.
- Blood grouping and crossmatching: It is important as the requirement of blood may be immediate.
- Radiology
 - ❖ Chest X-ray is useful (e.g. aspiration pneumonitis, inhalation injuries of lungs)
 - ❖ Contrast studies
 - Barium swallow may be required at later stages to rule out esophageal strictures
 - Bronchogram also may be required to rule out strictures of tracheobronchial tree.
- Endoscopy
 - ❖ Esophagoscopy and bronchoscopy may be required in some cases to assess the esophageal and bronchial injuries respectively, especially when injuries occur due to inhalation of toxic fumes
 - ❖ Gastroscopy may be needed to diagnose stress ulcers of stomach.
- *Electrocardiogram (ECG):* To rule out disturbances of heart especially in electrical burns, and may have to be repeated frequently.

Treatment

Resuscitation

- Basic principles of the burns victim are the same as for any other patient.
- Problems are specifically related to thermal injuries to airways, large fluid losses and potential for infection:
 - ❖ Cover burn areas with sterile drapes or plastic film to reduce infection and fluid loss
 - ❖ Give humidified oxygen by mask
 - ❖ Endotracheal intubation is required for airway injuries
 - ❖ Monitor hematocrit and electrolytes
 - ❖ Blood may be required to maintain hematocrit

- ❖ Good IV access is needed for fluid administration
 - IV analgesia
 - IV fluids

Examples of fluid regimens for resuscitation of burn victims

<i>Mount Vernon formula</i>	<i>Parkland formula</i>
4.5% albumin	Ringer lactate
Volume (ml) = $0.5 \times \text{wt (kg)} \times \% \text{ burn}$	Volume (ml) = $4 \times \text{wt (kg)} \times \% \text{ burn}$
Over six consecutive periods of 4, 4, 4, 6, 6, and 12 hours each	Given over 24 hours

NECROTIZING FASCIITIS

Incidence and Etiology

- A rapidly spreading soft tissue infection, which can affect any part of the body
- It follows, usually a minor trauma but the exact etiology is not fully understood
- Causative organisms—Mixture of gram-negative and anaerobic organisms (bacteroides and *Clostridium* sp. anaerobic streptococci) and/or group A *streptococcus*.

Pathogenesis

The infection spreads along the fascial planes causing necrosis of skin and subcutaneous tissues. Muscle layers are usually spared. The infection may spread rapidly and can be fatal in few hours.

Symptom

Painful discolored area.

Sign

The affected area is discolored and indurated.

Relevant Investigations

- Isolation of organism in pus by culture
- Determination of blood sugar levels is mandatory.

Treatment

- Control of diabetes is necessary
- Emergency excision of slough and necrotic tissue under cover of appropriate antibiotics is curative.

Section VI

Special Procedures and Situations

31. Emergency Surgical Procedures
 - Pericardial Aspiration
 - Intubation of Trachea
 - Percutaneous Tracheostomy
 - Cricothyroidotomy/
Minitracheostomy
 - Insertion of Chest Drains
32. Preparing the Patient for
Emergency Surgery
 - Passing a Nasogastric Tube
 - Peritoneal Tap
33. Antibiotics
 - Antibiotics and Emergency Surgery
34. Death

Emergency Surgical Procedures

PERICARDIAL ASPIRATION

Pericardial aspiration (Pericardiocentesis) is indicated in:

- Cardiac tamponade
- Large pericardial effusion
- For diagnostic pericardial fluid.

Materials Required

- Sterile gowns and gloves
- 10 ml syringe with needle
- Pericardial aspiration kit
- Sutures
- Securing tapes.

Procedure

- Position: Supine with 20 degrees head-up
- Establish IV access
- Adequate sedation
- With full aseptic technique, infiltrate the point of needle entry (just below and left of xiphisternum, between the xiphisternum and left costal margin)
- Introduce the needle directed to the left shoulder, and keep aspirating (Fig. 31.1)

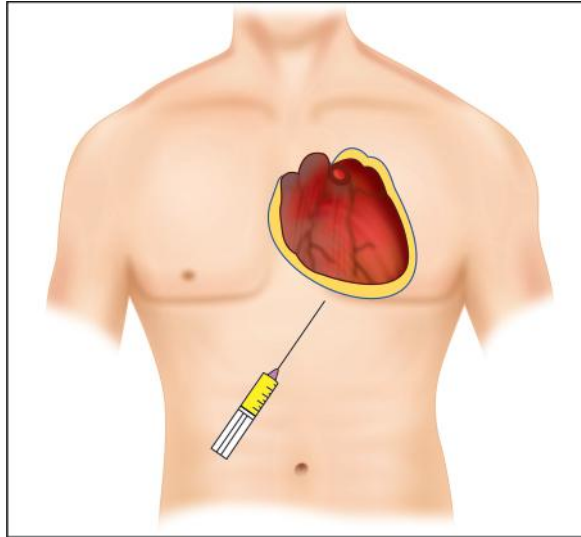


FIG. 31.1: Pericardial aspiration

- Once a straw colored fluid is drawn, keep the needle in that position, pass the guidewire and withdraw the needle
- Pass the catheter over the guidewire into the pericardial space and attach a 3-way tap
- Using a 50 ml syringe, aspirate the effusion or attach to a closed drainage system
- Suture the drain in place and strap.

Complications

- Pneumothorax
- Ventricular tachycardia
- Myocardial puncture
- Damage to coronary arteries.

- Small pericardial effusions not causing hemodynamic instability do not require pericardiocentesis
- Repeat chest X-ray and ECG

INTUBATION OF TRACHEA

Though this procedure is performed by the anesthetists it is better for the surgeons and surgical students to know, as this pertains to life-saving situation. They fall into three groups for which it is required.

- Relieving airway obstruction (e.g. tumors, head and neck trauma, surgery, airway edema)
- Protection of airway from aspiration (e.g. obtunded conscious level, impaired cough impulses)
- Facilitation of ventilation of airways (e.g. anesthesia and surgery, multiple organ failure, major trauma and brain injury).

Materials Required

- Self-inflating bags (Ambu bag)
- Face mask
- Oral/nasal airways
- Suction apparatus and suction catheters
- Laryngoscopes
- Endotracheal tubes (or various sizes)
- Sterile lubricant
- Syringe
- Anesthetic drugs/muscle relaxants
- Emergency drugs (atropine, adrenaline).

Procedure

- Oxygenate the patient well with 100 percent oxygen for about 3 to 4 minutes, which will wash out the nitrogen and fill the functional residual capacity with oxygen, thereby increasing the safety margin
- Keep the head in position (neck flexed, atlantoaxial joint extended on a firm pillow – ‘sniffing the morning air’ position)
- Give sedative/muscle relaxant as appropriate
- Hold the laryngoscope in the left hand and insert the blade into the right of the mouth sweeping the tongue under it. As the blade reaches the base of the tongue, the epiglottis is seen. Apply traction to gently draw the epiglottis forward exposing the V-shaped glottis behind
- Pass the endotracheal tube between the vocal cords so that cuff is distal to them
- Withdraw the laryngoscope gently
- Inflate the cuff of the endotracheal tube with air so that the tube snugly fits into the trachea
- Check the position of tube by auscultation and by observing the chest movements.

Immediate

- Hypoxia (due to repeated attempts)
- Misplacement of tube
- Obstruction of airway
- Trauma to airway, teeth.

Late

- Accidental displacement of tube
- Ventilator associated pneumonia
- Injury to vocal cords
- Tracheal stenosis.

Tracheal intubation is to done only if the student is familiar with this and has done with a senior in the past.

PERCUTANEOUS TRACHEOSTOMY

Conventional tracheostomy was performed only after patients had been intubated for about 10 – 14 days for fear of laryngeal and subglottic injury. Percutaneous tracheostomy has come into practice as the benefits are many.

Advantages of Tracheostomy

- More comfortable than the oro/nasotracheal tubes
- Need for muscle relaxants is less
- Switching from IPPV to T piece is easy without extubation
- Effective endotracheal suction can be done
- Speech is possible with cuff deflation or speaking tube.

Indications

- Actual or impending airway obstruction
- Difficult intubation
- Need for prolonged ventilator support.

Contraindications

- Distorted/abnormal anatomy
- Significant hemodynamic instability.

Materials Needed for Percutaneous Tracheostomy

- Skin disinfectant
- Local anesthetic
- Syringe
- 10 ml normal saline
- Basic surgical instruments
- Suture
- Securing tapes
- Emergency drugs
- Percutaneous tracheostomy kit (Fig. 31.2).

Procedure

- Position: supine with neck extended over a pillow
- Palpate the cricothyroid membrane and sternal notch



FIG. 31.2: Percutaneous tracheostomy kit

- Infiltrate with local anesthetic
- Make a 2 cm transverse incision over the membrane at the midline
- Puncture the trachea (between the first and third tracheal rings) with the introducer needle
- Confirm its position with a syringe aspiration (air/mucus)
- Pass the guidewire through the needle and remove the needle
- Dilate the trachea
- Insert the endotracheal tube
- Remove the guidewire
- Suck out all the secretions and blood clots in the trachea
- Inflate the cuff
- Place two stay sutures through the wings of tracheostomy tube, to prevent slippage, and secure with tapes
- Check the position of the tracheostomy tube (symmetrical chest expansion, X-ray).

Complications

Early

- Bleeding
- Tube misplacement
- Mucus plugging

Late

- Tracheal stenosis
- Tracheoesophageal fistula.

CRICOTHYROIDOTOMY/MINISTRACHEOSTOMY

Cricothyroidotomy is an emergency procedure to access the airway, when measures like intubation have failed. It involves the insertion of a small tube through the cricothyroid membrane for attaching to the ventilator.

Minitracheostomy involved the use of a small bored and noncuffed bore, with an internal diameter of 4 mm. Kits are available for both procedures.

Indications

- Cannot intubate
- Cannot ventilate
- Severe midfacial trauma

- Cervical spine injuries
- Chemical inhalation injuries.

Contraindications

- Inability to identify landmarks (cricothyroid membrane)
- Underlying anatomical abnormality (tumor)
- Acute laryngeal disease
- Tracheal transection
- Small children.

Materials Required

- Skin disinfectant
- Sterile drape
- Syringe with local anesthetic
- Cricothyroidotomy (Fig. 31.3)/Minitracheostomy kit
- Suture
- Securing tapes.

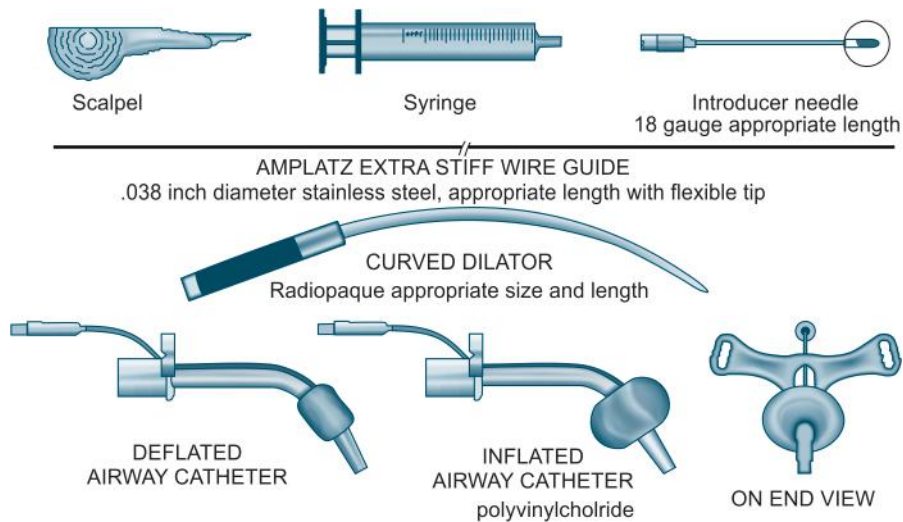


FIG. 31.3: Cricothyroidotomy kit

- Palpate the cricothyroid membrane
- Clean the skin
- Infiltrate over the cricothyroid membrane
- Make a skin incision
- Introduce needle into trachea and aspirate air
- Pass guidewire and remove needle
- Pass the introducer over the guidewire and slide the cricothyroidotomy/minitracheostomy tube off the introducer
- Remove the introducer and guidewire together, leaving the tube *in situ*
- Suck any blood or mucus
- Place two stay sutures through the wings of tracheostomy tube, to prevent slippage, and secure with tapes
- Check the position of the tracheostomy tube (symmetrical chest expansion, X-ray).

Complications

Early

- Bleeding
- Tube misplacement
- Mucus plugging

Late

- Tracheal stenosis
- Tracheoesophageal fistula.

INSERTION OF CHEST DRAINS

Insertion of a chest drain is required as an emergency treatment of life-threatening tension pneumothorax. A large bore tube preferably a 14-gauge cannula is used for this purpose.

Indications

Draining of:

- Pneumothorax
- Hemothorax
- Pleural effusion

- Empyema
- Chylothorax.

Type of Drain

Chest drains are of two types:

1. Large bore tubes
2. Seldinger drains.

Site of Drain

This is partly decided by the position of the collection clinically and radiographically.

- For draining the pneumothorax, the tube is placed in the 2nd intercostal space in the midclavicular line (Fig. 31.4).
- For draining the fluid collections, the tube is placed in the 5th intercostals space just anterior to the midaxillary line (Fig. 31.4).

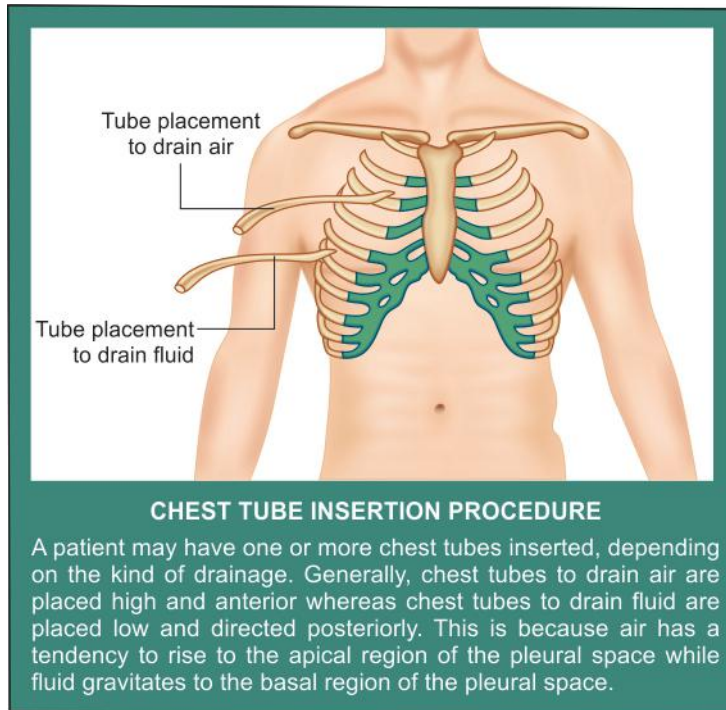


FIG. 31.4: Placing the chest drains

Materials Required

- Skin disinfectant
- Skin drapes
- Syringe
- Local anesthetic
- Basic instruments
- Chest drain
- Silk sutures
- Adhesive tapes
- Underwater seal.

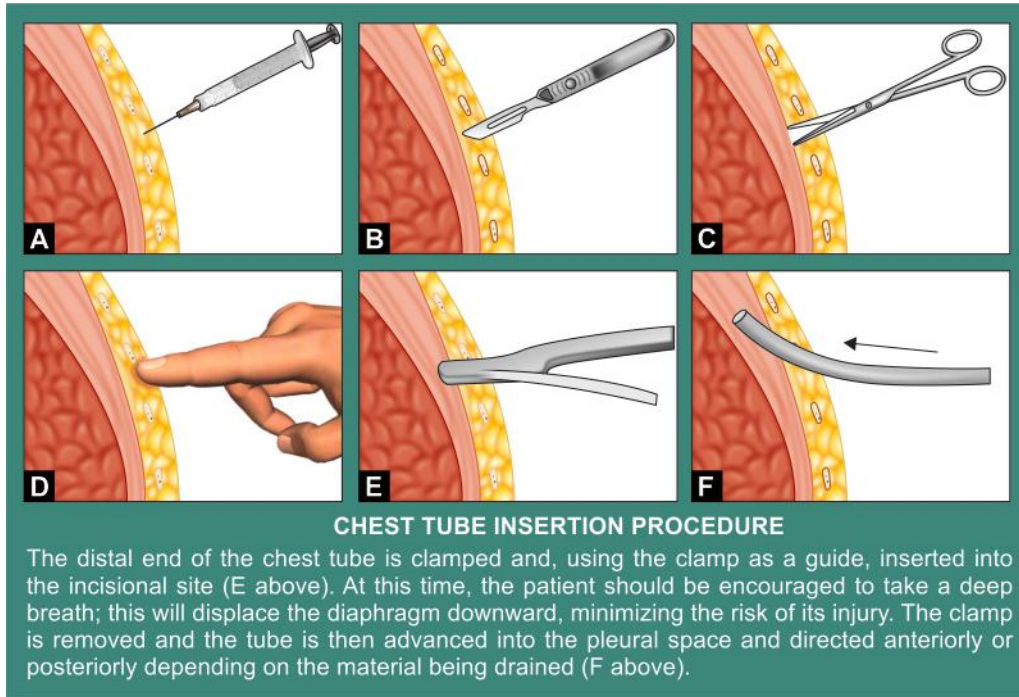
Procedures

Through Thoracostomy (Figs 31.5A to F)

- Position: supine with arm lifted, with a pillow behind the back
- Clean the field with sterile preparation
- Infiltrate the local anesthetic at the chosen site
- Make a small skin incision
- The intercostal muscles and the pleura are dissected out and plunged with artery forceps
- The chest drain is inserted into the pleural cavity
- The drain is connected to the underwater drain (Fig. 31.6)
- The drain is secured with sutures to the skin
- Adhesive strapping is done
- Order for a X-ray and the position checked.

Seldinger Technique

- Position: supine with arm lifted, with a pillow behind the back
- Clean the field with sterile preparation
- Infiltrate the local anesthetic at the chosen site
- Use the chest drain kit (Fig. 31.7)
- Advance the needle through chest until blood/fluid/air is aspirated
- Pass chest tube/pigtail catheter over guidewire
- Attach to the underwater seal
- Secure well and dress.



FIGS 31.5A to F: Method of placing the chest drain

- Never clamp the chest drain, as clamping may produce a tension pneumothorax
- While shifting the patient, the underwater drain should be kept below the level of chest

PASSING A NASOGASTRIC TUBE

Nasogastric tube is generally used to decompress the stomach and also for feeding.

Indications

- To deflate the stomach
- To aspirate gastric contents
- To provide enteral nutrition.

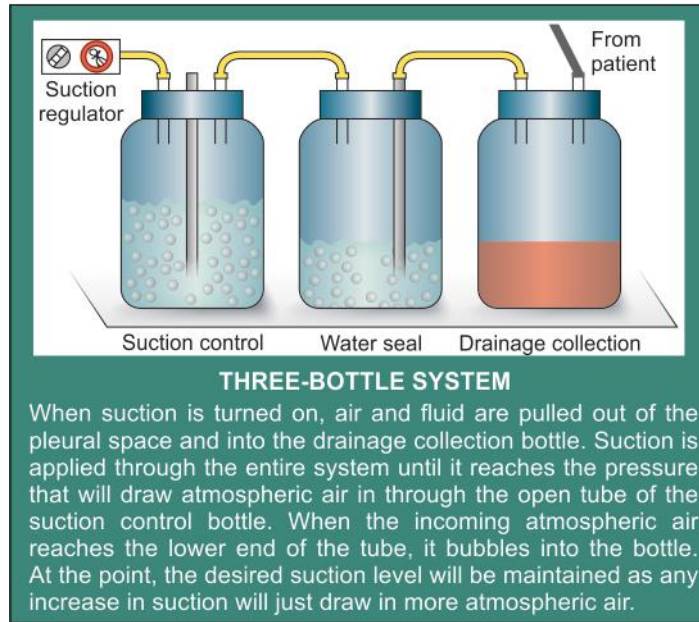


FIG. 31.6: Three chamber underwater drain

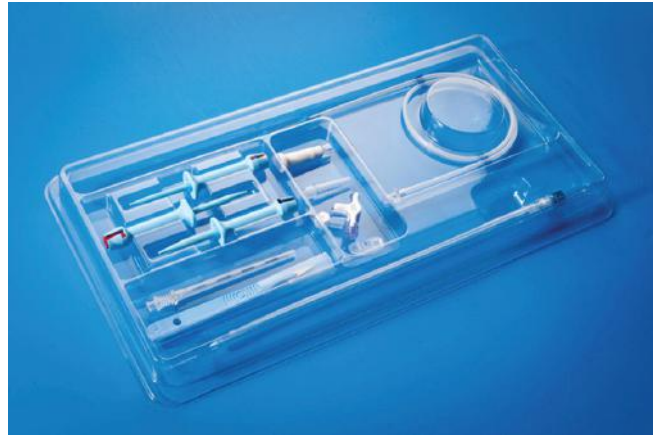


FIG. 31.7: Seldinger chest drain kit

Contraindications

Severe coagulopathy.

Materials Required

- Gloves and mask
- Nasogastric tube
- Lubricating jelly
- Laryngoscope (needed for unconscious patients).

Procedure (Fig. 31.8)

- Position: supine and head neutral or semirecumbent
- Lubricate the nasogastric (NG) tube well
- Keeping in alignment with the long axis of the patient, introduce the tube into the nostril
- If the patient is cooperative, ask him to swallow
- The tube generally flows down along with the swallowing movement
- Confirm the tip of NG tube in the stomach by auscultation or aspiration of contents
- Secure NG tube in position with adhesive tape.

PERITONEAL TAP

Peritoneal tap is required to obtain samples for diagnostic purposes and also relieve the tension of a tense ascites.

Materials Required

- Skin disinfectant
- Sterile drapes
- 10 ml syringe with local anesthetic
- Pig tail drain
- Sutures
- Adhesive strapping
- Ultrasound.

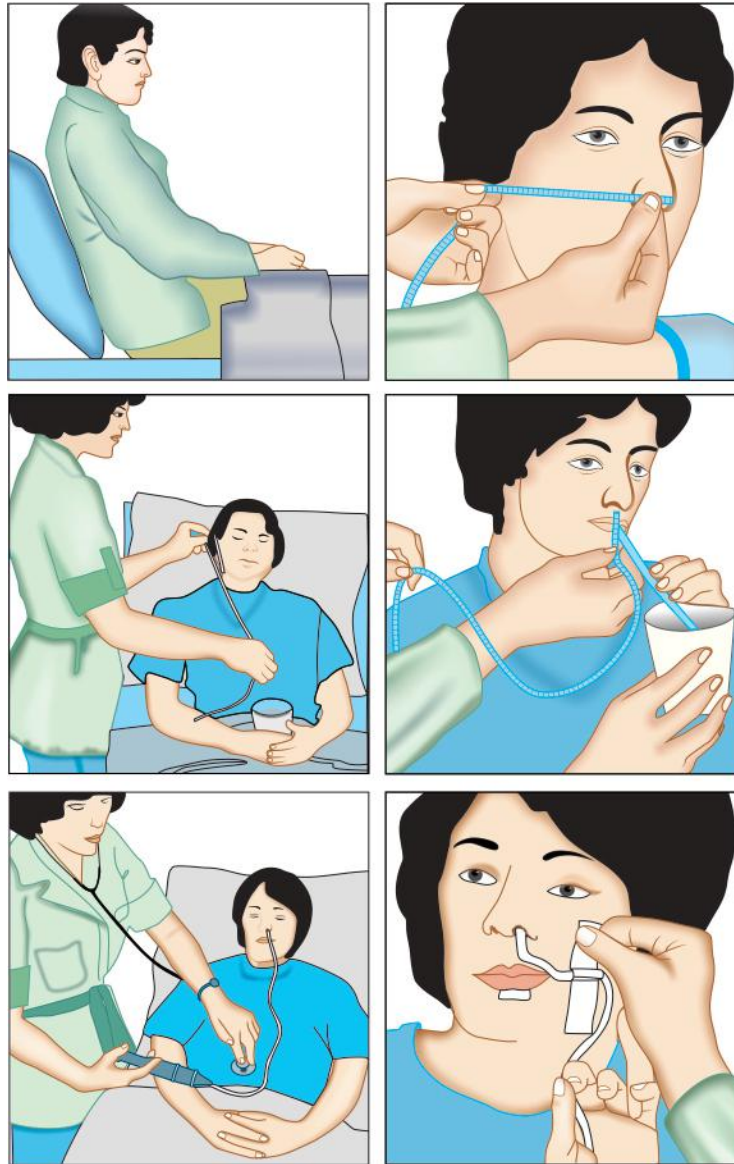


FIG. 31.8: Nasogastric tube insertion



FIG. 31.9: Peritoneal tap

Procedure

- Clean the skin with disinfectant
- Confirm the presence of fluid by percussion or by ultrasound
- At the chosen site, inject the local anesthetic
- Introduce the syringe and aspirate fluid to confirm its presence (Fig. 31.9)
- Introduce pig tail drain using Seldinger technique
- Connect the drain to collecting bottle
- Strap the drain.

Preparing the Patient for Emergency Surgery

Any patient for surgery needs optimization, especially so the patients for emergency surgery, as the parameters are never normal. This optimization becomes necessary to reduce the perioperative complications. The optimization requires:

- Fluid and electrolyte corrections
- Correction of hematological and metabolic disturbances.

General Measures

- Nil by mouth
- Good intravenous access
- Appropriate intravenous fluid replacement
- Appropriate antibiotics
- Adequate analgesia
- Measures for DVT prophylaxis
- NG tube insertion where stomach needs to be decompressed
- Urinary catheterization
- Central venous line access
- Informed consent.

Investigations Support

- Complete blood count (CBC)
- Urea and electrolytes
- Electrocardiogram (ECG) and Chest X-ray (CXR).

Situations which Need Special Attention

- Uncontrolled glycemic status
- Ketoacidosis
- Cardiovascular complications
- Altered conscious level
- Jaundiced patient with coagulation abnormalities
- Patient on steroids
- Patient on anticoagulants.

Antibiotics

ANTIBIOTICS AND EMERGENCY SURGERY

Antibiotics become necessary to prevent an infection or control an infection. Prevention becomes necessary so that sepsis related complications, which carry high morbidity and mortality are averted.

Prophylactic antibiotics are administered within 1 hour before the start of the surgical procedure. Second dose becomes necessary only when contamination is encountered or the blood loss exceeds 1 liter, since it is shown that antibiotic clearance is slower in patients undergoing surgery. Postoperative administration is only required when there is heavy contamination.

Which Emergency Surgical Procedure should have Antibiotic Prophylaxis ?

Antibiotic prophylaxis is recommended for the following emergency surgical procedures

- Colorectal surgery
- Appendicectomy
- Hernia surgery (use of mesh)
- Open biliary surgery
- Lower limb revascularization surgery.

The surgeries are classified based on the contamination at the time of surgery, and the infection rates vary differently (Table 33.1).

Surgical site infections (SSIs) are defined as infective complication occurring within 30 days of surgical procedure. The infection is caused by various organisms and some antibiotics are effective against these organisms (Table 33.2).

Table 33.1: Classification of surgeries related to contamination

<i>Classification of case</i>	<i>Type of surgery</i>	<i>Wound infection rate</i>
Clean	Surgery in noninflamed tissue, and bowel not opened	<2%
Clean contaminated	Surgery of hollow viscus other than colon, with minimal contamination	10%
Contaminated	Surgery through a hollow viscus with gross spillage	20%
Dirty	Fecal peritonitis, traumatic wound contaminated for >4 hrs, frank pus	40%

Table 33.2: Relationship of surgical procedures with potential organisms and sensitive antibiotics

<i>Surgical procedures</i>	<i>Organism</i>	<i>Antibiotics</i>
General surgical procedures	<i>Staphylococcus aureus</i>	Flucloxacillin, ciprofloxacin, clindamycin
	Beta – hemolytic streptococci	Ciprofloxacin, clindamycin
Upper GIT	Enteric Gram-negative bacilli	Cefuroxime, co-amoxyclav, gentamycin
Lower GIT	Enteric Gram-negative bacilli	Cefuroxime, co-amoxyclav, gentamycin
	Anaerobes	Metronidazole
Procedures using prosthesis	Coagulase + staphylococci	Vancomycin + removal of prosthesis

Table 33.3: Antibiotics related to Gram staining

<i>Gram stain</i>	<i>Antibiotic</i>
Gram-negative rods	Cefuroxime + / – gentamycin +/- metronidazole
Gram-positive cocci (Streptococci)	Amoxicillin
Gram-positive cocci (Staphylococci)	Flucloxacillin (Vancomycin / Teicoplanin)
Gram-negative cocci	Ceftriaxone

Prior to full culture reports becoming available, a Gram stain may provide information to the likely organism. Empiric antibiotics can be used on Gram stain with fair accuracy (Table 33.3).

- Infection prevention and control measures must be adhered to
- Sepsis is usually bacterial in origin, though fungi and viruses should be considered
- Obtaining appropriate microbiology samples will help to use the right antibiotic
- Choice of antibiotic should be carefully considered.

Death

A death is certified only when the following are documented:

- Absent respiratory rate for 3 minutes
- Absent pulse and heart sounds for 1 minute
- Fixed dilated pupils.

Breaking the News

Breaking a bad news especially of the impending death or after death is very tricky. This will upset the family which inherently is stressed. Well informed family will accept but ill informed or less informed will not take it very easy. To overcome this situation, the clinician should appear:

- Competent
- Honest
- Clear.

It is better to choose a suitable environment for discussion with the close relatives. Crowding of all family members and relatives should be politely discouraged. While discussing this it is better to:

- Review the case record in full
- Prepare the anticipated questions with your colleagues
- Keep a colleague or nurse alongwith
- Understand the current level of understanding
- Explain in detail

- Ask if there are any doubts and questions
- Be a good listener
- Do not lose your cool
- Be patient in answering all the questions, and this point of time no question is a 'dumb question'
- Assure them that you will be of help at anytime, which will take you a long way.

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