A text book of
SHALYA TANTRA

2

As per updated syllabus of CCIM New Delhi

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SHALYATANTRA

PAPER 2

PART A

BHAGNA

1. **Bhagna – Skeletal injuries: Prakara including pathological fracture, Samanya Lakshana, Upadrava and Chikitsa.** Description of fracture of following bones with Clinical features, Diagnosis, Complications and Management – scapula, clavicle, humerus, radius, ulna, femur, patella, tibia and pelvis bones. Sandimoksha - Dislocation: Dislocation of following joints with Clinical features, Diagnosis, Complications and Management of shoulder, elbow and hip.

Bhagna:
The word bhagna is derived from root "bhanj dhatu" and "kta" pratyaya meaning to break.

अस्थि विश्लेषो अत्र भङ्गे|
Discontinuity of bones or joints is called as Bhagna.

Sandhimukta: प्रसरणाकुञ्चनयोरशक्तिः संधिमुक्तता| (A. H. U. 27/1)
Incapability of extension, flexion movement is called as Sandhimukta.

Nidana:
पतनपीडनप्रहाराक्षेपणव्यालमृगदशनप्रभृतिभिरभिघातविशेषैरनेकविधमस्थ्नां भङ्गमुपदिशन्ति|| (Su. Ni. 15/3)

1. Patana: Falling from height
2. Peedana: Compression
3. Prahara: Strong blow from blunt instrument
4. Akshepana: Violent jerks, vigorous movements
5. Vyala mrugadashana: Bites, nail injuries or attack of wild animals
6. Balavad vigraha: Strong block from heavy built personality
7. Abhighata: Trauma

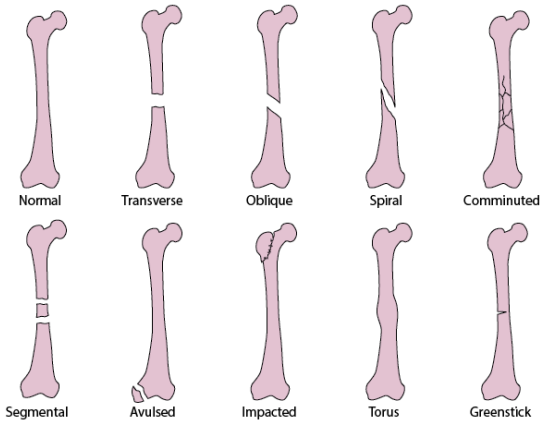
Bhagna prakara:
1. Kanda bhagna (Fracture)
2. Sandhimukta (Dislocation)

1. Kanda bhagna (Fracture):

काण्डभग्नमत ऊर्ध्वं वक्ष्यामः कर्कटकम्, अश्वकर्णं, चूर्णितं, पिच्चितम्, अस्थिच्छल्लितं, काण्डभग्नं, मज्जानुगतम्, अतिपातितं, वक्रं, छिन्नं, पाटितं, स्फुटितमिति द्वादशविधम्|| (Su. Ni. 15/8)

1) Karkaṭaka (Displaced fracture- separation of fractured end)
2) Ashvakarṇa (Oblique fracture- fractured bone elevated resembling ear of horse)
3) Chūrṇita (Comminuted fracture, crackling sound produced when hand moved over the fracture site.

4) Picchita (Compressed fracture, gets flattened due to pressure)
5) Asthicchallita (Periosteal avulsion)
6) Kāṇḍabhagna (Transverse fracture)
7) Majjānugata (Impacted fracture)
8) Atipātita (Complete fracture)
9) Vakra (Greenstick fracture)
10) Chinna (Incomplete fracture)
11) Pāṭita (Cracked fracture)
12) Sphuṭita (Fissured fracture)



1) Karkataka: Fracture resembles the shape similar to a 'crab' and so named as karkataka. There will be no displacement of fractured fragments. Fractured fragments may be elevated. On palpation it resembles a gland (Hematoma).
2) Ashwakarna: The fractured part has appearance of horse ear and is elevated and displaced.
3) Churnita: In this type of fracture, the bone breaks into multiple fragments and not able to regain normal shape. There will be greater injury palpation due to the presence of crepitus throughout the line of fracture.
4) Picchita: Fractured part gets separated from the body itself. There is flattening and marked swelling.
5) Asthichalita: Here the fracture is confined to any one surface of bone that is medial, lateral, anterior or posterior side.
6) Kandabhagna: Fractures of shaft of long bones. An attempted movement produces tremors.
7) Majjanugata: The fragments of broken bone are impacted in to the marrow cavity of another fragment. It is a fracture of old age.
8) Atipatita: Complete fracture. Fragments are completely separated and the separated fragments hangs or angulate.
9) Vakra: in children. The bone gets bend but not broken.
10) Chinna: Cortex of bone remain patent.
11) Patita: cracked in to multiple fissures resulting in severe pain in the affected part.
12) Sphutita: In which the fractured part is swollen, looks like tip of paddy bunch. There will be pricking pain due to sharp points of bristle.

2. Sandhimukta prakara:

तत्र सन्धिमुक्तम्- उत्पिष्टं, विश्लिष्टं, विवर्तितम्, अवक्षिप्तम्, अतिक्षिप्तं, तिर्यक्क्षिप्तमिति षड्विधम्||
(Su. Ni. 15/5)

1) Utapishta: (Fracture – dislocation)
Swelling on both sides of joints with pain, which specially occurs at night.
2) Vishlishta: (Subluxation)
Slight swelling but persistent pain and derangement of joint occur.

3) Vivartita: (Dislocation with lateral displacement)
   Due to lateral displacement of joint, pain and deformity occurs.
4) Avakshipta: (Dislocation with downward displacement)
   Separation of joint and severe pain occurs.
5) Atikshipta: (Dislocation with overriding)
   There is pain and overriding of both bones of joint.
6) Tiryakshipta: (Dislocation with oblique displacement)
   Here one of the bone gets obliquely displaced and excessive pain occurs.

Clinical features of Bhagna:

- Shavathu bahulya (Diffuse swelling at the site of fracture)
- Spandana (Throbbing or twitching sensation)
- Vivartana (Displacement of fractured fragment)
- Sparsha asahishnuta (Tenderness)
- Avapeedyamane shabda (Crepitus elicited on palpation)
- Srastangata (Flaccidity of muscles)
- Vividha vedana pradurbhava (Different types of pain which depend upon nature of trauma,
- bone fractured, displacement of fragments and nature of soft tissue injury)
- Sarva avastasu na shrama labha (Inability to get comfort in any position)

Clinical features of sandhimukta:

- Inability of extension, flexion, circumduction, or any movements
- Severe pain
- Hyperesthesia

Sādhyāsādhyatā:

Kṛcchrasādhya → Chūrṇita, Chinna, Atipātita, Majjānugata Bhagna
Asādhya → Kṛsha, Vṛddha, Bāla, Kṣatakṣīṇa, Kuṣṭha, Shvāsa, Sandhyupagata

Principles of Bhagna Chikitsa:

The line of treatment of bhagna comprises of three main steps (3R)

1. Bhagna sthapana (Reduction)
2. Kusha bandhana (Retention/Immobilization)
3. Karmavartana (Rehabilitation)

1. Bhagna sthapana:

There are two main techniques mentioned in Sushruta Samhita for bhagna sthapana: Anchana and Peedana.

a. Anchana (Traction):

It is a technique in which the wide gap between the fragments may be corrected. Anteriorly, medially, laterally, or posteriorly displaced fragments can be brought in alignment by the application of traction.

b. Peedana (Pressure):

It is another technique where in fractured fragments are approximated through gentle and controlled pressure. The Vinmana (Depressed) and Unmana (Elevated) technique can be incorporated within peedana only. In case vinmana fractures; the fragments should be carefully lifted. In case of unmana the raised fragments should be gently pressed down.

2. Kusha bandhan: It is used

- To prevents the movements that interfere with union
- To prevent re-displacement of fractured fragments
- To prevent angulations and
- To relieve pain

Splints:

The bark of madhuka, udumbara, ashvatha, palasha, kakubha, bamboo, sarja and banyan tree should be used for the purpose of splints. After the injury has been corrected, the joint regains its natural position. It should then be wrapped up all-round with a cloth impregnated with ghrita, over which kusha should be placed and then the limb bandaged properly.

Bandaging:

- The bandage should be changed according to doshas affecting the fracture and
- Weekly: In cold weather/Saumya rutu
- Every fifth day: In moderate weather/Sadharana rutu
- Every third day: In hot weather/Agneya rutu
- A very loose bandage does not immobilize the fracture site; whereas a very tight bandage produces inflammation, pain and suppuration in skin. Hence it is advised to tie moderately tight bandage for fractures.

3. Karmavartana:

The importance of physiotherapy in a limb injury was also appreciated by Sushruta. After proper union it is desirable that the joints or fractured parts must regain normal functions and shape. Various devices including exercises were being suggested by acharyas.

Factors causing delay in fracture healing:

- Persons who eat less
- Vata predominance
- Who shows lack of self-control
- Who has complications

Pathya:

- Shali rice
- Milk
- Pea soup
- Meat soup
- Ghrita
- Nourishing food & drink

Apathya:

- Avoid saltish, pungent, alkaline and citrous substance
- Should not have sexual intercourse
- Avoid exposure to sun
- Physical exercise
- Dry foods

Local application:

For local application manjistha, mashuka, rakta Chandan and shali grain powder mixed with shata dhauta ghrita should be used.

Orally:

Madhura rasa dravya processed with ksheera of just delivered cow ghrita along with laksha should be taken daily in the morning.

Time required for union of fracture:

Children → 1 month
Middle age → 2 month
Old age → 3 month

Malunion of fractures:

In case of transverse fracture, when it has united in a crooked position and even when consolidated, it should be refractured, set right and treated as an ordinary fracture. This method of ostoclasis is practiced even today for the treatment of malunion of long bone fractures where such correction is considered to be desirable.

Signs of proper union:

The fracture should be considered to have united well if the union is

- Painless
- Without any shortening of part
- Without any unevenness
- Allows of free and easy movements

Dislocation of hip joint:

The wise surgeon should reduce the dislocated thigh bone by a circular motion (Bigelow's maneuver for the reduction of hip dislocation by circumduction described in 1845). He should then bandage (thigh bone), as mentioned earlier for sphutita and picchita bhagna.

Dislocation of elbow, knee, ankle, and wrist joints:

When the elbow joint is dislocated, it should be massaged by the thumb. Later, the dislocated elbow joint should be pressed. Then, after applying extension it should be flexed and irrigation by oily substance carried out. The same procedure is applied for knee, ankle, and wrist joint dislocation.

MODERN CONCEPT OF FRACTURE

Fracture:

A fracture is a break in a bone. Most fractures result from a single, significant force applied to normal bone.

In addition to fractures, musculoskeletal injuries include

- Joint dislocations and subluxations (partial joint dislocations)
- Ligament sprains, muscle strains, and tendon injuries

Musculoskeletal injuries may occur in isolation or as part of multisystem trauma.

Most musculoskeletal injuries result from blunt trauma, but penetrating trauma can also damage musculoskeletal structures.

Classification:

1. Simple (Closed) or compound (Open):

- Simple / closed: The bone can break within its soft tissue envelop and may not communicate to the exterior through skin or mucus membrane.
- Compound / open: soft tissue itself may be damaged by the external forces, exposing the bone to the external atmosphere.

2. Based on extent of fracture line:

- Incomplete: It does not involve whole breadth of shaft and a portion remains intact. E.g., Greenstick fracture.
- Complete: A complete fracture could be un-displaced or displaced, where whole thickness of bone is discontinued.

3. Depend upon cause of fracture:

- Traumatic: It is due to some sort of load on normal bone like violence or injury which breaks it.
- Pathological fracture: It occurs in a diseased bone and is usually spontaneous. The force required to bring about a pathological fracture is trivial. E.g., tumour, infection etc.
- Stress or fatigue fracture: it is usually an incomplete fracture commonly seen in athletes and in bones subjected to chronic and repetitive stress. E.g., third metatarsal fracture, fracture of tibia etc.

4. Atypical fractures:

- Greenstick fractures: It is seen exclusively in children. Here the bone is elastic and usually bends due to buckling or breaking of one cortex when a force is applied.
- Impacted fractures: Here the fracture fragments are impacted into each other and are not separated and displaced.
- Hairline or crack fracture: It is very fine break in the bone that is difficult to diagnose clinically. Radiology usually helps or still better is CT scan.
- Torus fracture: This is just a buckling (bending) of the outer cortex.

Clinical features:

Symptoms:

- Pain: This is a very subjective symptom and is invariably the first and the most important complaint. It may be mild, moderate, and severe and may be due to tearing of periosteum (which contains the nerve endings), soft tissue injury, vascular injury, nerve injury, etc. It must be remembered that fracture pain is only felt during movement of fracture site.
- Swelling: It is due to soft tissue injury, medullary bleeding, and reactionary hemorrhage. Swelling is usually more in fractures and less in dislocations.
- Deformity: Patients with displaced fractures and dislocations usually present with deformity of varying severity.
- Loss of function: Inability to use the affected part is another frequent complaint.

Signs:

- **Tenderness:** This is an important clinical sign in bone and joint injuries and is usually seen after trauma.
- **Swelling:** The swelling is examined for shape, size (mild, moderate, severe), consistency (cystic, soft, hard), fluctuation, etc.
- **Abnormal mobility** between fracture fragments is a sure sign of fracture.
- **Loss of transmitted movements:** When one end of the limb is rotated, it automatically transmitted to the other end. Due to the break in the continuity, this is no longer possible in displaced fractures.
- **Crepitus:** This is an abnormal grating sensation produced by the friction between two torn surfaces of the fracture fragments. Obviously, it is electable only in displaced fractures. It should be elicited very gently at the end of the clinical examination.
- **Shortening:** Limb shortening of various degrees is common in bone and joint injuries.

Investigation: History, Physical examination, X-rays, Sometimes MRI, or CT scan

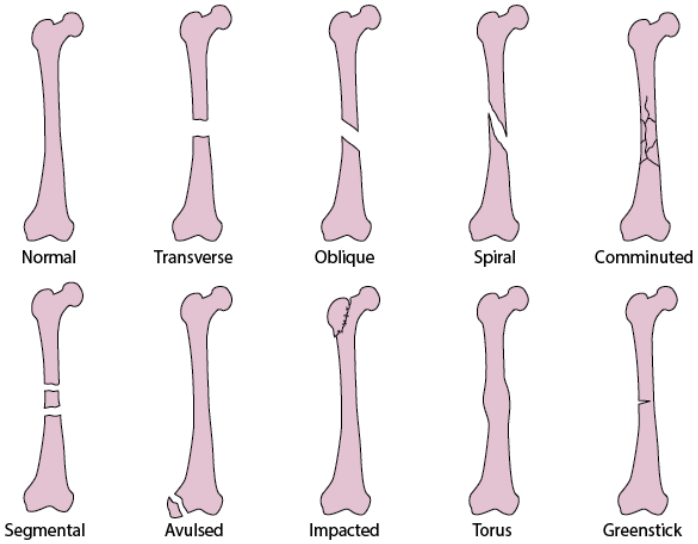
X-ray:

- Anteroposterior view: Shows sidewise displacement, external or internal fracture
- Lateral view: Shows displacement of lateral, anterior, or posterior

Common Types of Fracture Lines:

1. Transverse fractures are perpendicular to the long axis of a bone
2. Oblique fractures occur at an angle
3. Spiral fractures result from a rotatory mechanism; on x-rays, they are differentiated from oblique fractures by a component parallel to the long axis of bone in at least 1 view.
4. Comminuted fractures have > 2 bone fragments. Comminuted fractures include segmental fractures (2 separate breaks in a bone).
5. Avulsion fractures are caused by a tendon dislodging a bone fragment.

6. In impacted fractures, bone fragments are driven into each other, shortening the bone; these fractures may be visible as a focal abnormal density in trabeculae or irregularities in bone cortex.
7. Torus fractures (buckling of the bone cortex) and greenstick fractures (cracks in only 1 side of the cortex) are childhood fractures.



Management:

- Treatment of associated injuries
- Reduction as indicated, splinting, and analgesia
- RICE (rest, ice, compression, and elevation) or PRICE (including protection with a splint or cast) as indicated
- Usually, immobilization
- Sometimes surgery

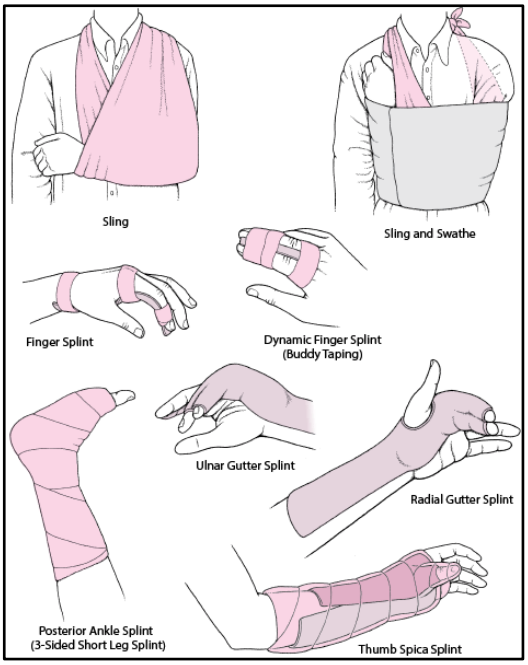
PRICE (protection, rest, ice, compression, elevation) may be beneficial.
Protection helps prevent further injury. It may involve limiting the use of an injured part, applying a splint or cast, or using crutches.

Rest may prevent further injury and speed healing.

Ice and **C**ompression may minimize swelling and pain. Ice is enclosed in a plastic bag or towel and applied intermittently during the first 24-48 hours (for 15-20 minutes, as often as possible). Injuries can be compressed by a splint, an elastic bandage, or, for certain injuries likely to cause severe swelling, a Jones compression dressing. The Jones dressing is 4 layers; layers 1 (the innermost) and 3 are cotton batting, and layers 2 and 4 are elastic bandages.

Elevating the injured limb above the heart for the first 2 days in a position that provides an uninterrupted downward path; such a position allows gravity to help drain edema fluid and minimize swelling.

Immobilization:



Immobilization decreases pain and facilitates healing by preventing further injury and keeping the fracture ends in alignment. Joints proximal and distal to the injury should be immobilized.

Most fractures are immobilized for weeks in a cast (a rigid, circumferential device). A few rapidly healing, stable fractures (e.g.: buckle wrist fractures in children) are not casted; early mobilization has the best results.

Patients with casts should be given written instructions, including the following:

- Keep the cast dry.
- Never put an object inside the cast.
- Inspect the cast's edges and skin around the cast every day and report any red or sore areas.
- Pad any rough edges with soft adhesive tape, cloth, or other soft material to prevent the cast's edges from injuring the skin.
- When resting, position the cast carefully, possibly using a small pillow or pad, to prevent the edge from pinching or digging into the skin.
- Elevate the cast whenever possible to control swelling.
- Seek medical care immediately if pain persists or the cast feels excessively tight.
- Seek medical care immediately if an odor emanates from within the cast or if a fever, which may indicate infection, develops.
- Seek care immediately for progressively worsening pain or any new numbness or weakness, which may indicate compartment syndrome.

A splint can be used to immobilize some stable injuries, including some suspected but unproven fractures and rapidly healing fractures that require immobilization for several days or less. A splint is non-circumferential; thus, it enables patients to apply ice and to move more than a cast does. Also, it allows for some swelling, so it does not contribute to compartment syndrome.
Some injuries that ultimately require casting are immobilized initially with a splint until most of the swelling resolves.

Other procedures:

- Joint replacement (arthroplasty) may be needed, usually when fractures severely damage the upper end of the femur or the humerus.
- Bone grafting may be done immediately if the gap between fragments of bone is too large. It may be done later if healing is delayed (delayed union) or does not occur (non-union).

Healing of fracture:

1. Stage of hematoma formation: Lasts for 7 days

Soon after fracture blood leaks out through torn vessels in the bone and forms hematoma in and around fracture.
The periosteum and local soft tissues are stripped from the fracture ends to form ischemia necrosis
Some osteocytes die and other are differentiate to produce daughter cells contributing to healing process.

2. Stage of granulation: Lasts for 2-3 weeks

Differentiated cells organize to provide blood vessels, fibroblasts, osteoblasts give rise to formation of soft granulation tissue.
Blood clot gives rise to lose fibrous mesh, which serves as framework for the growth of fibroblasts and new capillaries.
Clot is removed by macrophages and giant cells arise from granulation tissue.

3. Stage of callus: Lasts for 4-12 weeks

Osteoblasts are created by granulation tissue; they lay down an intercellular matrix which is impregnated by calcium salts forming callus.
It is visible on x-rays
It gives blood strength to fracture slower in adults and cortical bones faster in children.

4. Stage of remodeling: 1-4 years

Callus is replaced by mature bone with typical lamellar structure. The change is called multicellular unit based.

5. Stage of modelling: Many years

Bone is gradually strengthened, sharpening of cortices occur at endosteal and periosteal surface.

Complications:

Acute complications (associated injuries) include the following:

- Severe bleeding which leads to hemorrhagic shock
- Vascular injuries
- Nerve injuries
- Pulmonary embolism
- Fat embolism
- Compartment syndrome
- Infection

Long-term complications of fractures include the following:

- Joint instability
- Stiffness and impaired range of motion
- Non-union or delayed union, Malunion
- Osteonecrosis, Osteoarthritis
- Limb length discrepancy

SCAPULAR FRACTURE

Scapular fractures are uncommon injuries, representing about 3-5% of all shoulder fractures. Scapular fractures are often associated with other injuries due to the high energy trauma.

Etiology & Risk Factors:

- Participating in contact sports
- Activities with the potential of falling, such as rock climbing, bicycling, or horseback riding
- Decreased bone mass associated with osteoporosis
- Not wearing a seatbelt when driving

Up to 75% of all scapular fractures are due to traumatic events, such as motor vehicle or bicycle accidents.

Signs & Symptoms:
A person with a scapular fracture typically experiences severe pain.
This pain is often:

- Immediate
- Localized to the upper back, across the shoulder blade, and/or at the top of the shoulder
- Aggravated by arm movement or taking deep breaths, because chest wall movement may cause the fractured scapula to move.

Bruising and swelling in the shoulder and upper back
A grinding sensation during shoulder movement (if movement is possible)
The inability to lift the affected arm, and the desire to hold the arm to keep it and the shoulder still.
Weakness or persistent tingling in the arm
The shoulder may even appear flattened, drooped, or disfigured.

Treatment:

- Triangular sling
- Analgesic, Anti-inflammatory

CLAVICLE FRACTURES

It is a common fracture resulting due to falling on the ground / sometimes due to stretching of hand. It is usually displaced fracture; the outer fragment is displaced downward due to the gravity & pulled by pectoralis major muscle while inner fragment is displaced upward due to pull by the sternocleidomastoid muscles. The most common site is outer 1/3 and inner 2/3 junction.

Signs & Symptoms: Pain, swelling, tenderness & also neuro-deficiency in upper limb.

Treatment:

- Figure of 8 bandage
- Triangular sling

HUMERUS FRACTURE

Etiology:

Most of these fractures result from a fall on an outstretched arm; less often, a direct blow is involved.

Common sites:

- Surgical Neck
- Lesser tuberosity
- Lateral condyle
- Infra condyle
- Greater tuberosity
- Mid-shaft (middle 3rd commonly)
- Supra condyle

Diagnosis: X-ray & MRI

Treatment:

- Immobilization
- Hanging cast
- Reduction under anesthesia followed by application of POP in 'U' slab & it is maintained for 3 - 5 weeks.

Complications:

- Volkmann's Ischemic contracture.
- Myositis ossificans (Calcium deposition in Muscles)
- Cubitus valgus Deformity with internal rotation & slight flexion of elbow due to ulnar nerve palsy.
- Gun-stock deformity due to malunion in Supracondylar fracture
- Nerve palsy in fracture of shaft
- Non union
- Joint stiffness
- If injury includes elbow joint, carrying angle is decreased / increased. (Normally it is 11° in males & 14° in Females)

FRACTURE OF RADIUS

1. Colle’s fracture (Dinner fork deformity):

The fracture of the distal end of radius with dorsal site is known as “Colle’s fracture”

Common sites:

- Head, neck, shaft & styloid process.
- Mostly fractured site is distal end of radius with dorsal site.

Clinical features - In this fracture deformity looks like a dinner Fork

- Age - above 40 years
- Sex - mostly in females

Treatment:

- General treatment of fracture
- Colle's cast

Complication:

- Stiffness of joint
- Carpal tunnel syndrome
- Malunion
- Subluxation of inferior radio-ulnar joint

2. Smith's fracture:

It is a less common fracture & deformity is reverse of Colle's fracture, that is the distal end of radius with ventral site.
During POP both fractures require observation of fingers. Tight POP produces swelling of fingers & shows contracture of fingers. In such condition where swelling is present, plaster should be cut to reduce pressure.

3. Barton's fracture:

This fracture extends from distal articular surface of the radius to its anterior / posterior cortices.

Treatment:

- Close manipulation
- Plaster cast

ULNAR AND RADIAL SHAFT FRACTURES

Fractures of the radius and ulna are frequently caused by direct blows to the forearm (e.g.: during contact sports, falls, or defensive actions during an assault).
Concomitant dislocations can result from forces transmitted via the interosseous membrane between the radius and ulna.
Isolated midshaft radius or midshaft ulna fractures are common.

Monteggia fractures: are proximal ulnar fractures with a radial head dislocation.
Galeazzi fractures: are distal radial shaft fractures with a dislocation of the distal radioulnar joint.

Signs & Symptoms: Radial and ulnar shaft fractures can cause pain, deformity, ecchymosis, and swelling at the site of injury.

Diagnosis: Anteroposterior and lateral x-rays

Management:

- For isolated radial and ulnar shaft fractures, closed reduction and splinting with outpatient orthopedic follow up
- For Monteggia and Galeazzi injuries, urgent orthopedic consultation and usually open reduction with internal fixation (ORIF)

FRACTURE OF FEMUR

Fracture of neck of femur:

There are 2 types of Fracture

1. Intra Capsular
2. Extra Capsular

Clinical Features

- In this fracture, when patient is in supine position, leg rotates outwards which is a cardinal sign.
- SLR test is highly positive
- Pain in the groin.
- Inability to bear a weight on a limb.
- Swelling
- Inability to move the limb
- Tenderness
- This fracture is known as unsolved fracture. It does not cure early.

Treatment:

- Thomas splint
- Hip spica
- Internal fixation -

1. Multiple cancellous screws
2. Dynamic hip screw

- Multiple Knowle's pin.
- McMurray's osteotomy
- Hemiarthroplasty
- Meyer's procedure

Complications -

- Malunion
- Osteoarthritis
- Avascular necrosis

FRACTURE OF SHAFT OF FEMUR

A Fracture of shaft of femur is usually sustained by severe violence as it may occur in Road Traffic Accident. Shaft of femur requires more force to break. The fracture may occur at any site of shaft & nature of fracture is transverse, oblique, spiral depending upon force of fracture. The patient is having a history of severe violence.

Diagnosis - X-ray & MRI.

Conservative Treatment:

- Thomas splint
- Gallows' Traction
- Interlocking nailing
- Hip spica
- Closed infra-medullary nailing
- Plating

Complications:

- Shock
- Injury to femoral artery
- Infection
- Knee stiffness
- Fat embolism
- Injury to sciatic nerve
- Malunion

FRACTURES OF PATELLA

Patellar fractures are caused directly by trauma or a compressive force, or indirectly as the result of quadriceps contractions or excessive stress to the extensor mechanism. Indirect injuries are commonly associated with tears of the retinaculum and vastus muscles. Patella fractures make up about 1% of all skeletal injuries.

Complications:

- Injuries (sprain/rupture) to ligaments and tendons attached to the patella
- Avascular necrosis
- Post-traumatic arthritis
- Osteochondral damage to patellofemoral joint
- Stiffness
- Non-union, Malunion
- Concomitant injuries

Diagnosis: History, Physical examination, X-ray, MRI for associated injuries

Management:

- In acute cases, local anesthetics can be given to eliminate pain.
- Fracture immobilization with POP cylinder cast or range of motion brace locked in extension (4-6 weeks):

As healing takes place, knee flexion can gradually be increased.
Range of motion brace must be worn until union (on X-rays) and clinical signs of healing (not tender on palpation) are present.

- Crutch walking 6-8 weeks
- Rehabilitation
- Surgical intervention in case of significant displacement with extensor mechanism not intact.

TIBIA & FIBULA FRACTURES

Tibia and fibula fractures are characterized as either low-energy or high-energy.
Low-energy, non-displaced (aligned) fractures, sometimes called toddler's fractures, occur from minor falls and twisting injuries.
High-energy fractures, such as those caused by serious car accidents or major falls, are more common in older children.
Fractures of the tibia and fibula are typically diagnosed through physical examination and X-rays of the lower extremities.

Common Types:

1. Proximal Tibial Fractures:

These fractures occur in the knee end of the tibia and are also called tibial plateau fractures. Depending on the exact location, a proximal tibial fracture may affect the stability of the knee as well as the growth plate.

2. Tibial Shaft Fractures:

This type of fracture takes place in the middle, or shaft (diaphysis), of the tibia. There are three types of tibial shaft fractures:

i. Non-displaced: A fracture where the broken bones remain aligned. This type of fracture is usually seen in children under four. It can be caused by a mildly traumatic event or a twisting injury.

Often, the first symptom is a limp. Examination usually reveals tenderness or swelling at the lower part of the tibia.

Treatment typically involves immobilization in a short- or long-leg cast. The duration is 3-4 weeks for toddlers and 6-10 weeks for older children.

ii. Displaced, non-comminuted: A fracture where the bones are broken in not more than two pieces (non-comminuted) but are not aligned. This is an isolated fracture of the tibia with an intact fibula.

It is the most common tibial shaft fracture. It is caused by a rotational or twisting force such as a sports injury or a fall.

Treatment includes setting the bone without surgery and a long-leg cast with the knee bent. Unstable displaced fractures may require surgery.

iii. Displaced, comminuted: A fracture where the bones are broken in several fragments and are not aligned. This fracture can be caused by high-energy trauma, such as a car accident or being struck by a vehicle.

Treatment includes setting the bone without surgery and a long-leg cast worn for 4-8 weeks. A short-leg weight-bearing cast maybe also be needed in some patients. Unstable fractures may need surgery to maintain alignment.

3. Distal Tibial Fractures:

These fractures occur at the ankle end of the tibia. They are also called tibial plafond fractures. One of the common types in children is the distal tibial metaphyseal fracture. This is a fracture in the metaphysis, the part of tibia before it reaches its widest point.

These fractures are usually transverse (across) or oblique (slanted) breaks in the bone. Distal tibial metaphyseal fractures usually heal well after setting them without surgery and applying a cast. However, there is a risk of full or partial early closure of the growth plate. This may lead to a growth arrest in the form of leg length discrepancy or other deformity.

FRACTURE OF PELVIS

Pelvic fractures can involve the pubic symphysis, innominate bones, acetabulum, sacroiliac joint or sacrum.

They range from minimally displaced stable injuries caused by low energy falls to dramatically displaced and unstable injures that can cause massive hemorrhage. Genitourinary, intestinal, and neurologic injuries may also occur.

Diagnosis is by plain x-rays and usually CT.
Minor stable fractures require only symptomatic treatment.
Unstable fractures and fractures with significant hemorrhage usually require external fixation or open reduction with internal fixation (ORIF).

Etiology:
Most pelvic fractures result from high-energy injuries, most caused by motor vehicle crashes (including motor vehicle-pedestrian collisions) or a fall from a height. Some (e.g.: symphyseal or pubic ramus fractures) result from minor or low-energy injuries (e.g.: falls at home), especially in patients with osteoporosis.
Some pelvic fractures, typically in adolescents with open growth plates, are small avulsion fractures of the anterior or inferior iliac spine or of the ischial tuberosity.

Signs & Symptoms:
Most patients with a pelvic fracture have groin and/or lower back pain. Compression of the pubic symphysis or simultaneous compression of both anterior superior iliac spines is usually painful, particularly in severe fractures, and may indicate instability.
Depending on the severity of the fracture, patients may or may not be able to walk.

Signs of genitourinary and/or gynecologic (usually vaginal) injuries include:

- Blood at the urethral meatus
- Scrotal or perineal hematoma
- Hematuria, Anuria
- A high-riding prostate
- Vaginal bleeding

Intestinal or rectal injuries can cause:

- Abdominal or pelvic pain
- Rectal bleeding
- Later development of peritonitis

Neurologic injuries can cause:

- Weakness or loss of sensation and reflexes in the lower extremities, rectum, or perineum
- Incontinence
- Urinary retention

MODERN CONCEPT OF DISLOCATION

A dislocation is complete separation of the bones that form a joint.
Subluxation is partial separation.
In addition to dislocations, musculoskeletal injuries include the following:

- Fractures
- Ligament sprains, muscle strains, and tendon injuries

Complications:
Serious complications of dislocations are unusual but may threaten life or limb viability or cause permanent limb dysfunction.
Risk of complications is high with open dislocations (which predispose to infection) and with dislocations that disrupt blood vessels, tissue perfusion, and/or nerves.

Acute complications (associated injuries) of dislocations include the following:

- Fractures
- Bleeding
- Vascular injuries
- Nerve injuries
- Infection

Long-term complications of dislocations include the following:

- Instability
- Stiffness and impaired range of motion
- Osteonecrosis
- Osteoarthritis

Evaluation: History and physical examination, X-rays, Sometimes MRI or CT scan

Management:

- Treatment of associated injuries
- Reduction as indicated, splinting, and analgesia
- RICE (rest, ice, compression, and elevation) or PRICE (including protection) as indicated
- Usually, immobilization
- Sometimes surgery

Most joint dislocations can be reduced (returned to the normal anatomic position) without surgery. Occasionally, dislocations cannot be reduced using closed manipulative techniques, and open surgery is required.

SHOULDER DISLOCATIONS

In shoulder (glenohumeral) dislocations, the humeral head separates from the glenoid fossa; displacement is usually anterior.
Shoulder dislocations account for about half of major joint dislocations.

Shoulder dislocations may be

1. Anterior
2. Posterior
3. Inferior

1. Anterior Shoulder Dislocations

Shoulder dislocations are anterior in ≥ 95% of patients; the mechanism is abduction and external rotation.

Associated injuries can include:

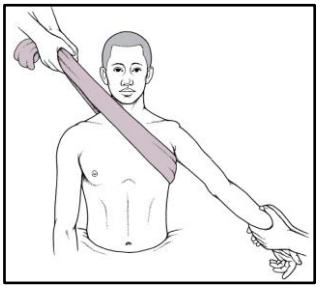
i. Brachial plexus injuries
ii. Rotator cuff tears (particularly in elderly patients)
iii. Fracture of the greater tuberosity
iv. Axillary nerve injury

Shoulder instability and thus recurrent dislocation are common in patients > 30 years.
The acromion is prominent, and the elbow is held slightly out from the side in abduction. The humeral head is displaced anteriorly and inferiorly and cannot be palpated in its usual position. Patients are unwilling to move the arm. They may have motor and sensory deficits (e.g., if the axillary nerve is injured, decreased sensation over the deltoid).

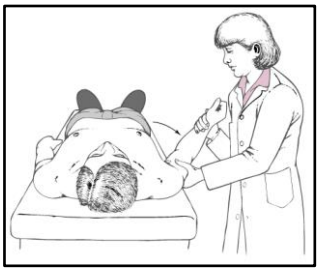
Diagnosis: True anteroposterior and axillary x-rays

Management:
Treatment of anterior shoulder dislocations is usually closed reduction using local anesthesia (intra-articular injection) or procedural sedation.
After reduction, the joint is immobilized immediately with a sling and swathe.
In patients age > 40 years, sling and swathe for 5-7 days and encourage early range of motion to help prevent complications (e.g.: frozen shoulder).

The traction-countertraction technique can be used to reduce anterior shoulder dislocations. For this procedure, the patient lies on a stretcher, and its wheels are locked. One practitioner pulls on a folded sheet wrapped around the patient's chest.
Another practitioner pulls the affected limb down and laterally 45°. After the humerus is free, slight lateral traction on the upper humerus may be needed.



Hennepin technique (external rotation) can be done with the patient supine or seated. The dislocated arm is adducted with the elbow held at 90°. The arm is then externally rotated slowly (e.g.: over 5-10 minutes) to allow time for muscle spasms to resolve. Reduction commonly occurs at 70-110° of external rotation. This technique is effective in about 80-90% of cases.



2. Posterior Shoulder Dislocations

Occasionally, shoulder dislocations are posterior; a commonly missed injury.
It is classically caused by seizures, electric shock, or electroconvulsive therapy done without muscle relaxants.

Deformity may not be obvious. The arm is held adducted and internally rotated. Typically, when the elbow is flexed, passive external rotation is impossible.
If such rotation is impossible, an anteroposterior (AP) shoulder x-ray should be taken.
If it shows no obvious fracture or dislocation, posterior shoulder dislocation should be considered. A clue to the diagnosis on the AP view is the light bulb or ice cream cone sign; the humeral head is internally rotated, and the tuberosities do not project laterally, making the humeral head appear circular.

Bony injuries occur in about 65% of posterior dislocations.

The axillary view or trans-scapular Y view is diagnostic. A posterior dislocation cannot be excluded without a Y view.

Reduction is often possible using longitudinal traction.

3. Inferior Shoulder Dislocations

Inferior dislocations (luxatio erecta) are rare and usually clinically obvious; patients hold their arm over their head (i.e., abducted to almost 180°), usually with the forearm resting on the head.
The arm is shortened; the humeral head is often palpable in the axilla.
The joint capsule is disrupted, and the rotator cuff may be torn.
The brachial artery is injured in < 5% of cases. The axillary nerve or another nerve is usually damaged, but deficits often resolve after reduction.

X-rays are diagnostic.

Reduction is done using traction-countertraction of the abducted arm. Closed reduction is usually successful unless there is a buttonhole deformity (humeral head is trapped in a tear of the inferior capsule); in such cases, open reduction is required.

ELBOW DISLOCATIONS

Most elbow dislocations are posterior and usually result from a fall on an extended arm.

Posterior elbow dislocations are common; it is the 2nd most common joint dislocation after shoulder dislocations.
Associated injuries may include:

- Fractures
- Injuries to the ulnar or median nerve
- Possibly injury to the brachial artery

The joint is usually flexed about 45°, and the olecranon is prominent and posterior to the humeral epicondyles; however, these anatomic relationships may be difficult to determine because of swelling.
Classically, patients with an elbow dislocation present with a shortened forearm and a very prominent olecranon.

X-rays are diagnostic.

For elbow dislocations, reduction is usually with sustained, gentle traction and correction of deformity after patients are sedated and given analgesics.
The following technique is commonly used:

- With the patient supine, the practitioner flexes the elbow to about 90° and supinates the forearm.
- An assistant stabilizes the upper arm against the stretcher.
- The practitioner grasps the wrist and applies slow, steady axial traction to the forearm while keeping the elbow flexed and the forearm supinated.
- Traction is maintained until the dislocation is reduced.

After reduction, the practitioner checks the elbow for stability by fully flexing and extending the elbow while pronating and supinating the forearm.
These movements should be easy after reduction.
After reduction, an x-ray should be taken to make sure no fractures were missed.

The joint is usually immobilized in a splint for up to 1 week until pain and swelling resolve; then active range-of-motion exercises are started, and a sling is worn for 2-3 weeks.

HIP DISLOCATIONS
Most hip dislocations are posterior and result from severe posteriorly directed force to the knee while the knee and hip are flexed (e.g.: against a car dashboard).
Associated injuries include:

- Patella fractures
- Posterior cruciate ligament injuries
- Acetabular and femoral head fractures

In patients with posterior dislocations, the leg is shortened, adducted, and internally rotated.
Anterior dislocations are rare and result in the leg being abducted and externally rotated.

Diagnosis: X-rays

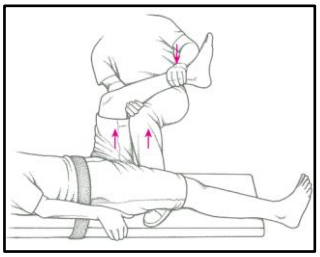
Management:
Treatment of hip dislocations is closed reduction as soon as possible, preferably in ≤ 6 hours; delay increases the risk of osteonecrosis.

The hip can be reduced using one of the following techniques:

- Allis technique
- Captain Morgan technique
- Rocket launcher technique

When any of these techniques is used, the patient requires sedation and muscle relaxation and is in the supine position.

For the Allis technique, the hip is gently flexed to 90°, and vertical traction is applied to the femur; this maneuver may be easiest and safest when the patient is temporarily placed on a rigid backboard that is put on the floor. A strap or brace is used to hold down the patient's hips (providing counter pressure to the vertical traction of the femur).



For the Captain Morgan technique, the patient's hips are held down by a sheet or belt, and the dislocated hip is flexed. Practitioners then place their knee under the patient's knee and lift up while applying vertical traction to the femur. The captain Morgan technique may have a better first-time success rate than the Allis technique.

For the rocket launcher technique, the practitioner stands on the side of the affected hip and faces the patient's feet. The dislocated hip and knee are flexed to 90°. The patient's hips are held down by a sheet or by a second practitioner (to provide countertraction to the pelvis). The practitioner squats, and the patient's knee is placed on the practitioner's shoulder; the practitioner essentially holds the leg like a rocket launcher. The patient's hip is adducted by pressing inward on the knee and is internally rotated by turning the foot out; the practitioner then gently applies traction to the femur by standing from the squat and pulling down on the patient's foot, using the practitioner's shoulder as a fulcrum.

DISEASES OF BONE

2. Diseases of bone: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital anomalies, Osteomyelitis, Cysts, Tumours and Tuberculosis.

CONGENITAL ANOMALIES OF BONE

Many diseases of the skeletal system are congenital in the sense that they become evident at or soon after birth. This does not imply that they all are genetically determined. Most are caused by factors operating during pregnancy, delivery or early infancy. Some of congenital anomalies of bone are

- Osteogenesis imperfecta (OI)
- Club foot
- Spina bifida

Osteogenesis imperfecta (OI):

It is also called as a Brittle bone disease, is a group of genetic disorders that mainly affects the bone.

Etiology:

The underlying mechanism is usually a problem with connective tissue due to lack of type I collagen. This occurs in more than 90% of cases due to mutations in COL1A1 or COL1A2 genes.

Symptoms:

- Bones that bend easily
- Short height and broad skull
- Scoliosis
- Blue tinge to the whites of eye (Blue sclera)
- Loose joints, weak teeth
- Bowed legs and arms

Diagnosis:

- DNA or collagen testing
- X-ray

Treatment:

- There is no cure, maintaining healthy life style (exercise, no smoking) can help prevent fractures
- Care of broken bones
- Pain medication and IV Pamidronate (Bisphosphonates): It reduce bone pain, prevented new vertebral fractures, reshaped previously fractured vertebral bodies and reduced of long bone fractures.
- Braces or wheel chairs
- Kebab treatment: In bend limb (malunited fracture) the bone is fractured in segments all the segments are held with intramedullary nail almost like a kebab.

Club foot / Congenital talipes equino varus (CTEV):
It is a rather vague term which has been used to describe the number of different abnormalities in the shape of foot, but over the years, it has become to be synonymous with the commonest congenital foot deformity, i.e., Congenital talipes equino varus (CTEV).

Definition:
It is a birth defect where one or both feet are rotated inwards and downwards. The affected foot, calf and leg may be smaller than the other.

Causes: Unknown

- Early amniocentesis
- Underdevelopment of bones and muscles
- Constriction of foot by the uterus contributed to occurrence of clubfoot.
- Genetics

Clinical features:

- Foot of child can be dorsiflexed
- Heel is small
- Outer side of foot is gently convex
- Calf muscles are wasted
- Size of foot is smaller
- Foot is in equinus, varus and adduction
- Callosities over lateral aspect of foot

Treatment:
Ponseti method: This involves moving the foot in to improved position followed by casting, which is repeated at weekly intervals. Once the inward bending is improved, the achilles tendon often cut and braces are worn until the age of four. Later minor surgery that corrects a persistent muscle imbalance while avoiding disturbance to the joints of foot.
French method: It involves daily manipulations of the feet along with stretching of feet, followed by tapping in order to maintain the range of motion gains achieved at the end of each session. It requires daily physical therapy for the first two months.
Posteromedial release (PMR) surgery: It is done at the age of 9-12 months.

Spina bifida:
It is a birth defect where there is incomplete closing of the backbone and membranes around the spinal cord.

Causes:

- After having one child with the condition, or if a parent has the condition, there is 4% chance the next child will also be affected.
- Failure of enfolding of nerve elements within the spinal canal during developmental period.
- Not having enough folate (folic acid and Vit B9) in the diet before and during pregnancy.

- Risk factors include certain antiseizure medications, obesity, and poorly controlled diabetes.
- Drinking alcohol often triggers macrocytosis which discards folate.

Types:
l. Spina bifida occulta:

- Commonest one
- It has no or mild signs.
- Here outer part of some vertebrae is not completely closed due to neural arch defect posteriorly.
- It may include hairy patch, dimple, and dark spot or swelling on the back at the site of gap in the spine.
- Sciatica like pain may start appearing at puberty
- Impulse on cough can be seen

2. Spina aperta: It is subdivided in to meningocele and Myelomeningocele.

a. Meningocele:

Typically causes mild problems with sac of fluid present at the gap in the spine.
A single developmental defect allows the meninges to herniate between the vertebrae.
As nervous system remains undamaged, individuals with meningocele are unlikely to suffer long term health problems.

b. Myelomeningocele:

It is also known as open spina bifida, is the most severe form.
Here the unused portion of the spinal column allows the spinal cord to protrude through opening, forming a sac enclosing the spinal elements such as meninges, CSF, and parts of spinal cord and nerve roots.

Clinical features:

- Leg weakens and paralysis
- Orthopedic abnormalities like club foot, hip dislocation, scoliosis etc.
- Bladder and bowel control problems including incontinence, UTI and poor kidney
- dysfunction
- Pressure sore and skin irritations
- Abnormal eye movements

Investigation:

- USG of fetal spine
- Amniocentesis of the mother’s amniotic fluid to test for alpha-fetoprotein (AFP)

Treatment:

- Surgery: Here spinal cord and its nerve roots are put back inside the spine and covered with meninges. In addition, a shunt may be surgically installed to provide a continuous drain for the excess CSF produced in the brain
- Development of limb function

OSTEOMYELITIS

Osteomyelitis is inflammation and destruction of bone caused by bacteria, mycobacteria, or fungi.

Etiology Risk Factors:

- Contiguous spread from infected tissue or an infected prosthetic joint
- Bloodborne organisms (hematogenous osteomyelitis)
- Open wounds (from contaminated open fractures or bone surgery)
- Trauma, ischemia, and foreign bodies predispose to osteomyelitis

Contiguous spread from adjacent infected tissue or open wounds causes about 80% of osteomyelitis; it is often polymicrobial. Staphylococcus aureus is present in ≥ 50% of patients; other common bacteria include streptococci, gram-negative enteric organisms, and anaerobic bacteria.

Osteomyelitis that results from contiguous spread is common in the feet (in patients with diabetes or peripheral vascular disease), at sites where bone was penetrated during trauma or surgery, at sites damaged by radiation therapy, and in bones contiguous to pressure ulcers, such as the hips and sacrum. A sinus, gum, or tooth infection may spread to the skull.

Pathophysiology:

Osteomyelitis tends to occlude local blood vessels, which causes bone necrosis and local spread of infection. Infection may expand through the bone cortex and spread under the periosteum, with formation of subcutaneous abscesses that may drain spontaneously through the skin.

In vertebral osteomyelitis, paravertebral or epidural abscess can develop.

If treatment of acute osteomyelitis is only partially successful, low-grade chronic osteomyelitis develops.

Signs & Symptoms:

- Patients with acute osteomyelitis of peripheral bones usually experience weight loss, fatigue, fever, and localized warmth, swelling, erythema, and tenderness.
- Vertebral osteomyelitis causes localized back pain and tenderness with paravertebral muscle spasm that is unresponsive to conservative treatment. More advanced disease may cause compression of the spinal cord or nerve roots, with radicular pain and extremity weakness or numbness. Patients are often afebrile.
- Chronic osteomyelitis causes intermittent (months to many years) bone pain, tenderness and draining sinuses.

Investigations:

- Erythrocyte sedimentation rate or C-reactive protein
- X-rays, MRI, or radio isotopic bone scanning
- Culture of bone, abscess, or both

Management:

1. Antibiotics:

Antibiotics effective against both gram-positive and gram-negative organisms are given after cultures have been done and until culture results and sensitivities are available.

2. Surgery:

If any constitutional findings (e.g.: fever, malaise, weight loss) persist or if large areas of bone are destroyed, necrotic tissue is debrided surgically.
Surgery may also be needed to drain coexisting paravertebral or epidural abscesses or to stabilize the spine to prevent injury.
Skin or pedicle grafts may be needed to close large surgical defects.
Broad-spectrum antibiotics should be continued for > 3 weeks after surgery. Long-term antibiotic therapy may be needed.

CYSTS

Benign cysts include aneurysmal bone cysts and unicameral bone cysts.
Fibrous dysplasia can also affect bones.

Aneurysmal Bone Cyst:

An aneurysmal bone cyst is an idiopathic expansile lesion that usually develops before age 25 years. This cystic lesion usually occurs in the metaphyseal region of the long bones, but almost any bone may be affected. It tends to grow slowly. A periosteal new bone shell forms around the expansile lesion and is often wider than the original bone. Pain and swelling are common.
Surgical removal of the entire lesion is the most successful treatment; regression after incomplete removal sometimes occurs.
Radiation should be avoided when possible because sarcomas occasionally develop. However, radiation may be the treatment of choice in completely surgically inaccessible vertebral lesions that are compressing the spinal cord.

Unicameral Bone Cyst:

Simple unicameral bone cysts occur in the long bones starting distal to the epiphyseal plate in children. The cyst is fluid-filled. It causes the cortex to thin and predisposes the area to a buckle-like pathologic fracture, which is usually how the cyst is recognized.
Smaller cysts sometimes heal without treatment. A non-displaced fracture through small cysts may be a stimulus for healing. Larger cysts, particularly in children, may require curettage and bone grafting; however, many respond to injections of corticosteroids, demineralized bone matrix, or synthetic bone substitutes.

Fibrous dysplasia:

Fibrous dysplasia involves abnormal bone development during childhood.
It weakens the bones. Fibrous dysplasia may affect one or several bones. Multiple fibrous dysplasias, cutaneous pigmentation, and endocrine abnormalities may be present.

The abnormal bone lesions of fibrous dysplasia commonly stop developing at puberty. They rarely undergo malignant degeneration.

TUMOURS

Benign bone tumours include benign giant cell tumours of bone, chondroblastomas, chondromyxoid fibromas, enchondromas, non-ossifying fibromas, osteoblastomas, osteochondromas, and osteoid osteomas.

Benign Giant Cell Tumours of Bone:

Benign giant cell tumours of bone, which most commonly affect people in their 20s and 30s, occur in the epiphyses and distal epiphyseal- metaphyseal area. These tumours are considered locally aggressive. They continue to enlarge and destroy bone and may eventually erode the rest of the bone and extend into the soft tissues.

They may cause pain. These tumours are notorious for their tendency to recur.

Rarely, a giant cell tumour of bone may metastasize to the lung, even though it remains histologically benign.

Most benign giant cell tumours of bone are treated by radical curettage and packing with methyl methacrylate or by bone graft.

Chondroblastoma:

Chondroblastoma is rare and occurs most commonly among people aged 10 to 20. Arising in the epiphysis, this tumour may continue to grow and destroy bone and the joint.

The tumour must be surgically removed by curettage, and the cavity must be bone grafted.

Local recurrence rate is about 10 to 20%, and recurrent lesions often resolve with repeated bone curettage and bone grafting.

Chondromyxoid fibroma:

Chondromyxoid fibroma is very rare and usually occurs before age 30.

The appearance on imaging studies, which is usually eccentric, sharply circumscribed, lytic, and located near the end of long bones, suggests the diagnosis of chondromyxoid fibroma.

The proximal tibia and iliac wing are typical locations.

Treatment of chondromyxoid fibroma after biopsy is surgical excision or curettage, often use of an adjuvant (e.g., phenol, liquid nitrogen, use of an argon beam), and bone grafting

Enchondroma:

Enchondromas may occur at any age but tend to manifest in people aged 10 to 40. They are usually located within the medullary bone metaphyseal-diaphyseal region.

These tumours are usually asymptomatic but may enlarge and become painful.

An asymptomatic enchondroma does not need biopsy, excision, or other treatment.

Non-ossifying fibroma (fibrous cortical defect, fibroxanthoma):

Non-ossifying fibroma is a benign fibrous lesion of bone that appears as a well-defined lucent cortical lesion on x-ray. A very small non-ossifying fibroma is called a fibrous cortical defect. These lesions are developmental defects in which parts of bone that normally ossify are

instead filled with fibrous tissue. They commonly affect the metaphysis, and the most affected sites are, in order, the distal femur, distal tibia, and proximal tibia. They can progressively enlarge and become multiloculated. Nonossifying fibromas are common among children. Most lesions eventually ossify and undergo remodeling, often resulting in dense, sclerotic areas. However, some lesions enlarge.
Small non-ossifying fibromas are asymptomatic. However, lesions that involve nearly 50% of the bone diameter tend to cause pain and increase the risk of pathologic fracture.
Non-ossifying fibromas are generally first noted incidentally on imaging studies (e.g., after trauma). They typically are radiolucent, single, < 2 cm in diameter, and have an oblong lucent appearance with a well-defined sclerotic border in the cortex. They can also be multiloculated.
Small non-ossifying fibromas require no treatment and limited follow-up. Lesions that cause pain or are close to 50% of the bone diameter may warrant curettage and bone grafting to decrease risk of a pathologic fracture through the lesion.

Osteoblastoma:
Osteoblastoma is a rare benign tumour that consists of tissue histologically similar to that of an osteoid osteoma. Some experts simply consider them large osteoid osteomas (> 2 cm).
Osteoblastoma is much more common among males and appears typically between ages 10 and 35. The tumour develops in the bone of the spine, legs, hands, and feet. It is a slow-growing tumour that destroys normal bone. This tumour is painful.
Treatment of osteoblastoma requires surgery, often curettage and bone grafting.

Osteochondroma:
Osteochondromas (osteocartilaginous exostoses), the most common benign bone tumour, may arise from any bone but tend to occur near the ends of long bones.
These tumours manifest most often in people aged 10 to 20 and may be single or multiple. Multiple osteochondromas tend to run in families.
Secondary malignant chondrosarcoma develops in well under 1% of patients with single osteochondromas, but in about 10% of patients with multiple osteochondromas.
Excision is needed if the tumour is compressing a large nerve or vessel; causes pain (especially when impinging on muscle and creating an inflammatory bursa); disturbs growth; or on imaging study has a destructive appearance, soft-tissue mass, or thickened cartilaginous cap (> 2 cm) suggesting transformation into malignant chondrosarcoma.

Osteoid Osteoma:
Osteoid osteoma, which tends to affect young people (commonly aged 10 to 35), can occur in any bone but is most common in long bones. It can cause pain (usually worse at night, reflecting increased nocturnal prostaglandin-mediated inflammation).
Pain is typically relieved by mild analgesics (particularly aspirin or other non-steroidal anti-inflammatory drugs [NSAIDs]) that target prostaglandins.
Most osteoid osteomas are treated by an interventional musculoskeletal radiologist using percutaneous techniques and anesthesia.

Less often, osteoid osteomas are surgically curetted or excised. Surgical removal may be preferred when the osteoid osteoma is near a nerve or close to the skin (e.g.: spine, hands, feet) because the heat produced by radiofrequency ablation may cause damage.

MALIGNANT TUMOURS OF BONES

Primary bone tumours are much less common than metastatic bone tumours, particularly in adults. Primary bone tumours include multiple myeloma, osteosarcoma, adamantinoma, chondrosarcoma, chordoma, Ewing sarcoma of bone, fibrosarcoma and undifferentiated pleomorphic sarcoma, lymphoma of bone, and malignant giant cell tumour.

Multiple Myeloma:

Multiple myeloma is the most common primary malignant bone tumour but is often considered a marrow cell tumour within the bone rather than a bone tumour because it is of hematopoietic derivation. It occurs mostly in older adults.

Signs & Symptoms:

- Persistent bone pain (especially in the back or thorax), renal failure, and recurring bacterial infections are the most common problems on presentation, but many patients are identified when routine laboratory tests show an elevated total protein level in the blood, proteinuria, or unexplained anemia or renal failure.
- Pathologic fractures (i.e., fractures that occur with minimal or no trauma) are common, and vertebral collapse may lead to spinal cord compression and paraplegia.
- Symptoms of anemia predominate or may be the sole reason for evaluation in some patients, and a few patients have manifestations of hyper viscosity syndrome.
- Peripheral neuropathy, carpal tunnel syndrome, abnormal bleeding, and symptoms of hypercalcemia (polydipsia, dehydration, etc.) are common.
- Patients may also present with renal failure.

Management:

- Chemotherapy for symptomatic patients
- Monoclonal antibodies, including elotuzumab and daratumumab
- Maintenance therapy with corticosteroids, thalidomide, and/or lenalidomide, and proteasome inhibitors, especially oral ixazomib
- Possibly autologous stem cell transplantation
- Possibly radiation therapy to specific symptomatic areas that do not respond to systemic therapy
- Treatment of complications (anemia, hypercalcemia, renal insufficiency, infections, and skeletal lesions; especially those associated with high risk of fracture)

Osteosarcoma (Osteogenic Sarcoma):

Osteosarcoma is the 2nd most common primary bone tumour and is highly malignant.
It is most common among people aged 10-25, although it can occur at any age.
Osteosarcoma produces malignant osteoid (immature bone) from tumour bone cells.

Osteosarcoma usually develops around the knee (distal femur more often than proximal tibia) or in other long bones, particularly the metaphyseal-diaphyseal area, and may metastasize, usually to lung or other bone. Pain and swelling are the usual symptoms.

Signs & Symptoms:
Symptoms of osteosarcoma vary depending on the location of the tumour. Common signs of this type of cancer include:

- Bone pain (in motion, at rest, or when lifting objects)
- Bone fractures
- Swelling, redness
- Limping
- Limitation of motion of joints

Management:
Treatment of osteosarcoma is a combination of chemotherapy and surgery.
After several courses of chemotherapy (over several months), limb-sparing surgery and limb reconstruction can proceed. On occasion, a surgical amputation is done before the start of chemotherapy for a fungating tumour.
More than 85% of patients can be treated with limb-sparing surgery without decreasing the long-term survival rate.
Continuation of chemotherapy after surgery is usually necessary. If there is nearly complete tumour necrosis (about 95%) from preoperative chemotherapy, 5-year survival rate is > 90%.

Metastatic Bone Tumours:
Any cancer may metastasize to bone, but metastases from carcinomas are the most common, particularly those arising in the following areas:
Breast, Lung, Prostate, Kidney, Thyroid, Colon
Prostate cancer in men and breast cancer in women are the most common types of cancers. Lung cancer is the most common cause of cancer death in both sexes. Breast cancer is the most common cancer to metastasize to bone. Any bone may be involved with metastases. Metastatic disease does not commonly spread to bone below the mid forearm or mid-calf, but when it occurs in those sites, it results most often from lung or sometimes kidney cancer.
Metastases manifest as bone pain, although they may remain asymptomatic for some time. Bone metastases may cause symptoms before the primary tumour is suspected or may appear in patients with a known diagnosis of cancer.
Bone biopsy is needed if the primary tumour is unknown after clinical and radiographic evaluation.
Most often, radiation therapy, bisphosphonates, and RANKL inhibitors are used to slow bone destruction.
Pathologic fractures may require treatment with surgery, kyphoplasty, or vertebroplasty.

BONE TUBERCULOSIS

Definition:

It is secondary and local manifestation of general disease. The primary site of infection is bronchial lymph gland from where it spread through blood and reaches bone and joints.

Predisposing factors:

Debility Malnutrition Anaemia Local trauma

Pathogenesis:

Disease may start from metaphysis of bone or directly from Synovial membrane → Finally synovial membrane become thick and edematous → Gradually disease spreads, it involves articular cartilage and erodes it → Gradually as bone get involved, hyperaemia washes away calcium, the bone become soft and rarified → Gradually capsules and ligaments also undergo degeneration leading to dislocation of affected joint → If timely intervention is not done → Tubercular caseation spreads to soft tissue around resulting in cold abscess → Gradually spreads across muscular plane and become subcutaneous → As skin gets perforated, it results in the formation of Tubercular sinus → Some times in TB of joint, cold abscess not seen, that condition is Called dry TB or carrier sicca → Here considerably there is atrophy of muscles around joints.

Features:

- Pain & swelling
- Disability & deformity

Investigation:

- Mantoux test is positive
- X-ray shows increased joint space due to effusion and thickening of synovial membrane
- and bony outline appears irregular.
- Biopsy of synovial membrane for bacteriological examination

Management:

- General
- Rest & Nutritious food

Anti tuberculosis treatment:

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol

Local treatment:

- Immobilized affected part (to subside local symptoms)
- Surgery not required in early stage (stage of synovitis)
- If despite ATT, cold abscess does not subside, it has to be aspirated with wide base needle, pus is sent for cultural and sensitivity. After withdrawn of needle, puncture is sealed.

- If pus is too thick, then abscess must be surgically opened and dead tissue, pus has to be excised.
- When articular cartilage has not destroyed much, after initial immobilization has taken off,
- gradual movement of joint is advised without much bearing of weight.

CRANIO-CEREBRAL INJURIES

3. Cranio-cerebral injuries: Mechanism, Pathology, Classification, Investigations, Complications and primary management.

Traumatic Brain Injury (TBI) is a disruption in the normal function of the brain that can be caused by a blow, bump or jolt to the head, the head suddenly and violently hitting an object or when an object pierces the skull and enters brain tissue.

Signs & Symptoms:
Signs and symptoms of a TBI can be mild, moderate, or severe, depending on the extent of damage to the brain. Mild cases may result in a brief change in mental state or consciousness. Severe cases may result in extended periods of unconsciousness, coma, or even death.
Any of the following may occur:

- Vomiting, Lethargy, Headache, Confusion, Paralysis, Coma, Loss of consciousness
- Dilated pupils, Vision changes (blurred vision or seeing double, unable to tolerate bright light, loss of eye movement, blindness)
- Cerebrospinal fluid (CSF) (clear or blood-tinged) appears from the ears or nose
- Dizziness and balance concerns
- Breathing problems, Slow breathing rate with an increase in BP, Slow pulse rate
- Ringing in the ears or changes in hearing
- Cognitive difficulties, Inappropriate emotional responses, Speech difficulties (slurred speech, inability to understand and/or articulate words)
- Dysphagia, Body numbness or tingling, Droopy eyelid, or facial weakness
- Loss of bowel control or bladder control

Types of Injuries:

1. **Hematoma:**

A hematoma is a blood clot within the brain or on its surface. Hematomas may occur anywhere within the brain. An epidural hematoma is a collection of blood between the dura mater (the protective covering of the brain) and the inside of the skull. A subdural hematoma is a collection of blood between the dura mater and the arachnoid layer, which sits directly on the surface of the brain.

2. **Contusion:**

A cerebral contusion is bruising of brain tissue. When examined under a microscope, cerebral contusions are comparable to bruises in other parts of the body. They consist of areas of injured or swollen brain mixed with blood that has leaked from arteries, veins, or capillaries.

3. **Intracerebral Hemorrhage:**

An intracerebral hemorrhage (ICH) describes bleeding within the brain tissue, and may be related to other brain injuries, especially contusions.

4. **Subarachnoid Hemorrhage:**

Subarachnoid hemorrhage (SAH) is caused by bleeding into the subarachnoid space. It appears as diffuse blood spread thinly over the surface of the brain and commonly after TBI. Most cases of SAH associated with head trauma are mild. Hydrocephalus may result from severe traumatic SAH.

5. **Diffuse Injuries:**

TBIs can produce microscopic changes that do not appear on CT scans and are scattered throughout the brain. This category of injuries, called diffuse brain injury, may occur with or without an associated mass lesion.

6. **Diffuse Axonal Injury:**

Axonal injury refers to impaired function and gradual loss of axons. These long extensions of nerve cells enable them to communicate with each other. If enough axons are harmed in this way, the ability of nerve cells to communicate with each other and to integrate their function may be lost or greatly impaired, possibly leaving a patient with severe disabilities.

7. **Ischemia:**

Another type of diffuse injury is ischemia or insufficient blood supply to certain parts of the brain. A decrease in blood supply to very low levels may occur commonly in a significant number of TBI patients. This is crucial since a brain that has just undergone a traumatic injury is especially sensitive to slight reductions in blood flow. Changes in blood pressure during the first few days after head injury can also have an adverse effect.

8. **Skull Fractures:**

Linear skull fractures or simple breaks or “cracks” in the skull may accompany TBIs. Possible forces, strong enough to cause a skull fracture may damage the underlying brain. Skull fractures may be alarming, if found on a patient evaluation. Fractures at the base of the skull are problematic since they can cause injury to nerves, arteries, or other structures. If the fracture extends into the sinuses, a leakage of cerebrospinal fluid (CSF) from the nose or ears may occur. Depressed skull fractures, in which part of the bone presses on or into the brain, can also occur.

Management of head injury:

1. Airway:

- Mouth gag to prevent tongue falling backwards
- Endotracheal intubation with positive pressure ventilation

2. General assessment of patient:

- To rule out abdominal injuries like splenic rupture
- Haemothorax, may need an intercostals tube
- Long bone fractures
- BP and pulse monitoring

3. Admission is indicated when:
- Definite WO unconsciousness
- Bleeding from ear or nose
- Persisting headache and vomiting
- Fracture of skull
- Alcoholic intoxication

4. Care of unconscious:
- Ryle's tube aspiration or feeding
- Catheter for drainage of urine
- Care of eyes Padding
- Change of position to avoid bed sores.

5. Drugs:
- IV fluids
- Sedation is avoided
- Analgesic and Anticonvulsants like phenytoin or phenobarbitone is started
- Diuretics are given to reduce cerebral oedema, either mannitol or frusemide. It should not be given in case of intracranial hematoma
- Antibiotics like penicillin, ampicillins are given to prevent the onset of meningitis
- Corticosteroids

6. Surgical management:
- Craniotomy
- Cranial flap is raised
- Clot is evacuated
- Followed by applying hitch stitches between dura layer and scalp
- Post op antibiotics, analgesics, anticonvulsants are given.

During surgery, the hair over the affected part of the head is usually shaved. After the scalp incision, the removed bone is extracted in a single piece or flap, then replaced after surgery unless contaminated. The dura mater is carefully cut to reveal the underlying brain. After any hematoma or contusion is removed, the neurosurgeon ensures the area is not bleeding. He/She then closes the dura, replaces the bone and closes the scalp. If the brain is very swollen, some neurosurgeons may decide not to replace the bone until the swelling decreases, which may take up to several weeks. The neurosurgeon may elect to place an ICP monitor or other types of monitors if these were not already in place. The patient is returned to the ICU for observation and additional care.

DISEASES OF SPINE

4. Diseases of Spine: Mechanism, Pathology, Classification, Investigations, Complications and primary management of Tuberculosis, Ankylosing Spondylitis and Disc prolapse.

TUBERCULOSIS OF SPINE

Spine is the most affected bone by tuberculosis infection. It primarily affects thoracic spine; however, tuberculosis of lumbar spine, cervical spine and sacrum are not uncommon.
Primary lesion in spine tuberculosis is infection of the disc and adjacent vertebral bones. This gradually involves vertebral bodies causing destruction and collapse of vertebra.
Occasionally, infection starts in the vertebral body or posterior part of the bony vertebral ring.
Spinal tuberculosis can also involve the spinal cord and meninges.

Incidence:

- Starts between 3-5 years age.
- In the spine, lower thoracic (T 10) or thoracolumbar junctional region is commonest site of affliction.
- The TB bacilli reach the vertebral bodies mainly through blood and lymphatic channels, especially abdominal gland and lymph vessels.

Risk Factors:

- Contact with persons afflicted with tuberculosis
- Poor nutrition
- Living in overcrowded places
- Debilitating medical illnesses
- Immunosuppressant therapy
- HIV and other illnesses suppressing the body's immunity

Varieties:

1. Central:
- Disease mainly affects body of vertebrae, in the forms of diffuse osteomyelitis.
- Gradually bone become soft and gets destructed, resulting in collapse of vertebral bodies.
- Leads to deformity of spine.
2. Metaphyseal:
- Disease involves body of epiphyses.
- Due to same blood supply, lower half of one vertebra and upper half of below vertebra, along with intervening intervertebral disc are involved.

Pathology:
TB settles → Causes tubercular endarteritis → Tubercular follicle developed in devitalized tissue, gradually forming a yellow, grey nodule visible to naked eye → As nodule increase in size, surrounding body lamellae disappear, resulting in collapse of vertebral bodies due to weight of vertebral column → Leads to spinal angular deformity called **Kyphosis/ Gibbus/ Hunchback**

Clinical features:

1. Pain at the site or along with spinal nerves which become worse during standing or jolting
2. Stiffness of joint due to painful movement and muscle spasm
3. Swelling
4. Attitude: In upper thoracic TB, typically military attitude with raised shoulder which is drawn backwards is seen to avoid sudden jerk
5. Tenderness and rigidity of spine
6. Weakness and decreased power in the limb muscles, with altered sensations
7. **Coin test:** Inability to pick up a coin is positive
8. General features like loss of weight, poor appetite, general weakness, lassitude, and evening rise of temperature

Investigation:

1. X-ray of spine shows
- Narrowing of disc space
- Soft tissue shadow of cold abscess
2. MRI (Ideal) or CT scan of spine

Treatment:

- Anti- tubercular drugs
- Rest with plaster jacket (SPICA)
- Drainage of cold abscess.

ANKYLOSING SPONDYLITIS
This is a chronic progressive inflammatory disease of the sacroiliac joints and axial skeleton. It is associated with HLA-B27 gene.

Incidence:
M: F = 10:1
Common in 15-35 years of age

Types:

1. True (due to intra articular lesion):

a. Fibrous: After damage of articular cartilage, fibrosis occurs between two articular surfaces. This limits the movements of joints, though not completely.

b. Bony: Due to major damage to articular cartilage, bony trabeculae are laid down between articular surfaces of bone. No movement is possible at joint.
2. False (Due to extra articular lesion): Lesion of extra articular results in limitation of movement of joints. Since joint itself is not directly involved it is called as false ankylosis.
   E.g., Contracture skin and fascia secondary to burn.

Pathology:
The initial inflammation of joints is followed by synovitis, arthritis, and cartilage destruction, fibrous and later bony ankylosis. The joints commonly affected are sacroiliac joints, spine, hip, and knee and manubrium sterni.

Clinical features:
- Malaise, fatigue, loss of weight & chest pain
- Early morning stiffness and pain in back
- Patient has a stiff spine like bamboo and loss of movements.
- Pain is worst at night or early morning which reduces after walking and some exercise.
- Tenderness at sacro-ileac joint
- SLR Test: Ask the patient to lift leg up with extended knee, will cause pain at sacro-ileac joint.

X-ray of spine: Bamboo spine appears with loss of lumbar lordosis.

Treatment:
1. General measures:
- Patient and family education
- Avoid smoking
- Regular exercise especially swimming
- Physiotherapy and spine exercise with deep breathing

2. Conservative therapy:
- Rest
- Drugs like phenyl butazone
- Radiotherapy as palliative measure

3. Surgery:
- Spinal osteotomy to correct spine
- Total hip replacement for hip ankylosis
- Total knee replacement of knee ankylosis

DISC PROLAPSE / SLIPPED DISC / INTERVERTEBRAL DISC PROLAPSE
It is protrusion of pulpy inner material of an intervertebral disc through the fibrous outer coat causing pressure on adjoining nerve roots, ligaments etc.

Causes:

1. External: Trauma (80%) which is common feature in athletes, workers, porters, trekkers etc.
2. Internal: Overweight, less toned muscle, thin body, hyper function of back bone, abnormal growth in vertebrae give rise to pathogenesis like lumbar strain, nerve irritation, lumbar radiculopathy, bony encroachment etc.
3. Physical illness and emotional stress
4. Swelling of pulp matter due to absorption of fluid, so that this bulges the fibrous sheath or burst out.
5. Disc degeneration (15%): Disc loses its elasticity and decreases fluid content. The weakened disc cannot resist body weight and is liable to herniated.

Common site: Disc between L4, L5 and L5, S1 & Disc between C5, C6 and C6, C7

Feature:

1. Low backache in distribution of root which gets aggravated by straining, coughing, twisting, stooping.
2. Pain radiates along the distribution of the nerve with tingling and numbness
3. Tenderness over interspinous ligament or lateral to spinous process over affected intervertebral space.
4. Lumbar scoliosis is significant
5. Restricted forward flexion but free lateral flexion
6. Wasting in the muscle with blunting of sensation, with absence knee and ankle jerk
7. Loss of bladder sensibility and retention of urine.
8. Positive SLR on affected side
9. Lasegue test (Modified SLR test): Hip is lifted to 90 degree with the knee bent. Knee is then gradually extended by examiner, if nerve stretch is present, it will not be possible to do. So, patient will feel pain in back of thigh or leg.

Investigation:

X-ray of spine	CT scan
Myelography or radiculography	MRI spine is choice of investigation

Treatment:

1. Posture training: Bed rest to align the vertebral column and minimize manipulation to recreate the tone of muscle and ligaments for 2-3 weeks
2. Hot fomentation and gentle exercise
3. Spinal jacket
4. Continuous or intermittent traction
5. Analgesics and muscle relaxants
6. Intradiscal injection of the chymopapain enzyme which dissolves the fibro-cartilaginous tissue and nucleus pulposus
7. TENS: Transcutaneous Electrical Nerve Stimulation
8. Surgery: Laminectomy or Discectomy.

DISEASES OF BREAST

5. Diseases of breast: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Sthana Vidradhi - Breast abscess and Sthana Arbuda - Breast tumours.

STANA VIDRADHI

There is no direct reference for Stanavidradhi in the classical Āyurvedic textbooks.
The following can be considered as a description of Stanavidradhi.

Saṁprāpti: The Doṣas which are vitiated due to the causative factors similar as for Bāhya Vidradhi, reach the Stanavaha srotas leading to Stanavidradhi lakṣhaṇa.

Nidana:

- Paryuṣita (stale), Ati Uṣṇa Rūkṣa Shuṣka Vidāhi Āhara
- Sleeping on an uneven bed
- Abnormal actions
- Rakta prakopaka nidāna (Ātapa, Agni, Kṣāra, etc.)

Bheda:

1. Vātaja
2. Pittaja
3. Kaphaja
4. Sannipātika
5. Raktaja
6. Abhigātaja

Lakshana:

1. Vātaja:

- Atyartha Vedanā, Paruṣa, Bhrama, Ānāha, Spandana
- Shyāva Aruṇa varṇa
- Viṣamasaṁsthiti (uneven swelling; increases & decreases)
- Chitra Utthāna Pāka (develops and suppurates in a variable manner)
- Tanu srāva

2) Pittaja

- Jvara, Dāha, Tṛṣṇā, Moha
- Rakta Tāmra Kṛṣṇa varṇa
- Kṣipra Utthāna Pāka (develops and suppurates quickly)
- Pīta srāva

3) Kaphaja

- Shīta, Stabdha, Alpavedanā, Kaṇḍū, Hṛllāsa, Jṛmbha, Aruchi, Gurutva
- Pīta Shveta varṇa
- Chira Utthāna Pāka (develops and suppurates slowly)
- Pāṇḍu srāva

4) Sannipātika
- Vātaja Pittaja Kaphaja Lakṣaṇa

5) Raktaja
- Kṛṣṇa Sphoṭa
- Shyāva varṇa
- Tīvra Dāha, Ruja, Jvara
- Pittaja Lakṣaṇa

6) Abhigātaja
Due to injury and uncongenial diet, heat generation occurs which spreads due to Vāyu. Pitta gets aggravated and withholds Rakta.
- Pittaja & Raktaja Lakṣaṇa

Chikitsā: Vraṇa & Shopha Chikitsā, Pāṭana, Jalaukāvacharaṇa

BREAST ABSCESS
Breast abscess is an acute inflammation / infection with collection of pus within the breast tissue. It may be a complication of infective mastitis.

Risk Factors:
- Lactation period, Breastfeeding
- Crack/fissure in the nipple
- Retracted nipple
- Improper cleaning of nipple
- Infection from nasopharynx of the baby

Types:
1. Lactational Breast Abscess
2. Non-lactational Breast Abscess

1. Lactational Breast Abscess
- Breast abscess in lactating women.
- Develops within first 6 weeks of breastfeeding.
- Causative organism: Staphylococcus aureus, Streptococci
- Cause: Drainage of milk is affected which leads to stagnation of milk and further to infection.
- Symptoms: Pain, Swelling, Tenderness, Cracked nipple

2. Non-lactational Breast Abscess
- Breast abscess in non-lactating women.
- Develops commonly around the age of 32.
- Sub-types:

a. Central infection
b. Peripheral infection

General Clinical Features:

1. Stage of Cellulitis
- Severe throbbing pain in the affected breast
- Breast is red, tense, tender, warm to touch with browny induration
2. Stage of Abscess
- High grade fever
- Chills
- Rigors
3. Untreated Abscess

Abscess may rupture through the skin, resulting in necrosis of the breast skin, ulceration and purulent discharge from the nipple.

Investigations:

- Breast ultrasound
- Diagnostic Needle Aspiration
- Nanogram

Management:

1. Advice
- Child should not be fed from the affected site
- If both breasts are affected, the breast should be emptied and the milk is boiled for 5 minutes. It can be given to the child when adequately warm.
- Nipple moisturizer for cracked nipple
- Comfortable breast support

2. Stage of Cellulitis
- Antibiotics: Metronidazole 5-7 days, Cephalosporines for 7-10 days
- Anti-inflammatory drugs such as aceclofenac
- Anti-pyretics like paracetamol

3. Stage of Abscess
- If pain and tenderness does not subside within 48 hrs after antibiotics, I & D might be indicated.
- Cone excision of duct in non-lactational breast abscess

I & D of breast abscess:

- Position: supine
- Anaesthesia: GA
- Part preparation: Painting & Drapping

Incision:
Incision in radial direction (Better cosmetic) about 5-6 cms is made over swelling where there is maximum tenderness.

Procedure:

1. Step 1

Prepare the surface of the abscess and surrounding skin with povidone-iodine or chlorhexidine solution and drape the abscess with sterile towels. Perform a field block by infiltrating local anesthetic around and under the tissue surrounding abscess.

The environment of an abscess is acidic, which may cause local anesthetics to lose effectiveness. Use an appropriate amount of anesthetic, and allow adequate time for anesthetic effect.

Avoid injecting into the abscess cavity, because it may rupture downward into the underlying tissues or upward toward the provider.

2. Step 2

Make a linear incision with a no. 11 or 15 blade into the abscess.

- The most common cause of abscess reoccurrence is an incision not wide enough to promote adequate drainage.
- Inform the patient before the procedure that scarring is possible.
- Contents of the abscess may project upward and outward when it is incised, especially if local anesthetic was inadvertently injected into (instead of around) the abscess. Use personal protective equipment to avoid self-contamination.

3. Step 3

Allow purulent material from the abscess to drain. Gently probe the abscess with the curved hemostats to break up loculations. Attempt to manually express purulent material from the abscess.

4. Step 4

Insert packing material into the abscess with hemostats or forceps. Dress the wound with sterile gauze and tape.

Closure:

- Do not close incision, if infection is very severe.
- Otherwise wound is sutured, drainage is removed after 48hrs or when discharge is minimal.

Post-op:

- NBM for 6hrs
- Antibiotics
- Dressing should be changed every day.

Complications:

- Inadequate anesthesia
- Pain during and after the procedure
- Bleeding
- Reoccurrence of abscess formation
- Septic thrombophlebitis
- Necrotizing fasciitis
- Fistula formation
- Damage to nerves and vessels
- Scarring

STANĀRBUDA

There is no direct reference for Stanārbuda in the classical Ayurvedic textbooks.
General description of Arbuda which develops in Stana pradesha should be followed.
Stanārbuda is generally compared to breast cancer; so, the following can be considered for Stanārbuda:

Saṁprāpti Ghaṭaka:
Doṣa → Tridoṣaja, Kapha pradhāna
Dūshyā → Rakta, Māṁsa, Meda
Srotāṇi → Raktavaha, Māṁsavaha, Medovaha, Stanavaha
Adhiṣthāna → Stana

Raktārbuda:

Lakṣaṇa

- Māṁsa-aṅkura (muscular sprout)
- Ashu vṛddha (fast growing)
- Praduṣta Raktasrāva (vitiated blood discharge)
- Chirapāka / Apāka (slow ripening with pus / No ripening with pus)

Upadrava: Raktakṣaya, Pāṇḍu
Sādhyāsādhyatā: Asādhya

Māṁsārbuda:

Lakṣaṇa:

- Avedana (painless)
- Snigdha (unctuous)
- Vaivarṇya (discolouration)
- Apāka (no suppuration / pus)
- Ashmopa (stony hard)
- Aprachalpa (immovable)

Nidana: Atimamsa bhojana, Mamsa dusti
Sadhya-Asadhyata: Asadhya

Adhyarbuda: One Arbuda develops over a previous one.

Dvirarbuda: Two Arbuda are growing simultaneously.

BREAST CARCINOMA

Breast carcinoma is a malignant proliferation of epithelial cells lining the duct / lobules of the breast.
It is the second most common cause of cancer death in woman (after lung cancer).

Aetiology:

- More common in developed western companies.
- Second most common carcinoma in females
- More common in 40–60-year age group
- Can be familial
- More common in nulliparous woman.
- Attaining early menarche and late menopause have high risk of breast malignancy.
- Early child bearing and breast feeding reduces the chances of malignancy. Early 1st child birth reduces the risk; late first child birth after 35 years increases the risk.
- It is more common in obese individuals.

Presentation:
Non-tender lump (upper outer quadrant)
Skin changes → Dimpling, ulceration, Peau d'orange (skin of an orange)
Nipple changes → Inversion, ulceration, distortion
Non-milky discharge; sometimes bloody
Palpable axillary / cervical lymph nodes

Types: - 2

1. In-Situ (non-spreading)
a. DCIS Ductal Carcinoma In-Situ
b. LCIS Lobular Carcinoma In-Situ

2. Invasive (spreading)
a. IDC Invasive Ductal Carcinoma (most common ~80%)
b. ILC Invasive Lobular Carcinoma (second most common)

Paget's Disease of the Nipple:
Paget's Disease of the Nipple is a rare form of breast cancer in which cancer cells collect in and around the nipple.

Diagnosis:
Biopsy, Blood cell counts, Blood marker test, Bone scan, Breast MRI, CT scan, PET scan, Chest X-ray, Ductal lavage, Mammograms

Treatment:
Surgery, Chemotherapy, Radiation therapy, Hormonal therapy, biological therapy

BREAST TUMOURS
Types of Breast Lumps:

1. Fibrocystic Breast Disease / Fibroadenosis
2. Fibroadenoma
3. Breast Cyst
4. Phyllode Tumour
5. Lipoma Breast
6. Mastitis
7. Breast Cancer
8. Papilloma
9. Hamartoma
10. Fat Necrosis

1) Fibrocystic Breast Disease / Fibroadenosis:

- Most common benign lesion of the breast.
- Occurrence: 25-45 years of age
- Cause: Fluctuation of hormones during normal menstrual cycle.
- Symptoms: Breast pain -> start prior to menstrual cycle; it may be dull, severe, intermittent, or continuous; Tenderness
- Features: Round & smooth borders of the breast; Rubbery, movable mass

2) Fibroadenoma:

- Most common benign tumour of the breast.
- Synonym: Breast mice
- Occurrence: 20-35 years of age
- Cause: Result of excess growth of glands and connective tissue.
- Symptoms: Asymptomatic
- Features: Well rounded, smooth & solid; Rubbery, movable mass

3) Breast Cyst:

- Fluid filled sac within the breast; normally resolves by itself after menopause.
- Occurrence: > 35 years of age
- Cause: Result of excess growth of glands and connective tissue.
- Symptoms: Breast pain or tenderness at the area of the breast lump
- Features: Smooth, easily movable, round or oval lump with distinct edges

4) Phyllode Tumour:

A benign tumour of the breast which grows rapidly and attains large size. It contains cystic spaces with leaf-like projections, hence the name phyllode.

- Occurrence: 30-50 years of age
- Sings &Symptoms: Unilateral, Smooth, soft, non-tender, necrosis of skin due to pressure, Skin over the breast is stretched, shiny and reddish with dilated veins, Tumour is not fixed to the skin, muscle wall, or chest cavity, Discharge of serous fluid

5) Lipoma Breast:

- A benign tumour of the breast composed of overgrowth of normal fat cells.
- Lipomas of the breast usually grow slowly and increase in size over a period of long time.
- Painless, palpable, mobile, soft

6) Mastitis:

Mastitis is the inflammation of breast tissue which may or may not be associated with bacterial infection.

7) Breast Carcinoma:
Breast carcinoma is a malignant proliferation of epithelial cells lining the duct / lobules of the breast.

8) Papilloma:

- A small solid benign tumour with a clear-cut border that projects above the surrounding tissue (e.g., wart).
- Intraductal papilloma is a mall benign wart-like growth in a milk duct of the breast; usually painless.

9) Hamartoma:
Hamartoma, also known as fibroadenolipoma, is a rare, benign formation that can develop in various organs, including the breast. It presents clinically as a soft, mobile nodular lesion and is generally asymptomatic.

10) Fat Necrosis:
A lump of dead or damaged breast tissue that sometimes appears after breast surgery, radiation, or other forms of trauma.

Stana Vṛddhi / Gynecomastia
Gynecomastia is an enlargement or swelling of breast tissue in males. It can affect one or both breasts.

Gynecomastia is most caused by an imbalance between the hormones estrogen and testosterone. Estrogen controls female traits, including breast growth. Testosterone controls male traits, such as muscle mass and body hair. Although each of these hormones produces the usual traits seen in males and females, males produce a small amount of estrogen and females produce a small amount of testosterone. Male estrogen levels that are too high or are out of balance with testosterone levels cause gynecomastia.

Gynecomastia occurs naturally at different times in a male's life. These phases are:

- After birth. Newborn boys are still under the effects of the estrogen they received from their mothers while developing in the womb. More than half of newborn males are born with enlarged breasts. The gynecomastia goes away within two to three weeks after birth.
- At puberty. Hormone levels are changing during puberty (usually 12 to 14 years of age). Breast enlargement usually goes away six months to two years after the start of puberty.
- At mid-life and beyond. Breast enlargement often peaks in men between the ages of 50 and 80. About one in four men in this age range have breast enlargement

- There are other conditions that can cause gynecomastia. These include:
  Obesity, Lack of proper nutrition, Tumours in the testicles or adrenal glands, Liver disease, Hyperthyroidism, Hypoandrogenism, Hypogonadism, Kidney failure

DISEASES OF CHEST

6. Diseases of chest: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Chest injury, Pleural effusion, Pleurisy and Tumours.

CHEST INJURY / THORACIC TRAUMA

Chest injuries are injuries to the chest wall (the bones, skin, fat, and muscles protecting the lungs and heart) or any of the organs inside the chest.

Causes:

- Road traffic accidents
- Industrial accidents
- Assault with blunt objects
- Fall from a height
- Crush injuries, stab injuries
- Gunshot injury

Pathophysiology:

Most morbidity and mortality due to chest trauma occurs because injuries interfere with respiration, circulation, or both.

Respiration can be compromised by

i. Direct damage to the lungs or airways
ii. Altered mechanics of breathing

Injuries that directly damage the lung or airways include pulmonary contusion and tracheobronchial disruption. Injuries that alter the mechanics of breathing include hemothorax, pneumothorax, and flail chest. Injury to the lung, tracheobronchial tree, or rarely esophagus may allow air to enter the soft tissues of the chest and/or neck (subcutaneous emphysema) or mediastinum (pneumomediastinum). This air itself rarely has significant physiologic consequence; the underlying injury is the problem. Tension pneumothorax impairs respiration as well as circulation.

Circulation can be impaired by

i. Bleeding
ii. Decreased venous return
iii. Direct cardiac injury

Bleeding, as occurs in hemothorax, can be massive, causing shock (respiration is also impaired if hemothorax is large). Decreased venous return impairs cardiac filling, causing hypotension. Decreased venous return can occur due to increased intrathoracic pressure in tension pneumothorax or to increased intrapericardial pressure in cardiac tamponade. Heart failure and/or conduction abnormalities can result from blunt cardiac injury that damages the myocardium or the heart valves.

Signs & Symptoms:

- Pain, which usually worsens with breathing if the chest wall is injured, and sometimes shortness of breath.

- Common findings include chest tenderness, ecchymoses, and respiratory distress; hypotension or shock may be present.
- Neck vein distension can occur in tension pneumothorax or cardiac tamponade if patients have sufficient intravascular volume.
- Decreased breath sounds can result from pneumothorax or hemothorax; percussion over the affected areas is dull with hemothorax and hyper resonant with pneumothorax.
- The trachea can deviate away from the side of a tension pneumothorax.
- In flail chest, a segment of the chest wall moves paradoxically - that is, in the opposite direction from the rest of the chest wall (outward during expiration and inward during inspiration); the flail segment is often palpable.

Diagnosis:

- Clinical evaluation
- Chest x-ray
- Sometimes other imaging studies such as CT scan, USG, aortic imaging studies

Management:

- Supportive care
- Treatment of specific injuries

- Immediately life-threatening injuries are treated at the bedside at the time of diagnosis:
- Respiratory distress with suspected tension pneumothorax → Needle decompression
- Respiratory distress or shock with decreased breath sounds and suspected hemothorax → Tube thoracostomy.
- Respiratory distress with suspected open pneumothorax → Partially occlusive dressing followed by tube thoracostomy.
- Respiratory distress with suspected flail chest → Mechanical ventilation
- Shock with suspected cardiac tamponade → Pericardiocentesis
- Suspected hypovolemic shock → Fluid resuscitation
- Immediate resuscitative thoracotomy can be considered for trauma victims if the clinician is proficient in the procedure and the patient has one of the following indications:
- Penetrating thoracic injury with a need for cardiopulmonary resuscitation (CPR) of < 15 minutes
- Penetrating non-thoracic trauma with a need for CPR of < 5 minutes
- Blunt trauma with a need for CPR of < 10 minutes
- Persistent systolic blood pressure of < 60 mmHg due to suspected cardiac tamponade, hemorrhage, or air embolism

PLEURAL EFFUSION

The disturbance in the balance of secretion and absorption may result in accumulation of fluid in pleural cavity, which is known as pleural effusion. This fluid is exudates by the visceral pleura and is absorbed by the parietal pleura.

Types:

1. Transudates: It develops when there is either excessive production of pleural fluid or the re-absorption capacity is reduced. It is clear with faint yellow tinge and no odour. Most common cause is congestive cardiac failure. Here protein content < 3g/100 ml
2. Exudates: It occurs when the pleura is damaged by trauma, infection, or malignancy. Here protein content > 3g/100 ml caused by block blood; vessels or lymph vessels.

Different forms:

- Hydrothorax (Serous fluid)
- Pyothorax (Pus)
- Chylothorax (Chyle)
- Hemothorax (Blood)
- Pneumothorax (Air)

Note: Hemorrhagic pleural effusion is not included in transudate or exudates group and it is usually caused by neoplastic invasion of pleura, pulmonary infarction, TB or unrevealed trauma.

Causes:

- Trauma
- Bacterial pneumonia
- Tuberculosis
- Congestive cardiac failure
- Pulmonary infarction

Clinical features:

- Breathlessness
- Decreased movement of chest on affected side
- Absent Or diminished breath sounds
- Percussion: Stony dullness
- Auscultation: Absent or decreased vocal resonance and fremitus
- Massive effusion, the mediastinum is shifted to opposite side causing compression to opposite lung.

Investigations:

- X-ray of chest PA view: Shows area of whiteness
- Pleural aspiration

Treatment:

- Treat the cause
- Antibiotics
- Thoracocentesis

PLEURISY

Definition:

Pleurisy, also known as pleuritis, is inflammation of the membranes that surrounds the lungs and lines the chest cavity (pleurae), The underlying mechanism involves the rubbing together of pleurae instead of smooth gliding. It is often associated with pleural effusion.

Causes:

1. Viral infections like coxsackie B virus, HRSV, CMV, adenovirus, influenza is most common
2. Bacterial infections associated with pneumonia and TB
3. Chest injuries
4. Post op heart surgery especially coronary artery bypass grafting
5. Cardiac problems like ischemia, pericarditis
6. Lung cancer and lymphoma
7. Pneumothorax

Clinical features:

1. Sudden sharp, stabbing, burning or dull pain in the right or left side of the chest during breathing, especially when one inhales and exhales.
2. It feels worst with deep breathing, coughing, sneezing, or laughing
3. Pleural friction rub: When doctor uses stethoscope to listen to the breathing, inflamed layers of pleurae make a rough, scratchy sound as they rub against each other during breathing.
4. Other symptoms may include shortness of breath, cough, fever or weight loss, depending upon underlying cause.

Investigations:

1. Chest x-ray: It may show, air or fluid in pleural space or underlying cause like pneumonia, fractured rib
2. Blood tests: To detect bacterial or viral infections

Treatment:

1. Treat the cause
2. Bed rest
3. Anti-inflammatory agents to control pain
4. Codeine based cough syrups to control the cough
5. Thoracocentesis to drain fluid, air, or blood from pleural space

CHEST WALL TUMOURS

Chest wall tumours are benign or malignant tumours that can interfere with pulmonary function. Primary chest wall tumours account for 5% of all thoracic tumours and 1 to 2% of all primary tumours. Almost half are benign.

The most common benign chest wall tumours are

1. Osteochondroma
2. Chondroma
3. Fibrous dysplasia

A wide range of malignant chest wall tumours exist. Over half are metastases from distant organs or direct invasions from adjacent structures (breast, lung, pleura, mediastinum).
The most common malignant primary tumours arising from the chest wall are sarcomas. About 45% originate from soft tissue, and 55% originate from cartilaginous tissue or bone. Chondrosarcomas are the most common primary chest wall sarcoma and arise from the anterior tract of ribs and less commonly from the sternum, scapula, or clavicle. Bone tumours include osteosarcoma and small-cell malignant tumours.

The most common soft-tissue primary malignant tumours are fibrosarcomas (desmoids, neurofibrosarcomas) and malignant fibrous histiocytomas.

Signs & Symptoms:

- Soft-tissue chest wall tumours often manifest as a localized mass without other symptoms.
- Some patients have fever.
- Patients usually do not have pain until the tumour is advanced.
- In contrast, primary cartilaginous and bone tumours are often painful.

Diagnosis:

- Chest X-ray, CT scan, MRI, Positron Emission Tomography (PET)
- Biopsy & Histologic evaluation

Management:

- Surgery
- Sometimes combination chemotherapy, radiation therapy, and surgery

Most chest wall tumours are treated with surgical resection and reconstruction.
Reconstruction often uses a combination of myocutaneous flaps and prosthetic materials.
In cases of multiple myeloma or isolated plasmacytoma, chemotherapy and radiation therapy should be the primary therapy.
Small-cell malignant tumours such as Ewing sarcoma and A skin tumour should be treated with a multimodality approach, combining chemotherapy, radiation therapy, and surgery.

LUNG CARCINOMA

Lung carcinoma is the leading cause of cancer-related death worldwide. About 85% of cases are related to cigarette smoking.

Etiology & Risk Factors:

- Cigarette smoking; the risk of cancer differs by age, smoking intensity, and smoking duration.
- The risk of lung cancer increases with combined exposure to toxins and cigarette smoking.

- Other confirmed or possible risk factors include air pollution, marijuana smoking, exposure to cigar smoke and second-hand cigarette smoke, and exposure to carcinogens (e.g.: asbestos, radiation, radon, arsenic, chromates, nickel, chloromethyl ethers, polycyclic aromatic hydrocarbons, mustard gas, coke-oven emissions, primitive cooking, heating huts).
- Chronic inflammation increases the risk of many cancers, including lung cancer. For example, COPD (chronic obstructive pulmonary disease), alpha-1 antitrypsin deficiency, and pulmonary fibrosis increase susceptibility to lung cancer.
- People whose lungs are scarred by other lung diseases (e.g.: tuberculosis) are potentially at increased risk of lung cancer.

Classification:

1. Small cell lung cancer (SCLC), about 15% of cases

SCLC is highly aggressive and almost always occurs in smokers. It is rapidly growing, and roughly 80% of patients have metastatic disease at the time of diagnosis.

2. Non–small cell lung cancer (NSCLC), about 85% of cases

The clinical behavior of NSCLC is more variable and depends on histologic type, but about 40% of patients will have metastatic disease outside of the chest at the time of diagnosis.

Signs & Symptoms:
About 25% of lung cancers are asymptomatic and are detected incidentally with chest imaging. Symptoms and signs can result from local tumour progression, regional spread, or distant metastases.

Local tumour can cause cough and, less commonly, dyspnoea due to airway obstruction, post obstructive atelectasis or pneumonia, and parenchymal loss due to lymphangitic spread.
Fever may occur with post obstructive pneumonia.
Up to half of patients report vague or localized chest pain.

Regional spread of tumour may cause pleuritic chest pain or dyspnoea due to development of a pleural effusion, hoarseness due to tumour encroachment on the recurrent laryngeal nerve, and dyspnoea and hypoxia from diaphragmatic paralysis due to involvement of the phrenic nerve.

Superior vena cava (SVC) syndrome results from compression or invasion of the SVC and can cause headache or a sensation of head fullness, facial or upper-extremity swelling, breathlessness when supine, dilated veins in the neck, face, and upper trunk, and facial and truncal flushing (plethora).
Metastases eventually cause symptoms that vary by location. Metastases can spread to the:

- Liver, causing pain, nausea, early satiety, and ultimately hepatic insufficiency
- Brain, causing behavioral changes, confusion, aphasia, seizures, paresis or paralysis, nausea and vomiting, and ultimately coma and death
- Bones, causing severe pain and pathologic fractures
- Adrenal glands, rarely causing adrenal insufficiency

Investigations:

- Chest x-ray
- CT or combined positron emission tomography (PET)-CT
- Cytopathology examination of pleural fluid or sputum
- Usually bronchoscopy-guided biopsy and core biopsy
- Sometimes open lung biopsy

Management:

- Surgery (depending on cell type and stage)
- Chemotherapy
- Radiation therapy
- Immunotherapy

DISEASES OF ESOPHAGUS

7. Diseases of esophagus: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital anomalies, Oesophagitis, Varices, Ulcer and Tumours.

CONGENITAL ANOMALIES OF ESOPHAGUS

The most common congenital anomaly of the esophagus is esophageal atresia, with or without tracheoesophageal fistula.

ESOPHAGEAL ATRESIA

Esophageal atresia is incomplete or abnormal formation of the esophagus, frequently associated with tracheoesophageal fistula.
Esophageal atresia is the most common gastrointestinal (GI) atresia. The estimated incidence is 1 in 3500 live births. Other congenital malformations are present in up to 50% of cases.
Two syndromes are associated with esophageal atresia:

1. VACTERL (Vertebral anomalies, Anal atresia, Cardiac malformations, Tracheoesophageal fistula, Esophageal atresia, Renal anomalies and radial aplasia, and Limb anomalies)
2. CHARGE (Coloboma, Heart defects, Atresia of the choanae, Retardation of mental and/or physical development, Genital hypoplasia, and Ear abnormalities)

Signs & Symptoms:
Characteristic signs are:

- Excessive secretions
- Coughing and cyanosis after attempts at feeding
- Aspiration pneumonia
- Esophageal atresia with a distal fistula leads to abdominal distension because, as the infant cries, air from the trachea is forced through the fistula into the lower esophagus and stomach.

Types:

1. Atresia with distal fistula (86%)
2. Isolated esophageal atresia (8%)
3. Isolated tracheoesophageal fistula (4%)
4. Atresia with double fistula (1%)
5. Atresia with proximal fistula (1%)

Investigations:

- Prenatal: Ultrasonography
- Postnatal: Nasogastric tube (NGT) or orogastric tube placement and x-ray

Management: Surgical repair
Preoperative management aims to get the infant into optimal condition for surgery and prevent aspiration pneumonia, which makes surgical correction more hazardous. Oral feedings are withheld. Continuous suction with an NGT in the upper esophageal pouch prevents aspiration of swallowed saliva. The infant should be positioned prone with the head elevated 30-40° and with the right side down to facilitate gastric emptying and minimize the risk of aspirating gastric acid through the fistula. If definitive repair must be deferred because of extreme prematurity, aspiration pneumonia, or other congenital malformations, a gastrostomy tube is placed to decompress the stomach. Suction through the gastrostomy tube then reduces the risk that gastric contents will reflux through the fistula into the tracheobronchial tree.

Surgical repair: When the infant's condition is stable, extra pleural surgical repair of the esophageal atresia and closure of the tracheoesophageal fistula can be done.
If a fistula is noted, it needs to be ligated.

ESOPHAGITIS
Esophagitis is inflammation of the esophagus.

Types:	Causes
1. Eosinophilic esophagitis	(Excessive eosinophils)
2. Reflux esophagitis	(GERD) → most common one
3. Infectious esophagitis	(Bacteria, viruses, fungi, parasites) Candida esophagitis is most common one.

General Symptoms:
- Dysphagia, Odynophagia
- Sore throat, Hoarse voice
- Heartburn, Acid reflux, Chest pain (worse with eating)
- Nausea, Vomiting
- Decrease in appetite
- Cough

1. Eosinophilic Esophagitis
Eosinophilic esophagitis is a chronic immune-mediated disease of the esophagus resulting in eosinophil-predominant inflammation of the esophagus.
Eosinophilic esophagitis is an increasingly recognized disease that can begin at any time between infancy and young adulthood; it occasionally manifests in older adults.
It is more common among males.

The cause of eosinophilic esophagitis is likely an immune response to dietary antigens in patients with genetic susceptibility; environmental allergens may also play a role. Untreated chronic esophageal inflammation ultimately can lead to esophageal narrowing and strictures.

Symptoms:

- Infants and children may present with food refusal, vomiting, weight loss, abdominal pain, and/or chest pain.
- In adults, esophageal food impaction is sometimes the first manifestation, and most patients have dysphagia.
- Symptoms of gastroesophageal reflux disease (GERD), such as heartburn, may occur.
- Patients often also have manifestations of other atopic disorders (e.g.: asthma, eczema, allergic rhinitis).

Investigations: Endoscopy with biopsy, sometimes a barium swallow

Management:

- Proton pump inhibitors
- Topical corticosteroids
- Elimination diet
- Sometimes esophageal dilation

2. Gastroesophageal Reflux Disease (GERD) / Reflux esophagitis:

Incompetence of the lower esophageal sphincter allows reflux of gastric contents into the esophagus, causing burning pain. Prolonged reflux may lead to esophagitis, peptic esophageal ulcers, stricture, and rarely metaplasia or cancer.
Gastroesophageal reflux disease (GERD) is common, occurring in 10-20% of adults. It also occurs frequently in infants, typically beginning at birth.

Etiology:
The presence of reflux implies lower esophageal sphincter (LES) incompetence, which may result from a generalized loss of intrinsic sphincter tone or from recurrent inappropriate transient relaxations (i.e., unrelated to swallowing). Transient LES relaxations are triggered by gastric distention or subthreshold pharyngeal stimulation.

Factors that contribute to the competence of the gastroesophageal junction include the angle of the cardio esophageal junction, the action of the diaphragm, and gravity (i.e., an upright position) and the patient's age.
Factors that may contribute to reflux include weight gain, fatty foods, caffeinated or carbonated beverages, alcohol, tobacco smoking, and drugs. Drugs that lower LES pressure include anticholinergics, antihistamines, tricyclic antidepressants, calcium channel blockers, progesterone, and nitrates.

Signs & Symptoms:

- The most prominent symptom of GERD is heartburn, with or without regurgitation of gastric contents into the mouth.
- Infants present with vomiting, irritability, anorexia, and sometimes symptoms of chronic aspiration.
- Both adults and infants with chronic aspiration may have cough, hoarseness, or wheezing.

- Esophagitis may cause odynophagia and even esophageal hemorrhage, which is usually occult but can be massive.
  Peptic strictures cause a gradually progressive dysphagia for solid foods.
  Peptic esophageal ulcers cause the same type of pain as gastric or duodenal ulcers, but the pain is usually localized to the xiphoid or high substernal region. Peptic esophageal ulcers heal slowly, tend to recur, and usually leave a stricture on healing.

Investigations: Clinical diagnosis, Endoscopy for patients not responding to empiric treatment, 24-Hour pH testing for patients with typical symptoms but normal endoscopy

Management:
Management of uncomplicated GERD consists of elevating the head of the bed about 15 cm (6 in) and avoiding the following:

- Eating within 3 hours of bedtime
- Strong stimulants of acid secretion (e.g., coffee, alcohol)
- Certain drugs (e.g., anticholinergics)
- Specific foods (e.g., fats, chocolate)
- Smoking

Weight loss is recommended for overweight patients and those who have gained weight recently.
Drug therapy is often with a proton pump inhibitor.
Anti reflux surgery (usually fundoplication via laparoscopy) is done in patients with more severe form of esophagitis, large hiatal hernias, hemorrhage, stricture, ulcers, large amounts of symptomatic non-acid reflux, or who cannot tolerate drug therapy. Esophageal strictures are most often managed by repeated endoscopic dilation.

3. Candida Esophagitis

Patients with Candida esophagitis usually complain of odynophagia and, less commonly, dysphagia. About 2/3 of patients have signs of oral thrush (thus its absence does not exclude esophageal involvement).
Patients with odynophagia and typical thrush may be given empiric treatment, but if significant improvement does not occur in 5-7 days, endoscopic evaluation is required. Barium swallow is less accurate.

Treatment of Candida esophagitis is with fluconazole 200-400 mg orally or IV once a day for 14-21 days. Alternatives include other azoles (e.g.: itraconazole, voriconazole, posaconazole) or echinocandins (e.g.: caspofungin, micafungin, anidulafungin).
Topical therapy has no role.

OESOPHAGEAL VARICES

Oesophageal varices are extremely dilated submucosal veins in the lower third of the oesophagus. They are most often a consequence of portal hypertension. They have strong tendency to develop bleeding.

Causes:

- Severe liver scarring (Cirrhosis)
- Blood clot (thrombosis) in portal vein
- Budd-Chiari syndrome

Pathophysiology:
The lower one third of the oesophagus is drained in to the superficial veins lining the oesophageal mucosa, which drains in to the left gastric vein (coronary vein), which in turn drains directly into the portal vein. These superficial veins (normally only approximately 1mm in diameter) become distended up to 1-2 cm in diameter in association with portal hypertension.

Normal portal pressure is approximately 9 mmHg compared to an inferior vena cava pressure of 2-6 mmHg. This creates a normal pressure gradient of 3-7 mmHg. If the portal pressure rises above the 12 mmHg, this gradient rises to 7-10 mmHg. A gradient greater than 5 mmHg is considered portal hypertension. At gradients greater than 10 mmHg, blood flow through the
hepatic portal system is redirected from the liver in to areas with lower venous pressure. This means that collateral circulation develops in the lower oesophagus, abdominal wall, stomach and rectum. The small blood vessels in these areas become distended, becoming thinner walled and
appear as varicosities.
In situation where portal pressure increases, such as with cirrhosis, there is dilation of veins in the anastomosis, leading to oesophageal varices.

Types:

1. Oesophageal (80%): Lower 1/3rd of oesophagus
2. Gastric (20%): Fundus and upper part of stomach

Clinical features:
It usually does not cause any features unless they bleed. The features of bleeding oesophageal varices include

- Vomiting and seeing significant amount of blood in vomitus
- Black, tarry, or bloody stool
- Features of shock
- Lightheadedness
- Loss of consciousness in severe cases

Investigation: Endoscopy, Evaluation for coagulopathy

Treatment:

- Emergency care is directed at stopping of blood loss, maintaining plasma volume, correcting disorders in coagulation induced by cirrhosis and appropriate use of antibiotics such as quinolones or ceftriaxone.
- Beta blocker: Medication to reduce pressure in portal vein like propranolol etc.
- Blood volume resuscitation should be done promptly and with caution.

- Therapeutic endoscopy with main two approaches as variceal ligation or banding and sclerotherapy.
- Oesophageal devascularization.

Ayurvedic treatment:
Treat the disease as that of haemorrhoids and varicose veins.

- Bolbhadra rasa or tab styplon
- Arshakuthara rasa, tab pilex
- Kaishore guggulu

OESOPHAGEAL ULCER

An esophageal ulcer is a distinct break in the margin of the esophageal mucosa. This mucosal damage to the esophagus is often caused by gastroesophageal reflux disease or from severe sustained esophagitis from other causes.

Causes:

- Gastroesophageal reflux disease
- Weakened Lower esophageal sphincter
- Repeated induced vomiting
- Infections of Candida species, Herpes simplex, and cytomegalovirus.
- Chronic consumption of acid rich foods

Clinical features:

- Trouble swallowing or painful swallowing
- Heartburn
- A feeling of stuck in the throat
- Blood in the vomit or black and tarry stools
- Abdominal or chest pain
- Dizziness
- Shortness of breath with activity
- Tiredness or fatigue
- Pale skin
- Weakness

Investigation:
History, Barium-contrast esophagram, Upper gastrointestinal endoscopy

Complications:

- upper gastrointestinal bleeding, in rare cases.
- recurrent peptic ulcers.
- esophageal strictures that narrow the esophagus.
- esophageal cancer.
- excessive weight loss due to appetite loss and difficulty swallowing.
- esophageal rupture.

Treatment:
The treatment of esophageal ulcers depends on the cause. Treatment for most ulcers uses proton pump inhibitors (PPIs), an acid-blocking medication.
If the ulcer is bleeding → injecting area with medication during endoscopy / applying heat to the area to stop bleeding.
Avoid NSAIDs
Antibiotics → If ulcer is linked to an infection

BENIGN ESOPHAGEAL TUMOURS
There are many types of benign esophageal tumours; many are found incidentally, remain asymptomatic, and warrant only observation. Some can cause swallowing symptoms and rarely ulceration or bleeding.

Evaluation typically is that of dysphagia, beginning with a barium esophagram or upper GI endoscopy (with or without endoscopic ultrasonography).
Once a lesion is visualized, tissue samples can be obtained with upper endoscopy.
A CT scan may be helpful in some cases.

Generally, treatment is recommended when patients become symptomatic.
Signs & Symptoms of benign esophageal tumours may include: - Bleeding in the esophagus

- Chest pain
- Dysphagia
- Food impaction
- Esophageal ulcers
- Regurgitation

Leiomyoma, the most common, may be multiple and can become large. Depending on its size and location, the tumour can be excised or enucleated.
Endoscopic submucosal dissection and video-assisted thoracoscopic surgery (VATS) have increasingly replaced open thoracotomy in many cases, thus reducing operative morbidity.
With treatment, this tumour usually has an excellent prognosis.
Esophageal papillomas and granular cell tumours, although rare, may become malignant and their complete endoscopic removal is recommended.

ESOPHAGEAL CANCER
The most common malignant tumour in the proximal two thirds of the esophagus is squamous cell carcinoma; adenocarcinoma is the most common in the distal one third.

Risk Factors:

- Alcohol ingestion & Tobacco use in any form (primary risk factors)
- Achalasia, human papillomavirus infection, lye ingestion (resulting in stricture), sclerotherapy, esophageal webs, and irradiation of the esophagus.
- Genetic causes are unclear, but 50% of patients with tylosis (hyperkeratosis palmaris et plantaris), an autosomal dominant disorder, have esophageal cancer by age 45, and 95% have it by age 55.

Adenocarcinoma of the Esophagus:
Adenocarcinoma occurs in the distal esophagus.
It is 4 times more common among whites than blacks.
Alcohol is not an important risk factor, but smoking is contributory. Adenocarcinoma of the distal esophagus is difficult to distinguish from adenocarcinoma of the gastric cardia invading the distal esophagus.

Most adenocarcinomas arise from Barrett esophagus, which results from chronic gastroesophageal reflux disease and reflux esophagitis.
Barrett esophagus is replacement of normal squamous epithelium of the distal esophagus with metaplastic columnar epithelium during the healing phase of acute esophagitis in the continued presence of stomach acid.

Obesity is associated with a 16-fold increased risk of esophageal adenocarcinoma, probably because obesity is a contributing factor to reflux.

Other Malignant Tumours of the Esophagus:
Less common malignant tumours include spindle cell carcinoma (a poorly differentiated variant of squamous cell carcinoma), verrucous carcinoma (a well-differentiated variant of squamous cell carcinoma), pseudosarcoma, mucoepidermoid carcinoma, adenosquamous carcinoma, cylindroma (adenoid cystic carcinoma), primary oat cell carcinoma, choriocarcinoma, carcinoid tumour, sarcoma, and primary malignant melanoma.

Metastatic cancer constitutes 3% of esophageal cancer. Melanoma and breast cancer are most likely to metastasize to the esophagus; others include cancers of the head and neck, lung, stomach, liver, kidney, prostate, testis, and bone. These tumours usually seed the loose connective tissue stroma around the esophagus, whereas primary esophageal cancers begin in the mucosa or submucosa.

Signs & Symptoms:

- Early-stage esophageal cancer tends to be asymptomatic.
- When the lumen of the esophagus becomes constricted to about < 14 mm, dysphagia commonly occurs. The patient first has difficulty swallowing solid food, then semisolid food, and finally liquid food and saliva; this steady progression suggests a growing malignant process rather than a spasm, benign ring, or peptic stricture. Chest pain may be present, usually radiating to the back.
- Weight loss, even when the patient maintains a good appetite, is almost universal.
- Compression of the recurrent laryngeal nerve may lead to vocal cord paralysis and hoarseness. Nerve compression may cause spinal pain, hiccups, or paralysis of the diaphragm.
- Malignant pleural effusions or pulmonary metastasis may cause dyspnea.
- Intraluminal tumour involvement may cause odynophagia, vomiting, hematemesis, melena, iron deficiency anemia, aspiration, and cough.

- Fistulas between the esophagus and tracheobronchial tree may cause lung abscess and pneumonia. Other findings may include superior vena cava syndrome, malignant ascites, and bone pain.
- Lymphatic spread to internal jugular, cervical, supraclavicular, mediastinal, and celiac nodes is common. The tumour usually metastasizes to lung and liver and occasionally to distant sites (e.g.: bone, heart, brain, adrenal glands, kidneys, peritoneum).

Investigations:
Endoscopy with biopsy, CT and endoscopic ultrasonography, Basic blood tests, including complete blood count, electrolytes, and liver function, should be done.

Prognosis:
Prognosis depends greatly on stage, but overall is poor (5-year survival: < 5%) because many patients present with advanced disease. Patients with cancer restricted to the mucosa have about an 80% survival rate, which drops to < 50% with submucosal involvement, 20% with extension to the muscularis propria, 7% with extension to adjacent structures, and < 3% with distant metastases.

Management:
- Surgical resection, often combined with chemotherapy and radiation
- Immunotherapy plus chemotherapy for certain advanced cancers

GULMA ROGA

8. Gulma Roga - Nidana, Prakara, Lakshana, Upadrava and Chikitsa.

Definition:

हृद्वस्त्योरन्तरे ग्रन्थिः सञ्चारी यदि वाऽचलः |

चयापचयवान् वृत्तः स गुल्म इति कीर्तितः || (Su. U. 42/4)

A spherical, mobile, or fixed lump (in the abdominal cavity) between the hridaya (Epigastrium)
and basti (Hypogastric), which may increase or decrease in size is known as gulma. According to
Sushruta, we can compare gulma with batti roga (Palpation of round mass like structure around
umbilicus) or may resembles gaseous tumour of abdomen.

Nature of gulma:

गुल्मवद्वा विशालत्वाद्गुल्म इत्यभिधीयते | (Su. U. 42/6)

- Originated from the vitiated vayu
- Does not give clue about its site of origin
- Large appearance like a cluster of shrubs

Gulma does not undergo paka:

स यस्मादात्मनि चयं गच्छत्यप्स्विव बुद्बुदः ||

अन्तः सरति यस्माच्च न पाकमुपयात्यतः | (Su. U. 42/6-7)

The gulma grows within its constituents (doshas) themselves just as a bubble form (a knobbly swelling) in water and as it keeps moving within (the abdominal cavity), it does not suppurate.

Location of gulma:

पञ्च गुल्माश्रया नृणां पार्श्वे हृन्नाभिबस्तयः | (Su. U. 42/5)

1. Dakshina parshwa
2. Vama parshwa
3. Hrudaya
4. Nabhi
5. Basti

Types:

1. Vataja
2. Pittaja
3. Kaphaja
4. Sannipataja
5. Raktaja

Poorvaroopa:

- Malaise
- Borborygmi
- Inability to eat to full satisfaction
- Eructation
- Weak digestion
- Obstruction to passage of faeces, urine, and flatus
- Dislike for food

Symptoms:

Vataja Gulma	Pittaja gulma	Kaphaja gulma
• Pain in precordial region and abdominal cavity • Dryness of mouth and throat • Suppression of flatus • Capricious digestion	• Perspiration • Fever • Heart burn on taking food • Burning sensation • Thirst • Flushing of body • Acrid taste in mouth	• A feeling as if covered with wet clothes • Dislike for food • Tiredness of the limbs • Vomiting • Excessive salivation • Sweetish taste in mouth

Raktaja gulma:

- In a woman who takes harmful diet just after delivery, after abortion in early pregnancy or else during the menses, vayu engulf the (menstrual blood) and produces a painful swelling (in relation to the uterus) with a burning sensation.
- General features as that of pittaja gulma.

Avastha anusara gulma:
Amavastha: Heavy, hard, situated beneath the muscles without change in colour and fixed firmly
pachyamana: Burning, painful, causing agony, loss of sleep, irritant and fever
pakvavastha: Burning, bluish red in colour, palpable like bladder and severe painful.

Sadhya Asadhyata:
Sadhya → Ekdoshaja
Krachrasadhya → Dvidoshaja
Asadhya → Sannipataja

Vataja gulma chikitsa:

- Snehana and swedana
- Virechana by eranda snehapana
- Snehapana
- Basti (Anuvasana and niruha)
- Raktamokshana: When all the other treatment modalities fail to pacify the condition, then
  one should go for raktavasechana at baahu.
- Shadanga ghrita, Chitrakadi ghrita, hingvadhya ghrita
- Svarjikadi churna

Pittaja gulma chikitsa:

- Snehapana by kakolyadigana siddha ghrita
- Virechana by aaragvadhadi gana dravya
- Niruha basti
- Trunamuladi ghrita

Kaphaja gulma chikitsa:

- Snehapana and abhyanga with pippalyadi ghrita
- Virechana by teekshana drugs
- Teekshana niruha basti
- Raktamokshana by Ghati yantra (Cupping therapy)
- Trini ghrita

Raktaja gulma chikitsa:

- Same as pittaja gulma
- Ghrita processed with water of palasha kshara to drink
- Vaginal douche with pippalyadi gana ghrita
- Raktapradara chikitsa

Raktamokshana in gulma:

सशूले सोन्नतेऽस्पन्दे दाहपाकरुगन्विते ||

गुल्मे रक्तं जलौकोभिः सिरामोक्षेण वा हरेत् | (Su. U. 42/52)

A prominent, non-pulsatile localised abdominal swelling (gulma) with colicky pain, burning sensation and painful suppuration should be treated with bloodletting by the application of jalauka or siravyadha.

Hard faeces:

बद्धवर्चोनिलानां तु सार्द्रकं क्षीरमिष्यते ||

कुम्भीपिण्डेष्टकास्वेदान् कारयेत् कुशलो भिषक् | (Su. U. 42/55-56)

- Milk with ginger
- Swedana (Kumbhi/Pinda/Ishtika)

Absolute constipation:

वातवर्चोनिरोधे तु सामुद्गार्द्रकसर्षपैः ||

कृत्वा पायौ विधातव्या वर्तयो मरिचोत्तराः | (Su. U. 42/59-60)

- Drink ardraka boiled in milk
- Suppository prepared from common salt, ginger and mustard as well as black pepper should be introduced per rectum.

In general:

- Dadhika ghrita, rasonadi ghrita, shadanga ghrita, chitrakadi ghrita, hingavadi ghrita
- Kshara avaleha
- Vrushchivadhya arishta
- Pathadi churna
- Hingavadi churna with tilvaka ghrita in tridoshaja gulma
- Jangala mamsa rasa with trikatu and saindhava

SHOOLA VYADHI

9. **Shoola vyadhi -** Nidana, Prakara, Lakshana, Upadrava and Chikitsa.

Nirukti:

शङ्कुस्फोटनवत्तस्य यस्मात्तीव्राश्च वेदनाः |

शूलासक्तस्य लक्ष्यन्ते तस्माच्छूलमिहोच्यते || (Su. U. 42/81)

Shula is the condition where patient feels severe pain as being pierced by nail making him appear listless.

Nidana & Samprapti:

- Withholding flatus, urine and faeces
- Indigestion
- Excessive physical labour
- Drinking fluid when hungry
- Over-eating
- Eating before the previous meal has been digested
- Intake of incompatible food
- Intake of germinating grains
- Use of food prepared from pasted cereals and of dry meat

Vata vitiated by these factors produces severe colicky pain in the abdomen due to which the patient suffering from agony gets respiratory distress.

Types:

1. Vataja
2. Pittaja
3. Kaphaja
4. Sannipataja

Vataja shoola:

- When person gets severe abdominal colic on an empty stomach.
- Develops rigidity of whole body
- Respiratory distress
- Difficulty in passing flatus, urine & faeces

Pittaja shoola:

- Thirst
- Intoxication symptoms
- Burning sensation
- Fainting due to excruciating abdominal colicky pain
- Desire for cold which gets relieved by the application of cooling things

Kaphaja shoola:

- Nauseating tendency
- Heaviness of body
- Excessive fullness of abdomen

Sannipataja shoola:
Combination of all above symptoms

Vataja shoola chikitsa:

- Swedana with payasa, krashara or of fatty minced meat
- Diet: Warm and oily food with cooked vegetable of trivruta, sprouts of chirabilva fried in oil, meat juice of birds and wild animals
- Drinks: Sura, sauviraka, sukta, mastu, udvasit and ghrita mixed with black salt

Pittaja shoola chikitsa:

- Emesis should be induced easily after giving patient cold water as much as necessary to drink.
- Pots made up of copper filled with water should be placed over the site of abdominal colic
- Diet: Jaggery, shali rice, barley, milk, draughts of ghrita, purgatives and meat of wild animals.
- Drink: Soup of palasha with sugar, parushaka, grapes, date palm fruits and aquatic products

Kaphaja shoola chikitsa:

- Emesis with pippali water
- Pippali and shringavera
- Dry fomentation
- Churna of patha, vacha, trikatu and katukarohini taken with chitrak kwath.
- Bhasma from equal parts of pippali, svarjika, kshara, yava, chitraka and sevya.

ACUTE ABDOMEN
An acute abdomen refers to a sudden, severe abdominal pain.

Nature of pain:

- Continuous: Acute pancreatitis, acute appendicitis
- Episodic: Acute hyperacidity, acute cystitis, ruptured ectopic gestation
- Colicky: Biliary colic, appendicular colic, ureteric colic, dysmenorrhoea
- Associated with fever: Acute cholecystitis, acute pancreatitis, colitis, appendicitis, cystitis, perforated duodenal ulcer
- Associated with loose stool: Acute diverticulitis, colitis
- Pain aggravating factor: Lying supine (Acute pancreatitis), deep breath (Acute cholecystitis)
- Pain relieving factor: Leaning forward while sitting (Acute pancreatitis), food (Acute hyperacidity)
- Referred pain: Stomach (chest), Appendix (umbilicus), Bladder (penis), Ureter (testis)

Causes:

Right hypochondrium: • Acute cholecystitis • Acute cholangitis • Hepatitis • Hyperacidity • Perforated duodenal ulcer	Epigastrium: • Acute hyperacidity • Acute Pancreatitis • Perforated duodenal ulcer • Acute hepatitis (left lobe)	Left hypochondrium: • Acute hyperacidity • Acute pancreatitis • Splenic infarct
Right iliac fossa: • Acute or perforated appendicitis • Acute mesenteric adenitis • Acute Meckel's diverticulitis • Torsion of testis (R) • PID • Ureteric colic (R)	Hypogastrium: • Acute cystitis • Acute congestive dysmenorrhoea • Acute Meckel's diverticulitis • Uterine fibroid	Left iliac fossa: • Acute diverticulitis • Torsion of testis (L) • PID • Incarcerated left inguinal hernia • Ureteric colic (L)
Right lumbar: • Right renal pathology • CA of ascending colon	Umbilicus: • Gastric malignancy • Dissection of abdominal aorta	Left lumbar: • Left renal pathology • CA of descending colon

Abdominal distension:
General: Perforated duodenal ulcer, perforated appendicitis with peritonitis
Right upper abdomen: Hepatomegaly
Right lower abdomen: Ruptured appendicitis, torsion of right ovary
Epigastrium: CA stomach
Hypogastrium: Distended urinary bladder in cystitis
Left upper abdomen: Splenomegaly
Left lower abdomen: Torsion of left ovary.

Tenderness:
All quadrants: General peritonitis
Right upper quadrant: Acute hepatitis, acute cholecystitis
Right lower quadrant: Acute hepatitis, acute mesenteric adenitis
Epigastrium: Acute hepatitis, acute gastritis
Hypogastrium: CA cystitis
Left upper quadrant: Acute gastritis, acute pancreatitis
Left lower quadrant: Acute colitis, diverticulitis

Investigation:

- Routine blood
- Plain x ray abdomen
- Endoscopy
- Stool examination
- USG
- CT Scan

Treatment:

- Nil by mouth
- Depend upon cause
- Non perforated pathology: Medical treatment
- Perforated pathology: Early surgical treatment
- Exploratory laparoscopy: If diagnosis is not clear and not responded to medical treatment

UDARA ROGA

10. Udara Roga: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Jalodara - Ascites, Chidrodara – Perforation, Peritonitis and Badhagudodara-Intestinal obstruction.

JALODARA

Nidana & Samprapti:

Drinking cold water soon after snehana, anusasana, vamana or virechana or after taking basti, leads to vitiation of channels. And even if (channels) are lined by oily substance dakodara occurs.

Lakshana:

- The abdomen becomes very smooth
- Abdominal distension
- Umbilicus gets everted as if full of water. Just as leather bag full of water and air shakes, fluctuates and makes sounds.

Treatment (abdominocentesis):

- The patient should first be managed with the vatahara tailas and subjected to swedana with warm water.
- He should be made to stand and held firmly in the armpits by dependable persons surrounding him.
- A deep puncture as deep as a thumb breadth should be made by a vreehimukha shastra below the naval four angulas beyond the hairline (midline) on the left side.
- Then, a tubular instrument made up of tin or of any similar metal, which open at both ends should be introduced and the collected fluid removed.
- Thereafter, the nadiyantra should be removed, the wound anointed with taila and lavana and then bandaged.
- If fluid removed all at once, it causes thirst, fever, body ache, diarrhoea, asthma, cough and burning sensation in the foot; therefore, the fluid should be drained little by little at intervals of 3, 4, 5, 6, 8, 10, 12 or 16 days.
- After the dosha has been drained, firm bandaging by sheep's wool, silk or leather should be done over.
- For 6 months food should be taken with milk or meat juices of wild animals.
- Then for next 3 months food should be given with milk, diluted with an equal quantity of water or with citrous fruit juices or with the meat juices of wild animals.
- For the remaining 3 months light and wholesome food should be taken. Thus, in a year the patient gets free from disease.

ASCITIS

It is pathological collection of fluid in peritoneal cavity.

Types:

Mild: Up to 150 ml amount required to demonstrate sonologically

Moderate: 1500-2000 ml causes clinical dullness in flanks

Severe: More than 2000 ml (Gross ascites/Marked abdomen distension)

SAAG:

- Serum ascites albumin gradient
- Subtracting ascitic fluid albumin level from serum albumin level
- If SAAG > 1.1 then its Portal hypertension

Portal hypertension:

- Causes renal sodium retention due to rennin angiotensin aldosterone pathway
- Increases hydrostatic pressure in hepatic sinusoids and splanchnic vessels, leading to ascites.

Clinical features:

1. Specific features related to cause
2. Gradual abdominal distension evenly, with fullness of flanks, which are dull to percussion.
3. In severity, respiratory embarrassment
4. Right sided pleural effusion

Signs:

1. Mild: Puddle sign (Tapping around umbilicus in knee elbow position elicit dullness)
2. Moderate: Positive shifting dullness in abdomen
3. Severe: Positive fluid thrill, Tanyol sign* (umbilicus shifted downwards) and smiling horizontal umbilicus

Investigation:

- USG abdomen
- Ascitic tap

Treatment:

Treat the cause

Spironolactone 100 mg/day (diuretic, prevent salt absorbs, vit K)

Paracentesis abdominis

Bladder should empty before tapping

Puncture of peritoneum is carried out under local anaesthetic using a moderate sized Trocar and cannula

Site of tap is below umbilicus, lateral to rectus muscle

Slow gradual tapping is important, otherwise patient undergoes fluid & electrolyte imbalance

Up to 5 litres can tapped at a time

CHIDRODARA

Chidrodara, also known as Kṣatodara, is compared with intestinal perforation.

Nidāna:

- Ingestion of a sharp foreign body along with food (sand particles, star, pieces of wood, bone, thorns, glass, etc.)
- Jṛmbha
- Atyshana

Saṁprāpti:

Nidāna sevana → Anta Bhidhyate (perforation of intestine) → Pāka Gacchati (suppuration occurs the place of perforation)→ Āhārarasa flows out through the opening into the adjacent area → Chidrodara

Lakṣhaṇa:

- Distension of the abdomen mostly below the umbilicus due to accumulation of Āhārarasa
- Lohita-Nīla-Pīta-Picchila-Kuṇapagandha Purīṣa
- Hikā, Shvāsa, Kāsa, Tṛṣṇā, Moha, Arochaka, Avipāka, Daurbalya

Chikitsā:

- Basic treatment is same as for Baddhagudodara.
- After suturing, apply black clay mixed with Madhuka and bandage the area.
- Sneha Avagāha
- Basic treatment protocol is same as that of baddhagudodara
- Identify the site of perforation
- Perforated area will be bitten by black ants
- When the ants have bitten the intestines, their body should be chopped off and removed leaving the heads behind.
- Then suturing should be done as before and other reparative measures shou
- Apply black clay mixed with madhuka and bandaged
- Ask the patient to sit in through full of oil or ghrita.
- Milk diet should be given.

PERFORATION OF PEPTIC ULCER

It is dreaded complication of duodenal ulcer
More common in males (M: F = 8: l)
Anterior duodenal ulcer and posterior gastric wall perforates (80%)
It may be precipitated by excessive smoking, alcohol or NSAIDs etc
Rarely a silent ulcer can also perforate especially those patients treated with cortisones

Stages of duodenal ulcer perforation:

1. Stage of chemical peritonitis: 2-4 hours

Immediately after perforation acid peptic juice, bile and pancreatic juice come out in to general peritoneal cavity, which results in peritoneal irritation or chemical peritonitis. The features are

- Acute agonizing pain in epigastrium initially, later in right side of abdomen and finally becomes generalised. Pain may radiate to scapular region which becomes more on movements.
- There may be episodes of coffee-ground vomiting followed by ascites
- Fever. The patient is pale and anxious
- Per abdomen: There is guarding and rigidity of the abdominal wall
- Tenderness and rebound tenderness are present all over abdomen (Blumberg's sign)
- On percussion, liver dullness is obliterated because of collection of free air (gas) under the right dome of diaphragm
- Bowel sounds are usually absent

2. Stage of reaction: 3-6 hours

Due to chemical irritants, peritoneum reacts by secreting peritoneal fluid (sterile). As result Of this HCI and bile are diluted by peritoneal secretions (reaction of peritoneum to the insult) resulting in reduction of pain. Hence it is also called stage of illusion or delusion. The features are

- Tachycardia
- Hypotension
- Evidence of dehydration due to loss of fluid in to peritoneal cavity
- Shifting dullness is present
- Abdominal distension is due to fluid and paralytic ileus
- Guarding and rigidity are worsened

3. Stage of bacterial peritonitis: after 12 hours

As acid secretions of stomach abolished reflexly, there is no acid barrier. The bacterial invasion becomes easy from intestine which causes diffuse peritonitis. The features are

- Severely ill, sunken eye, cold periphery and shallow rapid breath
- Features of hypovolaemic and septic shock
- Gross abdominal distension, guarding, rigidity, abdominal tenderness all over suggests generalised peritonitis.

Investigation:

1. Plain X-ray chest or abdomen in erect / sitting position shows collection of free gas under right dome of free diaphragm.
2. USG and CT scan
3. Aspiration of peritoneal cavity may show bile-stained fluid.

Management:

- Aspiration with Ryle's tube of stomach contents to reduce further contamination, to decrease biliary and pancreatic juice.
- Fluids are given pre-operatively to treat dehydration and post-op for 3-4 days till paralytic ileus settle down.
- Nil by mouth and catheterisation
- Drugs:
  Injection Ampicillin 500 mg IV 6th hourly against gram +ve
  Injection Genta 80 mg IV 8th hourly against gram -ve
  Injection metronidazole 500 mg IV 8th hourly to treat anaerobic organisms
- Rosoe-Graham operation: Emergency laparotomy is done through a midline incision. Infected fluid sucked out. The perforation is identified and closed with interrupted absorbable silk sutures, which is strengthened by placement of omentum. Peritoneal wash is given to avoid residual abscess. Abdomen is closed with drain which is removed after days. Post operatively patient is put on anti-pyloric regime with PPI for 3 months.

PERITONITIS

Peritonitis is inflammation of the peritoneal cavity.

Etiology:

- The most serious cause is perforation of the gastrointestinal tract, which causes immediate chemical inflammation followed shortly by infection from intestinal organisms.
- Peritonitis can also result from any abdominal condition that causes marked inflammation, such as appendicitis, diverticulitis, strangulating intestinal obstruction, pancreatitis, pelvic inflammatory disease, mesenteric ischemia
- Intraperitoneal blood from any source, such as ruptured aneurysm, trauma, surgery, ectopic pregnancy, is irritating and results in peritonitis.
- Barium causes severe caking and peritonitis and should never be given to a patient with suspected gastrointestinal tract perforation. However, water-soluble contrast agents can be safely used.
- Peritoneosystemic shunts, drains, and dialysis catheters in the peritoneal cavity predispose a patient to infectious peritonitis, as does ascitic fluid.

Signs & Symptoms:

Abdominal pain, Cloudy peritoneal fluid, Fever, Nausea, Tenderness to palpation.

Complications:

Peritonitis causes fluid to shift into the peritoneal cavity and bowel, leading to severe dehydration and electrolyte disturbances. Acute respiratory distress syndrome can develop rapidly. Kidney failure, liver failure, and disseminated intravascular coagulation follow. Without treatment, death occurs within days.

Diagnosis:
Diagnosis of peritonitis is made by clinical criteria and testing. A sample of peritoneal fluid is obtained for Gram stain, culture, and white blood cell (WBC) count with differential.
Peritonitis is present if a patient has at least 2 of the following 3 criteria:

i. Clinical features consistent with peritonitis (abdominal pain, tenderness, and/or cloudy dialysis effluent)
ii. Dialysis effluent WBC > 100/mcL with > 50% polymorphonuclear cells after a dwell time of at least 2 hours
iii. Positive peritoneal fluid culture

Management:
Antibiotic therapy is usually given IV or intraperitoneally (IP) for peritonitis and orally for exit-site infections. Patients with peritonitis are admitted to the hospital if IV treatment is necessary or if hemodynamic instability or other significant complications arise. Most cases of peritonitis respond to prompt antibiotic therapy.

BADDHAGUDODARA
Baddhagudodara, also known as Baddhodara, is compared with a form of intestinal obstruction; similar like a clogged drainage.

Nidāna:

1. Obstruction due to intake of food mixed with feathers or hairs
2. Udāvarta
3. Arsha
4. Antra Sammūrcchana (intussusception)
5. Apāna Mārgāvarodha

Saṁprāpti:
Nidāna sevana → Apāna Vāyu duṣṭi → Agnimāndya → Varcha Pitta Kapha avarodha → Baddhagudodara

Lakṣaṇa:

- Tṛṣṇā, Dāha, Jvara, Mukhashoṣa, Tālushoṣa, Urusāda, Kāsa, Shvāsa, Daurbalya, Arochaka, Avipāka, Vit-Mūtra saṅga, Ādhmāna, Chardi, Kṣavathu, Shiraḥshūla, Uraḥshūla, Nābhī-Guda shūla, Udara mūḍhatva
- Sthira Aruṇa Nīla Rāji Sirā (abdominal wall is covered with reddish and bluish stretch marks and visible veins)
- Purīṣa gets obstructed in Guda and may not come out at all or only in small amounts with difficulty.
- Hṛd-Nābhī Madhye Parivṛddhi (distension between heart and umbilicus)
- Viṭ-Samagandhika Pracchardayan (faecal smell is present in vomitus)

Chikitsā:
Abhyaṅga & Svedana
Incision should be taken on the left side, 4 aṅgula below the umbilicus, beyond the midline. Inspect the intestine after removing 4 aṅgula of intestine at a time. Any obstruction is removed. Apply Madhu and Ghṛta over the intestine and put it back to its normal position. Close the abdomen by suturing.

Intestinal obstruction
When Intestinal contents are prevented from travelling distally is called as intestinal obstruction.

Classification:

1. **Depend on nature of obstruction**

a. Mechanical/ dynamic obstruction: There is peristalsis working against obstructive agent,
b. Adynamic like Paralytic ileus (No peristalsis), electrolyte imbalance, spinal injuries

2. **Depend on cause of obstruction**

In lumen of gut	**In wall of gut**	**Outside wall of gut**
• Gall stone ileus • Round worm mass • Trichobezoar (Mass of hairs trapped in GIT system specially stomach)	• Stricture e.g., tuberculosis • Crohn's disease • Cancer (15%) • Atresia	• Adhesions (40%, from outside) • Volvulus • Intussusception • Meckel's diverticulum • Obstructed hernia (25%)

3. **Depend on severity of infection**

a. Acute: Affects small bowel
b. Chronic: Affects large bowel
c. Acute on chronic obstruction

4. **Depend on blood supply**

a. Simple obstruction: Blood supply is not seriously impaired
b. Strangulated obstruction: Blood supply is seriously impaired especially mesenteric vessels
c. Closed loop obstruction: When bowel is obstructed at both proximal and distal points.

Pathophysiology:

1. Changes proximal to the bowel obstruction:

Intestinal obstruction → Increased peristalsis → Becomes vigorous → Obstruction not relieved → Peristalsis not relieved → Peristalsis ceases → Flaccid, paralysed, dilated bowel

2. Changes at the site of obstruction:

Initially venous return is impaired → Congestion, oedema of bowel wall occurs (turn into purple) → Later this jeopardizes the arterial supply → blackish discolouration → loss of peristalsis → Gangrene → Perforation occurs → Bacteria and toxins migrates into the peritoneum → Peritonitis

Clinical features:

- Abdominal pain
- Vomiting
- Abdominal distension
- Dehydration
- Tachycardia
- Tachypnoea
- Fever
- Cold periphery
- Constipation and obstipation
  Constipation → Bowel does not evacuate faeces but passes flatus
  Obstipation → Neither faeces nor flatus are evacuated from bowel
- Features of strangulation:
  Blumberg's sign → Continuous severe pain, tenderness and rebound tenderness

Investigations:

1. CBC
2. Sr. electrolytes, USG – Whole abdomen
3. Plain X-ray abdomen – In erect and supine position
4. CT Scan
5. Two enema test – (for intestinal obstruction)
   After 1st enema – faeces
   After 2nd enema – (2 hours later) – if no faeces, then it is suggestive of intestinal obstruction

Management: Nil by mouth

- Nasogastric aspiration: To decrease distension, prevent vomiting & respiratory complications
- Fluids: To correct electrolytes, dehydration, and shock
- Drugs: Gram positive, negative & anaerobes
- Blood transfusion
- ICU critical care

Surgical treatment:

- General anaesthesia
- Abdomen exploration through midline vertical Incision
- Open peritoneum, if cleared straw coloured fluid (simple obstruction), blood-stained fluid (strangulated obstruction)
- If empty & collapsed caecum (small intestine obstruction), caecum distended (large bowel obstruction)
- Affected bowel is resected

DISEASES OF STOMACH AND DUODENUM

11. Diseases of stomach and duodenum: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Pyloric Stenosis, Peptic Ulcer and Tumours.

CONGENITAL HYPERTROPHIED PYLORIC STENOSIS / PYLORIC STENOSIS

It is hypertrophy of musculature of pyloric antrum, especially the circular muscle fibres, causing primary failure of pylorus to relax, Mucosa is compressed such an only a probe can be inserted.

Here duodenum is normal.

Etiology & Risk Factors:

- The exact etiology of hypertrophic pyloric stenosis is uncertain, but a genetic component is likely because siblings and offspring of affected people are at increased risk, particularly monozygotic twins.
- Maternal smoking during pregnancy also increases risk.
- Infants exposed to certain macrolide antibiotics (e.g.: erythromycin) in the first few weeks of life are at significantly increased risk.
- Some studies have noted increased risk in bottle-fed infants compared to breastfed infants, but it is not clear whether this risk is associated with a change in feeding method or with the type of feeding.

Signs & Symptoms: Symptoms of hypertrophic pyloric stenosis typically develop between 3 weeks and 6 weeks of life.

- Projectile vomiting (without bile) occurs shortly after eating.
- Until dehydration sets in, children feed avidly and otherwise appear well, unlike many of those with vomiting caused by systemic illness.
- The baby is usually quite hungry and eats or nurses eagerly. The milk is sometimes curdled in appearance, because as the milk remains in the stomach and does not move forward to the small intestine, the stomach acid "curdles" it.
- Gastric peristaltic waves may be visible, crossing the epigastrium from left to right.
- A discrete, 2- to 3-cm, firm, movable, and olive-like pyloric mass is sometimes palpable deep in the right side of the epigastrium.
- With progression of illness, children fail to gain weight, become malnourished, and develop dehydration.

Investigation:

1. Barium meal x-ray: String sign or rail road track sign (narrowing & elongation obstruction of pyloric canal) or double track sign with pyloric obstruction
2. USG: Doughnut sign (pyloric muscle 4mm or more in thickness, length of pyloric more than 1.8cms)

Treatment:
Medical:

1. Correct the electrolyte imbalance and dehydration.
2. Atropine methyl nitrate orally 1-2 ml half an hour before each food, to relax the pylorus muscle temporary.
3. Small frequent feeds should be advised.

Surgical:

- Pyloromyotomy, which leaves the mucosa intact and separates the incised muscle fibres. Postoperatively, the infant usually tolerates feeding within a day.

Non-surgical:

- Non-surgical therapy by using a feeding tube passed beyond the pylorus is not considered a good alternative because of the efficacy and safety of pyloromyotomy.

PEPTIC ULCER

Peptic ulcers are open sores that develop on the inside lining of your stomach and the upper portion of your small intestine. The most common symptom of a peptic ulcer is stomach pain.
Peptic ulcers include:

1. Gastric ulcers that occur on the inside of the stomach
2. Duodenal ulcers that occur on the inside of the upper portion of your small intestine (duodenum)

GASTRIC ULCERS

Causes:
Stomach ulcers are usually caused by Helicobacter pylori (H. pylori) bacteria or non-steroidal anti-inflammatory drugs (NSAIDs).
Helicobacter pylori:

- H. pylori bacteria live in the stomach lining. The bacteria can irritate the stomach lining and make it more vulnerable to damage from stomach acid.

Non-steroidal anti-inflammatory drugs:

- Common NSAIDs include:
  ibuprofen
  aspirin
  naproxen
  diclofenac
- Many people take NSAIDs without having any side effects. But there's always a risk the medication could cause problems, such as stomach ulcers, particularly if taken for a long time or at high doses.
- You may be advised not to use NSAIDs if you currently have a stomach ulcer or if you've had one in the past. Paracetamol is a safer painkiller to use.

Clinical features:

- Abdominal pain
- Indigestion
- Heartburn
- Loss of appetite
- Feeling and being sick
- Weight loss

Investigation:

- Urea breath test
- Stool antigen test
- Blood test
- Gastroscopy

Complication:

- internal bleeding
- perforation - the lining of the stomach splits open
- gastric outlet obstruction - the stomach ulcer obstructs the normal passage of food through your digestive system

Treatment:
Antibiotics

- If you have an H. pylori infection, you'll usually be prescribed a course of 2 or 3 antibiotics.
- The most used antibiotics are:
  amoxicillin
  clarithromycin
  metronidazole

Ulcers caused by NSAIDs
If your stomach ulcers caused by taking NSAIDs:

- You will be given a course of PPI (Proton pump inhibitor) medication
- Your use of NSAIDs will be reviewed, and you may be advised to use an alternative painkiller.

DUODENAL ULCER
A duodenal ulcer is a sore that forms in the lining of the duodenum.

Causes:

- The main cause of this damage is infection with bacteria called Helicobacter pylori, or *H. pylori*. The bacteria can cause the lining of your duodenum to become inflamed and an ulcer can form.
- Some medications can also cause duodenal ulcers, particularly anti-inflammatory medicines such as ibuprofen and aspirin. It is rare that other medicines or medical conditions cause an ulcer.
- There are some lifestyle factors that may make you more likely to get a duodenal ulcer, such as:
  smoking
  drinking a lot of alcohol
  experiencing stress

Clinical features:

- Pain in stomach or abdomen
- Indigestion
- Feeling full and bloated after eating
- Nausea
- Weight loss

Diagnosis:

- blood tests
- stool sample
- breath tests

Treatment:

- Antibiotics
- Antacids
- Drinking less alcohol
- Losing weight if you are overweight
- Quitting smoking
- Relief can also come by reducing your intake of:
  hot drinks
  fatty foods
  spicy foods
  acidic foods (such as tomato)

STOMACH CANCER

Stomach cancer or gastric cancer is a disease in which malignant cells form in the lining of the stomach.

Etiology & Risk Factors:

Etiology of stomach cancer is multifactorial, but Helicobacter pylori plays a significant role.

- Helicobacter pylori infection
- Autoimmune atrophic gastritis
- Smoking
- Gastric polyps
- Genetic factors
- The World Health Organization (WHO) International Agency for Research on Cancer (IARC) has reported a positive association between consumption of processed meat and stomach cancer.

Classification:

Gastric adenocarcinomas can be classified by gross appearance:

1. Protruding: The tumour is polypoid or fungating.
2. Penetrating: The tumour is ulcerated.
3. Superficial spreading: The tumour spreads along the mucosa or infiltrates superficially within the wall of the stomach.

4. Linitis plastica: The tumour infiltrates the stomach wall with an associated fibrous reaction that causes a rigid “leather bottle” stomach.
5. Miscellaneous: The tumour shows characteristics of ≥ 2 of the other types; this classification is the largest.

Prognosis is better with protruding tumours than with spreading tumours because protruding tumours become symptomatic earlier.

Signs & Symptoms:

- Initial symptoms of stomach cancer are non-specific, often consisting of dyspepsia suggestive of peptic ulcer.
- Later, early satiety (fullness after ingesting a small amount of food) may occur if the cancer obstructs the pyloric region or if the stomach becomes non-distensible.
- Dysphagia may result if cancer in the cardiac region of the stomach obstructs the esophageal outlet.
- Loss of weight or strength, usually resulting from dietary restriction, is common. - - Massive hematemesis or melena is uncommon, but secondary anemia may follow occult blood loss.
- Physical findings may be unremarkable or limited to heme-positive stools. Late in the course, abnormalities include an epigastric mass; umbilical, left supraclavicular, or left axillary lymph nodes; hepatomegaly; and an ovarian or rectal mass. Pulmonary, central nervous system, and bone lesions may occur.

Investigations: Endoscopy with biopsy, CT scan and endoscopic USG

Treatment:

- Surgical resection, sometimes combined with chemotherapy, radiation, or both.
- Curative surgery involves removal of most or all the stomach and adjacent lymph nodes and is reasonable in patients with disease limited to the stomach and perhaps the regional lymph nodes. Adjuvant chemotherapy or combined chemotherapy and radiation therapy after surgery may be beneficial if the tumour is resectable.
- Metastasis or extensive nodal involvement precludes curative surgery, and, at most, palliative procedures should be undertaken. However, the true extent of tumour spread often is not recognized until curative surgery is attempted.
- Palliative surgery typically consists of a gastroenterostomy to bypass a pyloric obstruction and should be done only if the patient’s quality of life can be improved.

DUODENAL CANCER

Duodenal cancer is a rare type of cancer that forms in the first part of the small intestine; the duodenum.

There are four main types of small intestine cancer:

1. Adenocarcinoma initially develops in the glandular cells that line the inside of the small intestine. Adenocarcinomas are the most common type of small intestine cancer, accounting for approximately 1 in 3 cases.
2. Sarcoma begins in the muscle and other supporting tissues of the small intestine. Around 10% of small intestine cancers are sarcomas
3. Carcinoid tumours are slow-growing and develop in the neuro-endocrine cells of the small intestine. Neuro-endocrine cells produce hormone-like substances.
4. Lymphomas form in cells called lymphocytes. These are part of the immune system and are present in most parts of the body, including the intestines.

Small intestine cancers are rare, accounting for fewer than 1 in 100 of all cancers and fewer than 1 in 10 cancers that occur in the digestive tract.
This type of cancer is more common in older people, particularly in those aged over 60 years.

Risk Factors:
Risk factors for developing a type of small intestine cancer, such as duodenal cancer, include:

- Age. Small intestine cancer is more common in older people.
- Inherited conditions. Those that may increase the risk of developing small intestine cancer include familial adenomatous polyposis, Lynch syndrome, Peutz-Jeghers syndrome, cystic fibrosis
- Gastrointestinal disorders. Crohn's disease or celiac disease can increase the risk of cancer in the small intestine.
- Colon cancer can increase the risk of developing small intestine cancer.
- Smoking and alcohol can increase the risk of small intestine cancer.
- Diet. Some studies indicate that people who eat a lot of red meat, salt, or smoked foods may have a higher risk of small intestine cancer.

Signs & Symptoms:
Small intestine cancer, including duodenal cancer, can cause a variety of symptoms which are also commonly seen in other conditions.
Symptoms include unexplained weight loss, abdominal pain, bloody stools, diarrhoea, a lump in the abdomen, nausea, vomiting, weakness and fatigue, anemia, jaundice.

Diagnosis:
It can be difficult to diagnose duodenal cancer, due to the natural folds of the small intestine and because symptoms can be like those of several other conditions.
History taking, Blood tests, MRI, CT scans, Upper endoscopy, Biopsy, Capsule endoscopy

Management: Surgery, Radiotherapy, Chemotherapy, Biologic therapy

DISEASES OF SMALL & LARGE INTESTINE

12. **Diseases of small intestine:** Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Tuberculosis, Obstruction and Perforation.

13. **Diseases of large intestine -** Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Tuberculosis, Obstruction, Perforation, Tumours, Appendicitis, Crohn's disease and Ulcerative Colitis.

INTESTINAL TUBERCULOSIS

It is a chronic disease of the walls of the intestine, which are characterized by tubercle deposits.

Causes:

1. Secondary to pulmonary tuberculosis (Ulcerative T B)
2. Primary: Infection due to bovine strain of Mycobacterium tuberculosis and results from ingesting infected milk.

Common sites: Ileo-caecal region is commonly involved in TB due to following reasons

- Organism gets trapped in rich layers of payer's patch
- It favours growth of organism due to alkaline media
- Stasis due to presence of ileo-caecal valve
- Terminal ileum is area of maximum absorption

Types:

1. Ulcerative type (60%)
2. Hyperplastic / Obstructive (10%)
3. Mixed (30% Ulcero – hyperplastic)

Clinical features:

1. Age: 20-40yrs
2. Pain abdomen: In right lower quadrant, which can be dull vague or colicky type (stricture) It increases after taking food (due to gastro-colic reflux, spasm of ileum & peristalsis strengthens) & relieved by vomiting (because of obstruction).
3. Diarrhoea: Paste like, watery, small quantity with abnormal foul smell. It may alternate with constipation
4. Anaemia (Vitamin 1312), loss of appetite and loss of weight
5. Abdominal distension: Due to ascites & sub-acute intestinal obstruction
6. Fever

Signs:

1. Patients are malnourished & pale
2. Visible intestinal peristalsis
3. Mass in right ileac fossa: Hard, nodular, non-mobile, non-tender with impaired resonance

Investigation:

1. Chest x-ray
2. Hb ↓, ESR ↑,
3. Abdomen USG
4. Colonoscopy and biopsy
5. Stool examination: Mycobacterial culture positive

Management:

1. Bed rest, nutrition supplement, IV fluids
2. Anti TB drugs (if no evidence of obstruction)
3. Obstruction: Stricturoplasty or limited resection is choice. It includes removal of terminal 8-10cm of diseased ileum, caecum with appendix & diseased portion of ascending colon. Then ileo-colic anastomosis. Later ATT for 12-18 months

INTESTINAL OBSTRUCTION

Intestinal obstruction is significant mechanical impairment or complete arrest of the passage of contents through the intestine due to pathology that causes blockage of the bowel.

Mechanical obstruction is divided into obstruction of the small bowel (including the duodenum) and obstruction of the large bowel. Obstruction may be partial or complete. About 85% of partial small-bowel obstructions resolve with non-operative treatment, whereas about 85% of complete small-bowel obstructions require surgery.

Etiology:

Overall, the most common causes of mechanical obstruction are:

- Adhesions
- Hernias
- Tumours

Other general causes include:

- Diverticulosis, Diverticulitis
- Foreign bodies (including gallstones)
- Volvulus (twisting of bowel on its mesentery)
- Intussusception (telescoping of one segment of bowel into another)
- Faecal impaction

Diverticulosis, Diverticulitis:

Diverticulosis occurs when small, bulging pouches (diverticula) develop in the lining of the digestive tract. When one or more of these pouches become inflamed or infected, the condition is called diverticulitis.

Volvulus:
Volvulus is the twisting of a loop of intestine around its mesenteric attachment, resulting in a closed loop bowel obstruction. The affected bowel can become ischaemic due to compromised blood supply, rapidly leading to bowel necrosis and perforation.
Volvulus often has an abrupt onset. Pain is continuous, sometimes with superimposed waves of colicky pain.
Occasionally, the rotation can be reduced non-invasively with an endoscope.

Intussusception (Antrāntra Pravesha):
Intussusception is a form of bowel obstruction in which one segment of intestine telescopes inside of another, causing intestinal obstruction. Although it can occur anywhere in the gastrointestinal tract, it usually occurs where the small and large intestines meet.
The obstruction can cause swelling and inflammation that can lead to intestinal injury.
The exact cause of intussusception is unknown. In most cases, it is preceded by a virus that produces swelling of the lining of the intestine, which then slips into the intestine below.
The main symptom of intussusception is severe, crampy abdominal pain alternating with periods of no pain. Painful episodes may last 10 to 15 minutes or longer, followed by periods of 20 to 30 minutes of no pain, after which the pain returns. Nausea, vomiting or rectal bleeding (red jelly-like stools), sometimes mixed with mucus, may also occur.

Pathophysiology:
In simple mechanical obstruction, blockage occurs without vascular compromise. Ingested fluid and food, digestive secretions, and gas accumulate above the obstruction. The proximal bowel distends, and the distal segment collapses. The normal secretory and absorptive functions of the mucosa are depressed, and the bowel wall becomes oedematous and congested. Severe intestinal distension is self-perpetuating and progressive, intensifying the peristaltic and secretory derangements and increasing the risks of dehydration and progression to strangulating obstruction.

Strangulating obstruction is obstruction with compromised blood flow; it occurs in nearly 25% of patients with small-bowel obstruction. It is usually associated with hernia, volvulus, and intussusception. Strangulating obstruction can progress to infarction and gangrene in as little as 6 hours. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall.
The ischemic bowel becomes oedematous and infarcts, leading to gangrene and perforation.
In large-bowel obstruction, strangulation is rare (except with volvulus).

Signs & Symptoms:
Obstruction of the small bowel causes symptoms shortly after onset:

- Abdominal cramps cantered around the umbilicus or in the epigastrium
- Vomiting
- In patients with complete obstruction – obstipation (severe constipation; neither stool nor gas can be passed)
- Patients with partial obstruction may develop diarrhoea.
- Severe, steady pain suggests that strangulation has occurred.

- In the absence of strangulation, the abdomen is not tender.
- Hyperactive, high-pitched peristalsis with rushes coinciding with cramps is typical. - Sometimes, dilated loops of bowel are palpable.
- With infarction, the abdomen becomes tender and auscultation reveals a silent abdomen or minimal peristalsis.
- Shock and oliguria are serious signs that indicate either late simple obstruction or strangulation.

Obstruction of the large bowel usually causes milder symptoms that develop more gradually than those caused by small-bowel obstruction.

- Increasing constipation leads to obstipation and abdominal distension.
- Vomiting may occur (usually several hours after onset of other symptoms) but is not common.
- Lower abdominal cramps unproductive of faeces occur.
- Physical examination typically shows a distended abdomen with loud borborygmi. There is no tenderness, and the rectum is usually empty. A mass corresponding to the site of an obstructing tumour may be palpable.

Investigations: Abdominal series; Supine and upright abdominal x-rays should be taken and are usually adequate to diagnose obstruction.
Abdominal CT is being used more often in suspected small-bowel obstruction.

Management:

- Nasogastric suction
- IV fluids
- IV antibiotics if bowel ischemia suspected

Patients with possible intestinal obstruction should be hospitalized. Treatment of acute intestinal obstruction must proceed simultaneously with diagnosis. A surgeon should always be involved.

Supportive care is similar for small- and large-bowel obstruction:

- Nasogastric suction, IV fluids (0.9% saline or lactated Ringer’s solution for intravascular volume repletion), and a urinary catheter to monitor fluid output.
- Electrolyte replacement should be guided by test results, but, in cases of repeated vomiting, serum sodium and potassium are likely to be depleted.
- If bowel ischemia or infarction is suspected, antibiotics should be given (e.g.: a 3rd-generation cephalosporin, such as cefotetan 2 g IV) before operative exploration.

Specific Measures

- Obstruction of the duodenum in adults is treated by resection or, if the lesion cannot be removed, palliative gastrojejunostomy.

- Complete obstruction of the small bowel is preferentially treated with early laparotomy, although surgery can be delayed 2 or 3 hours to improve fluid status and urine output in a very ill, dehydrated patient. The offending lesion is removed whenever possible.
- If a gallstone is the cause of obstruction, it is removed through an enterotomy, and cholecystectomy need not be done.
- Procedures to prevent recurrence should be done, including repair of hernias, removal of foreign bodies, and lysis of the offending adhesions.
- Obstructing colon cancers can sometimes be treated by a single-stage resection and anastomosis, with or without a temporary colostomy or ileostomy. When this procedure is not possible, the tumour may be respected, and a colostomy or ileostomy is created; the stoma may possibly be closed later.
- Fecal impaction usually occurs in the rectum and can be removed digitally and with enemas.

ACUTE PERFORATION OF THE GASTROINTESTINAL TRACT

Any part of the gastrointestinal tract may become perforated, releasing gastric or intestinal contents into the peritoneal space.
Mortality is high, varying with the underlying disorder and the patient's general health.

Etiology:
Both blunt and penetrating trauma can result in perforation of any part of the gastrointestinal tract.
Swallowed foreign bodies, even sharp ones, rarely cause perforation unless they become impacted, causing ischemia and necrosis from local pressure.
Foreign bodies inserted via the anus may perforate the rectum or sigmoid colon.

Signs & Symptoms:
Esophageal, gastric, and duodenal perforations tend to manifest suddenly and catastrophically, with abrupt onset of acute abdomen with severe generalized abdominal pain, tenderness, and peritoneal signs. Pain may radiate to the shoulder.
Perforation at other gastrointestinal sites often occurs in the setting of other painful, inflammatory conditions. Because such perforations are often small initially and frequently walled off by the omentum, pain often develops gradually and may be localized. Tenderness also is more focal. Such findings can make it difficult to distinguish perforation from worsening of the underlying disorder or lack of response to treatment.
In all types of perforation, nausea, vomiting, and anorexia are common.
Bowel sounds are quiet to absent.

Investigations:
Abdominal series, Abdominal CT, Barium should not be used if perforation is suspected

Management: Surgery, IV fluids and antibiotics
If a perforation is noted, immediate surgery is necessary because mortality caused by peritonitis increases rapidly the longer treatment is delayed. If an abscess or an inflammatory mass has formed, the procedure may be limited to drainage of the abscess. A nasogastric tube is sometimes inserted before operation. Patients with signs of volume depletion should have urine output monitored with a catheter. Fluid status is maintained by adequate IV fluid and electrolyte replacement. Broad-spectrum IV antibiotics effective against intestinal flora should be given.

COLORECTAL CANCER
Colorectal cancer (CRC) is extremely common; incidence rises sharply around age 40 to 50. Overall, more than half of the cases occur in the rectum and sigmoid, and 95% are adenocarcinomas. Colorectal cancer is slightly more common among men than women.

Etiology:
Colorectal cancer most often occurs as transformation within adenomatous polyps. About 80% of cases are sporadic, and 20% have an inheritable component. Predisposing factors include chronic ulcerative colitis and Crohn's colitis; the risk of cancer increases with the duration of these disorders.
Patients in populations with a high incidence of CRC eat low-fiber diets that are high in animal protein, fat, and refined carbohydrates.

Signs & Symptoms:
Colorectal adenocarcinomas grow slowly, and a long interval elapse before they are large enough to cause symptoms. Symptoms depend on lesion location, type, extent, and complications.

The right colon has a large calibre and a thin wall and its contents are liquid; thus, obstruction is a late event. Bleeding is usually occult. Fatigue and weakness caused by severe anemia may be the only complaints.
Tumours sometimes grow large enough to be palpable through the abdominal wall before other symptoms appear.

The left colon has a smaller lumen, the feces are semisolid, and cancer tends to cause obstruction earlier than in the right colon. Partial obstruction with colicky abdominal pain or complete obstruction may be the initial manifestation. The stool may be streaked or mixed with blood. Some patients present with symptoms of perforation, usually walled off (focal pain and tenderness), or rarely with diffuse peritonitis.

In rectal cancer, the most common initial symptom is bleeding with defecation. Whenever rectal bleeding occurs, even with obvious haemorrhoids or known diverticular disease, coexisting cancer must be ruled out.
Tenesmus or a sensation of incomplete evacuation may be present.
Pain is common with perirectal involvement.

Investigations:

- Colonoscopy
- Fecal occult blood testing
- Sometimes: Flexible sigmoidoscopy, Fecal DNA testing, CT colonography

Management:
Surgical resection, sometimes combined with chemotherapy, radiation, or both

APPENDICITIS
Appendicitis is an acute inflammation of the vermiform appendix.

Etiology:
Appendicitis is thought to result from obstruction of the appendiceal lumen, typically by lymphoid hyperplasia but occasionally by a fecalith, foreign body, or even worms. The obstruction leads to distension, bacterial overgrowth, ischemia, and inflammation. If untreated, necrosis, gangrene, and perforation occur. If the perforation is contained by the omentum, an appendiceal abscess results.

Signs & Symptoms:
Epigastric or periumbilical pain followed by brief nausea, vomiting, and anorexia
After a few hours, the pain shifts to the right lower quadrant.
Pain increases with cough and motion.

Diagnosis: Clinical evaluation, Abdominal CT scan, USG, Laparoscopy

Prognosis:

- Without surgery or antibiotics, the mortality rate for appendicitis is > 50%.
- With early surgery, the mortality rate is < 1%, and convalescence is normally rapid and complete.
- With complications (rupture and development of an abscess or peritonitis) and/or advanced age, the prognosis is worse: Repeat operations and a long convalescence may follow.

Management:

- Surgical removal of the appendix; open or laparoscopic appendectomy
- IV fluids and antibiotics

CROHN'S DISEASE
Crohn's disease is a chronic transmural inflammatory bowel disease that usually affects the distal ileum and colon but may occur in any part of the gastrointestinal tract.

Signs & Symptoms:

- Chronic diarrhoea with abdominal pain, fever, anorexia, and weight loss
- The abdomen is tender, and a mass or fullness may be palpable.

- Gross rectal bleeding is unusual except in isolated colonic disease, which may manifest similarly to ulcerative colitis.
- Some patients present with an acute abdomen that simulates acute appendicitis or intestinal obstruction.
- About 33% of patients have perianal disease (especially fissures and fistulas), which is sometimes the most prominent or even initial complaint.

Complications:
There is an increased risk of cancer in affected small-bowel segments. Patients with colonic involvement have a long-term risk of colorectal cancer equal to that of ulcerative colitis, given the same extent and duration of disease.
Chronic malabsorption may cause nutritional deficiencies, particularly of vitamin D and B12.

Investigations:

- Barium x-rays of the small bowel
- Abdominal CT scan
- Sometimes barium enema, magnetic resonance (MR) enterography, upper endoscopy, colonoscopy, and/or video capsule endoscopy
- Laboratory tests should be done to screen for anemia, hypoalbuminemia, and electrolyte abnormalities.

Prognosis:
Established Crohn's disease is rarely cured but is characterized by intermittent exacerbations and remissions. Disease-related mortality is very low.
GI cancer, including cancer of the colon and small bowel, is the leading cause of excess Crohn's disease-related mortality.
About 10% of people are disabled by Crohn disease and the complications it causes.

Treatment:
General:

- Rest
- High protein diet and vitamins supplement
- Stop smoking

Medical

- Immuno-suppressive therapy using Azathioprine and Infliximab (Monoclonal antibody) IV (help in closure of fistulae)
- Corticosteroids (Same as ulcerative colitis)
- Antibiotics for fistula and colitis while metronidazole to reduce anal and colonic pathology
- Acupuncture (In China)

Surgery:

- Resection: Ileo-caecal or segmental resection. In obstruction, perforation, intraabdominal abscess, internal fistulae, bleeding & malignancy
- Stricturoplasty: In stricture
- Colectomy with Ileo-rectal anastomosis

ULCERATIVE COLITIS

Ulcerative colitis is a chronic inflammatory and ulcerative disease arising in the colonic mucosa, characterized most often by bloody diarrhoea.

Pathophysiology:

Lesion in base of crypts of lieberkuhn → Crypt abscess → Pus in lieberkuhn → Abscess ultimately ruptures to form tiny ulcers → Proctitis and colitis → Multiple, small, irregular, shallow, superficial Pinpoint ulcer → Inflammation spread into submucosa of colon → Attempt of healing may produce polyp like structure (Pseudopolyposis) which is surrounded by heaped of granulation tissue & oedematous mucosa → Gut spasm → Epithelial hypertrophy healing with fibrosis resulting in narrow, contracted colon (Pipe stem colon) → Stricture of colon

Toxic Colitis:

Toxic colitis or fulminant colitis occurs when transmural extension of ulceration results in localized ileus and peritonitis. Within hours to days, the colon loses muscular tone and begins to dilate. Toxic colitis is a medical emergency that usually occurs spontaneously in the course of very severe colitis.

Signs & Symptoms:

- Attacks of bloody diarrhoea of varied intensity and duration interspersed with asymptomatic intervals
- Usually an attack begins insidiously, with increased urgency to defecate, mild lower abdominal cramps, and blood and mucus in the stools. Some cases develop after an infection (e.g.: amebiasis, bacillary dysentery).
- When ulceration is confined to the rectosigmoid, the stool may be normal or hard and dry, but rectal discharges of mucus loaded with red and white blood cells accompany or occur between bowel movements.
- If ulceration extends proximally, stools become looser and the patient may have > 10 bowel movements per day, often with severe cramps and distressing rectal tenesmus, without respite at night. The stools may be watery or contain mucus and frequently consist almost entirely of blood and pus.
- Toxic or fulminant colitis manifests initially with sudden violent diarrhoea, fever up to 40° C, abdominal pain, signs of peritonitis, and profound toxemia.
- Systemic symptoms and signs, more common with extensive ulcerative colitis, include malaise, fever, anemia, anorexia, and weight loss.

Investigation: Barium enema, Colonoscopy, Plain X-ray abdomen

Management:
Conservative:

1. Hospitalization & bed rest
2. Sedatives and tranquilisers with psychological counselling
3. Anti-diarrhoeal drugs like Lomotil etc.
4. Antibiotics: Salazopyrines 2ml/day. It is anti-microbial drug help in chronic cases
5. Corticosteroids:
   Oral prednisolone 60mg/day. Dose is tapered off over 3-4 weeks (they decreases frequency of stools)
   In acute attack IV hydrocortisone 100mg
   Prednisolone retention enema, 20mg in 200ml saline. Advice for 7-10 days
6. Cyclosporines: IV 4 mg/kg/day
7. Diet: It should be milk free. Avoid too hot & too cold items. Fruits are helpful. Vitamin A, B, C, D with supplements of iron and potassium.

II. Surgery:

1. Total proctocolectomy followed by permanent ileostomy and connected to ileostomy bag
2. Restorative proctocolectomy: Proctocolectomy with ileo-anal anastomosis with pouch as reservoir (J, S, W pouches)

DISEASES OF RECTUM AND ANAL CANAL

14. Diseases of Rectum and Anal Canal – Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital disorders, Arshas - Haemorrhoids, Parikartika - Fissure-in-ano, Bhagandara - Fistula-in-ano, Guda Vidradi - Anorectal abscesses, Gudabhramsa - Rectal prolapse, Sanniruddaguda - Anal stricture, Incontinence, Rectal Polyp and Tumours.

CONGENITAL DISORDERS

IMPERFORATE ANUS (PROCTOTRESIA) / ANORECTAL MALFORMATION (ARM)

The congenital defect of fusion between hindgut (post allantoic gut) and proctoderm leads to partial or complete obstruction of anus.

Post allantoic gut: Gives rise to rectum and upper 2 cm of anal canal

Proctoderm: Form lower part of anal canal below dentate line

Wingspread classification:

1. Low anomaly
2. High anomaly

Low Anomalies:

1. Covered Anus:
   Anus is covered by a tag of skin so that the anal opening is not situated in its normal position. The skin needs to be incised and anal dilatation is done.
2. Anterior Ectopic Anus:
   Anal opening is present anteriorly near the perineum.
   It is treated by a plastic cut back operation.
3. Anal Stenosis / Stricture:
   Congenital anal stenosis is narrowing of the anal opening and canal. It requires regular dilatation.
4. Membranous Anus:
   Anus is covered by a thin membrane. It is treated by cruciate incision followed by anal dilatation.

High Anomalies:

1. Anorectal Agenesis:
   The rectum ends above the pelvic floor and is usually connected with bladder or posterior fornix of vagina with fistulous connection. Anal canal is not developed.
2. Rectal Atresia:
   The anal canal is normal but ends blindly just below the pelvic floor, and the rectum ends just above the pelvic floor. There is no formation of fistula.

3. Cloaca:

It presents only in females. Bowel, urinary bladder and genital tract open into a single chamber.

Clinical features:

- Inability to pass meconium
- Features of obstruction
- Passing meconium per urethra
- Abdominal distension
- Improper anal dimple

Diagnosis:

- Diagnosis: Wangensteins invertogram:
- Should perform after 6-12 hours of birth (to collect sufficient air in large intestine)
- Child hold upside down (3-4 minutes), so that gas reaches rectum
- Metal coin kept over anus
- X-ray
- If Gas shadow: Above pubo-cocygeal line (High), Below pubo-cocygeal line (Low), Or difference between metal coin and shadow: >2.5 cm (High) <2.5 cm (Low)

Treatment:

Low anomaly: Dealt while explaining earlier

High: Initial colostomy, pull through operation through pubo-rectalis and anastomosis of rectal

Pouch to create anal canal. Closure of colostomy is done later.

ARSHAS

Arshas are protrusions of Māṁsa which obstructs Gudamārga and torture the person. According to Ā. Charaka, Arshas is formation of Aṅkura (muscular sprouts) in Gudavalli.

Paryāya: Gudāṅkura, Gudakīla, Māṁsakīla, Durnamaka, Anamaka, Payūroga

Nidāna:

- Viruddhāshana
- Adhyashana
- Strīprasaṅga (excessive coitus)
- Utkaṭukāsana (sitting for long time on irregular surfaces)
- Pṛṣṭhayāna (riding/travelling on the back of animals)
- Vegavidhāraṇa

Saṁprāpti: Nidāna sevana → Doṣa prakopa Rakta pradhāna → Gudavali Traya pradesha duṣṭi → Māṁsāṅkura → Arshas

Vargīkaraṇa:

1. According to Origin
a. Sahaja
b. Janmottara kālaja
2. According to General Character
a. Shuṣka (Vāta-Kaphaja)
b. Arda (Pitta-Raktaja)
3. According to Site
a. Bāhya (Arshas forming at Bāhyavali -> Saṁvariṇī)
b. Abhyantara (Arshas forming at Abhyantaravali -> Visarjinī & Pravahinī)
4. According to Doṣa
a. Vātaja
b. Pittaja
c. Kaphaja
d. Dvandvaja
e. Sannipātaja
f. Raktaja

Pūrvarūpa:
Anne ashraddhā, Kṛcchrāt pakti, Amlīkā, Paridāha, Viṣṭambha, Pipāsā, Sakthisadana, Āṭopa, Kārshya, Udgāra bāhulya, Akṣṇo Shvayathu, Antra kūjana, Guda parikartana, Pāṇḍuroga, Grahaṇīdoṣa, Shoṣa, Kāsa, Shvāsa, Balahāni, Bhrama, Tandrā, Nidranāsha, Indriya daurbalya

Lakṣhaṇa:

1. Vātaja Arshas
- Aṅkura appears dry and reddish (Rūkṣa Aruṇa)
- Arshas resembles Kadamba puṣpa
- Kaṭhina purīṣa
- Kaṭi-Pṛṣṭha-Pārshva-Vṛṣaṇa-Nābhī shūla
- Blackish discolouration of Nakha, Akṣi, Danta, Mukha, Mutra, Viṭ
- Gulma, Plīhā, Udara

2. Pittaja Arshas
- Aṅkura appears slender, with bluish tips and discharge
- Arshas resembles Jalaukavaktra
- Purīṣa pravṛtti Sarakta Sadāha
- Yellowish discolouration of Nakha, Akṣi, Danta, Mukha, Mutra, Viṭ
- Jvara, Dāha, Pipāsā, Mūrcchā

3. Kaphaja Arshas
- Aṅkura appears pale, broad at the base, rounded and does not discharge any fluid or blood
- Arshas resembles Gostana
- Whitish discolouration of Nakha, Akṣi, Danta, Mukha, Mutra, Viṭ
- Shopha, Shītajvara, Arochaka, Avipāka, Shirogaurava

4. Raktaja Arshas
- Arshas resembles Nyagrodha / Guñja
- Pittaja Lakṣaṇa
- Hard stool with significant amount of Duṣṭa Rakta

5. Sahaja Arshas

- Duṣṭa Shukra Shoṇita janya
- Durdarshana (difficult to see as it is deeply situated)
- Paruṣa, Pāṅshu (grey), Dāruṇa (severely painful)
- Kṛsha, Alpabhukta, Sirā santata gātra, Svarakṣaya, Āṭopa
- Ghrana-Nāsa-Akṣi-Shiroroga

Sādhyāsādhyatā:

1. Sādhya → Ekadoṣa, Bāhyavali sthita, < 1 year duration
2. Kṛcchrasādhya → Dvidoṣaja, Madhyamavali sthita, > 1 year duration
3. Yāpya → Tridoṣaja (Manda lakṣaṇa)
4. Asādhya → Tridoṣaja, Sahaja, Antarvali sthita, Upadrava

Asādyha Lakṣaṇa:
Tṛṣṇā, Aruchi, Shūla, Atishoṇita srāva, Shotha, Atisāra, Chardi, Jvara, Gudapāka, Sammoha

Upadrava: Udāvarta, Vāta-Viṭ-Mūtra saṅga, Nābhī-Pārshva-Ura shūla

Chikitsā:
Sthānika Chikitsā:

- Svedana & Abhyaṅga
- Avagāha Svedana with Kvātha, Uṣṇodaka, Gomūtra
- Raktamokṣaṇa with Shastra/Jalauka in Kaṭhina Arshas & Duṣṭa Rakta
- Arshoghna Lepa (Snuhī kṣīra and Haridrā chūrṇa)
- Haridrādi Lepa (Haridrā and Jālinī chūrṇa with Sarṣapa taila)

Bheṣaja:

- Guḍa Harītakī, Dashamūla Guḍa, Chaṅgeri Ghṛta
- Harītakī with Gomūtra, Shatāvarī mūla Kalka with Kṣīra
- Dashamūla kvātha, Pippalyādi yoga
- Abhayāriṣṭa, Dhātryariṣṭa, Dantyariṣṭa, Takrāriṣṭa
- Arshakuṭhāra rasa, Vyoṣādya chūrṇa, Chandraprabhā vaṭī,
- Takra, Takra with Pañchakola

Doṣa Chikitsā:

- Vātaja Arshas → Kakkolādya chūrṇa, Hiṅgvādi chūrṇa
- Pittaja Arshas → Dhattūrādi chūrṇa, Bhallātaka modaka,
- Kaphaja Arshas → Ghṛta prepared with Kvātha of Surasādi gaṇa & Dīpanīya Jalaukāvacharaṇa followed by Lepa with Arkapatra svarasa, Shuṇṭhī kvātha pāna
- Raktaja Arshas → Chirabilvādi chūrṇa, Pittaja Chikitsā, Raktapitta Chikitsā

Kṣārakarma:

- Patient is asked to lie in lithotomy position.
- The anal canal is well lubricated with Ghṛta (or xylocaine jelly nowadays).
- Arshoyantra (proctoscope) lubricated with Ghṛta is carefully introduced.
- The pile mass which is prolapsing into the lumen of the proctoscope is carefully observed and cleaned with a cotton swab.
- Using a scoop (Tala yantra), Kṣāra is applied over the pile mass, without allowing any leakage onto healthy mucosa.
- Kṣāra is left undisturbed for 100 Mātrakāla and Arshas is burned by Kṣāra.
- Observe Samyak Lakṣaṇa as Arshas takes on the colour of Pakva Jambu, reduction of mass, depression in mucous membrane instead of mass, and formation of a smooth ulcer.
- Kṣāra is removed from the pile mass by washing it with Dhānyamla, Dadhimastu or Amla phala svarasa.
- Ghṛta & Yaṣṭīmadhu are applied to the site.
- Kṣārakarma for each Arshas should be carried out at an interval of 7 days.
- (Kṣārasūtra with Haridrā chūrṇa and Snuhī kṣīra may also be done).

Shastrakarma & Agnikarma:

- Pile mass which has prolapsed needs to be excised. Afterwards, Agnikarma should be done.

HEMORRHOIDS / PILES

Hemorrhoids are dilated vessels of the hemorrhoidal plexus in the anal canal.
Increased pressure in the veins of the anorectal area leads to haemorrhoids.
This pressure may result from pregnancy, frequent heavy lifting, or repeated straining during defecation (e.g.: due to constipation).
Hemorrhoids may be external or internal. In a few people, rectal varices result from increased blood pressure in the portal vein, and these are distinct from haemorrhoids.

External haemorrhoids are located below the dentate line and are covered by squamous epithelium.
Internal haemorrhoids are located above the dentate line and are lined by rectal mucosa.
Hemorrhoids typically occur in the right anterior, right posterior, and left lateral zones.

Signs & Symptoms:

- Hemorrhoids are often asymptomatic, or they may simply protrude.
- External haemorrhoids may become thrombosed, resulting in a painful, purplish swelling.
  Rarely, they ulcerate and cause minor bleeding.
  Cleansing the anal region may be difficult.

- Internal haemorrhoids typically manifest with bleeding after defecation; blood is noted on toilet tissue and sometimes in the toilet bowl.
  Internal haemorrhoids may be uncomfortable but are not as painful as thrombosed external haemorrhoids.
  Internal haemorrhoids sometimes cause mucus discharge and a sensation of incomplete evacuation.
  Strangulated haemorrhoids occur when protrusion and constriction occlude the blood supply. They cause pain that is occasionally followed by necrosis and ulceration.

Diagnosis:

- Anoscopy
- Sometimes sigmoidoscopy or colonoscopy

Most painful haemorrhoids, thrombosed, ulcerated or not, are seen on inspection of the anus and rectum. Anoscopy is essential in evaluating painless or bleeding haemorrhoids.
Rectal bleeding should be attributed to haemorrhoids only after more serious conditions are excluded (i.e., sigmoidoscopy or colonoscopy).

Classification of Internal Hemorrhoids:

1. Grade I → No prolapse; Prolapse after a Valsalva manoeuvre
2. Grade II → Prolapse reduces spontaneously; Prolapse after Valsalva manoeuvre
3. Grade III → Prolapse needs manual reduction; Chronic prolapse
4. Grade IV→ Manual reduction of prolapse ineffective

Management:

- Symptomatic: Stool softeners, sitz baths, analgesics
- Occasionally excision for thrombosed external haemorrhoids
- Injection sclerotherapy, rubber band ligation, or infrared photocoagulation for internal haemorrhoids

Symptomatic Treatment:
Symptomatic treatment of haemorrhoids is usually all that is needed. It is accomplished with stool softeners (e.g.: docusate, psyllium), warm sitz baths (i.e., sitting in a tub of tolerably hot water for 10-15 minutes) after each bowel movement and as needed, anaesthetic ointments containing lidocaine, or witch hazel (hamamelis) compresses (which soothe by an unknown mechanism).
Pain caused by a thrombosed external haemorrhoid can be treated with non-steroidal anti-inflammatory drugs.
Infrequently, simple excision of the external haemorrhoid is done, which may relieve pain rapidly; after infiltration with 1% lidocaine, the thrombosed portion of the haemorrhoid is excised, and the defect is closed with an absorbable suture.

Office-based Procedures:
Patients with grades I and II internal haemorrhoids and some patients with grade III internal haemorrhoids who do not respond to symptomatic treatment can often be treated effectively with the following office-based procedures.

1. Injection sclerotherapy with 5% phenol in vegetable oil or other sclerosing agents can be used to treat bleeding internal haemorrhoids. Bleeding should cease at least temporarily.
2. Rubber band ligation is used for larger, prolapsing internal haemorrhoids, bleeding internal haemorrhoids, or those that do not respond to conservative management. With mixed internal and external haemorrhoids, only the internal component should be rubber band ligated. The internal haemorrhoid is grasped and withdrawn through a stretched ½-cm diameter band, which is released to ligate the haemorrhoid, resulting in its necrosis and sloughing. Typically, one haemorrhoid is ligated every 2 weeks; 3-6 treatments may be required.
   Sometimes, multiple haemorrhoids can be ligated at a single visit, but this may cause more pain. External haemorrhoids should not be banded.
3. Infrared photocoagulation is useful for ablating nonprolapsing, bleeding internal haemorrhoids, or haemorrhoids that are not cured with rubber band ligation.

Surgical Hemorrhoidectomy:
Surgical hemorrhoidectomy is required for patients who do not respond to other forms of therapy and for those who have grade IV internal haemorrhoids. Significant postoperative pain is common, as are urinary retention and constipation.
Stapled hemorrhoidopexy is an alternative procedure for circumferential haemorrhoids and causes less postoperative pain but has higher recurrence and complication rates than conventional surgical hemorrhoidectomy.

PARIKARTIKĀ
Excruciating cutting type of pain all around Guda, Basti and Nābhī is known as Parikartikā.

Paryāya: Kṣatapāyu, Kṣātaguda

Nidāna:

- Virechana Vyāpat → Ati-Rūkṣa-Tīkṣṇa-Lavaṇayukta Virechana Dravya given to a patient who has Mṛdukoṣṭha, Mandāgni or who is debiliated.
- Basti Vyāpat → Atyuṣṇa & Lavaṇa Basti given to Vāta-Pittaja Rogī

Lakṣhaṇa:

- Parikartana (excruciating cutting type of pain) in Guda, Basti, Nābhī and Meḍhra.
- Sadāha, Picchāsra (slimy-bloody discharge)
- Anila saṅga
- Vāyu viṣṭambha
- Aruchi

Chikitsā:

- Dīpana Pāchana, Vātānulomana, Vāta-Pitta shamana, Bāhya Shīta Lepana
- Picchābasti mixed with Yaṣṭīmadhu kalka, Kṛṣṇa taila, Madhu & Ghṛta
- Anuvāsana Basti with Yaṣṭīmadhu Siddha Ghṛta in Pittolbana Parikartikā
- Anuvāsana Basti with Yaṣṭīmadhu Siddha Ghṛta in Vātolbana Parikartikā
- Shītāmbu Pariṣeka
- Kṣīrapāna
- Jātyādi Ghṛta Pichu
- Agnituṇḍī vaṭī, Chitrakādi vaṭī, Triphalā guggulu

FISSURE IN ANO / ANAL FISSURE / ANAL ULCER

An anal fissure is an acute longitudinal tear or a chronic ovoid ulcer in the squamous epithelium of the anal canal.

Anal fissures are believed to result from laceration by a hard or large stool or from frequent loose bowel movements. Constipation. Repeated child birth. Ischemia.

Trauma (e.g.: anal intercourse) is a rare cause.

The fissure may cause internal sphincter spasm, decreasing blood supply and perpetuating the fissure.

Signs & Symptoms:

- Anal fissures usually lie in the posterior midline but may occur in the anterior midline.
- Those off the midline may have specific etiologies, particularly Crohn's disease.
- An external skin tag (the sentinel pile) may be present at the lower end of the fissure, and an enlarged (hypertrophic) papilla may be present at the upper end.
- Fissures cause pain and bleeding. The pain typically occurs with or shortly after defecation, lasts for several hours, and subsides until the next bowel movement.

Diagnosis: Clinical evaluation / Inspection

Management:

- Stool softeners
- Protective ointments, sitz baths
- Nitroglycerin ointment, topical calcium channel blocker, or botulinum toxin type A injection

  Fissures often respond to conservative measures that minimize trauma during defecation (e.g.: stool softeners, psyllium, fiber).
  Healing is aided by use of protective zinc oxide ointments or bland suppositories (e.g.: glycerin) that lubricate the lower rectum and soften stool.
  Topical anesthetics (e.g.: benzocaine, lidocaine) and warm (not hot) sitz baths for 10-15 minutes after each bowel movement and as needed give temporary relief.

Topical nitroglycerin 0.2% ointment, nifedipine cream 0.2%, 2% diltiazem gel, and injections of botulinum toxin type A into the internal sphincter relax the anal sphincter and decrease maximum anal resting pressure, allowing healing.
When conservative measures fail, surgery (internal anal sphincterotomy) is needed to interfere with the cycle of internal anal sphincter spasm.

BHAGANDARA

Bhagandara is the condition in which there will be Dāraṇa (splitting with severe pain) of Bhaga (vagina), Guda or Basti.
If there is no opening, it is called Bhagandara Piḍikā. It is a deep rooted Piḍikā around the anus in 2 aṅgula circumference associated with pain and fever. If Piḍikā ruptures, Bhagandara is formed.

Nidāna:

- Hasti-Ashva Pṛṣṭha gamana (riding on the back of horses or elephants)
- Kaṭhina Utkaṭaka Asana (sitting for long periods on hard and uneven surfaced)
- Arsha Nidāna
- Pūrvajanma Pāpa

Saṁprāpti:
Nidāna → Piḍika formation, 1 or 2 aṅgula from anus; internally (Arvachina/Antarmukha) or externally (Prachina/Bahirmukha) → Rakta Māṁsa vikṛti → Nāḍīvraṇa → Pūya srāva → Dāraṇa → Bhagandara

Bheda: (Ā. Sushruta = 5; Ā. Vāgbhaṭa = 8)

1. Shataponaka (Vātaja)
2. Uṣṭragrīva (Pittaja)
3. Parisrāvi (Kaphaja)
4. Shambukāvarta (Sannipātaja)
5. Unmārgi (Āgantūja)
6. Parikṣepi (Vāta-Pittaja)
7. Ṛju (Vāta-Kaphaja)
8. Arsho-Bhgandara (Pitta-Kaphaja)

Pūrvarūpa: Kaṭī-Kapāla vedanā, Kaṇḍū, Dāha, Guda Shopha

Lakṣhaṇa:

1. Shataponaka (Vātaja) Bhagandara:

- Aruṇa varṇa Piḍikā, Todādi Vedanā
- multiple openings like a sieve occur if Piḍikā ruptures
- Vishāda Phenila Srāva
- Tādana, Bhedana, Chedana, Ruja
- Gudavraṇa
- If left untreated, Vāta-Mūtra-Purīṣa-Retasa Srāva from multiple openings

2. Uṣṭragrīva (Pittaja) Bhagandara:
- Rakta Tanu Piḍikā, Dāha
- If Piḍikā ruptures, it produces Agni-Kṣāravat Dāha
- Gudavraṇa with Durgandha Uṣṇa Srāva

3. Parisrāvi (Kaphaja) Bhagandara:
- Shukla Sthira Piḍikā, Kaṇḍū
- If Piḍikā ruptures, Guḍavraṇa occurs with Picchila Srāva and Kaṇḍū

4. Shambukāvarta (Sannipātaja) Bhagandara:
- Piḍikā resembles Gostana
- Toda, Dāha, Kaṇḍū
- Gudavraṇa is curved and deep
- Srāva has various colours

5. Unmārgi/Kṣātaja (Āgantūja) Bhagandara:
- Bones which were ingested along with meat do not get digested and mix with faecal matter, causing trauma to Guda
- Kotha and Kṛmi develop which destroy the rectal wall and produce multiple openings

6. Parikṣepi (Vāta-Pittaja) Bhagandara:
- A circular fistula around the anus is formed
- Shyāva Tāmra varṇa
- Dāha, Oṣa, Ghora ruja

7. Ṛju (Vāta-Kaphaja) Bhagandara:
- Straight track is formed
- Kṛcchrapāka
- Pāṇḍu, Kiñchit Shyāva

8. Arsho-Bhgandara (Pitta-Kaphaja) Bhagandara:
- Shopha, Kaṇḍū, Dāha
- Shīghra Pāka, Arshasa mūla Kledayan

Sādhyāsādhyatā:
1. Kṛcchrasādhya → Ekadoṣaja, Dvidoṣaja
2. Asādhya → Tridoṣaja, Unmārgi

Chikitsā:

- Balavān Rogī → Virechana → Eṣaṇa → Pāṭana → Shodhana → Taila dāha → Vraṇavat Chikitsā
- Daurbalya Rogī → Eṣaṇa → Kṣārasūtra → Vraṇavat Chikitsā
- Bhagandara Piḍikā → Jalaukāvacharaṇa
- Bhagandara → Chedana → Kṣārakarma / Agnikarma
- Bhagandharanāshaka taila, Madhuyaṣṭyādi taila
- Viḍaṅgādi Leha, Guḍūchyādi Leha
- Shigru guggulu, Abhayāriṣṭa
- Āragvadhādi varti (Āragvadha, Haridrā, Tagara, Madhu & Ghṛta)
- Apathya → Vyāyāma, Maithuna, Krodha, Prṣṭāyana, Guru Āhāra

FISTULA IN ANO / ANORECTAL FISTULA

An anorectal fistula is a tubelike tract with one opening in the anal canal and the other usually in the perianal skin

Fistulas arise spontaneously or occur secondary to drainage of a perirectal abscess. Most fistulas originate in the anorectal crypts.

Other causes include Crohn ‘s disease, Tuberculosis, Diverticulitis, Cancer, and Trauma.

Fistulas in infants are congenital and are more common among boys.

Rectovaginal fistulas may be secondary to Crohn’s disease, obstetric injuries, radiation therapy, or cancer.

Signs & Symptoms:

- A history of recurrent anorectal abscess followed by intermittent or constant discharge is usual.
- Discharge material is purulent, serosanguineous, or both.
- Pain may be present if there is infection.
- On inspection, one or more secondary openings can be seen.
- A cordlike tract can often be palpated.

Diagnosis:

- Clinical evaluation
- Sometimes anoscopy, sigmoidoscopy, or colonoscopy

Goodsall’s rule:

If external opening is anterior to imaginary line drawn across the midpoint of anus, the fistula runs straight directly in to anal canal.

If external opening is situated posterior to that line, the track usually will curve and internal opening will be on midline posterior of anal canal.

Exception to this rule is when external opening is anterior to imaginary line but situated more than 1.5 inches/3.75cms away from anus. In this case track will curve posteriorly and end in posterior midline.

Management:
Various surgical procedures
Medical treatment if caused by Crohn's disease

In the past, the only effective treatment was surgery, in which the primary opening and the entire tract are unroofed and converted into a "ditch."
Partial division of the sphincters may be necessary. Some degree of incontinence may occur if a considerable portion of the sphincteric ring is divided.
Alternatives to conventional surgery include advancement flaps, biologic plugs, and fibrin glue instillations into the fistulous tract.
More recently, the ligation of intersphincteric fistula tract (LIFT) procedure, where the fistula tract is divided between the sphincter muscles, has gained acceptance as an alternative more likely to preserve continence.

If diarrhoea or Crohn's disease is present, fistulotomy is inadvisable because of delayed wound healing.

For patients with Crohn disease, metronidazole, other appropriate antibiotics, and suppressive therapies can be given. Infliximab is effective in closing anal fistulas caused by Crohn's disease.

ANORECTAL ABSCESS
An anorectal abscess is a localized collection of pus in the perirectal spaces.
Abscesses usually originate in an anal crypt.

An abscess may be in various spaces surrounding the rectum and may be superficial or deep.
A perianal abscess is superficial and points to the skin.
An ischiorectal abscess is deeper, extending across the sphincter into the ischiorectal space below the levator ani; it may penetrate to the contralateral side, forming a "horseshoe" abscess.
An abscess above the levator ani (i.e., supralevator abscess) is quite deep and may extend to the peritoneum or abdominal organs; this abscess often results from diverticulitis or pelvic inflammatory disease.

Crohn's disease (especially of the colon) sometimes causes anorectal abscess.
A mixed infection usually occurs, with Escherichia coli, Proteus vulgaris, Bacteroides, streptococci, and staphylococci predominating.

Signs & Symptoms:

- Superficial abscesses can be very painful; perianal swelling, redness, and tenderness are characteristic. Fever is rare.
- Deeper abscesses may be less painful but cause toxic symptoms (e.g.: fever, chills, malaise). There may be no perianal findings, but digital rectal examination may reveal a tender, fluctuant swelling of the rectal wall.

- High pelvirectal abscesses may cause lower abdominal pain and fever without rectal symptoms. Sometimes fever is the only symptom.

Diagnosis:

- Clinical evaluation
- Sometimes examination under anesthesia or rarely CT
  CT scan is useful when a deep abscess or Crohn's disease are suspected.
  Higher (supralevator) abscesses require CT to determine the intra-abdominal source of the infection.
  Patients with any findings suggestive of a deeper abscess or complex perianal Crohn 's disease should have an examination under anesthesia at the time of drainage.

Management:

- Incision and drainage (I & D)
- Antibiotics for high-risk patients

GUDABHRAṀSHA

Gudabhraṁsha is displacement of Guda from its normal site.

Nidāna:

- Pravāhaṇa (excessive straining during defecation)
- Atisāra
- Dehasya Rūkṣa Durbala

Lakṣhaṇa: Prolapse of Guda

Chikitsā:

- Snehana & Svedana of Guda and repositioning it to its normal site.
- Gophana Bandha (T-bandage)
- Chaṅgeri Ghṛta pāna

RECTAL PROLAPSE & PROCIDENTIA

Rectal prolapse is painless protrusion of the rectum through the anus.
Procidentia is complete prolapse of the entire thickness of the rectum.
Transient, minor prolapse of just the rectal mucosa often occurs in otherwise normal infants.
Mucosal prolapse in adults persists and may progressively worsen.
The primary cause of procidentia is unclear. Most patients are women > 60.

Signs & Symptoms:
The most prominent symptom of rectal prolapse and procidentia is protrusion.
It may only occur while straining or while walking or standing.
Rectal bleeding can occur, and incontinence is frequent.
Pain is uncommon unless incarceration or significant prolapse occurs.

Diagnosis:

- Clinical evaluation
- Sigmoidoscopy, colonoscopy, or barium enema
  To determine the full extent of the prolapse, the clinician should examine the patient while the patient is standing or squatting and straining.
  Rectal procidentia can be distinguished from haemorrhoids by the presence of circumferential mucosal folds. Anal sphincter tone is usually diminished.
  Sigmoidoscopy, colonoscopy, or barium enema x-rays of the colon must be done to search for other disease.
  Primary neurologic disorders (e.g.: spinal cord tumours) should be considered.

Management:

- Elimination of causes of straining
- For infants and children: Sometimes strapping buttocks together
- For adults: Usually surgery

In infants and children, conservative treatment is most satisfactory. Causes of straining should be eliminated. Firmly strapping the buttocks together with tape between bowel movements usually facilitates spontaneous resolution of the prolapse.

For simple mucosal prolapse in adults, the excess mucosa can be excised.
For procidentia, rectopexy, in which the rectum is mobilized and fixed to the sacrum, may be required in patients who can tolerate a laparotomy.
In patients who cannot tolerate a laparotomy, perineal operations (e.g.: Delorme or Altemeier procedure) can be considered.

SANNIRUDDHA GUDA

Sanniruddha Guda is mentioned under Kṣudra Roga by Ā. Sushruta and Ā. Vāgbhaṭa.
It is narrowing of the anal passage causing difficulty in defecation. Hence, it can be compared with anal stenosis.

Saṁprāpti: Vegavarodha → Vata vikruti → Guda ashraya→ Obstruct mahasrotas → Narrowing of ano-rectal lumen (Vagbhata: from both externally and internally) → Difficulty in defecation → Sannirudha guda

Chikitsā: Repeated dilatation in interval of 3 days is done with increasing size of Loha Nāḍī Yantra covered with Ghṛta. Pariṣeka with Vātahara Kvātha & Taila.

Anal stricture
Stricture: Narrowing of lumen

Causes:

1. Spasmodic: Chronic anal fissure (fibrosis) and habit of taking purgatives
2. Congenital
3. CA
4. Senile
5. Post-op: Haemorrhoidectomy
6. IBD (Inflammatory bowel disease): Ulcerative colitis and Crohn's disease.
7. Irradiation: After 1 to 2 yrs
8. Lymphogranuloma inguinale: A sexually transmitted disease affecting female patients. Initially para-rectal lymph nodes are enlarged followed by development of rectal stricture.

Clinical features:

1. Progressive constipation
2. Strip shape hard stool with pain and bleeding
3. Per abdomen: Loaded colon with scybalous mass (mass of hard faeces)
4. Rectal examination: Stricture as tight ring.

Investigation: Barium enema x-ray and Colonoscopy

Treatment:

1. Treat the cause
2. Bulk purgatives and veg diet
3. Regular dilatation (under G.A)
4. Resection in severe and recurrent condition
5. Anoplasty (Anal reconstruction)

ANAL INCONTINENCE

Definition:

Inability to hold faeces in rectum due to failure of voluntary control over the anal sphincter is called as anal incontinence.

Mechanism of anal incontinence:

Distension of rectum causes tonic contraction of anal sphincter. This is controlled by cerebrum and the centre is in the lumbosacral region of spinal cord. Faeces in contact with anal canal stimulate the specialised nerve endings. High pressure in the anal canal (25 - 120 mm of Hg) and angle between rectum and anal canal (80°) are the important factors which maintain anal continence.

Types:

1. Partial: A person loses only small amount of liquid waste.
2. Complete: The entire solid bowel movement cannot be controlled.

Causes: 5D

1. Denervation: Spinal injury, spina bifida
2. Damage: Childbirth (Pudendal nerve damage due to chronic straining), wounds. Surgeries like Lord's anal dilatation etc
3. Descent: Rectal prolapse (temporary), perineal descents
4. Debility: Old age, malnutrition
5. Destruction: Radiotherapy, Malignancy

Treatment:
Temporary incontinence:

- Reassurance
- Perineal exercise to improve the tone of internal and external sphincter
- Kegel exercise: Strengthen pelvic floor muscle

Permanent incontinence:

- Treat the cause
- Sphincteroplasty
- Sphincter replacement
- Dynamic graciloplasty: Gracilis muscle can be used to create a new anal sphincter by transposing it followed by electrical stimulation using a pacemaker
- Colostomy

RECTAL POLYP

These are tissue growth that arise from the wall of rectum and protrude into it.

Cause: Unknown

- Diet high in animal fat, red meat and low in fibre encourage polyp formation.
- Some are hereditary
- IBD

Clinical features:

- Mostly asymptomatic and are discovered on routine digital or endoscopic examination of rectum.
- Rectal bleeding
- Abdominal cramp
- Pain when prolapsed
- Mucus filled or watery diarrhoea

Diagnosis:
Proctoscopy, Sigmoidoscopy, Colonoscopy

Treatment: Polypectomy

- Polyps should be removed completely with a snare or biopsy forceps during total colonoscopy. If colonoscopic removal is unsuccessful, laparotomy should be done.
- Follow-up surveillance colonoscopy

CARCINOMA OF RECTUM

Incidence:

1. Colorectal CA is the fourth most common variety of malignant tumour found in women.
2. It is the second most CA in western countries.
3. It is common in females.
4. Usually originates from a pre-existing adenoma or papilloma
5. In 3% of cases, it occurs in multiple sites (synchronous)
6. Any tumour within 15 cm proximal to anal margin can say as rectal cancer

Aetiology:

Precancerous condition	Risk factors:
1. FAP (Familial adenomatous polyp) 2. Villous adenoma 3. Ulcerative colitis 4. Crohn's disease	1. Diet: Red meat and saturated fatty acids (oil material) 2. Smoking and alcohol 3. Family history of rectal cancer 4. Risk of developing other cancers like 5. Endometrium (40%), Stomach (20%), Billiary tree (20%, Ovary (10%)

Clinical feature:

1. Bleeding Per anal: Earliest symptom
    - Occurs with stool or at end of defecation or stained underclothing.
    - Painless (Mimics with Haemorrhoids)
2. Early morning spurious diarrhoea: Due to accumulation of mucus overnight in ampulla of rectum which causes an urgency to pass stool but results in only mucus with minimal stools (cauliflower growth)
3. Sense of incomplete defecation (growth at lower half of rectum)
4. Tenesmus (painful incomplete defecation are with bleeding) which is common with stricturous growth.
5. Bloody slime: Blood mixed with mucus
6. Alteration in bowel habit:
    - Constipation: Annular growth in rectosigmoid junction
    - Early morning diarrhoea: Annular growth in ampulla (cauliflower growth)

Staging of CA:

Dukes Staging:

Stage A: Growth limited to rectal wall (mucosa and submucosa)

Stage B: Growth has extended beyond rectal wall but no involvement of regional lymph nodes.

Stage C: Lymph nodes involved

TNM Classification:

Tumour	Lymph node	Metastasis
T_0: No Primary tumour	N_0: No node spread	M_0: No distinct spread
T_{1s}: CA in situ	N_1: 1-3 nodal spread	M_1: Distant spread present
T_1: Invasion to submucosa	N_2: 4 or more spread	
T_2: Invasion to muscularis propria		
T_3: Invasion to non-peritonealised peri rectal tissue		
T_4: Involvement of visceral peritoneum, other organs or structures		

Investigation:

1. Proctoscopy
2. Sigmoidoscopy
3. Barium enema
4. Colonoscopy
5. TRUS (Transrectal ultrasound)
6. EUS (Endorectal ultrasonography)
7. USG
8. CT scan

Treatment:

Radiotherapy: Neutron beam radiation is used in dose of 4000-5000 cGy units (when growth is below peritoneal reflection)

Types:

1. Pre-op radiotherapy: Indicated when tumour is extended through bowel wall. It reduces size of tumour and thus tumour may be operable. Dose is 45cGy units.
2. Post-op radiotherapy: To reduce local reoccurrence
3. Papillons interactive radiation: Indicated for small, localized, well differentiated and exophytic cancer as curative radiotherapy. Dose is 4000-5000 cGy units in 3 minutes.

Chemotherapy:

- Injection 5FU (5 fluorouracil) IV for 5 days with injection leucovorin (immunomodulator) for 5 days
- 3 such courses are given at 4 weekly intervals

Surgery:

- HAR (High Anterior Resection): Growth between I I-15cm from anal verge
- APR (Abdomino-Perineal Resection): Growth within 7cm from anal verge
- LAR (Low Anterior Resection): Growth between 7-1 lcm from anal verge
- Hartmon's operation: Indicated in old and debilitated patients who may not withstand APR

15. Abdominal injuries and their management.

The abdomen can be injured in many types of traumas; injury may be confined to the abdomen or be accompanied by severe, multisystem trauma. The nature and severity of abdominal injuries vary widely depending on the mechanism and forces involved, thus generalizations about mortality and need for operative repair tend to be misleading.

Types: Injuries are often categorized by type of structure that is damaged:

1. Abdominal wall
2. Solid organ (liver, spleen, pancreas, kidneys)
3. Hollow viscus (stomach, small intestine, colon, ureters, bladder)
4. Vasculature

Etiology: Abdominal trauma is typically also categorized by mechanism of injury:

1. Blunt
2. Penetrating

Blunt trauma may involve a direct blow (eg, kick), impact with an object (e.g.: fall on bicycle handlebars), or sudden deceleration (e.g.: fall from a height, vehicle crash). The spleen is the organ damaged most, followed by the liver and a hollow viscus (typically the small intestine). Penetrating injuries may or may not penetrate the peritoneum and if they do, may not cause organ injury. Stab wounds are less likely than gunshot wounds to damage intra-abdominal structures; in both, any structure can be affected. Penetrating wounds to the lower chest may cross the
diaphragm and damage abdominal structures.

Classification:
Injury scales have been devised that classify organ injury severity from grade 1 (minimal) to grades 5 or 6 (massive); mortality and need for operative repair increase as grade increases.

1. Grade 1 → Minor / Minimal
2. Grade 2 → Moderate
3. Grade 3 → Serious
4. Grade 4 → Severe
5. Grade 5 → Critical
6. Grade 6 → Maximum / Massive

Complications:

- Delayed consequences of abdominal injury include:
- Hematoma rupture
- Intra-abdominal abscess
- Bowel obstruction or ileus
- Biliary leakage and/or biloma
- Abdominal compartment syndrome
- Abscess, bowel obstruction, abdominal compartment syndrome, and delayed incisional hernia also can be complications of treatment.

Signs & Symptoms:

- Abdominal pain typically is present; however, pain is often mild and thus easily obscured by other, more painful injuries (e.g.: fractures) and by altered sensorium (e.g.: due to head injury, substance abuse, shock).
- Pain from splenic injury sometimes radiates to the left shoulder.
- Pain from a small intestinal perforation typically is minimal initially but steadily worsens over the first few hours.
- Patients with renal injury may notice hematuria.
- On examination, vital signs may show evidence of hypovolemia (tachycardia) or shock (eg, dusky color, diaphoresis, altered sensorium, hypotension).
- Not all penetrating abdominal injuries originate from wounds on the abdominal wall; entrance wounds may be located on the back, buttocks, flank, perineum, and lower chest.

Diagnosis:

- Clinical evaluation
- USG, CT scan, X-ray
  Ultrasonography is the first imaging method for screening patients with blunt abdominal trauma. It can demonstrate variety of post traumatic abdominal organ pathologies including hematomas, contusions, lacerations, and hemoperitoneum.

- Diagnostic laparoscopy or exploratory laparotomy
- As in all patients experiencing significant trauma, clinicians do a thorough, organized trauma evaluation simultaneous with resuscitation. Because many intra-abdominal injuries heal without specific treatment, the clinician's primary goal is to identify injuries requiring intervention.

General Management:

- Stabilizing the patient by ensuring adequate airway, breathing and circulation
- Surgery to repair damaged organs
- Laparotomy in blunt traumas
- Blood transfusion if necessary
- IV fluids
- Management of shock

SHALYATANTRA

PAPER 2

PART B

DISEASES OF LIVER

1. **Diseases of Liver:** Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Yakrit Vidradhi - Abscess, Neoplasia, Portal hypertension and Yakritdalyodar –Hepatomegaly.

LIVER ABSCESS

Definition: It is pus filled mass inside the liver.

Types:

1. Pyogenic liver abscess (800/0)
2. Amoebic liver abscess (10%)
3. Fungal abscess (less than 100/0)

1. Pyogenic liver abscess:

Incidence:
It is more common in elderly diabetics, alcoholic and immune-suppressed (TB, HIV etc) persons.
M: F = 2: 1

Causes:

- Bacteroides fragilis
- E. coli, Klebsiella pneumoniae
- Staphylococcus aureus
- Hepatic trauma
- Sub – diaphragmatic abscess
- Emphysema thoracis
- Stricture of common bile duct
- Acute appendicitis
- Acute diverticulitis
- Ulcerative colitis
- Neonatal umbilical sepsis

Clinical factors:
Multiple abscess → acute condition
Single abscess → chronic condition
Shooting type fever with chills and sweating
Pain
Nausea
Vomiting
Anorexia
On palpation → enlarged and tender liver

Investigation:
Blood examination → Leucocytosis, increased alkaline phosphate
USG
CT Scan
FNAC: Yellow coloured pus

Treatment:
Conservative: Antibiotics like cephalosporines, metronidazole with analgesics and antipyretics
Percutaneous drainage
Open (surgery)

2. Amoebic liver abscess:

Incidence:
M: F = 10: 1
Common age is 20-40 years
More in male alcoholic
More in low socio-economic peoples

Etiopathology:
As a complication of amoebic dysentery
Organism: Entamoeba histolytica
Entamoeba histolytica present in colonic lesion (harmless) → Through portal vein it enters in to liver → Causes destruction of hepatocyte by releasing cyto-toxic enzyme resulting in liquefactory necrosis → Infection begins with intra-hepatic portal thrombosis and infarction → Coalescence of such small necrotic area results in formation of large single abscess.

Symptoms:
Severe pain in right hypochondrium
High grade fever with chills and rigors
Profuse sweating
Weakness, anorexia
Thoracic symptoms like non-productive cough and shoulder pain when abscess presents in superior surface

Signs:
Enlarged & tender liver in right hypochondrium
Anaemia
Emaciation & toxic look
Intercostals tenderness (DD with acute cholecystitis)
Anchovy sauce pus → chocolate brown (mixture of broken RBC, hepatocytes, and broken liver cells.)

Investigation:
Blood examination: Leucocytosis and anaemia
Serologic test: Indirect haemagglutination test positive
Stool examination: For ova and cyst of entamoeba histolytica
USG: To locate site of abscess and to aspirate the pus.
Treatment:
Conservative:
Tab metronidazole 400-800 mg TDS for 14 days

Analgesics
If not improved then inj. emetine l mg/kg body weight. Total 60mg/day deep 1M for 6 days

Needle aspiration:
Choice:
When metronidazole is contraindicated like in 1st trimester of pregnancy.
Can repeat if pus collects

Indication:
Single abscess
Uncertain diagnosis
Abscess with high risk of rupture
Failure to respond to antibiotics in 3-5 days

Position: Semi-reclined or supine

Anaesthesia: Local anaesthesia (5ml of 2% lignocaine infiltrated from skin to peritoneum, after
cleaning the selected area with antiseptic solution)

Procedure:

- Aspiration can be done with large bore needle no 12 to 18 or vein flow no 18 or by lumbar puncture needle.
- 50cc syringes attached to needle with three-way canula.
- Needle inserted at the site, depth and direction is noted by sonography guidance.
- Amount and character of pus noted and pus sent for culture and sensitivity through antiseptic technique.
- Puncture site is sealed with benzoin plug.
- Patient shifted to ward and kept under observation.
- Post procedure chest x ray is done.

Follow up:
At the end of first week, and then 1^{st}, 2^{nd}, 3^{rd} month after aspiration.

Surgery:
Indications:
USG failure FNAC
Ruptured abscess with peritonitis

Procedure:

1. Laparotomy
2. Abscess identification
3. Contents evacuation
4. Thorough peritoneal wash
5. Keep self-retaining Malecot's catheter which is connected to bag outside

Post operative:
Up to 3 to 5 (lays, necrotic liver tissue, chocolate pus and blood drained out
Once draining becomes minimal, pulled out catheter.

Complication:

- Bronchus rupture
- Pleural rupture
- Pericardial space rupture
- Peritoneum rupture

YAKRUT VIDRADHI
Symptoms:
Shwasa, trishna

Chikitsa:
Ushakadi gana / Varunadi gana kashaya

Hydatid cyst (यकृत पूटि / ग्रन्थि):
Definition:
Watery fluid cavity in liver.

Organism:
Echinococcus granulosus (dog tapeworm)

Pathology:
Dog (Tapeworm in jejunum) → passes in to stool (ova viable for weeks in stool & soil) → eaten by cow's & sheep → human eats them → ova swallowed in to stomach→ it penetrates gastric mucosa (so no lesion in colon) → reach retro-peritoneal structure → penetrate portal vein → enter liver → organism grow & developed own protective layer → cyst

Incidence:
More in sheep rearing area like Australia, America, New Zealand etc
Less in India
It may present in lungs, kidney, spleen & brain with liver

Layers of hydatid cyst:

1. Adventia: Given by host to ghost, adhere to liver, cannot separated
2. Ectocyst: Layer which get peeled off during surgery, white & elastic produced by endocyst
3. Endocyst: Secrets hydatid fluid inside & ectocyst outside.

Clinical features:

- Asymptomatic, accidently found on routine examination
- Pain in upper abdomen, which is dragging in nature due to hepatomegally
- Liver is enlarged, non-tender with smooth surface
- **Hydatid thrill:** Rare, keep 3 finger over liver, percuss over mid finger & get impulse by other two fingers

Investigations:

- USG to detect and aspirate cyst
- Plain x-ray abdomen shows speckled calcification
- CT scan/ ERCP

Treatment:
Calcified cyst/dried cyst & asymptomatic cysts are left alone
Symptomatic & size more than 5cms are treated
Try Albendazole/Fluconazole 400 mg BD for 6 weeks, if no improvement then goes for surgery.

Surgery:
Laparotomy & isolation of cyst
Aspirate contents & inject scolicidal agent like savlon
Incise cyst, peel off ectocyst
Inj. hydrocortisone before & after surgery to avoid anaphylactic shock

NEOPLASM OF LIVER
Classification:

1. Benign
2. Cancerous / malignant

1. Benign:

a. Haemangioma
b. Hepatic adenoma
c. Focal nodular hyperplasia

a. Haemangioma:

These are the most common type of benign liver tumour, found in up to 7% of autopsy specimens. This tumour is more commonly seen in females than in males (5: 1). They start in blood vessels. The compressibility of the tumour and bruit heard on the lump can make the diagnosis possible. Most of these tumours do not cause symptoms and do not need treatment. Some may bleed and need to be removed.

b. Hepatic adenoma:

They occur in young women due to excessive use of OCP. These benign epithelial liver tumours are in most cases located in the right hepatic lobe and are frequently seen as solitary The size of adenomas ranges from I to 30 cm. The prognosis of these tumours has still not mastered. The main complications are haemorrhage and necrosis causing rupture.

c. Focal nodular hyperplasia:

It is the second most common tumour of liver. It is 8 times more common in females. This tumour is the result of arteriovenous malformation hepatocyte response. This process is one in which all normal constituents of the liver arc present, but the pattern by which there are presented is abnormal.

2. Cancerous / malignant tumous:
a. Hepatocarcinoma (hepatoma) – 80 %
b. Cholangiocarcinoma – 20 %

a. Hepatocarcinoma:

Hepatomas arc about 8 times more common in men than in women. The predisposing factors are hepatitis B, alcoholic cirrhosis (60%), haemochromatosis and parasitic infestation with the liver fluke clonorchis sinensis (More in China and Japan).
The most common symptoms arc weakness, malaise, upper abdominal pain, and weight loss. Hepatomegaly is the only diagnostic criteria clinically. The liver is hard, irregular but not tender.
Treatment includes excision (lesion must be localized with no metastasis) and chemotherapy (5-FU). Radiotherapy has not achieved good results.

b. Cholangiocarcinoma:

Carcinoma arising from the small ducts or ductules is often associated with cirrhosis, haemochromatosis, chronic cholestasis and congenital cystic diseases of liver.
Women are more affected in the ratio of 2: l. Vague abdominal pain, fever, pruritus, and jaundice are the usual symptoms. Slight hepatomegaly is quite common.
Surgical excision is the treatment of choice. If the tumour is not resectable a bypass by an intubation procedure may provide good palliation.

PORTAL HYPERTENSION

- Portal hypertension is elevated pressure in the portal vein.
- The portal vein, formed by the superior mesenteric and splenic veins, drains blood from the abdominal GI tract, spleen, and pancreas into the liver.
- Within reticuloendothelium lined blood channels (sinusoids), blood from the terminal portal venules merges with hepatic arterial blood. Blood flows out of the sinusoids via the hepatic veins into the inferior vena cava.
- Normal portal pressure is 5-10 mmHg, which exceeds inferior vena caval pressure by 4-5 mmHg (portal venous gradient). Higher values are defined as portal hypertension.

Etiology:
Portal hypertension results mainly from increased resistance to blood flow in the portal vein. A common cause of this resistance is disease within the liver.
Portal hypertension is caused most often by cirrhosis (in developed countries), schistosomiasis (in endemic areas), or hepatic vascular abnormalities.

Pathophysiology:

- In cirrhosis, tissue fibrosis and regeneration increase resistance in the sinusoids and terminal portal venules. However, other potentially reversible factors contribute; they include contractility of sinusoidal lining cells, production of vasoactive substances, various systemic mediators of arteriolar resistance, and possibly swelling of hepatocytes.

- Over time, portal hypertension creates portosystemic venous collaterals. They may slightly decrease portal vein pressure but can cause complications.
- Engorged serpentine submucosal vessels (varices) in the distal esophagus and sometimes in the gastric fundus can rupture, causing sudden, catastrophic gastrointestinal bleeding. Bleeding rarely occurs unless the portal pressure gradient is > 12 mmHg.
- Gastric mucosal vascular congestion (portal hypertensive gastropathy) can cause acute or chronic bleeding independent of varices.
- Visible abdominal wall collaterals are common; veins radiating from the umbilicus (caput medusae) are much rarer and indicate extensive flow in the umbilical and periumbilical veins.
- Collaterals around the rectum can cause rectal varices that can bleed.

Signs & Symptoms:

- Portal hypertension is asymptomatic; symptoms and signs result from its complications. The most dangerous is acute variceal bleeding.
- Patients typically present with sudden painless upper gastrointestinal bleeding, often massive. Bleeding from portal hypertensive gastropathy is often subacute or chronic. Ascites, splenomegaly, or portosystemic encephalopathy may be present.

Diagnosis: Usually clinical evaluation

- Portal hypertension is assumed to be present when a patient with chronic liver disease has collateral circulation, splenomegaly, ascites, or portosystemic encephalopathy.
- Imaging may help when cirrhosis is suspected. Ultrasonography or CT often reveals dilated intra-abdominal collaterals, and Doppler ultrasonography can determine portal vein patency and flow.
- Esophagogastric varices and portal hypertensive gastropathy are best diagnosed by endoscopy, which may also identify predictors of esophagogastric variceal bleeding (e.g.: red markings on a varix).

Management:

- Ongoing endoscopic therapy and surveillance
- Non-selective beta-blockers with or without isosorbide mononitrate
- Sometimes portal vein shunting
- When possible, the underlying disorder is treated.

YAKRUTDALYODAR

Nidana: Vidahi and abhishyandi ahara sevana

Samprapti: Does vitiation of Rakta and Kapha, leads to enlargement of liver on right side of abdomen.

Lakshana:

- Glani (Lassitude)
- Mandagni (Impaired digestion)
- Pandu (Anaemia/jaundice)
- Mandajwara (Low fever)
- Ksheena bala (Loss of strength)

Chikitsa:
After proper snehana and Swedana, the vein inside the elbow of patient's right hand should be duly opened.
Shodhana karma
Samudra shukti kshara with milk
Yava with suvarchika and hingu.
The alkali of parijata, ikshuka, and apamarga with oil.

HEPATOMEGALY
Hepatomegaly is a general medical term for an abnormally enlarged liver.
Liver enlargement has many different causes, and by itself does not suggest a specific diagnosis or disease. Instead, it could indicate a variety of different conditions.

Etiology:
Infective → Hepatitis, Hepatic abscess, Malaria, Hydatid cyst, Actinomycosis
Metabolic → Fatty infiltration, Amyloidosis, Gaucher's disease
Neoplastic → Hepatoma, Haemangioma, Myeloma, Lymphoma
Cirrhotic → Portal, Biliary, Cardio
Congenital → Haemolytic anaemia, Polycystic disease
Drugs & Toxins → Alcoholism, Poisoning
Miscellaneous → Budd-Chiari Syndrome (BCS), Hunter Syndrome, Sarcoidosis

Signs & Symptoms:
Enlargement of liver, tenderness
Lethargy, Malaise
Loss of appetite
Loss of weight
Jaundice

Investigations:
Physical evaluation
Abdominal ultrasound
CT scan, MRI
Blood tests, Liver function tests
Sometimes liver biopsy

Management depends on the underlying disease since hepatomegaly is only a symptom or complication, and not a separate diagnosis.

DISEASES OF GALLBLADDER

2. **Diseases of Gallbladder:** Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Cholecystitis, Cholelithiasis, Obstructive jaundice and Tumours.

CHOLECYSTITIS

Definition:

Inflammation of gall bladder with or without stone is called as cholecystitis.
Common between 4th and 5th decade.

Types:

1. Acute:

Calculus (obstructive)
Acalculus (recovering from major illness – 10%)

2. Chronic

1. Acute cholecystitis:

Cystic duct blockage with gall stone → Leads to build up of bile in gall bladder & increases pressure within gall bladder → Leads to right upper abdomen pain → Concentrated bile, pressure & bacterial infection irritate & damage gall bladder wall → Inflammation & swelling of gall bladder → This reduces normal blood flow to area of gall bladder → This can lead to cell death due to inadequate oxygen.

causative organisms: E coli (Common), streptococci, klebsiella, pseudomonas, clostridial welchii etc

Risk factors: Female sex, increasing age, diabetes mellitus, pregnancy, oral contraceptives. obesity & rapid weight loss.

Clinical features:

1. H/o chronic cholecystitis or cholelithiasis
2. Onset is sudden & follows heavy fatty meal
3. Severe pain in right hypochondria, which may refer back to inferior angle of right scapula or to tip of right shoulder
4. Nausea & vomiting with low grade fever
5. Mild jaundice
6. Murphy's sign: Keep finger in right hypochondrium & ask patient to take, deep inspiration. At height of inspiration there is sudden catch in inspiration. It is due to inflamed gall bladder coming in contact with fingers & producing pain. This is called positive sign.
7. Boa's sign: An area of hyperesthesia between 9th & I lth rib posterior on right side.
8. Upper abdomen guarding and rigidity
9. Palpable, tender, smooth, soft gall bladder

Complications:

- Gall bladder perforation leads to biliary peritonitis
- Peri-cholecystic abscess
- Cholangitis and septicaemia

Investigation:

- USG: Calculus (Post acoustic shadow), Acalculus (Inflammed, thick gall bladder)
- Plain x-ray abdomen: 10% stones are radio-opaque
- CT scan

Treatment:

- Conservative treatment first
- After gap of 6 weeks to 3 months elective cholecystectomy

Conservative management:

- NBM
- Naso-gastric aspiration for 3-5 days
- IV fluids
- Drugs to reduce gastric & pancreatic secretions
- Antibiotics, analgesics & antispasmodics
- Gradually once sign of inflammation subsides, gastric aspiration & IV fluids stopped
- Fluids advised orally, later soft diet.

Surgery: Cholecystectomy (It should be done in following conditions)

- Pain & tenderness spread across abdomen
- Size of lump increases
- Increased fever & pulse rate
- If patient appears very ill or elder

2. Chronic cholecystitis:

May occur secondary to acute cholecystitis, which converts gall bladder in to contracted, fibrosed, non-functioning and shrunken.

Clinical features:

- Intolerance to fatty food (Classical symptom)
- Belching
- Nausea & vomiting
- Pain appears after food
- Recurrent attack of pain in right upper quadrant or epigastric region
- Murphy’s sign positive (Sitting position), while Moynihan's sign positive (Lying down)

Flatulent dyspepsia: Stomach upset with frequent eructations of swallowed air.
Treatment: Cholecystectomy

GALL STONES / CHOLELITHIASIS (PITTASHAYA ASHMARI)

Derivation:
Cholecyst → Gall bladder
Cholelith → Gall stone
Lithiasis → Stone formation

Incidence:
Common in 5^{th} – 6^{th} decade.
M: F = 1: 4

Aetiology:

1. Metabolic aspect:

Normal ratio of bile acids: cholesterol = 25:1
When it drops to 13: l, the cholesterol gets precipitated.

2. Infection aspect (80%)

Organisms like E coli etc. from infected tooth, tonsil etc. reaches the gall bladder via blood and form focus, around which cholesterol and bile get precipitated, later which results in mixed stones.

3. Stasis aspect:

Due to pregnancy, oestrogen, followed vagatomy & prolonged TPN leads to bile stasis and caused mixed stones

4. Reflux aspect:

Reflux of pancreatic enzymes in to gall bladder leads to cholesterol precipitation.

5. Saint's triad:

Gall stones
Hiatus hernia
Diverticulosis of colon

6. Risk factors (4F*):

Female, Fatty, Fertile & Forty yrs of age

7. Lack of melatonin:

It inhibits cholesterol secretion from gall bladder, enhances conversion of choles to bile & is anti-oxidant which can reduce oxidation stress to gall bladder.

Patho-physiology:
Increases concentration of cholesterol
Inability to gall bladder to contract & evacuate all bile, leads to over concentration.
Protein in liver & bile that either promote or inhibit cholesterol crystallization in to gall stones.
Oestrogen (OCP) increases cholesterol level in bile & also decreases gall bladder movement.

Types:

1. Cholesterol stones (10%)

Occurs in patient with increased cholesterol level
Single, solitary occurs in aseptic bile
Radiolucent
Light pale yellow in colour or chalk white having tiny dark central spot

2. Mixed stones (80%)

Containing alternate layer of cholesterol & pigment
Multiple, small faceted by mutual pressure
Radio graphically visible because of calcium content

3. Pigmented stones (5-10%)

They are calcium bilirubinate stones commonly occurs due to haemolysis
Black, multiple, small & irregular, often sludge like

Clinical features:

- Asymptomatic (Silent stones)
- Gall stone attack: Intense pain in upper right abdomen often with nausea & vomiting this steadily increases for approximate 30 mts to several hours
- Clinical features specially seen after taking fatty meal, almost night & after drink
- Murphy's sign positive

Effects / complications of gall stone:
In gall bladder →

- Silent stone
- CA of gall bladder
- Gall stone colic
- Mucocele of gall bladder
- Empyema
- Acute and chronic cholecystitis

In CBD →

- Obstructive jaundice
- Liver failure
- Cholangitis
- Acute/recurrent pancreatitis

In intestine →
Intestinal obstruction due to gall stone ileus.

Treatment:

1. Emergency early cholecystectomy
2. Open / laproscopic cholecystectomy

Cholesterol stones:
Medicines are used when patient have functioning gall bladder, young and thin consistency.

- Oral dissolution treatment: CDCA (Chemo Deoxy Cholic Acid) for 2 yrs. or UDCA (Urso Deoxy Cholic Acid)
- Direct contact dissolution: MTBE (Methyl Terbutyl Ether) given through catheter placed in gall bladder percutaneously.

OBSTRUCTIVE JAUNDICE

Jaundice is a yellowish discoloration of the skin and mucous membranes caused by hyperbilirubinemia. Jaundice becomes visible when the bilirubin level is about 2-3 mg/dL (34-51 micromole/L).
Obstructive jaundice is a specific type of jaundice, where symptoms develop due to a narrowed or blocked bile duct or pancreatic duct, preventing the normal drainage of bile from the bloodstream into the intestines.

Etiology:

- Cholelithiasis
- Cholangitis
- Lymph node enlargement near the bile duct
- Pancreatic cancer
- Pancreatic cysts
- Other pancreatic duct obstructions such as scarring

Signs & Symptoms:

- Abdominal pain (typically occurs in the right upper quadrant of the body)
- Yellow discoloration of the eyes and skin
- Dark-colored urine
- Pale stools
- Diarrhoea
- Easy bleeding or bruising
- Fever and chills
- Itching
- Anorexia
- Malaise or lethargy
- Weight loss

Serious symptoms that might indicate a life-threatening condition:

- Abdominal swelling, distension and bloating
- High fever (38.3° C)
- Nausea with or without vomiting
- Severe abdominal pain

Investigations:

- CT scan, MRI
- Blood tests to examine bilirubin levels
- Endoscopic retrograde cholangiopancreatography (ERCP)
- Endoscopic ultrasound (EUS)
- Choledochoscopy
- Probe-based confocal endomicroscopy
- Narrow band imaging of the bile duct

Management:
Treatment options for obstructive jaundice depend on the exact cause and severity of the disease. Examples include:

- Antibiotic therapy (if indicated for infection)
- Endoscopic retrograde cholangiopancreatography (ERCP), an imaging procedure that allows treatment of some bile duct problems, including removal of gallstones that are causing obstruction
- Intravenous fluids and pain medications
- Nutritional support
- Surgery or other procedures to repair anatomical defects or create alternative pathways for the flow of bile
- Transplantation of the liver (if all other methods are unsuccessful and all of the liver is damaged)
- Treatment for cancer, if present, which may include surgery, chemotherapy, or radiation therapy

TUMOURS OF GALL BLADDER
Incidence:
It is common in northern eastern India
Patients are over 50 years.
M: F = 1: 3

Aetiology:
Cholelithiasis: 90% of cases are associated with gall stones, which is 7-10 times more than general population
Chemicals: Who work in rubber industries
Gall bladder polyp > 1 cm or > 3 in number
Diet: Adulterated mustard oil for cooking is found to precipitate CA gall bladder
Porcelain gall bladder is more prone for malignant transformation

Pathology:
90% adenocarcinoma
35% lymph node spread
25% localized disease

Clinical features:
Significant weight loss with anorexia
Jaundice
Pain and mass in right upper quadrant which is hard and non-tender.
Obstructive jaundice, bleeding and ascites are late features.
Features of acute cholecystitis

Spread:
Direct: Liver, bile duct, duodenum, kidney, colon
Lymph: Peripancreatic and periduodenal node
Blood: Liver, lungs, bone

Investigation:
USG
CT scan
FNAC
ERCP

Treatment:
Cholecystectomy: If mucosa only involved
Extended cholecystectomy: If bladder wall is involved
Radiation has very small benefits
Chemotherapy (5FU)

DISEASES OF PANCREAS

3. Diseases of Pancreas: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Pancreatitis, Cysts of Pancreas and Tumours.

PANCREATITIS

- Pancreatitis is classified as either acute or chronic.
- Acute pancreatitis is inflammation that resolves both clinically and histologically.
- Chronic pancreatitis is characterized by histologic changes that are irreversible and progressive and that result in considerable loss of exocrine and endocrine pancreatic function. Patients with chronic pancreatitis may have a flare-up of acute disease.
- Pancreatitis can affect both the exocrine and endocrine functions of the pancreas. Pancreatic cells secrete bicarbonate and digestive enzymes into ducts that connect the pancreas to the duodenum at the ampulla of Vater (exocrine function).
- Pancreatic beta cells secrete insulin directly into the bloodstream (endocrine function).

Acute Pancreatitis:

Acute pancreatitis is acute inflammation of the pancreas (and, sometimes, adjacent tissues). The most common triggers are gallstones and alcohol intake.
Acute pancreatitis is a common disorder and a major healthcare concern.

Etiology:

- Gallstones and alcohol consumption account for ≥ 70% of acute pancreatitis cases.
- Other causes include several genetic mutations predisposing to pancreatitis. An autosomal dominant mutation of the cationic trypsinogen gene causes pancreatitis in 80% of carriers; an obvious familial pattern is present.
- Other mutations have lesser penetrance and are not readily apparent clinically except through genetic testing. The gene that causes cystic fibrosis increases the risk of recurrent acute pancreatitis as well as chronic pancreatitis.
- Acute pancreatitis is a complication that develops after endoscopic retrograde cholangiopancreatography (ERCP) in about 5% of patients.

Types:

1. Interstitial pancreatitis is defined by the presence of an enlarged pancreas on imaging. Peripancreatic stranding may be seen and is a sign of inflammation. Most patients develop this type of pancreatitis. Most cases are self-limiting.
2. Necrotizing pancreatitis is defined by the presence of pancreatic and/or peripancreatic necrosis. It is best seen on contrast-enhanced cross-sectional imaging. Necrotizing pancreatitis occurs in 5-10% of patients with acute pancreatitis and is associated with a prolonged and more severe disease course.

Classification:

1. Mild pancreatitis: Inflammation is confined to the pancreas and its close vicinity. Patients do not have organ failure or systemic or local complications.
2. Moderately severe pancreatitis: Patients have local or systemic complications but no organ failure, or only transient organ failure (resolves within 48 hours).
3. Severe pancreatitis: There is persistent single or multiorgan failure (> 48 hours). Most patients have one or more local complications.

Complications:

1. Local: Pancreatic and peripancreatic fluid collections, splenic vein thrombosis, pseudoaneurysm formation, and gastric outlet dysfunction
2. Systemic: Shock, organ failure

Signs & Symptoms:

- An acute pancreatitis attack causes steady upper abdominal pain, typically severe enough to require parenteral opioids.
- The pain radiates through to the back in about 50% of patients.
- Pain usually develops suddenly in gallstone pancreatitis; in alcoholic pancreatitis, pain develops over a few days.
- The pain usually persists for several days. Sitting up and leaning forward may reduce pain, but coughing, vigorous movement, and deep breathing may accentuate it.
- Nausea and vomiting are common.
- The patient appears acutely ill and sweaty.
- Pulse rate is usually 100-140 beats/minute.
- Respiration is shallow and rapid.
- Blood pressure may be transiently high or low.
- Temperature may be normal or even subnormal at first but may increase to
- 37.7-38.3° C within a few hours.
- Scleral icterus is occasionally present because of obstruction of the bile duct by a gallstone or inflammation and swelling of the pancreatic head.
- Decreased bowel sounds and abdominal distention.
- Marked abdominal tenderness occurs, most often in the upper abdomen.
- Infection in the pancreas or in an adjacent fluid collection should be suspected if the patient has a generally toxic appearance with fever and an elevated white blood cell count or if deterioration follows an initial period of stabilization.
- Patients with severe disease can develop multiorgan failure (cardiovascular, renal, and respiratory).

Diagnosis:

- Serum markers (amylase, lipase)
- Pancreatitis is suspected whenever severe abdominal pain occurs, especially in a patient with significant alcohol use or known gallstones.
- To exclude other causes of abdominal pain and to diagnose metabolic complications of acute pancreatitis, a broad range of tests is usually done at initial evaluation. These include laboratory and imaging tests.

Management:
The basic treatment of acute pancreatitis includes:

- Early goal-directed fluid resuscitation
- Analgesia
- Nutritional support

Treatment of severe acute pancreatitis and complications includes:

- ICU (intensive care unit) care
- Enteral nutrition preferred over parenteral nutrition
- Antibiotics for extrapancreatic infections and infected necrosis
- Necrosectomy (removal of necrotic tissue) for infected necrosis
- Endoscopic retrograde cholangiopancreatography (ERCP) for acute pancreatitis and concurrent acute cholangitis
- Drainage of pseudocysts

Chronic Pancreatitis:
Chronic pancreatitis is persistent inflammation of the pancreas that results in permanent structural damage with fibrosis and ductal strictures, followed by a decline in exocrine and endocrine function (pancreatic insufficiency).
Drinking alcohol and smoking cigarettes are two of the major risk factors.

Signs & Symptoms:

- Abdominal pain and pancreatic insufficiency are the primary manifestations of chronic pancreatitis.
- About 10-15% of patients have no pain but present with malabsorption.
- Clinical manifestations of pancreatic insufficiency include flatulence, abdominal distention, steatorrhea, undernutrition, weight loss, and fatigue.
- Glucose intolerance may appear at any time, but overt diabetes mellitus usually occurs late in the course of chronic pancreatitis.
- Patients also are at risk of hypoglycemia because pancreatic alpha cells, which produce glucagon (a counter-regulatory hormone), are lost.

Diagnosis → X-ray, CT scan, Pancreatic function test, MRI coupled with magnetic resonance cholangiopancreatography (MRCP)

Management → Pain control, Pancreatic enzyme supplements, Management of diabetes and other complications

CARCINOMA OF PANCREAS

Incidence:

- Common at age of 60 years with higher rate in men
- Adeno-carcinoma of duct cell origin (70%)
- 4th leading cause of death due to cancer in males, after lung, colon, prostate

Aetiology: DOEP

Diet: Coffee, alcohol, smoking, westernization of diet (fatty food rich in animal proteins)

Occupation: Industrial carcinogens like benzidine, gasoline agents etc.

Precancerous condition: Chronic tropical pancreatitis, Haemochromatosis (extensive calcification of pancreas) and hereditary pancreatitis are associated with pancreatic cancer.

Endocrine cause: Diabetic patients are 10 times more vulnerable to develop CA of pancreas

Site:

Head of pancreas (70%)

Body and tail (30%)

Symptoms:

- Loss of appetite and generalized weakness
- Gross weight loss in 3-6 months
- Jaundice (CA of head pancreas): Short duration, severe, progressive associated with pruritis
- Severe pain in upper abdomen, radiating to the back in the region of L1 and L2. Due to infiltration of retroperitoneal nerve plexus or pancreatic duct obstruction. It is relieved by leaning forward.
- Silvery stool (Due to mixing of undigested fat with metabolized blood oozes from periampullary growth)

Signs:

- Per abdomen: Mass felt in upper abdomen which is fixed
- Trousseau's sign': Migrating thrombophlebitis of the legs can occur in visceral malignancies
- particularly from CA of pancreas, rarely stomach, colon etc. It is due to sluggish blood flow resulting in thrombus formation in superficial veins.
- Left supra-clavicular node may be palpable

Investigation:

USG

CT scan

ERCP

Treatment:

- Whipple procedure (pancreaticoduodenectomy)
- Adjuvant chemotherapy and radiation therapy
- Symptomatic treatment, analgesics (opioids), pancreatic enzyme supplementation
- Ultimately, most patients experience pain and die. Thus, symptomatic treatment is as important as controlling disease. Appropriate end-of-life care should be discussed.

Radiotherapy:
4000 – 6000 cGy units
Response rate is 5 – 10 %

Chemotherapy:
Inj. 5 FU in 5% dextrose
Immunotherapy

PSEUDOCYST OF PANCREAS

Collection of pancreatic fluid in the lesser sac, due to attack of pancreatitis. It is called Pseudocyst because it has no epithelial lining. It is lined by fibrin layers.

Contents:
Brownish fluid with sludge like necrotic material
Albumin, mucin, cholesterin, blood cells

Aetiology:
Following an attack of acute pancreatitis (usually appears after 3 weeks)
Trauma at upper abdomen leads to laceration of pancreas, pancreatic secretions and blood escape in to lesser sac and formed cyst.
Post op of pancreatolithotomy, pancreatectomy etc.

Sites:
Between stomach and transverse colon
Between stomach and liver
Behind or below the transverse colon

Clinical features:

1. Epigastric swelling:

- Tensley cystic mass in the epigastrium, umbilical region or in left hypochondrium.
- Mass feels firm on palpation.
- Classically upper border of the mass is not felt.
- Smooth, soft, resonant on percussion
- Infected: Tender, fever, chills
- Transmitted pulsation from aorta can be felt.

2. Anorexia and weight loss
3. Baid sign: If a Ryle's tube is passed, it can be felt over the swelling, because stomach

Investigation:

- USG
- ERCP (To see communications)
- CT scan (Size, shape, number, wall thickness, content, pancreatic duct size and extent of necrosis)

Treatment:

- Conservative: Majority of the pseudocyst followed by acute pancreatitis resolve by itself within 3-4 weeks. Follow up using repeat USG at regular intervals.
- USG guided drainage by ERCP
- Cystogastrostomy: Surgery is done after 6 weeks because that is the time required for the wall to become fibrous. Size of the cyst should be at least 6 cm.
- Distal pancreatectomy (Cyst at tail of pancreas)

DISEASES OF SPLEEN

4. Diseases of Spleen – Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Pleehodara – Splenomegaly and Splenic rupture.

PLEEHODARA

Nidana:

- Overeating
- Travelling by vehicle
- Excessive sexual intercourse
- Vomiting
- Excessive jerking
- Excessive movements
- Heavy weight lifting

Samprapti:

- Due to above said causes the spleen located in left side gets displaced from its normal place and thus, is enlarged or due to collection of blood and rasa also enlarges it.
- The enlarged spleen is hard and like a small stony piece in the beginning, gradually increases and becomes like a tortoise.

Chikitsa:

- Snehana
- Virechana
- Siravyadha in left arm
- Swedana
- Niruha and anuvasana basti

SPLENOMEGALY

Enlargement of spleen due to many reasons is called as splenomegaly.

Causes:

- Infection: Bacterial (Typhoid, T B, splenic abscess, septicemia), viral (HIV related thrombocytopenia), protozoal and parasitic (Malaria, kala azar, schistosomiasis)
- Blood diseases: Acute and chronic leukemia, myelofibrosis, polycythemia, haemolytic anaemia, sickle cell disease
- Metabolic: Gaucher 's disease, amyloidosis, porphyrias, rickets
- Portal hypertension, circulatory infarction
- Collagen diseases
- Malignancy: Hodgkin 's lymphoma, splenic sarcomas

Clinical features:

- Pallor
- Recurrent fever
- Enlargement of spleen and liver
- Jaundice
- Pain abdomen
- Mass in left hypochondrium, moves with respiration, dull to percuss, directed towards right ileac fossa
- Hook sign: Inability to hook under left costal margin

Grades of splenomegaly:
Grade 0 → Normal, Impalpable spleen
Grade 1 → Spleen palpable only on deep inspiration
Grade 2 → Spleen palpable on mid clavicular line, half way between umbilicus and costal margin.
Grade 3 → The spleen expands towards the umbilicus.
Grade 4 → The spleen goes past the umbilicus.
Grade 5 → The spleen expands towards the symphysis pubis.

Investigation:

- Fragility test: Here increased fragility of the erythrocytes is the typical feature
- LFT: Elevated serum bilirubin
- Reticulocyte count is increased significantly (25%)
- Faecal urobilinogen is increased
- USG

Treatment:

- Blood transfusion to improve hemoglobin status. Later splenectomy is done
- Accessory spleen should be removed
- Pneumococcal vaccine should be given to all patients before elective splenectomy (3 weeks prior to surgery and 3 weeks after the surgery)
- If there are gall stones, cholecystectomy done

SPLENIC INJURY AND RUPTURE
Splenic injury usually results from blunt abdominal trauma.
A ruptured spleen is a medical emergency that occurs as a result of a break in the spleen's surface.

Etiology:

- Significant impact (e.g.: motor vehicle crash) can damage the spleen, as can penetrating trauma (e.g.: knife wound, gunshot wound).
- Splenic enlargement as a result of fulminant Epstein-Barr viral disease predisposes to rupture with minimal trauma or even spontaneously.
- Splenic injuries range from subcapsular hematomas and small capsular lacerations to deep parenchymal lacerations, crush injury, and avulsion from the pedicle.

Signs & Symptoms:

- The main immediate consequence is hemorrhage into the peritoneal cavity.
- The amount of hemorrhage ranges from small to massive, depending on the nature and degree of injury. Many small lacerations, particularly in children, cease bleeding spontaneously. Larger injuries hemorrhage extensively, often causing hemorrhagic shock.
- A splenic hematoma sometimes ruptures, usually in the first few days, although rupture can occur from hours to even months after injury.
- The manifestations of major hemorrhage, including hemorrhagic shock, abdominal pain, and distention, are usually clinically obvious.
- Lesser hemorrhage causes left upper quadrant abdominal pain, which sometimes radiates to the left shoulder. Patients with unexplained left upper quadrant pain, particularly if there is evidence of hypovolemia or shock, should be asked about recent trauma. Maintain a high index of suspicion for splenic injury in patients who have left rib fractures.

Investigations: CT scan, USG, Exploratory laparotomy

Management:

- Observation
- Angioembolization
- Sometimes surgical repair or splenectomy

DISEASES OF KIDNEY AND URETERS

5. Diseases of Kidney and Ureters - Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital anomalies, Polycystic kidney, Injuries, Perinephric abscess, Calculus and Neoplasms.

CONGENITAL ANOMALIES

1. **Agenesis of kidney:**

The condition in which a new born is missing one or both kidneys. Absence of kidney on one side is often associated with absence of ureter. It occurs when the uretic bud (Kidney bud) fails to develop at early stage of fetal growth. In unilateral agenesis single kidney becomes hypertrophied
and functions almost double the normal to make good the absence of one kidney. Dialysis and kidney transplant is choice of treatment.

2. **Hypoplasia and dysplasia:**

Hypoplasia defined as abnormally small kidney with normal morphology and reduced nephron numbers. It is due to failure of meta nephrogenic cap.

In dysplasia the internal structures of one or both kidneys do not develop normally. Fluid filled sacs replace normal kidney tissue. It is due to abnormally development of secretory part of the kidney.

3. **Supernumerary kidney:**

There may be more than one kidney on one or both sides.

4. **Duplex kidney:**

Duplex kidney, also called duplicated ureters, is a problem with the urinary tract where there are two ureters draining urine from a single kidney. It is more common in females than males and it’s an unpreventable birth defect. There are surgeries available to fix the problem, but most issues simply resolve on their own. Treatment is rarely needed.

5. **Ectopic kidney:**

Defect in the position of kidney is called as ectopic kidney. The left kidney is often seen to be ectopic than the right one.

Pelvic kidney: This occurs when kidney docs not ascend from its original location to its final location, during fetal development and get stuck in brim of pelvis. It is usually detected by chance and hardly requires any treatment. Usually, the opposite kidney lies in normal position.

Crossed renal ectopia: It is said to be present when the kidney is seen in the opposite retroperitoneal space at loin below the level of L2 and become fused yet retain their oven vessels and ureter. The ureter of lower kidney crosses the midline to enter the bladder on the contralateral side. Both renal pelvis can lie one above each other. Sometimes the ureter of the ectopic kidney may open in to the same side of bladder as the normal one and then there is often chance of reflux of urine leading to hydro-ureter and hydro-nephrosis of the crossed kidney.

Rotated kidney: The kidney may rotate abnormally so that its pelvis faces forwards instead of medially. It does not produce any symptoms as such.

6. Horseshoe kidney:

Definition: A congenital disease when kidney fuse together to form a horse shoe shape during development in womb. It is also called as renal fusion or super kidney.

Causes:

- The failure of complete ascent of kidneys with the fusion of lower (Classical horseshoe kidney) or upper poles (Reverse horseshoe kidney). It is due to fusion of subdivisions of mesonephric duct, when the embryo is as early as 30 – 40 days old.
- Inferior mesenteric artery crosses the isthmus (part in front of vertebrae) at the level of L3 – L4. Hence, horseshoe kidney cannot ascend. It is felt lower in the abdomen.

Incidence:

- This condition is more common in males (l in 500 peoples)
- Most common site is in front of the 4th lumbar vertebrae

Clinical features:

- Asymptomatic for many years
- Presence of fixed, non-mobile, firm mass in midline at the level of 4th lumbar vertebra
- Recurrent UT I: Due to angulation of ureters or isthmus result in stasis and infection, this later produces recurrent UTI and stones in kidney.
- Rovsing sign: Hyperextension of the spine results in abdominal pain, nausea or vomiting due to stretching of the capsule.

Investigation:

- USG
- IVU: Lower calyx is directed medially where there is fusion and curving of ureter like flower vase.
- CT scan

Treatment:

- Treat the complications like UTI and kidney stones
- Repair and reconstruction of the hydro-nephrosis are done in usual manner.

7. POLYCYSTIC KIDNEY: (वृक्क ग्रन्थि)

This is an autosomal dominant disease transmitted through chromosomes from any one of the parents. It is more common in females and almost always bilateral (95%)

Pathophysiology:

- During development, some of the uriniferous tubules fail to join with the collecting ducts. Such uriniferous tubules develop in to cysts.
- They enlarge often to 3 —4 times the normal size
- The kidneys are studded with multiple large cysts with clear or brownish fluid
- When the cyst ruptures in to the pelvis of kidney, it results in hematuria.

Clinical features:

- Asymptomatic
- Mass per abdomen: Both kidneys are enlarged, surface in nodular, bosselated, firm to hard and sometimes cystic, moves with respiration.
- Loin pain (dull ache) due to stretching of the renal capsule or hemorrhage in to cyst.
- Hypertension (75%) is due to renal ischemia which stimulates juxtaglomerular apparatus to secret renin.
- Hematuria in 25% of cases due to over distending cyst rupture
- Infection: Fever and chills due to stasis
- Features of renal failure: Thirst, vomiting, abdominal distension due to paralytic ileus and uremia.

Investigation:
Serum urea and creatinine to rule out renal failure
Plain x-ray KUB
Abdominal USG/CT scan
IVU: The spider leg deformity of the calyces.

Treatment:
Conservative:

- Asymptomatic: Does not require any treatment other than follow up
- Infected cyst: Antibiotics, if necessary, cyst should be aspirated using USG
- Polycystic kidney with hypertension: Control of hypertension with drugs. If not controlled then bilateral nephrectomy followed by renal transplantation should be done

Surgery:

- Polycystic disease with renal failure: Emergency dialysis followed by renal transplantation.
- Rovsing operation: The kidney is exposed. The cyst is opened. The fluid is evacuated. The cut edge is marsupialised.

RENAL TRAUMA

The kidney is injured in up to 10% of patients who sustain significant abdominal trauma.
Overall, about 65% of genitourinary (GU) injuries involve the kidney.
It is the most injured GU organ from civilian external trauma.

Most renal injuries (85-90% of cases) result from blunt trauma, typically due to motor vehicle crashes, falls, or assaults. Most injuries are low grade.
The most common accompanying injuries are to the head, central nervous system, chest, spleen, and liver. Penetrating injuries usually result from gunshot wounds and are usually associated with multiple intra-abdominal injuries, most commonly to the chest, liver, intestine, and spleen.

Renal injuries are classified according to severity into 5 grades:
Grade 1: Subcapsular hematoma and/or renal contusion
Grade 2: Laceration ≤ 1 cm in depth without urinary extravasation
Grade 3: Laceration > 1 cm without urinary extravasation
Grade 4: Laceration involving the collecting system with urinary extravasation; any segmental renal vascular injury; renal infarction; renal pelvis laceration and/or ureteropelvic disruption
Grade 5: Shattered or devascularized kidney with active bleeding; main renal vascular laceration or avulsion

Investigations:

- Clinical evaluation, including repeated vital sign determination
- Urinalysis and hematocrit (Hct)
- If a high-grade renal injury is suspected, contrast-enhanced CT with delayed images (done about 10-15 minutes after the initial study)

Management:
Most blunt renal injuries, including all grade 1 and 2 and most grade 3 and 4 injuries, can be safely managed non-operatively. Patients should be maintained on strict bed rest until the gross hematuria has resolved.

Prompt intervention is required for patients with the following:

- Persistent bleeding (i.e., enough to necessitate repeated transfusions)
- Expanding perinephric hematoma
- Renal pedicle avulsion or other significant renovascular injuries
- Ureteropelvic junction disruption

Intervention can include surgery, stent placement, or selective angiographic embolization. Penetrating trauma usually requires surgical exploration, although observation may be appropriate for patients in whom the renal injury has been accurately staged by CT scan, blood pressure is stable, and no associated intra-abdominal injuries require surgery.

URETERAL TRAUMA

Most ureteral injuries occur during surgery.
Procedures that most often injure the ureter include ureteroscopy, hysterectomy, low anterior colon resection, and open abdominal aneurysm repair.
Mechanisms include ligation, transection, avulsion, crush, devascularization, kinking, and electrocoagulation.
Non-iatrogenic ureteral injuries account for only about 1-3% of all genitourinary trauma.
They usually result from gunshot wounds and rarely from stab wounds.
In children, avulsion injuries are more common and occur at the ureteropelvic junction.
Complications include peritoneal or retroperitoneal urinary leakage; perinephric abscess; fistula formation; and ureteral stricture, obstruction, or both.

Investigations: Imaging, exploratory surgery, or both

Management:

- For minor injuries, percutaneous nephrostomy tube or ureteral stent
- For major injuries, surgical repair

PERINEPHRIC ABSCESS

Definition:
Collection of pus in the peri-renal area is termed as perinephric abscess.

Causes:

- Haematogenous spread
- Extension of appendicular abscess in
- perinephric area
- Infection of peri-nephric haematoma
- Ruptured pyonephrosis
- TB perinephric abscess
- Organisms like streptococci, E coli

Clinical features:

- H/O prolonged urinary infection
- High fever
- Pain in the flank
- Tenderness over kidney or lies just beneath the lower rib

Investigation:

- Leukocytosis
- Plain x-ray KUB
- IVU: Mathe's sign (Lack of downward displacement of kidney in the erect posture with respiration)
- USG/CT scan

Treatment:

- Bed rest, local heat application
- Antibiotics
- I and D under GA through lumbar incision.

RENAL CALCULUS / KIDNEY STONES / NEPHROLITHIASIS / (वृक्क अश्मरी)

Nephrolithiasis refers to the presence of stones within the kidneys.
Cystoliths refers to stones in the urinary bladder. Ureterolithiasis, on the other hand, refers to stones within the ureter, and urolithiasis refers to stones in any part of the urinary tract (kidneys, ureter, bladder and urethra).

Nephrolithiasis is one of the most common kidney diseases in adults.

Stone formation occurs when there is an excess of crystal-forming substances that cannot be dissolved in the urine.
High urinary excretion of certain substances; for instance, calcium, oxalate, uric acid, and cysteine; can promote stone formation, whereas the excretion of others, such as citrate, has a protective effect.
Disturbances in urinary pH, a low urine volume, and a lack of protective substances that prevent the crystals from sticking together may contribute to stone formation. Additionally, environmental factors such as decreased fluid intake, hot climates, and dietary factors play an important role in the development of nephrolithiasis.

Types of Nephrolithiasis:

1. Hypocitraturiç Stones:

Most kidney stones are made from calcium salts.
Calcium oxalate stones are the most common type, followed by calcium phosphate stones. The main risk factor for developing calcium stones is an increased excretion of calcium (hypercalciuria) and oxalate (hyperoxaluria). These abnormalities can be idiopathic or can arise due to other systemic diseases, such as hyperparathyroidism, renal tubular acidosis, and malabsorption.

2. Struvite Stones:

Struvite stones are sometimes referred to as infectious stones, since they can be associated with infections of the urinary tract, especially those caused by urea-splitting organisms (Proteus mirabilis, Klebsiella, Staphylococcus, etc.) These bacteria split urea molecules into ammonium and CO2, thereby raising the urine's pH to neutral or alkaline values, and ultimately leading to the precipitation of solutes, to which the bacteria can adhere.

3. Uric Acid Stones:

Uric acid stones generally develop due to increased excretion of uric acid (hyperuricosuria) and low urine pH. Risk factors include high-protein diets, gout, inflammatory bowel disease, genetic diseases, and diabetes. Uric acid stones are one of the few kidney stones that can be managed successfully through medical treatment that consists of adequate hydration and supplements to raise the urine's pH.

4. Cystine Stones:

Cystine stones are generally caused by cystinuria, a hereditary disease that causes increased excretion of cystine in the urine, as well as low urinary pH. Cystine stones are very hard and can be seen as thin hexagonal crystals in a urine analysis.

5. Drug-induced Stones

Drug-induced stones can develop by two mechanisms. In some cases, excessive use of laxatives or diuretics can contribute to metabolic abnormalities that ultimately lead to stone formation.
On the other hand, certain medications, such as indinavir or ciprofloxacin, can crystallize in the urine and create stones.

Symptoms:

- Pain in abdomen: Dull aching to pricking pain feel at renal angle, lumbar region (Fixed pain) due to capsular and parenchymal distension. Pain may radiate to groin and testis in males (Referred).
- Nausea and vomiting due to pylorospasm
- Fever
- Haematuria: Is common. The quantity of blood loss is small and makes urine dirty or smoky.
- Burning micturition during attack of pain, pyuria may occur along with increased frequency of micturition.

Signs:

- Tenderness at renal angle between sacrospinalis and 12th rib
- Rigidity of muscles over kidney
- Swelling at flank (Hydronephrosis)

Investigation:

- Plain X-ray KUB: 90% of renal stones are radio opaque. Enlarged renal shadow can be seen.
- USG: Exact size and location of the stone can be evaluated.

Treatment:
Conservative:
Flush therapy (IV fluids with inj. frusemide)
Anti-inflammatory and anti-spasmodic drugs
Plenty of water (< 5 cm)

Non operative treatment:

- Extracorporeal shock wave lithotripsy (ESWL):

After cystoscopy, a ureteric stunt (Double J stent) is placed in to the ureter on the side of large renal stone. Shock waves are generated (around 500 1500 shock waves) which blast the stone. The stones get crushed and most of the stones will come out by the side of stent.

- Endoscopic procedures:

Percutaneous nephrolithotomy (PCNL)

- Laser lithotripsy:

Helpful in fragmented cystine stones. Holmium-YAG laser lithotripsy occurs primarily through a photothermal mechanism that causes stone vaporization.

Operative treatment:

- Pyelolithotomy: When stone is at extrarenal pelvis
- Nephrolithotomy: Intra renal pelvis
- Extended pyelolithotomy: Calyx stone and large Staghorn calculus
- Pyelonephrolithotomy: Pelvis as well as renal parenchyma
- Partial nephrectomy: Stone impacted at lower most calyx

Staghorn calculus:

It is the stone occupying the renal pelvis and calyces.
The stone tend to grow in alkaline urine, especially when proteus organisms are present, which split urea to ammonium, as a result calculus may enlarge to fill all or most of renal collecting system forming a staghorn calculus.
They are most often composed of struvite (Magnesium ammonium phosphate) and or calcium carbonate. It is white in colour, soft and smooth.
Often referred as infection stone since they are strongly associated with UTI.

Clinical features:

- Pain in flanks
- Fever
- Burning micturition
- Haematuria

Treatment:

- Antibiotics
- Oral chemolysis by AHA (Aceto hydroxamic acid). They are high renal clearance; penetrate bacterial cell wall and acts synergistically.
- Unilateral stone is removed by nephropyelolithotomy.
- In bilateral cases, IVU is very essential. The kidney which is functioning better should be treated first. After 3 months the other side kidney should be operated upon.
- PCNL and ESWL

URETERIC CALCULUS

Clinical features:

- Ureteric colic: Pain is severe, colicky, and intolerable and lasts for a few hours. Pain feels loin to groin when stone descends in to lower ureter, pain radiates to the testicles, tip of penis (in males). labia majora (in females) and to the upper portion of thigh due to irritation of genitor-femoral nerve. During attack of pain, patient is unable to get relief in any position.

- Nausea, vomiting and sweating due to pain and reflux pylorospasm
- Haematuria or pyuria
- Strangury, dysuria
- Right sided ureteric colic is mimics with acute appendicitis due to presence of tenderness and rigidity at RIF.

Complication:
This may lead to obstruction, hydroureteronephrosis, renal parenchymal atrophy, infection and pyonephrosis.

Treatment:
Conservative:

- Plenty of water
- Flushing therapy: About 2 liter of IV fluid, with 20 — 40 mg inj. frusemide. It can be repeated for a few days
- Antibiotics to control infection, NSAID and muscle relaxants

Surgery:

- Stone in upper ureter: ESWL
- Middle ureteric stone: ESWL ureteroscopy basketing or ureterolithotomy
- Lower ureteric stone: Ureteroscopic removal
- Vesicoureteric junction: Ureteroscopic removal or endoscopic meatotomy of vesicoureteric junction

RENAL PELVIC & URETERAL CANCERS
Cancers of the renal pelvis and ureters are usually transitional cell carcinomas (TCCs) and occasionally squamous cell carcinomas.
TCC of the renal pelvis accounts for about 7-15% of all kidney tumors.
TCC of the ureters accounts for about 4% of upper tract tumors.

Risk factors are the same as those for bladder cancer (smoking, excess phenacetin use, long-term cyclophosphamide use, chronic irritation, exposure to certain chemicals).
Also, inhabitants of the Balkans with endemic familial nephropathy are inexplicably predisposed to develop upper tract TCC.

Signs & Symptoms:
Most patients present with hematuria; dysuria and frequency may occur if the bladder also is involved. Colicky pain may accompany obstruction. Uncommonly, hydronephrosis results from a renal pelvic tumor.

Investigations:

- USG or CT with contrast
- Cytology or Histology

Management:

- Radical nephroureterectomy, including excision of a cuff of bladder and regional lymphadenectomy
- Neoadjuvant chemotherapy prior to nephroureterectomy is recommended for high-grade and high-stage lesions because other treatments can decrease renal function, often preventing subsequent use of adjuvant chemotherapy.
- Post-treatment surveillance with cystoscopy

RENAL CELL CARCINOMA / ADENOCARCINOMA OF THE KIDNEYS

Renal cell carcinoma (RCC) is the most common renal cancer. RCC, an adenocarcinoma, accounts for 90-95% of primary malignant renal tumors. RCC metastasizes most often to the lymph nodes, lungs, adrenal glands, liver, brain, and bone.
Less common primary renal tumors include transitional cell carcinoma, Wilm's tumor (most often in children), and sarcoma.

Risk factors include:

- Smoking
- Obesity
- Excess use of phenacetin
- Acquired cystic kidney disease in dialysis patients
- Exposure to certain radiopaque contrast agents, asbestos, cadmium, and leather tanning and petroleum products
- Some familial syndromes, particularly von Hippel–Lindau disease

Signs & Symptoms:

- Symptoms usually do not appear until late, when the tumor may already be large and metastatic.
- Gross or microscopic hematuria is the most common manifestation, followed by flank pain, fever of unknown origin (FUO), and a palpable mass.
- Sometimes hypertension results from segmental ischemia or pedicle compression. - Paraneoplastic syndromes occur in 20% of patients.
- Hypercalcemia is common and may require treatment.

Investigations: CT with contrast or MRI

Management:

- Radical nephrectomy (removal of kidney, adrenal gland, perirenal fat, and Gerota fascia) is standard treatment for localized RCC and provides a reasonable chance for cure. Results with open or laparoscopic procedures are comparable; recovery is easier with laparoscopic procedures.
- Nephron-sparing surgery (partial nephrectomy) is possible and appropriate for many patients, even in patients with a normal contralateral kidney if the tumor is < 4 to 7 cm.

- Partial nephrectomy is gaining popularity because it results in a lower incidence of chronic kidney disease than radical nephrectomy.
- Palliation can include nephrectomy, tumor embolization, and possibly external beam radiation therapy. For some patients, drug therapy reduces tumor size and prolongs life

Wilm's tumour / Nephroblastoma:
Wilm's tumor is an embryonal cancer of the kidney composed of blastemal, stromal, and epithelial elements. Genetic abnormalities have been implicated in the pathogenesis, but familial inheritance accounts for only 1-2% of cases.

Wilm's tumor usually manifests in children < 5 year of age but occasionally in older children and rarely in adults. Wilm's tumor accounts for about 6% of cancers in children < 15 years of age.

Signs & Symptoms:

- The most frequent finding is a painless, palpable abdominal mass.
- Less frequent findings include abdominal pain, hematuria, fever, anorexia, nausea, and vomiting.
- Hematuria can be microscopic or gross.
- Hypertension may occur and is of variable severity.

Investigations: Abdominal ultrasonography, CT, or MRI; During surgery, locoregional lymph nodes are sampled for pathologic and surgical staging.

Management: Surgery and chemotherapy, Radiation therapy for patients with higher stage/risk disease.

DISEASES OF URINARY BLADDER

6. Diseases of Urinary bladder – Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital anomalies, Injuries, Ashmari - Vesical Calculus, Cystitis and Neoplasms.

CONGENITAL ANOMALIES OF URINARY BLADDER

Congenital urinary bladder anomalies often occur without other genitourinary abnormalities. They may cause infection, retention, incontinence, and reflux. Symptomatic anomalies may require surgery.

Bladder Diverticulum:

- A bladder diverticulum is a herniation of the bladder mucosa through a defect in bladder muscle. It predisposes to urinary tract infections (UTIs) and may coexist with vesicoureteral reflux.
- It is usually discovered during evaluation of recurrent UTIs in young children.
- Diagnosis of bladder diverticulum is by voiding cystourethrography.
- Surgical removal of the diverticulum and reconstruction of the bladder wall may be necessary.

Bladder Exstrophy:

- In exstrophy, there is a failure of midline closure from the umbilicus to the perineum, resulting in bladder mucosa continuity with the abdominal skin, separation of the pubic symphysis, and epispadias or bifid genitalia.
- The bladder is open suprapubically, and urine drips from the open bladder rather than through the urethra. Despite the seriousness of the deformity, normal renal function usually is maintained. The bladder can usually be reconstructed and returned to the pelvis, although vesicoureteral reflux invariably occurs and is managed as needed. Additional surgical intervention may be necessary to treat a bladder reservoir that fails to expand sufficiently or has sphincter insufficiency. Reconstruction of the genitals is required.

Megacystis Syndrome:

- In this syndrome, a large, thin-walled, smooth bladder without evident outlet obstruction develops, usually in girls. Megacystis syndrome is poorly understood. The syndrome may be a manifestation of a primary myoneural defect, especially when intestinal obstruction (e.g.: megacystis-microcolon, intestinal hypoperistalsis syndrome) is also present.
- Symptoms are related to UTIs, and vesicoureteral reflux is common.
- Ultrasonography with the bladder empty may disclose normal-appearing upper tracts, but voiding cystourethrography may show reflux with massive upper tract dilation. Ureteral reimplantation may be effective, although some patients benefit from antibacterial prophylaxis, timed voiding with behavioral modification, intermittent catheterization, or a combination.

Neurogenic Bladder:

- Neurogenic bladder is bladder dysfunction caused by neurologic disorders, including spinal cord or central nervous system abnormalities, trauma, or the sequelae of pelvic surgery (e.g.: for sacrococcygeal teratoma or imperforate anus). The bladder may be flaccid, spastic, or a combination. A flaccid bladder has high-volume, low-pressure, and minimal contractions.
- A spastic bladder has normal or low-volume, high-pressure, and involuntary contractions. When present, chronically elevated bladder pressure often causes progressive kidney damage, even without infection or reflux.
- Manifestations include recurrent UTIs, urinary retention and/or incontinence, and potentially renal insufficiency.
- Management goals include lowering risk of infection, maintaining adequate bladder storage pressure and volume, effective bladder emptying, and achieving social continence. Treatment of neurogenic bladder includes drugs (e.g.: anticholinergics, prophylactic antibiotics), intermittent catheterization, and/or surgical intervention (e.g.: augmentation cystoplasty, appendicovesicostomy, botulinum toxin injections, neurostimulation). Children with neurogenic bladder often also have a neurogenic bowel with constipation and stool incontinence that also require proper management.

INJURIES OF URINARY BLADDER

Types:

1. Extra-peritoneal (80%)
2. Intra-peritoneal

1. Extra peritoneal:

Aetiology:

- Fracture of pelvis (Road trauma accidents), fall over manhole
- Others: Stab wounds, gunshot wounds, during passage of cystoscope, pelvic surgeries etc.

Clinical features:

- The diagnosis of pelvic fracture is easily made by lateral compression on the bony pelvis, which will evoke pain and crepitus at the fracture site.
- Diffuse pain in lower abdomen with fullness and tenderness (Collection of urine and blood in extraperitoneal space)
- Dullness on percussion at suprapubic region
- Strangury and inability to pass urine

Investigation:
X ray: pelvic fracture
Retrograde cystourethrogram: to confirm the site

Treatment:

- Shock and haemorrhage should be treated by proper resuscitation.

- Surgery: A lower midline abdominal incision is made. Before reaching the bladder, one may see pelvic haematoma and extravasations of urine. The haematoma and urine are cleaned and bladder is opened in the midline. Repair extra-peritoneal rupture intravesically

2. Intra-peritoneal rupture:

Aetiology:
Blow or kick or fall on to a fully distended bladder (Drunk individuals)

Clinical features:

- Sudden, agonising pain in hypogastrium followed by hypotension, shock, and syncope.
- Patient does not feel the desire to pass urine.
- Lower abdominal guarding and rigidity after few hours of injury, which indicates peritonitis
- Distension of abdomen (Due to urine leakage)
- If there is a considerable amount of urine in the peritoneal cavity, shifting dullness may be elicited.

Investigation:

- X-ray: Ground glass appearance of the lower abdomen due to presence of urine in peritoneal cavity.
- Peritoneal tap to confirm the urine
- Cystogram

Treatment:
Surgery: A lower midline abdominal incision is made. Before reaching the bladder, one may see pelvic haematoma and extravasations of urine. The haematoma and urine are cleaned and bladder is opened in the midline. Then urethral catheter is introduced and midline bladder wound is closed, while intra-peritoneal rupture is repaired through trans-peritoneal approach.

ASHMARI

Nirukti:
अश्मानं रति ददति अश्मरि |
“Ashma” means “stone.”
“Rati” means “to present”
Ashmari means the formation and presentation of a substance like stone.

Nidana:
तत्र असंशोधनशीलस्य अपथ्यकारिणाः प्रकुपितः श्लेष्म मुत्र सम्प्रक्तोऽनुप्रविश्य बस्ति अश्मरी जनयति |
Those who neglect the Samshodhana of internal channels
Those who are engaged in unwholesome dietary habits become the victim of Ashmari.

Samprapti:
Nidana causes kapha prakopa, which mixes with mutra, entered in basti and formed ashmari.

Similes given by sushruta:
A new pitcher filled with clear water can also show settling down of muddy particles in due course of time. In the same way the calculi are formed in Basti.
As air & fire of electricity in the sky consolidate water (to form hail storms) similarly Pitta located in the bladder, along with Vayu consolidates Kapha to form calculi.

Purvarupa:

- Basti peeda (Pain in hypogastric region)
- Aruchi (Anorexia)
- Mutrakricchra (Dysuria)
- Bastishirovedana (Pain in Suprapubic region)
- Mushka Vedana
- Shepha Vedana
- Jwara (fever)
- Avasada
- Bastigandhatwa (Concentrated urine smell like goat's urine)
- Sandra Mutra (Sedimentation of urine)
- Avila Mutra (Turbidity of urine)

Rupa:

- Nabhi Vedana
- Mutra Dhara Sanga
- Gomeda Prakasha
- Dhavan, Plavan etc.
- Basti Vedana
- Mehana Vedana
- Atyavilam
- Sarudhira Mutra
- Sevani Vedana
- Mutra Vikirana
- Sasiktam

Classification of ashmari:

1. Shleshmaja ashmari
2. Pittaja ashmari
3. Vataja ashmari
4. Shukraja ashmari

Kaphaja	Pittaja	Vataja	Shukraja
Heavy and cold sensation in bladder area	Burning hot sensation and inflammatory changes in urinary tract	Severe bladder pain, umbilical and pain in the anus	Dysuria
Cutting, incising, pricking pain	Reddish / yellowish – black or honey like in colour	Frequent passage of flatus	Scrotal swelling
White, slimy, big like kukkutanda	Resembles bhallataka seed	Urethral burning	Lower abdominal pain

Colour – madhuka pushpavata		Dysuria	Special characteristic feature is, it can be crushed into powder by pressure
Heavy in weight		Difficulty in defecation. Dusty coloured, hard, irregular, rough and nodular like kadambapushpa	

Sadhyasadhyata:
In our classics Acharyas have described about 'Ashta Mahagadas' and these mahagadas are not easy to treat and they are not having good prognosis. As Ashmari is mentioned as one of them, so it requires attention for its cure.

Ashmari in balaka:
Ashmari in balaka is easily curable because of following reasons.

- Smaller space occupying lesion
- Less fat in subcutaneous and peri-nephric region the prognosis is better
- Can easily catch and remove

Upadrava:

- Daurbalya
- kukshi Shula
- Ushnavata
- Vamana
- Sadana
- Aruchi
- Trishna
- karshya
- Pandu
- Hruta peeda

Chikitsa:
Chikitsa in purva rupa:

1. Snehana
2. Swedana
3. Vamana
4. Virechana etc.

Ayurvedic formulations:

- Varunadi kashaya
- Chandraprabha vati
- Tab crush
- Gokshuradi kashaya
- Tab cystone
- Tab clacurosin

Panchakarma chikitsa:

1. Virechana: Tilvak ghrita etc
2. Basti: Especially Uttara basti
3. Niruha basti
4. Anuvasana basti: Peetadaru siddha taila

Shalya karma chikitsa:
Purvakarma:

1. Snehana & swedana 7 days before operation
2. Virechana 2 days before operation
3. Abhyanga & sweda on day of operation
4. Laghu bhojana 1 hr before operation
5. Bali-mangala & swastivachana
6. Aashawasan (Counselling & assurance)

Pradhana karma:

1. Nabhi snehana with left thumb to bring down Ashmari
2. Lubricate left mid & index finger with goghrita & introduced them in to patient's rectum with the palmar aspect upward to go behind bladder
3. Now pass the bladder in between the fingers in rectum & thumb on perineum.
4. The manipulation should be so tactful; the Ashmari must come to his grip & must be able to press ashmari accurately & exactly between fingers and thumbs.

Pashchata karma:

1. Basti shodhana by pancha valkal kashaya
2. Tub bath
3. Apply lodhra, yashti, manjishta, haridra & goghrita from 4th day onwards.
4. Agnikarma after 1 week.

VESICAL CALCULI / BLADDER STONE / CYSTOLITHIASIS

Vesical calculi are caused by a build-up of minerals. They can occur if the bladder is not completely emptied after urination. Eventually, the leftover urine becomes concentrated and minerals within the liquid turn into crystals.
Sometimes, these stones will be passed with urine while they are still small. Other times, bladder stones can get stuck to the wall of the bladder. If this happens, they gradually gather more mineral crystals, becoming larger over time.

The smallest bladder stones are barely visible to the naked eye, but some can grow to an impressive size. The largest bladder stone, according to Guinness World Records, weighed almost 1.9 kg and measured 17.9 x 12.7 x 9.5 centimetres.

Etiology & Risk Factors:

- Bladder stones start to grow when urine is left in the bladder after urinating.
  This is often due to an underlying medical condition that prevents complete evacuation, such as neurogenic bladder, prostate enlargement, cystitis, nephrolithiasis, bladder diverticula, cystocele.
- Males are at higher risk of developing bladder stones, especially with age.

Signs & Symptoms:
Bladder stones may not produce symptoms straight away. But, if the stones grow large enough, they may irritate the bladder or urethra.

Symptoms can include the following:

- Discomfort or pain in the penis for males
- Urination with discomfort and pain
- Lower abdominal pain
- Frequent urination
- Difficult urination or interrupted flow
- Starting a stream during urination takes longer
- Hematuria
- Cloudy or abnormally dark urine

Diagnosis: Physical examination, Urinalysis, CT scan, USG, X-ray, IVP

Management:

- Intake of plenty water is sufficient to expel small bladder stones.
- Large calculi may require cystolitholapaxy or surgical removal.
- During a cystolitholapaxy, an instrument called a cystoscope is inserted into the bladder to locate the bladder stone or stones. A laser is used to break up the stones into smaller fragments which are then removed.

CYSTITIS

Definition: Inflammation of the urinary bladder mucosa is called as cystitis.

Causes:

Common .in females, because of short urethra in females may cause ascending infection and cystitis

Honeymoon cystitis → Initial period of sexual contact in females can cause diffuse form of cystitis.

Causative organisms → E coli, klebsiellae, pseudomonas, staphylococcus aureus (Acute cystitis)

Predisposing factors:

- Catheters, instrumentation
- Bladder stone, bladder diverticulum, bladder neck obstruction, bladder tumours

Clinical features:

- Frequency, urgency, and dysuria are hallmark of cystitis
- Low backache
- Fever, chills, and rigors
- Suprapubic pain and tenderness

Investigation:

- Leucocytosis
- Urine Microscopy: More than 5 WBC (females) and 2-3 WBC (Males)
- Culture study to know organism
- Cystoscopy: Bladder inflammation, mucosal changes

Treatment:

- Plenty of water intake to flush the bacteria from bladder
- Urine alkalisers like Neeri, Chitraka
- Local hot and Sitz bath
- Appropriate antibiotics
- Antipyretics, analgesics and antispasmodics

URINARY BLADDER CANCER

Bladder cancer is usually transitional cell (urothelial) carcinoma.
Patients usually present with hematuria (most commonly) or irritative voiding symptoms such as frequency and/or urgency; later, urinary obstruction can cause pain.

Risk factors include:

- Smoking (the most common risk factor, causing ≥ 50% of new cases)
- Excess phenacetin use (analgesic abuse)
- Long-term cyclophosphamide use
- Chronic irritation (e.g.: in schistosomiasis, by chronic catheterization, or bladder calculi)
- Exposure to hydrocarbons, tryptophan metabolites, or industrial chemicals, notably aromatic amines (aniline dyes, such as naphthylamine used in the dye industry) and chemicals used in the rubber, electric, cable, paint, and textile industries.

Types:

1. Transitional cell carcinomas (urothelial carcinoma), which account for > 90% of bladder cancers. Most are papillary carcinomas, which tend to be superficial and well differentiated and to grow outward; sessile tumors are more insidious, tending to invade early and metastasize.
2. Squamous cell carcinomas, which are less common and usually occur in patients with parasitic bladder infestation or chronic mucosal irritation.
3. Adenocarcinomas, which may occur as primary tumors or rarely reflect metastasis from intestinal carcinoma. Metastasis should be ruled out.

Signs & Symptoms:

- Most patients present with unexplained hematuria (gross or microscopic).
- Some patients present with anemia, and hematuria is detected during evaluation. - Irritative voiding symptoms (dysuria, burning, frequency) and pyuria are also common at presentation.
- Pelvic pain occurs with advanced cancer, when a pelvic mass may be palpable.

Investigations: Cystoscopy with biopsy, Urine cytology

Management:

- Transurethral resection and intravesical immunotherapy or chemotherapy (for superficial cancers)
- Cystectomy or radiation with chemotherapy (for invasive cancers)

MUTRAGHATA AND MUTRAKRICHRA

7. Mutraghata and Mutrakrichra - Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management. Retention of urine.

MUTRAGHATA

Nidana:

- Vegavarodha (suppression of urges)
- Ashmari (Stones)
- Excessive intake of ruksha, ushna and teekshana materials
- Vibandha (Constipation)
- Abhighata (trauma)

Types: 12

1. Vatakundalika
2. Vatasthila
3. Vata vasti
4. Mutratita
5. Mutra jathara
6. Mutra utsanga
7. Mutra kshaya
8. Mutra granthi
9. Mutra shukram
10. Ushnavata
11. Mutrauksada (2 types)

1. Vatakundalika:

Nidana leads to aggravation of vata, which localises in bladder causing severe pain in this region and passes the urine in drops.

2. Vatashtila:

The aggravated Vata produces an enlargement like a stone, leading to distension of bladder and rectum, severe pain and obstruction to passage of urine and faeces.

3. Vatavasti:

Vegavarodha etc for long time, aggravate vata, which localising in bladder outlet, blocks the urinary passage, resulting in retention of urine and pain over region of bladder.

4. Mutratita:

When patient tries to pass urine, he passes it slowly in drops.

5. Mutra jathara:

Vegavarodha makes apana vayu to move upwards, causing distension of abdomen below region of umbilicus associated with severe pain and inability to pass urine.

6. Mutrotsanga:

Irrespective of site of obstruction, whenever the person tries to pass urine, he has to strain and when he passes, the urine is mixed with blood and is in small quantities. This may or may not be associated with pain.

7. Mutra kshaya:

In person who takes dry foods and does excessive physical activity, pitta and vata get aggravated, localise in bladder, leading to passage of urine in small quantities with pain and burning sensation.

8. Mutra granthi:

Sudden development of swelling (round) inside the bladder (internal urethral orifice), which is fixed and produces symptoms like ashmari is termed as mutra granthi.

9. Mutra shukra:

Shukra vegavarodha makes semen to get mixed with urine, making urine appear like water mixed with ash (chyleuria).

10. Ushnavata:

Excessive physical exercise, exposure to sun etc aggravate pitta. which along with causes burning sensation in bladder, genitalia and perineum, makes the urine yellow and mixed blood.

11. Mutrauksada:

a. Pittaja: Burning micturition which is clear and yellow. On drying urine resembles gorochana powder
b. Kaphaja: Dysuria, which is slimy, concentrated and whitish. On drying urine resembles shankha churna

Chikitsa:

- Different forms of medication depend upon their uses (Kashaya, kalka, sarpi, leha, payasa, kshara, madhu, asava, swedana)
- Snehana and swedana
- Uttar basti
- Virechana
- Kalka of bala, svadamshtra, kraunch asthi, Kokilaksha, devadaru, chitraka with sura – orally

URINARY RETENTION

Urinary retention is incomplete emptying of the bladder or cessation of urination.
It may be acute or chronic.
Causes include impaired bladder contractility, bladder outlet obstruction, detrusor-sphincter dyssynergia (lack of coordination between bladder contraction and sphincter relaxation), or a combination.

Retention is most common among men, in whom prostate abnormalities or urethral strictures cause outlet obstruction. In either sex, retention may be due to drugs (particularly those with anticholinergic effects), severe faecal impaction (which increases pressure on the bladder), or neurogenic bladder in patients with diabetes, multiple sclerosis, Parkinson ‘s disease, or prior pelvic surgery resulting in bladder denervation.

Symptoms & Complications:
Urinary retention can be asymptomatic or cause urinary frequency, a sense of incomplete emptying, and urge or overflow incontinence.
It may cause abdominal distention and pain. When retention develops slowly, pain may be absent.
Long-standing retention predisposes to UTI and can increase bladder pressure, causing obstructive uropathy.

Diagnosis: Diagnosis is obvious in patients who cannot void. In those who can void, incomplete bladder emptying is diagnosed by postvoid catheterization or ultrasonography showing an elevated residual urine volume. A volume < 50 mL is normal; < 100 mL is usually acceptable in patients > 65 years but abnormal in younger patients. Other tests (e.g.: urinalysis, blood tests, ultrasonography, urodynamic testing, cystoscopy, cystography) are done based on clinical findings.

Management: Urethral catheterization and treatment of specific cause

MUTRA KRUCHCHRA
Types:

1. Vataja
2. Pittaja
3. Kaphaja
4. Sannipataja
5. Abhighataja
6. Shakruda
7. Ashmari
8. Sharkara

1. Vataja mutra kruchchra:

- Passes urine in small amount with difficulty producing pain
- Tearing sensation in the scrotum, penis, and urinary bladder

Treatment:
Shavadamshtradi taila or ghrita used as orally, anuvasana basti and uttar basti
Oil should be processed with the expressed juice of shvadamshtra, along with jaggery, milk and ginger; it should be administered as above.

2. Pittaja mutra kruchchra:

- Passes turmeric coloured or bloody urine with scalding sensation
- Feels his scrotum, penis, and urinary bladder as if burning with fire

Treatment:
Ghee or milk processed with trina panchmula, utpala, kakoli and nygrodha gana should be taken orally and for urethral irrigation.
Fat medicated with above said drugs also administered as basti.
Laxatives with milk along with juices of grapes and sugarcane

3. Kaphaja mutra kruchchra:

- Passes slimy and colourless urine which is not warm
- Horripilation
- Feeling of heaviness in the scrotum, penis and the urinary bladder

Treatment:
Taila and yava processed with surasa, ushaka, musta and varuna.

4. Sannipataja mutra kruchchra:

- Passes urine of various colours again and again with difficulty, pain and either warm or a cold sensation
- Feels as if sinking into darkness

5. Abhighataja mutra kruchchra:

- An injury to the urinary system after surgery or trauma produces very painful obstructive
- uropathy of its channels Clinical features are similar to those of vatabasti.

Treatment:
Sadhyo vrana chikitsa

6. Shakrud pratighata mutra kruchchra:

- When there is faecal retention, movement of apana vayu get reversed
- Produces aadhmana, Shula and mutrasanga

Treatment:
Vatahara treatment
Swedana, avagaha, basti, churna kriya

7. Sharkaraja mutra kruchchra:

- The disintegrated particles of kapha, transformed by pitta and then broken up by vayu are known as sharkara.
- Precordial pain, rigor, colicky pain in the flanks, impaired digestive power, fainting and dysuria.
- When sharkara are passed with the flow of urine, pain subsides and this period of remission lasts as long as another sharkara does not obstruct the urinary passage.

DYSURIA

Dysuria is painful or uncomfortable urination, typically a sharp, burning sensation.
Dysuria results from irritation of the bladder trigone or urethra. Inflammation or stricture of the urethra causes difficulty in starting urination and burning on urination. Irritation of the trigone causes bladder contraction, leading to frequent and painful urination.
Dysuria most frequently results from an infection in the lower urinary tract, but it could also be caused by an upper urinary tract infection (UTI). Impaired renal concentrating ability is the main reason for frequent urination in upper UTIs.

DISEASES OF PROSTATE

8. Diseases of Prostate - Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Prostatitis, Prostatic abscess, Benign Enlargement of Prostate and Carcinoma of Prostate.

PROSTATITIS / SHUKRA GRANTHI SHOTHA

Inflammation of prostate is called as prostatitis. In this condition addition to prostate, seminal vesicles and posterior urethra arc involved.

Types:

1. Acute
2. Chronic

1. Acute prostatitis:

Causes:

- Haematogenous: Causative organisms are E coli, klebsiella, staphylococcus faecalis and staphylococcus albus reach through blood
- Instrumentation
- Ascending infection from below or descending infection from above

Clinical features:

- High grade fever, chills and rigors
- Burning micturition
- Perineal heaviness or pain on defecation and urethral discharge
- Bodyache specially low backache
- Rectal examination: Tender, boggy, enlarged prostate.

Treatment:

- IV fluids, analgesics, antipyretics
- Hospitalisation, Sitz bath
- Antibiotics like norfloxacin should be given for 2-3 weeks to prevent recurrence.

Note: avoid alcohol and sexual intercourse for 6 weeks.

2. Chronic prostatitis:

Common in elderly men

Chronic prostatitis results due to inadequately treated acute prostatitis.

May develop secondary to cystitis and pyelonephritis

Clinical features:

- Low grade fever
- Urethral discharge
- Pain in perineum, rectum and low back
- Pain on sexual intercourse
- Rectal examination may reveal boggy and tender prostate

Diagnosis:
Prostate massage is given by bidigital method, index finger in the rectum and thumb in the perineum to one side. Now the patient is asked to void the urine. Presence of prostatic threads in urine is diagnostic of chronic prostatitis.

Treatment:
Prolonged antibiotic therapy (Trimethoprim + Sulfamethoxazole)

PROSTATIC ABSCESS
Prostate abscesses are focal purulent collections that develop as complications of acute bacterial prostatitis.
The usual infecting organisms are aerobic gram-negative bacilli or, less frequently, Staphylococcus aureus.

Symptoms:
Common symptoms:

- Urinary frequency
- Urinary retention
- Dysuria

Less common symptoms:
Perineal pain, fever, evidence of acute epididymitis, hematuria, and purulent urethral discharge.

Diagnosis: Prostate ultrasonography and possibly cystoscopy

Management: Antibiotics, Drainage
Treatment involves appropriate antibiotics plus drainage by transurethral evacuation or transperineal aspiration and drainage.

BENIGN PROSTATIC HYPERPLASIA / HYPERTROPHY (BPH)
Benign prostatic hyperplasia (BPH) is non-malignant adenomatous overgrowth of the periurethral prostate gland

Pathophysiology:
Multiple fibroadenomatous nodules develop in the periurethral region of the prostate, probably originating within the periurethral glands rather than in the true fibromuscular prostate (surgical capsule), which is displaced peripherally by progressive growth of the nodules.

As the lumen of the prostatic urethra narrows and lengthens, urine outflow is progressively obstructed. Increased pressure associated with micturition and bladder distention can progress to hypertrophy of the bladder detrusor, trabeculation, cellule formation, and diverticula.

Incomplete bladder emptying causes stasis and predisposes to calculus formation and infection. Prolonged urinary tract obstruction, even if incomplete, can cause hydronephrosis and compromise renal function.

Clinical features:

1. Frequency: Occurs due to visceral introversion of sensitive prostatic mucus membrane. Started with day time, later day and night time (5-10 times during night). It is due to irritability of bladder and amount of residual urine.
2. Urgency: Internal sphincter mechanism is deranged due to invasion of prostate into bladder. This result in few drops of urine tickling down the post urethra resulting in urgent desire to pass urine.
3. Hesitancy: Patient must wait due to obstruction of internal urethral orifice by median lobe
4. Difficulty in micturition with weak stream and dribble
5. Retention of urine (Acute/chronic)
6. Haematuria

Diagnosis:

- Digital rectal examination
- Urinalysis and urine culture
- Prostate-specific antigen level
- Sometimes uroflowmetry and bladder ultrasonography
- Transrectal biopsy is usually done with ultrasound guidance and is usually only indicated if there is suspicion of prostate cancer. Transrectal ultrasonography is an accurate way to measure prostate volume.

Treatment:
Conservative management:

- Avoid heavy alcohol consumption
- Patient should void as soon as he feels the urge to do
- Correction of electrolyte, urea, and creatinine
- Catheterisation in acute retention. If fails then suprapubic catheterisation
- Drugs: Finasteride acetate 5mg daily for 6 months (to decrease the size) and Alpha-
- adrenergic blocker to relax internal sphincter for better drainage of bladder.

Surgical treatment:
Indications:

- Acute urine retention
- Chronic urine retention with post void residual urine > 200ml
- Prostatitism (Frequency, dysuria, urgency)
- Complication like haematuria, hydro-uretero-nephrosis etc

Methods:

1. Transurethral resection of prostate (TURP)
2. Transvesical suprapubic prostatectomy (Freyers)
3. Retropubic prostatectomy (Millins)
4. Perineal prostatectomy (Youngs)

PROSTATE CANCER

- Prostate cancer is usually adenocarcinoma.
- Adenocarcinoma of the prostate is the most common non-dermatologic cancer in men 50 in the US. Incidence increases with each decade of life; autopsy studies show prostate cancer in 15-60% of men aged 60-90 years old, with incidence increasing with age. The lifetime risk of being diagnosed with prostate cancer is 1 in 6. Median age at diagnosis is 72, and > 75% of prostate cancers are diagnosed in men > 65.
- Risk is highest for black men.

- Sarcoma of the prostate is rare, occurring primarily in children. Undifferentiated prostate cancer, squamous cell carcinoma, and ductal transitional carcinoma also occur infrequently. Prostatic intraepithelial neoplasia is considered a possible premalignant histologic change.
- Hormonal influences contribute to the course of adenocarcinoma but almost certainly not to other types of prostate cancer.

Signs & Symptoms:
Prostate cancer usually progresses slowly and rarely causes symptoms until advanced. In advanced disease, haematuria and symptoms of bladder outlet obstruction (e.g.: straining, hesitancy, weak or intermittent urine stream, a sense of incomplete emptying, terminal dribbling) may appear.
Bone pain, pathologic fractures, or spinal cord compression may result from osteoblastic metastases to bone (commonly pelvis, ribs, vertebral bodies).

Diagnosis:

- Screening by digital rectal examination (DRE) and prostate-specific antigen (PSA)
- Assessment of abnormalities by transrectal needle biopsy
- Grading by histology
- Staging by CT and bone scanning

Management:

- For localized cancer within the prostate, surgery, or radiation therapy
- For cancer outside of the prostate, palliation with hormonal therapy, radiation therapy, or chemotherapy
- For some men who have low-risk cancers, active surveillance without treatment
- Treatment is guided by prostate-specific antigen (PSA) level, grade and stage of tumour, patient age, coexisting disorders, and life expectancy.

DISEASES OF URETHRA

9. Diseases of Urethra – Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Urethritis, Stricture and Rupture.

URETHRITIS

Urethritis is inflammation (swelling and irritation) of the urethra. It is classified under lower urinary tract infections.

Etiology & Risk Factors:
Both, bacteria, and viruses may cause urethritis.
Some of the bacteria that cause this condition include E. coli, Neisseria gonorrhoeae, Chlamydia trachomatis. These bacteria also cause UTIs and STDs.
Viral causes are herpes simplex virus (HSV) and cytomegalovirus (CMV).

Other causes include:

- Injury
- Sensitivity to the chemicals used in spermicides, contraceptive jellies, or foams

Sometimes the cause is unknown.
Females are at higher risk in general, males at age 20-35 as well. Multiple sex partners, history of STD or high-risk sexual behaviour (e.g.: anal sex without protection) also increase the risk of contracting urethritis.

Signs & Symptoms:

1. In men

- Hematuria, Hematospermia
- Dysuria, Urinary frequency
- Abnormal penile discharge (purulent / whitish / mucoid)
- Itching, tenderness, or swelling
- Enlarged lymph nodes in the groin area
- Pain during sexual intercourse or with ejaculation
- Fever

2. In women

- Abdominal pain
- Dysuria, Urinary frequency
- Abnormal vaginal discharge
- Fever and chills
- Pelvic pain, Dyspareunia

Investigations:
Physical evaluation, Digital rectal exam, Cystoscopy, CBC, C-reactive protein test, Pelvic ultrasound, Pregnancy test, Urinalysis, Urine cultures, Urethral swab

Management:
- Antibiotics or Antivirals
- Analgesics
- Avoidance of sexual intercourse, or with protection (condom)
- Avoidance of chemicals in case of sensitivity

Complications:
Men with urethritis are at risk for the following:
- Cystitis
- Epididymitis
- Orchitis
- Prostatitis
- Long-term damage to the urethra may cause urethral stricture.

Women with urethritis are at risk for the following:
- Cystitis
- Cervicitis
- Pelvic inflammatory disease (PID; infection of the uterus, fallopian tubes, ovaries)

URETHRAL STRICTURE
Urethral stricture is scarring that obstructs the anterior urethral lumen.
Urethral stricture can be congenital or acquired.
Anything that damages the urethral epithelium or corpus spongiosum can cause acquired stricture.

Common causes include:
- Trauma
- STDs such as gonorrhoea
- Unknown causes (idiopathic strictures)

Trauma, the most common cause, may result from a straddle injury or, occasionally, an iatrogenic injury (e.g.: after traumatic endoscopy or catheterization).
Less common causes include:
- Lichen sclerosus
- Urethritis (usually chronic or untreated)
- Malignancy (urethral cancer)

Signs & Symptoms:
Symptoms may not develop until the urethral lumen has been decreased considerably.
Strictures may cause:

- Double urine stream
- Obstructive voiding symptoms (e.g.: weak urinary stream, hesitancy, incomplete emptying)
- Recurrent urinary tract infections (including prostatitis)

Diagnosis: Retrograde urethrography or cystoscopy

Urethral stricture is usually suspected when urethral catheterization is difficult. It should also be considered in males with gradual onset of obstructive symptoms or recurrent urinary tract infections, particularly if they have risk factors or are young.

Management:

- Dilation or internal urethrotomy
- Self-catheterization
- Open urethroplasty

Urethral trauma & rupture

- Urethral injuries usually occur in men. Most major urethral injuries are due to blunt trauma. Penetrating urethral trauma is less common, occurring mainly as a result of gunshot wounds, or, alternatively, due to inserting objects into the urethra during sexual activity or because of psychiatric illness.

- Urethral injuries are classified as contusions, partial disruptions, or complete disruptions, and they may involve the posterior urethra (membranous and prostatic) or anterior urethra (bulbous and penile urethra).
- Posterior urethral injuries occur almost exclusively with pelvic fractures.
- Anterior urethral injuries are often consequences of a perineal blow, motor vehicle crash, or perineal straddle injury due to a fall. Iatrogenic injuries occur during transurethral instrumentation (e.g.: catheter placement or removal, cystoscopy).

- Complications include infection, incontinence, erectile dysfunction, and stricture or stenosis ("stenosis" is narrowing of the posterior urethra whereas "stricture" refers exclusively to the anterior urethra

Signs & Symptoms:

- Pain with voiding or inability to void.
- Blood at the urethral meatus is the most important sign of a urethral injury.
- Additional signs include perineal, scrotal, penile, and labial ecchymosis, edema.

Diagnosis: Retrograde urethrography

Management:

- Usually urethral catheterization (for contusions) or suprapubic cystostomy
- Sometimes endoscopic realignment or surgical repair (for select injuries)
- Delayed definitive surgery

URETHRAL RUPTURE

- Rupture of the urethra is an uncommon result of penile injury, incorrect catheter insertion, straddle injury, or pelvic girdle fracture.
- When urethral rupture occurs, urine may extravasate (escape) into the surrounding tissues. The membranous urethra is most likely to be injured in pelvic fractures, allowing urine and blood to enter the deep perineal space and subperitoneal spaces via the genital hiatus.
- The spongy urethra is most likely to be injured with a catheter or in a straddle injury, allowing urine and blood to escape into the scrotum, the penis, and the superficial peritoneal space.
- Urethral rupture may be diagnosed with a cystourethrogram.
- Due to the tight adherence of the fascia lata, urine from a urethral rupture cannot spread into the thighs.

DISEASES OF PENIS

10. Diseases of Penis: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Congenital anomalies, Niruddhaprakasha -Phimosis, Parivartika -Paraphimosis, Avapatika - Prepuceal ulcer, Arbuda- Tumours and Lingarsha - Penile Warts.

CONGENITAL ANOMALIES OF PENIS

1. Aphallia:
- Agenesis of penis caused by failure in embryologic development of genital tubercle.
- Incidence of 1 per 10 million male births
- Associated with other genito-urinary abnormalities and with musculoskeletal and cardiopulmonary defects

2. Chordee:
- This anomaly is ventral, lateral, and/or rotational curvature of the penis, which is most apparent with erection and is caused by fibrous tissue along the usual course of the corpus spongiosum, or by a size difference between the two corpora cavernosa. Chordee may be associated with hypospadias.
- Severe deformity may require surgical correction.

3. Concealed penis:
- Also called hidden or buried penis
- Penis is normally developed but hidden under fat in supra-pubic region, scrotum, perineum and thigh.

4. Diphallia:
- Duplication of penis
- Occurs in 1 per 5 million male births
- Associated with hypospadias, bifid scrotum, bladder duplication and renal agenesis.

5. Lateral curvature:
- Due to hypo/hyperplasia of one corpora cavernosa
- Surgical treatment is often effective

6. Epispadias:
- In this condition urethra opens on dorsum of penis, proximal to the glans.
- Most common site is at the abdomino-penile junction
- Treatment is correction of incontinence of urethra and urinary diversion.

7. Hypospadias:

A congenital malformation of urethra, where external meatus is situated at point on under surface of penis.

Types:
Depending upon abnormal position of external meatus:

1. Glandular variety:
- In this external meatus is situated few mm away from normal site within the glans.

2. Coronal variety:
- In this urethra opens at the corona glandis, junction of glans and body of penis
3. Penile hypospadias:
- In this external opening is situated at any part of under surface of body of penis.
4. Peno scrotal/perineal hypospadias:
- Urethral opening is seen at the junction of penis and scrotum is called as peno-scrotal.
- Scrotum is split and urethra opens between its two halves is said to be perineal one. It is associated with bilateral undescended testis

Clinical features:
Incidence of 1 per 350 males
Micturition: Normal, urine deflected downwards thus spoiling the underwears and may cause infection and dermatitis.
Penile variety is associated with chordee (bending of penis) who causes difficulty in erection and sexual intercourse.

Treatment:
Correction of chordee at the age of 1.5 years
Urethroplasty (5-7 years)
MAGPI repair: Meatal advancement glandular repair (Glandular variety)
Note: In hypospadias, circumcision is contraindicated, as prepuce is necessary for future urethroplasty.

NIRUDDHA PRAKASHA
Obstruction to exposure of light over glans penis is called as niruddha prakasha.

Nidana → vata vikruti → vata get shelter in prepuce and glans → obstructed urinary passage → patient passes thin stream of urine without pain and glans cannot be exposed.

Treatment:
- Gentle dilatation by Nāḍī Yantra after applying Ghṛta should be done every third day.
- Pariṣeka over Shishna Maṇi with Chakra taila or Vātahara taila
- Snigdhānna
- Shastra karma; removal of Shishna Charma (circumcision).

It should then be treated as Sadyovraṇa.

PHIMOSIS

Inability to retract preputial skin over the glans penis.

Aetiology:

1. Congenital
- Pin hole meatus
2. Acquired:

a. Inflammatory: It is common in diabetes patients (Due to presence of glucose in them urine giving rise to infection in the prepuce)

- Balanitis: Inflammation of glans
- Posthitis: Inflammation of prepucial skin
- Balano-posthitis: Both

b. Traumatic: Vigorous stretching during masturbation or forceful trauma → prepucial fibrosis narrowing of opening of prepuce → phimosis

c. Neoplastic: CA of penis

Clinical features:

- Inability to retract the prepuce
- Difficulty in micturition: Mother complains that when child micturate the prepuce balloons out (Second bladder*) and urine comes out in thin stream (Pin hole meatus)
- Pain and purulent discharge coming out through preputial orifice in old cases.

Treatment:

- Steroid creams application (70% success): Loosens the tight skin, reducing the body's inflammatory and immune response and by thinning the skin.
- Manual stretching
- Dorsal slit (Super-incision) is a single incision along the upper length of foreskin from the tip to corona, exposing the glans without removing any tissue.
- Circumcision

Complication:

- Recurrent balanoposthitis
- Preputial calculi: Due to phimosis more amount of smegma secreted which mixed with salt of urine and get collected within preputial layers giving rise to stone appearance.
- Para-phimosis
- Retention of urine
- CA of penis

Circumcision:

Removal of preputial skin

Indications:
Ritual → religious
Phimosis, paraphimosis, balanoposthitis of diabetes, early CA of prepuce

Contraindication:
Hypospadias

Anaesthesia:
Children → GA
Adults → LA (Root of penis)

Procedure:
Adults:

- Hold skin of tip of penis
- Separate prepuce from glans
- Dorsal skin is cut up to corona glandis
- Trim preputial layer parallel to corona
- Precautions: Less skin cut at ventral side to prevent Chordee and avoid damage to frenular artery
- Frenular artery is transfixed and ligated
- Apply interrupted chromic catgut suture to layers of prepuce
- Dressing

Children:

- Hold prepuce by 2 artery forceps
- Apply gentle traction
- Apply artery clamp distal to glans
- Skin distal to clamp is removed
- Identify and ligate bleeding points

Post operative:

- Antibiotics
- Analgesics and sedatives
- Removal of suture is very painful, so use absorbable sutures

Post operative complications:

- Injury to glans penis can occur when there is extensive adhesion between prepuce and glans
- Reactionary haemorrhage due to slippage of ligature
- Chordee: If more skin is cut on ventral aspect

PARIVARTIKA

Excessive rubbing, squeezing and trauma → vyana vayu vikruti → It reaches the penis and retracts the skin leaving the glans below → then prepuce swells up like a knot and hangs down from glans penis → parivartika

Clinical features:
Pain, burning and suppuration
Itching

Treatment:

- Ghrita abhyanga & upanaha sweda followed by vatahara chikitsa
- Try manual reduction gently
- Then once again upanaha sweda
- Vataghna basti & Snigdha anna

PARAPHIMOSIS

Inability to place back the retracted preputial skin over the glans is called paraphimosis.

Etiology:
Follow sexual intercourse
Professional cause: During catheterization if retracted prepuce is not pull forwards
Trauma: Direct forceful retraction of phimotic prepuce

Pathology:
Causes → prepuce get stuck in corona glandis → obstruction of venous blood flow → edema and congestion of glans → swelling of prepuce → severe pain → neglected → necrosis → gangrene → glans will fall off

Clinical features:
Severe pain and swelling in glans penis
Normal micturition as urethra not compressed
Edema at corona glandis

Treatment:

1. Manual gentle reduction can be tried
2. Inj. hyaluronidase in to constricted preputial skin, it reduces oedema later gentle manipulation is possible
3. Dundee technique: Needle pricking on prepuce
4. Dorsal slit of prepuce
5. Circumcision
6. Antibiotics and analgesics

AVAPATIKA

Avapāṭikā is the condition in which Shishna Charma gets torn (prepuceal laceration).

Nidāna:

- Sexual intercourse with an adolescent girl (Bālā) having narrow vaginal orifice.
- Hastābhighāta (injury by hand) in which Shishna Charma gets forcibly retracted.
- Mardanāt Pīḍanāt (excessive forceful rubbing or squeezing)
- Shukravega vidhāraṇa

Chikitsā: Parivartikā & Sadyovraṇa Chikitsā

PREPUCEAL ULCER

Genital ulcers, defined as single or multiple vesicular, ulcerative or erosive lesions of the genital tract, with or without inguinal lymphadenopathy, should lead to consideration of sexually transmitted infection.
The principal causative organisms are Treponema pallidum (syphilis), Haemophilus ducreyi (chancroid) and Herpes simplex (genital herpes).
Chlamydia trachomatis (lymphogranuloma venereum) and Calymmatobacterium granulomatis (donovanosis) are less frequent.

BALANTITIS, POSTHITIS & BALANOPOSTHITIS

Balanitis is inflammation of the glans penis, posthitis is inflammation of the prepuce, and balanoposthitis is inflammation of both.

Inflammation of the head of the penis has both infectious and non-infectious causes. Often, no cause can be found.

Infectious: Candidiasis, Chancroid, Chlamydial urethritis, Gonococcal urethritis, Herpes simplex virus infection, Molluscum contagiosum, Scabies, Syphilis, Trichomoniasis
Non-infectious: Balanitis xerotica obliterans, Contact dermatitis, Fixed drug eruptions, Lichen planus, Psoriasis, Reactive arthritis, Seborrheic dermatitis

Balanitis usually leads to posthitis except in circumcised patients.
Balanoposthitis is predisposed by:

- Diabetes mellitus
- Phimosis

Phimosis interferes with adequate hygiene. Subpreputial secretions may become infected with anaerobic bacteria, resulting in inflammation.
Chronic balanoposthitis increases the risk of Balanitis xerotica obliterans, Phimosis, Paraphimosis, and Cancer.

Signs & Symptoms: Pain, irritation, and a subpreputial discharge often occur 2 or 3 days after sexual intercourse. Phimosis, superficial ulcerations, and inguinal adenopathy may follow.

Diagnosis: Clinical evaluation and selective testing

History should include investigation of latex condom use. The skin should be examined for lesions that suggest a dermatosis capable of genital involvement. Patients should be tested for both infectious and non-infectious causes, especially candidiasis. Blood should be tested for glucose.

Management:

- Hygiene and treatment of specific causes
- Sometimes subpreputial irrigation
- Sometimes circumcision

LINGARBUDA / SHISHNARBUDA / PENILE CANCER

- Most penile cancers are squamous cell carcinomas; they usually occur in uncircumcised men, particularly those with poor local hygiene.
- Human papillomavirus (HPV) plays a role in etiology.
- Premalignant lesions include erythroplasia of Queyrat, Bowen disease, and bowenoid papulosis. Erythroplasia of Queyrat (affecting the glans or inner prepuce) and Bowen disease (affecting the shaft) progress to invasive squamous cell carcinoma in 5-10% of patients; bowenoid papulosis does not appear to do so.
- The 3 lesions have different clinical manifestations and biologic effects but are virtually the same histologically; they may be more appropriately called intraepithelial neoplasia or carcinoma in situ.

Signs & Symptoms:

- Most squamous cell carcinomas originate on the glans, in the coronal sulcus, or under the foreskin. They usually begin as a small erythematous lesion and may be confined to the skin for a long time.
- These carcinomas may be fungating and exophytic or ulcerative and infiltrative.
- The latter type metastasizes more commonly, usually to the superficial and deep inguinofemoral and pelvic nodes.

- Most patients present with a sore that has not healed, subtle induration of the skin, or sometimes a pus-filled or warty growth.
- The sore may be shallow or deep with rolled edges. Many patients do not notice the cancer or do not report it promptly. Pain is uncommon. Inguinal nodes may be enlarged due to inflammation and secondary infection.

Diagnosis: Biopsy; CT or MRI helps in staging localized cancer, checking for invasion of the corpora, and evaluating lymph nodes.

Management:

- Untreated penile cancer progresses, typically causing death within 2 years. Treated early, penile cancer can usually be cured.

- Topical treatment with 5-fluorourcil or imiquimod and laser ablation are effective for small, superficial lesions.
- Circumcision is done for lesions of the foreskin.
- Wide excision is preferred for recurrent lesions or in patients who cannot reliably follow up. Mohs surgery, when available, can be done instead of wide excision.

- Invasive and high-grade lesions require more radical resection. Partial penectomy is appropriate if the tumour can be completely excised with adequate margins, leaving a penile stump that permits urination and sexual function.
- Total penectomy is required for large infiltrative lesions. If tumors are high-grade or invade the corpora, bilateral ilioinguinal lymphadenectomy is required. If there is suspicion for bilateral node-positive disease or bulky unilateral lymphadenopathy, then neoadjuvant chemotherapy prior to lymphadenectomy is advised.

LINGARSHA / GENITAL WARTS

Liṅgārsha is the condition in which Māṁsa grows excessively on the genital area.

Genital warts / Veneral warts / Condyloma acuminata manifest as discrete flat to broad-based smooth to velvety papules on the perineal, perirectal, labial, and penile areas. Infection with high-risk HPV types (most notably types 16 and 18) is the main cause. These warts are usually asymptomatic. Perirectal warts often itch.

Diagnosis: Clinical evaluation, rarely biopsy
A cardinal sign of warts is the absence of skin lines crossing their surface and the presence of pinpoint black dots (thrombosed capillaries) or bleeding when warts are shaved.

Prognosis:
Many warts regress spontaneously; others persist for years and recur at the same or different sites, even with treatment. Factors influencing recurrence appear to be related to the patient's overall immune status as well as local factors. Patients subject to local trauma (e.g.: athletes, mechanics, butchers) may have recalcitrant and recurrent HPV infection. Genital HPV infection has malignant potential, but malignant transformation is rare in HPV-induced skin warts, except among immunosuppressed patients.

Management:

- Topical irritants (e.g.: salicylic acid, cantharidin, podophyllum resin)
- Destructive methods (e.g.: cryosurgery, electrocautery, curettage, excision, laser)

DISEASES OF SCROTUM AND TESTIS

11. Diseases of Scrotum and Testis: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Epididymo-orchitis, Epididymal cyst, Scrotal filariasis, Shukrashmari - Seminal calculus, Torsion of testis, Ectopic testis, Undescended testis and Tumours.

EPIDIDYMO-ORCHITIS

Inflammation of epididymis and testis is called as epididymo-orchitis. In fact, epididymis is first inflamed (Epididymitis) and later infection spread to testis (Orchitis).

Aetiology:

- A complication from urine infection like E. coli, track down the vas deference to cause epididymo-orchitis.
- Sexually transmitted infection (Chlamydia and gonorrhoeal infection)
- Mumps
- Urethral instrumentation associated with prostatitis
- An operation to prostate or urethra

Symptoms:

- Pain and swelling of epididymis
- Fever and malaise
- UTI with frequency, urgency, and dysuria
- Scrotal wall become red, oedematous, and glossy and may discharge pus (Abscess)

Treatment:

- Depend upon underlying cause
- Antibiotics (Doxycycline) for 2 weeks
- Local massage or ice pack or pain killers
- Drink plenty of water
- Scrotum is supported on sling made up of broad adhesive tape attached between thighs.

EPIDIDYMAL CYST

Cysts which may occur in connection with the epididymis can be divided in to two broad groups.

1. Epididymal cyst
2. Spermatocele

	Epididymal cyst	Spermatocele
Aetiology	Cystic degeneration of appendages of epididymis – congenital	Obstruction to the sperm conducting mechanism – acquired, retention cyst
Site	Behind the body of testis	Behind and above the testis
Loculi	Multi-locular	Uni-locular
Contents	Crystal clear, watery	Like barley water
Appearance	Bunch of grapes	Looks like 3rd testis
Trans-illumination	Brilliant (Chinese lantern pattern)	Poor or negative
Aspiration	Recurrence as the cyst is multi-locular	May cure as the cyst is unilocular
Excision	Avoid in young	May be excised if aspiration is not successful

SCROTAL FILARIASIS

Scrotal filariasis is a manifestation of filariasis and refers to scrotal involvement from parasitic nematodes of the super family.
Causative organism: W. Bancrofti

Pathology:
parasite → obstruction of pelvic lymphatics → lymphangitis → fibrosis of lymph vessels lymphatic obstruction → exudation of lymph in the CT of scrotum → enlarged scrotum and penis may get buried in it → subcutaneous tissue of penis replaced by fibrous tissue → make penile enlargement and curved.

Features:
Fever, chills, rigors
Scrotal swelling and skin become thick, rough with loss of hair due to which testis, epididymis and spermatic cord are not palpable.
Ulceration of vesicular eruption of scrotal skin lead to clear or milky discharge

Ultrasound:
Filarial dance sign: Dilated lymphatic channels (average diameter 6mm) containing curvilinear
echogenic undulating structures representing the microfilariae (usually 5-6).

Treatment:

- Diethylcarbamazine (DEC) 6 mg/kg body. weight in combination with albendazole
- Cleaning and dressing of ulcer
- Surgery

SHUKRASHMARI

The stone developed in seminal vesicle due to suppression of shukra. If shukra vega is suppressed it takes vimargagamana and lodged in between medhra and vrushana. At this stage vata dries up the shukra and shukrashmari is formed. It is compared to spermolith.

Shukrashmari lakshana – Basti shoola, mutrakruchra, vrishana shotha.

SEMINAL CALCULUS

Seminal vesicle stones or calculi refer to solid mineralized pieces of material within the seminal vesicles.
Seminal vesicle calculi are rare and have been mainly reported after the age of 40 years.

Etiology:
The exact etiology of seminal vesicle stones seems to be unclear, possible etiological factors include the following:

- Diverse inflammatory or infectious processes; e.g.: seminal vesiculitis
- Ejaculatory duct obstruction and neoplasms
- Diabetes mellitus
- Hyperparathyroidism
- Congenital anomalies

Signs & Symptoms:

- Hematospermia, Hematuria, Dysuria
- Perineal, testicular or ejaculatory pain
- Spermolithiasis

Investigations: TRUS, CT, MRI

Management: Surgical intervention for stone removal.

Different surgical approaches include transurethral resection of ejaculatory ducts (TURED), transurethral seminal vesiculoscopy (TRU-SVS) endoscope laser lithotripsy, laparoscopic, and open approaches.

TORSION OF TESTIS (TORSION OF SPERMATIC CORD)

Emergency condition of testis, where testis rotates in its axis compromising its blood supply. If not treated within 6-12 hours, testicular gangrene may occur.

Rotation:

- Left testis → anticlock
- Right testis → clockwise

Predisposing causes:

- Inversion of testis (Testis lies horizontal or upside down)
- High investment of tunica vaginalis (Here testis hangs like a clapper in bell)
- When body of testis is separated from the epididymis
- Sudden contraction of spirally attached cremasteric muscles leads to rotation of testis around the vertical axis during straining at stools, lifting heavy weights, coitus.
- Undescended or ectopic testis

Clinical features:

- Childrens and adolescents
- Sudden agonising pain in the scrotum, groin, and lower abdomen
- Nausea and vomiting
- Scrotum is empty and its skin become red, oedematous, and tender
- Elevation of scrotum increases pain (Relieves pain in epididymo-orchitis)
- The affected testis is positioned high (Deming's sign)
- Angell's sign: Because of presence of mesorchium, opposite testis lies horizontally.
- Mild pyrexia

Investigation:
Scrotal doppler and USG

Management:

- In the first hour untwist the testis manually
- If this is not successful do urgent exploration of the scrotum and undo the torsion and viable testis should be fixed to the scrotum to prevent recurrence.
- Orchidectomy (Gangrenous testis)

ECTOPIC TESTIS

When testis fails to descend in to scrotum and present at ectopic site (not the route through which is descends)

Lockwood's theory:
Gubernaculum testis has 5 tails viz scrotal, pubic, perineal, inguinal and femoral. Scrotal tail is strongest one and other tails normally disappears and that is why testis normally descends to scrotum. However, if this is weak, the other scrotal tail may pull it in a different direction, resulting in ectopic testis. Size and function of testis will be normal but more prone to injury.

Sites:

- Superficial inguinal pouch
- Root of penis
- Perineum
- Femoral triangle (thigh)

Treatment: Orchidopexy in new scrotal pouch.

CRYPTORCHIDISM / UNDESCENDED TESTIS

Cryptorchidism is failure of one or both testes to descend into the scrotum; in younger children, it is typically accompanied by inguinal hernia.
Undescended testes are almost always idiopathic. About 10% of cases are bilateral.

Cryptorchidism affects about 3% of term infants and up to 30% of preterm infants; two thirds of undescended testes spontaneously descend within the first 4 months of life.

80% of undescended testes are diagnosed at birth. The remainder are diagnosed during childhood or early adolescence; these are usually caused by an ectopic gubernacular attachment and become apparent after a somatic growth spurt.

Pathophysiology:

- Normally, the testes develop at 7-8 weeks gestation and remain cephalad to the internal inguinal ring until about 28 weeks, when they begin their descent into the scrotum guided by condensed mesenchyme (the gubernaculum).
- Onset of descent is mediated by hormonal (e.g.: androgens, mullerian-inhibiting factor), physical (e.g.: gubernacular regression, intra-abdominal pressure), and environmental (e.g.: maternal exposure to estrogenic or antiandrogenic substances) factors.

- A true undescended testis remains in the inguinal canal along the path of descent or is less commonly present in the abdominal cavity or retroperitoneum.
- An ectopic testis is one that descends normally through the external ring but diverts to an abnormal location and lies outside the normal course of descent
- (e.g.: suprapubically, in the superficial inguinal pouch, within the perineum, or along the inner aspect of the thigh).

Signs & Symptoms:

- In about 80% of cases, the scrotum on the affected side is empty at birth; in the remainder of cases, a testis is palpable in the scrotum at birth but appears to ascend with linear growth because of an ectopic gubernacular attachment that restrains it from following the normal “descent” of the scrotum.
- Inguinal hernia associated with cryptorchidism is rarely symptomatic, but the patent process is often detectable, especially in infants (but less commonly in those with ectopic undescended testes).
- Rarely, an undescended testis manifests acutely because of testicular torsion.

Investigations: Clinical evaluation, sometimes laparoscopy, Rarely USG or MRI

Management: Surgical repair
For a palpable undescended testis, treatment is surgical orchiopexy, in which the testis is brought into the scrotum and sutured into place; an associated inguinal hernia is repaired if present.

For a non-palpable undescended testis, abdominal laparoscopy is done; if the testis is present, it is moved into the scrotum. If it is atrophic (usually the result of prenatal testicular torsion), the tissue is removed.

Surgery should be done at about 6 months of age in term infants and at 1 year of age in preterm infants because early intervention improves fertility potential and may reduce cancer risk. Also, the shorter the child, the shorter the distance necessary to place the testis into the scrotum.

TESTICULAR CANCER

Testicular cancer begins as a scrotal mass, which is usually not painful.
The cause of testicular cancer is unknown.

Most testicular cancers originate in primordial germ cells. Germ cell tumours are categorized as seminomas (40%) or non-seminomas (tumors containing any non-seminomous elements). Non-seminomas include teratomas, embryonal carcinomas, endodermal sinus tumors (yolk sac tumours), and choriocarcinomas.

Tumours originating in the epididymis, testicular appendages, and spermatic cord are usually benign fibromas, fibroadenomas, adenomatoid tumors, and lipomas. Sarcomas, most commonly rhabdomyosarcoma, occur occasionally, primarily in children.

Signs & Symptoms:
Most patients present with a scrotal mass, which is painless or sometimes associated with dull, aching pain. In a few patients, haemorrhage into the tumour may cause acute local pain and tenderness.

Investigations:

- Ultrasonography for scrotal masses
- Exploration if testicular mass is present
- Staging by abdominal, pelvic, and chest CT as well as tissue examination
- Serum tumour markers such as alpha-fetoprotein and beta-HCG (human chorionic gonadotropin)

Management:

- Radical inguinal orchiectomy
- Radiation or chemotherapy for seminomas
- Chemotherapy or retroperitoneal lymph node dissection for nonseminomas

VRIDDHI ROGA

12. Vriddhi Roga: Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Mutravriddhi – Hydrocele.

VRIDDHI

Vṛddhi is the condition in which vitiated Doṣa in the lower abdomen enter the channels of scrotum and produce swelling.

Purvarupa:

- Pain in bladder, testis, and penis
- Obstruction to passage of flatus
- Swelling of scrotum

Bheda	Lakshana	Chikitsa
kaphaja	- Swelling resembles a distended bladder filled with air - Parusa, ruja	- Trivṛtta Sneha, Svedana & Virechana - Snehana, Upanāha & Vātahara Pradeha - Vātahara Basti - After attaining Pāka, incision should be made by avoiding the median raphe. wound is treated like Sadyovraṇa.
Pittaja	- Swelling resembles Pakva Udumbara phala - Jvara, Dāha, Ūṣmā - Āshupāka	- Apakvāvasthā → Pittaja Granthi Chikitsā - Pakvāvasthā → Incision; then clean with Ghṛta & Madhu. Taila & Kalka are applied to promote healing of the wound.
Kaphaja	- Kaṭhina, Alpavedanā - Shīta, Kaṇḍū	- Pralepa with Uṣṇa Dravya and Gomūtra - Dāruharidrā kvātha with Gomūtra for Pānārtha - Kaphaja Granthi Chikitsā (except Vimlāpana) - Incision and wound care
Raktaja	- Kṛṣṇa Vṛtta Sphoṭa - Pittavṛddhi Lakṣaṇa	- Jalaukāvacharaṇa, Virechana - Pittavṛddhi Chikitsā
Medoja	- Mṛdu, Snigdha, Alpavedanā - Tālaphala prakāsha (shines like palm fruit)	- Svedana - Pralepa with Surasādi Gaṇa Dravya and Gomūtra, and bandaging until Pakvāvasthā. - Incision, removal of Meda, and wound care with Kāsisa and Saindhava Lavaṇa
Mutraja	Refer to following topic	
Antraja	Refer to following chapter	

MUTRA VRIDDHI

Mūtravṛddhi is the condition in which Vṛuṣaṇa (scrotum) gets swollen due to accumulation of fluid (hydrocele).

Nidāna: Mūtravega vidhāraṇa
Lakṣaṇa:

- Vṛuṣaṇa swings like a bag filled with water while walking
- Mutrakricchra
- Vṛṣaṇayo Vedanā
- Koshayo Shvayathu

Chikitsā:

- Svedana followed by wrapping the swelling with a cloth
- Puncturing by Vrīhimukha Shastra (trocar) on the most dependent part lateral to the raphe, and fluid is drained.
- Sthāgika bandha should be applied.

HYDROCELE
Abnormal collection of serous fluid in the tunica vaginalis sac is called as Hydrocele.

Types:

1. Primary (idiopathic)
2. Secondary (secondary to disease in testis, epididymis)

1. Primary hydrocele:

Causes:

- Defective absorption of fluid due to damage to the endothelial wall by low grade infection
- Defective lymphatic drainage

Types:

1. Vaginal hydrocele
2. Infantile hydrocele
3. True congenital hydrocele
4. Encysted hydrocele of the cord
5. Hydrocele-en-Bissac
6. Hydrocele of canal of neck

Clinical features:

- Swelling of scrotum associated with slight discomfort
- On examination: Enlarged scrotum on one or both side with notch at middle of affected side of scrotum
- Get above swelling + ve: At the root of scrotum, can feel only cord structures
- Fluctuation test + ve
- Transillumination + ve
- Reducibility - No
- Percussion: Dull
- Palpation of testis – No

2. Secondary hydrocele:

Causes:

- Acute and chronic epididymo-orchitis
- Malignant disease of testis
- Syphilitic infection of testis
- Trauma

Clinical features:

- Swelling of scrotum (small)
- Transillumination – usually -ve
- Palpation of testis – easily
- Consistency – lax and cystic

	Primary hydrocele	Secondary hydrocele
Aetiology	Defective absorption of fluid	Excessive production of fluid
Examples	Vaginal hydrocele, infantile hydrocele	Filarial hydrocele, secondary to malignant tumour of testis
Size	Moderate, big	Small
Palpation on the testis	Difficult	Easily palpable
Transillumination	Positive in majority cases	Usually, negative
Consistency	Tensely cystic	Lax, cystic
Treatment	Partial excision and eversion	Treat the primary case

Complication:

- Haematocele: Occurs due to trauma
- Calcification of hydrocele sac
- Infertility
- Pyocele: Infected haematocele
- Atrophy of testis

Treatment:

1. Aspiration: Temporary method, there is chance of introducing infection. It can be done only in high-risk patients. Not advised
2. Lord's plication: Indicated in small hydroceles. The sac is opened and cut edges of the sac are plicated to tunica albuginea. As a result, the sac gets scrumpled up near the testis. The testicular secretions get absorbed by subcutaneous lymphatics and venous system.
3. Jaboulay's operation (Partial excision and eversion of sac): This is indicated in large hydroceles. The thick, large sac is excised and is sutured behind testis.

ANTRA VRIDDHI

13. Antra Vriddhi – Aetiopathogenesis, Classification, Clinical features, Diagnosis, Complications and Management of Hernia - Inguinal, Femoral, Epigastric, Umbilical, Incisional and rare forms of Hernia.

ANTRA VRIDDHI

Antravṛddhi is the condition in which Vṛuṣaṇa gets swollen due to downward protrusion of abdominal contents (hernia).

Due to carrying heavy weight, fighting with a strong opponent, falling from a tree, and other such straining factors, Vāta gets vitiated and aggravated, afflicting a part of the Antra (bowel). This reaches Vaṅkṣaṇa (inguinal region), and protrudes. It causes swelling of the scrotum like a distended urinary bladder. Upon squeezing, swelling reduces with a gurgling noise; after releasing, swelling reappears.

Chikitsa:

- Aprapta phalakosha: Vatahara chikitsa
- Vankshanottha shotha: Agnikarma by ardhendu vaktra shalaka
- Koshaprapta: Asadhya
- Agnikarma: The skin of the thumb of the contralateral limb should be incised in the middle and dahana should be done.

HERNIA

Hernia means to bud or to protrude (Greek) and to rupture (Latin)

Abnormal protrusion of viscus (Organs of abdominal cavity) or part of it through opening of weak point in body is known as hernia.

Aetiology:

1. Weakness of abdominal muscle
2. Increased abdominal pressure which forces contents out through normal abdominal musculature.

Contents of hernia:

1. Sac: Pouch of peritoneum which comes out through abdominal musculature
- Mouth: Opening through which contents enter the sac
- Neck: Constricted part which passes through abdominal muscles
- Body: Main portion of sac
- Fundus: Most redundant part of sac

2. contents: Viscus lies within the sac of hernia
3. coverings: Skin and muscles of abdomen

Classification:

1. Reducible hernia
2. Irreducible hernia
3. Obstructed hernia
4. Inflamed hernia
5. Strangulated hernia

1. Reducible hernia:

Hernia gets reduce by its own or by patient or by surgeon.
Intestine reduces with gurgling and it is difficult to reduce the first portion.
Omentum is doughy and it is difficult to reduce the last portion
Reducibility and impulse on coughing are positive

2. Irreducible hernia:

Here contents cannot be return to abdomen due to narrow neck, adhesions, overcrowding
It predisposes to strangulation
Femoral and umbilical Hernia are generally irreducible.

3. Obstructed hernia:

Irreducibility + intestinal obstruction
Though there is intestinal obstruction (due to blocked faeces) there is no alteration in blood supply to hernial contents.

4. Inflamed hernia:

Due to inflammation of contents of sac Eg: appendicitis
Here hernia is tender but no tense
Overlying skin is red and oedematous.

5. Strangulated hernia:

Irreducible hernia with obstruction to flow of blood
Swelling is tense, tender, with no impulse on coughing and with features of intestinal obstruction.
It develops when neck of sac is very constricted.

Maydl's hernia: Retrograde strangulation
The hernial sac contains two loops of bowel with another loop of bowel being intra-abdominal.
A loop of bowel in the form of W lies in the hernial sac and the centre portion of the W loop may become strangulated, either alone or in the combination with the bowel in the hernial sac.
It is more often seen in men and predominantly on the right side.

Hernia sites:

1. Epigastric
2. Paraumbilical
3. Umbilical
4. Lumbar
5. Spigelian
6. Femoral
7. Inguinal

INGUINAL HERNIA

There is protrusion of abdominal contents through inguinal region of abdominal wall.

Anatomical classification:

1. Indirect / oblique
2. Direct

1. Indirect inguinal hernia:

Contents of abdomen enter the deep inguinal ring passes through inguinal canal and come out through superior inguinal ring.

2. Direct inguinal hernia:

Contents of abdomen protrude through a defect in the transversalis fascia in the posterior wall of inguinal canal (i.e., through Hesselbach's triangle)

Clinical features:

1. Pain and swelling in inguinal region or pain radiating to testis on doing strenuous work.
2. Shape: oval (incomplete) or pyriform (complete)
3. Generally, does not reduce spontaneously and has to be reduced by patient himself or by physician.
4. Cough impulse: Ask the patient to cough, can see expansile impulse on coughing.
5. Get above swelling: This test is to differentiate scrotal swelling from inguino-scrotal swelling. In standing position, palpate root of scrotum for spermatic cord. It is palpated in incomplete hernia while not in complete one.
6. Zieman's technique: This is a variation in impulse on coughing to see if the hernia is direct or indirect, or femoral. Place index finger over deep inguinal ring (1/2 inch above mid-inguinal point), middle finger over superficial inguinal ring & ring finger over saphenous opening, and ask the patient to cough.
   Impulse on index finger: Indirect hernia.
   Impulse on middle finger: Direct hernia.
   Impulse on ring finger: Femoral hernia
7. Ring invagination test: In recumbent position, little finger should be invaginated through the skin from the bottom of the scrotum till the pulp feels the ring. The patient is asked to cough. If the impulse is felt on pulp of the finger- it is direct hernia, if it is felt on the tip of the finger, the hernia could be indirect.

8. Internal ring occlusion test: Done in standing position. Hernia is reduced and then the thumb is placed on deep inguinal ring and the patient is asked to cough. A direct hernia will show a bulge medial to occluding finger, but indirect hernia will not find access.

Investigation: USG

Treatment: operation is treatment of choice
Conservative management:

- When patient refuses surgery
- Has poor health and short life expectancy
- Treat the cause
- TRUSS can be used
- Taxis: Patient is placed in supine position with hip and knee flexed and hip internally rotated. Contents are pushed with one hand directing with other hand.

TRUSS:

- Used to prevent hernia from protruding out of superficial inguinal ring.
- This is never curative method
- Used only when hernia is reducible
- It should be used all throughout day except night.
- Can be used only in small/ medium size hernia
- If truss is not worn correctly (when worn, it should prevent appearance of swelling), there is great risk of obstruction or strangulation.
- It is absolutely contra indicated in femoral and sliding Hernia

Operative methods:
Herniotomy: Excision of hernia Sac (Children)
Herniorrhaphy (Young patients) and hernioplasty (Old patients): Herniotomy with surgical repair of post wall of inguinal canal.

Herniotomy:
Indication: Indirect/direct hernia with good muscle tone
Position of patient: Supine
Anaesthesia: Regional or G.A
Part Preparation: Parts are cleaned with iodine and spirit, from level of umbilicus above to upper part of thigh below.
Incision: 6-8 cm incision is made parallel to inguinal ligament at level of deep ring in medical $2/3^{rd}$ of inguinal ligament.

Layers opened and procedure:

- Skin
- Two layers of superficial fascia
- External oblique is incised in line of direction of fibres, till external ring is slit open
- Thin cremasteric box is opened
- Identification of sac glistening white colour
- Isolation of cord from sac by blunt and sharp dissection and cord is held separately by using cord holding fercep.
- Mobilize the sac upto deep ring. Mobilization is complete when inferior epigastric artery pulsation and extraperitoneal pad of fat are seen.
- Open the sac and see for contents.
- Reduce the contents
- Twist the sac to avoid injury to contents of sac
- Transfixation ligature is applied as high as possible at the neck of sac and it is tightened.
- Excision of sac. After excision sees the excised sac whether omentum or intestine has been injured or not.

Closure:

- External oblique is sutured with chromic catgut / silk.
- Subcutaneous fat with absorbable catgut suture
- Skin with silk

Post-op management:

- NBM for 6-8 hours
- Oral fluids and soft diet later
- Analgesics
- Antibiotics
- Scrotal support if dissection is more (complete)
- Suture removal after 7-10 days

Advice at discharge:
Not to strain or lift heavy weights (bucketful of water) or to carry load on shoulders for 3 months.
If there is any precipitating cause such as chronic cough or difficulty in passing urine, they have to be treated first; otherwise, hernia will recur once again.

Herniorrhaphy:
This means Herniotomy and approximation of conjoined tendon to inguinal ligament to strengthen the posterior wall of inguinal canal.

Bassini's Repair:
Conjoined tendon above is approximated to inguinal ligament by using non-absorbable suture such as nylon, silk or polypropylene. Nonabsorbable suture is used to that strength remains for long time. This repair is called as Bassini's Herniorrhaphy.

Precautions:
Ilio-inguinal nerve should not be caught in ligature
Conjoined muscles should not be strangulated.
There should not be any tension on suture lines.

Hernioplasty:

- The procedure is herniotomy along with re inferred repair of posterior wall of inguinal canal
- Generally indicated in all indirect hernias where patients have poor muscle tone.
- In all direct hernia
- In recurrent hernia
- The patients who do strenuous job etc.
- First herniotomy, then
- As the gap between conjoint tendon and inguinal ligament is large, the gap is darned with a graft of fascia lata or strip of skin or polypropylene mesh.

Complication of hernia:

- Irreducibility
- Strangulated Hernia
- Inflamed Hernia
- Obstructed Hernia
- Incarcerated Hernia

FEMORAL HERNIA
Definition: Herniation of intra-abdominal contents through femoral canal is called Femoral hernia

- Never congenital
- Common in women (M: F = 1: 2)
- It is important because it cannot be managed by conservatively using TRUSS and is associated high risk of strangulation (because of rigidity femoral ring) Common between age group 20-40 years
- More common on right side at the age of 60-80 years

Aetiology:
Repeated pregnancies
Wide femoral canal

Pathology:
Abdominal contents pass through femoral ring, femoral canal and comes out through saphenous openings.

Clinical features:

1. Swelling (below and lateral to pubic tubercle and below inguinal ligament) and dragging pain are common complaints.
2. Impulse on coughing and reducibility is present (Gurgling sound during reduction)
3. Gaur sign: Dilatation of superficial epigastric ileac veins due to compression by hernia sac circumflex
4. Generally small, when large they are shaped like retort, with its bulbous part looking upward.
5. Are generally firm and dull on percussion.

Treatment: inguinal operation (Lotheissen operation)

- Fascia transvalis is divided
- Hernia sac is visualized
- Sac is retracted, neck is ligated and distal part is excised
- Femoral ring is now obliterated by stitching the conjoint tendon or inguinal ligament down to pectineal ligament, bladder, and pubic branch of obturator artery
- Avoid injury to femoral Vein

EPIGASTRIC HERNIA / FATTY HERNIA

Herniation of abdominal contents through gap/defect in linea alba between xiphisternum and umbilicus.
Here hernia begins as a protrusion of extraperitoneal fat, so called as fatty hernia of linea alba.

Precipitating factors:
Sudden straining or heavy exercise results in tearing of few fibres of linea alba.
Opening is very narrow so viscera do not herniate.

Clinical features:

- Common in muscular man, manual labours
- Swelling (in between xiphisternum and umbilicus)
- Expansile impulse on cough
- Dull aching pain due to fatty contents which are partially strangulated.
- Tenderness is important feature
- Many cases are associated with peptic ulcer

Treatment:

- Long midline incision over swelling
- Deepened incision, identify fatty herniation
- Pedicle of fatty profusion is ligated and excised
- Repair defect in linia alba and closed skin
- If peritoneal sac is protruded, it is opened and omentum is examined.
- If normal, push back into abdominal cavity, if partially strangulated, affected portion is excised.

UMBILICAL HERNIA

It is herniation through weak umbilicus scar. This is more in males. It can be discussed under two headings:

Congenital →
Umbilical hernia of new-born
Umbilical hernia of infants and children
Acquired →
Umbilical hernia of adults
Umbilical hernia of new-born (exomphalos):
Failure of midgut as a whole or part to return into abdominal cavity during embryonic life results in exomphalos.

1. Exomphalos minor:

- Here umbilical cord attached to the summit of the sac
- Sac is small and defect less than 5 cm
- It is treated by twisting the cord and ligating the sac. Care should be taken to avoid damage to the intestine.

2. Exomphalos major:

- Here umbilical cord is attached to the inferior aspect of the sac, containing intestine, abdominal structures, e.g., liver, bowel.
- This type of hernia is usually associated with absent abdominal musculature.
- The operation should be done before the rupture of the sac as the morbidity increases greatly in the event of a rupture of the sac.

Comparison between umbilical hernia in infants and adults:

Features	Infants	Adults
Age	0 – 3 years	50 – 60 years
Sex	Common in male child	Common in females
Cause	Neonatal sepsis	Obesity, weak muscles, pregnancy
Defect	A small defect in umbilical scar	Above or below the umbilicus
Symptoms	Symptom less, swelling on umbilical region whenever the child cries	Swelling on umbilical region which increases on straining, expansile impulse on cough, dragging pain and reducible
Strangulation	Rare	Very common
Treatment	Conservative (strapping) Surgery (rare)	Mayo's repair (excision of umbilicus, reduction of content and excision of sac)

INCISIONAL HERNIA

Herniation occurs through acquired Scar in the abdominal wall caused by previous surgery or accidental trauma. This is common in females.

Causes:

1. Post-op: • Constipation, cough, abdominal distension • Early removal of suture • Infection • Conditions which increase intra-abdominal pressure like BPH • Steroids in early post-op	2. Incision: • Wrongly placed incision • When nerve cuts • Midline, infra-umbilical incision • Improper closes of deep layers of abdomen, imperfect haemostasis

Clinical features:

- Scar of previous surgery can be observed
- Serosanguinous discharge on 4th day of post-op through main suture line is signal of development of wound dehiscence
- H/O any of precipitates factor
- Swelling in relation to scar
- Impulse on cough and reducibility
- Scar is thin and secondary, healing in the form of irregular scar may present
- After reduction of contents, defect can be palpated through scar which depends upon number of stitches that have given way.

Treatment:
Preventive measures:

1. Patients with chronic cough, bronchitis are treated before surgery
2. Weight reduction in obese before surgery
3. During surgery, due care should take to adequately repair deep layer
4. Avoid post-op wound infection

Surgery:

- Elliptical incision around swelling
- Edges are undermined, deepened till aponeurosis
- Unhealthy skin is gradually directed off the sac
- If contents are adhered to sac, sac is opened and contents are separated.
- Layers are separated carefully and sutured separately.

HIATUS HERNIA

Hiatus hernia is a protrusion of the stomach through the diaphragmatic hiatus.
Etiology of hiatus hernia is usually unknown, but a hiatus hernia is thought to be acquired through stretching of the fascial attachments between the esophagus and diaphragm at the hiatus (the opening through which the esophagus traverses the diaphragm).

Types:

1. Sliding Hiatus Hernia (most common): Gastroesophageal junction and a portion of the stomach are above the diaphragm.

2. Paraesophageal Hiatus Hernia: Gastroesophageal junction is in the normal location, but a portion of the stomach is adjacent to the esophagus in the diaphragmatic hiatus.

Signs & Symptoms:
Most patients with a sliding hiatus hernia are asymptomatic, but chest pain and other reflux symptoms can occur.
A paraesophageal hiatus hernia is generally asymptomatic but, unlike a sliding hiatus hernia, may incarcerate and strangulate. Occult or massive gastrointestinal haemorrhage may occur rarely with either type.

Diagnosis: Chest x-ray, Barium swallow, Upper endoscopy
Management: Sometimes surgical repair, sometimes a proton pump inhibitor
An asymptomatic sliding hiatus hernia requires no specific therapy. Patients with accompanying GERD should be treated with a proton pump inhibitor.
For a paraesophageal hernia, repair should be considered because of the risk of strangulation.

Hurray!
You are one more book closer to your B.A.M.S. degree.