Summary File

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Feedback

Editing File

Summary Progress Tracking

Click on the desired lecture for quick access

Lecture name	Progress
Jaundice	Completed
Gastric outlet, small and large bowel obstruction	Completed
Abdominal pain (central , lower , acute)	Refer to the lecture
Abdominal pain (Generalized and Upper)	Refer to the lecture
Approach to wound healing & Burn case scenario	Completed
Neck Swellings	Completed
GI hemorrhage	Completed
Abdominal wall and umbilical hernia (types, surgical anatomy, predisposing factors, clinical features and complications)	Completed
Perforation and infarction of viscus (etiology, clinical features and complications)	Completed
Breast Disease	Completed

IMP Drs' Notes

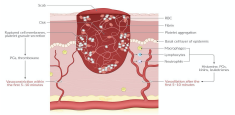
Approach to wound healing & Burn

Wound Healing

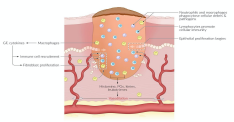
Obj 1: List and explain the phases of Normal Wound Healing

- Heamostasis (5-10min): **Main cells are: platelets**:
form the platelet plug, degranulation of platelets and recruitment of neutrophils stimulated to release:

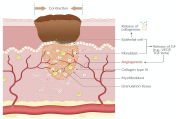
- **Platelet derived growth factor (PDGF)**
- **Transforming growth factor β (TGFβ)**: has a major role in wound healing (Excess causes abnormal scars) produced by **α granules**
- **Fibroblast growth factor**



- Inflammatory /Migratory "lag" phase (1-4 Days): Presented by rubor (redness) , tumour (swelling), calour (heat) and dolour (pain).
Main cells are:
First 24 hours → **PMNs (neutrophils)**
After 24 hours (Key cell) → **Macrophages**: Phagocytosis, wound debridement, activation of fibroblast, angiogenesis and matrix synthesis regulation

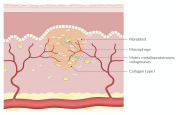


- Proliferative / Fibroplasia "incremental" (3 days - 3 weeks): Main cells are: Fibroblasts
Dominant cell type peaking at 7-14 days
Re-Epithelization, angiogenesis, collagen synthesis and ECM formation, In this phase, the scar changed from red to pale
Arterial oxygen tension **(PaO2)** is a key determinant of the rate of collagen synthesis.



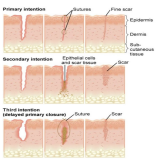
- Remodeling / Maturation (3 weeks - one year): **cross linking of the collagen**

- Type III collagen replaced by type I collagen and become organized and the scar became mature, (no net increase in collagen content).
- Re-establishing normal 4:1 ration (I:III): Duration depends on age, genetics, type of wound, location. Tensile strength increases to 80% of pre-injured skin.



Obj 2: List and describe the types of Wound Healing

- 1° intention: Primary closure by suturing the edges together. Never try to close wound by 1° intention **if** : more than 24h passed since the injury , **if** it's from humans or animals bite , **if** any chronic wound
- **2° intention:** Wound left open to heal by processes of granulation, contraction and epithelialization
- **3° intention:** delayed primary healing (a combination of primary healing and secondary healing). Desired for contaminated wounds



Obj 3: Identify and differentiate between the factors Affecting Wound Healing

- Local Factors:

- **Blood supply:** Decreased tissue perfusion results in decreased wound oxygenation, Fibroblasts are oxygen-sensitive and their function is reduced in hypoxic states.
- **Radiation**
- **Wound site:** The presence of persistent pressure or recurrent trauma at wound site may compromise the healing of it.
- **Wound contamination:** Such wound require debridement, removal of all dead tissues and foreign bodies, and cleaning.
- **Hematoma formation in the wound:** Bleeding must be controlled because hematoma formation in the wound predisposes to wound infection and it separates the wound edges and prevents wound healing.
- **Mechanism of wounding:** Incisional wounds will follow the normal phases of Wound healing whereas crushing or avulsion wounds may not heal in normal process and may have a prolonged healing process
- **Tissue loss:** This will delay the slow wound healing and predispose to infection
- **Pressure in the wound area:** Pressure in the wound will reduce the blood supply, which causes

General (patient): ★DID NOT HEAL Local (wound):

1. Nutrition
2. Drugs/Toxins
3. Age
4. DM
5. Smoking
6. Vascular disease
7. Obesity
8. Systemic diseases
9. Idiopathic
10. Inherited diseases
11. Surgical technique

1. Oxygen (Hypoxia)
2. Infection
3. Acidity
4. Radiation
5. Loss of growth factors
6. Denervation (in diabetics)
7. Iatrogenic
8. Edema
9. Cancer
10. Foreign body

Chemotherapy treatment can be started after 14 days.

Approach to wound healing & Burn

Obj 3: Identify and differentiate between the factors Affecting Wound Healing	**Systemic Factors:** • **Malnutrition:** Nutrition is extremely important for protein and collagen synthesis, and metabolic energy for wound healing, **Vitamin A** → formation of the extracellular matrix deficiency in vit A delays wound healing, **Vitamin E** → formation of the extracellular matrix, **Vitamin C** → Collagen synthesis , has a role in the cross-linkage, **Zinc** is an essential cofactor for many enzymes and the synthesis of RNA and DNA. Deficiency causes impared epithelial and fibroblast proliferation. • **Chronic diseases:** such as liver disease, jaundice, malignancy, uremia, will dispose to malnutrition, wound infection, and delay wound healing. • **Pharmacological:** Steroids decrease inflammation and subsequent wound healing, NSAIDS decrease collagen synthesis and inhibit platelet aggregation, Antineoplastic agents decrease fibroblasts proliferation and wound contraction. • **(Endocrine abnormalities) Uncontrolled diabetes:** Diabetics often have delayed wound healing. Neuropathy rather than small vessel occlusive disease may be responsible for delayed healing. • **Smoking:** Nicotine causes vasoconstriction decreasing perfusion, CO shifts the oxygen dissociation curve to the left and reduces tissue oxygenation. • **Age:** Rate of cell multiplication decreases with age, All stages of healing are protracted in elderly, Healed wounds have less tensile strength in elderly. • **Immunosuppression** • **Decreased oxygen delivery to tissue reduce:** Collagen formation, Extracellular matrix deposition, Angiogenesis and Epithelialization. • Nutrition, Drugs/Toxins Age, DM, Smoking, Vascular disease, Obesity, Systemic diseases, Idiopathic, Inherited diseases, Surgical technique
Obj 4: List and discuss the types of wounds	**- Acute** (1st week)**:** wounds such as clean incised, avulsion, abrasion puncture and crushing. • **Tidy:** Incised, clean wound in healthy tissue without tissue loss such as a wound from a clean sharp object, Can be closed immediately and Heals by primary intention. • **Untidy:** Crushed or avulsed tissues with contamination, devitalized tissues and tissue loss, such as wounds caused by explosion, missile or bullet injury, Requires debridement, excision of devitalized tissues, and cleaning several times before definitive repair is performed and Usually heals by secondary or tertiary intention. **- Subacute:** 1-6 weeks **- Chronic (> 6 weeks):** wounds such as venous, ischemic, pressure and diabetic ulcers.
Obj 5: Classify Wounds According to the Mechanism of Wounding	**- Clean:** New wound (<12 hours), minimal contamination, may need debridement, usually closed primarily, the edges approximated with an appropriate method. **- Avulsion(Degloving):** Shearing force causing such wound, with skin flap or total skin loss, and exposure of underneath structures. 2 Management includes several debridement sessions, cleaning, with a pressure dressing to prevent accumulation of blood and serum under the skin flap of sometimes a skin graft or flap is required when there is significant blood loss. **- Abrasion:** These result from friction damage and are characterized by superficial bruising and superficial loss of epithelial cells and portions of the dermis with intact deep structures. Management includes cleaning to prevent traumatic tattoo and and to allow the wound to heal by regeneration of epithelial cells. **- Puncture:** Such wound does not need closure, but assessment for deep structures injury or deep foreign bodies by imaging and clinical follow up to detect the development of infection is required. **- Crushing:** This injury is associated with significant loss of tissues including different structures that may initially appear viable. Non-viable tissues need debridement, cleaning of the wound, closure with a skin graft or myocutaneous flap depending on the exposed underlying structures.
Obj 6&7: Explain the Management plan of Acute & Chronic Wound	**- Acute wound management:** • Management of the patient should follow the acute trauma life support (ATLS) principles. • Complete examination. • Wound examination include possible damaged structures, assess the severity and tetanus status. • Skin cover by graft or flap may be required if there is skin loss. **- Chronic wound management:** • Chronic wounds develop when the normal wound healing process fails to repair the tissue injury. • Debridement, cleaning, daily dressing changes, and negative pressure devices have been the main method to enhance the healing process in chronic wounds. • Judicious intervention is essential in countering these factors to enhance the normal wound healing. E.g, debridement can change the wound to an acute state or condition that will accelerate normal healing.
Obj 8: Explain the mechanism of Compartment Syndrome	**- Compartment Syndrome:** This occurs due to increased pressure in the muscles compartment after trauma. It occurs most commonly after closed trauma (crushing injury). The typical clinical feature is severe pain in the affected compartment 2 particularly with passive muscles movement, distal sensory deficit, and the late sign is absent distal pulses and the presence of clinical features with persistent pressure of 30 mmHg or higher is an indication for fasciotomy.
Obj 9: Describe Degloving Injury	**- Degloving Injury:** This occurs when the skin and subcutaneous tissue are avulsed from the underlying fascia with exposed neurovascular structures, bones, and tendons. It can be an open or closed injury. The trial of replantation or revascularization is extremely challenging.

Approach to wound healing & Burn

Obj 10: Demonstrate the points regarding leg ulcer	- **Venous Ulcer:** The usual site is above the medial malleolus it results from venous hypertension due to varicose veins or DVT. - **Ischemic Ulcer:** This is due to arterial insufficiency or vasculitis. It occurs commonly at the tip of the toes in the shin area of the leg. Blood supply insufficiency is obvious, and the ulcer is dry with minimal granulation tissue. - **Traumatic Ulcer:** This is due to repeated trauma and can be self-inflicted. - **Neoplastic Ulcer:** It could be squamous or basal cell carcinoma or sarcoma. Biopsy should be obtained from any chronic ulcer which is persistent and unresponsive to appropriate wound care to rule out malignant changes (squamous cell carcinoma called "Marjolin's ulcer"). It usually affects the area with chronic or prolonged scar (chronic burn), and it has poor prognosis. Treatment of chronic leg ulcer includes treatment of underlying cause, appropriate wound care, but skin graft is required in some cases. - **Pressure Ulcer:** Localized tissue damage usually over bony prominences as a result of prolonged pressure and consequently reduced blood supply to local tissue. The most commonly affected sites in the body are sacrum, greater trochanter, heel, ischium, occiput and lateral malleolus. - **Diabetic Ulcer:** Diabetic patients are at risk of skin ulcer for the following reasons: Neuropathy, Hyperglycemia and Arterial disease The commonest sites of dibetic ulcers are found at the plantar surface of the metatarsal, heels and dorsum of foot but it can occur anywhere in the foot or leg. - **Chronic Infection:** This is due to Tuberculosis or Syphilis.
Obj 11: Explain how different abnormal scars are formed **Very important**	- **Atrophic scar:** This is a flat, pale and stretched in appearance. It can be traumatized easily as the epidermis and dermis are thinned. Excision and resuturing may improve such scar. - **Contracture scar:** This is when the scar form across a joint or flexion or extension skin creases. It may cause restriction of the movement at the joint , flexion or extension deformity. Contracture can be prevented be postoperative splints and intensive physiotherapy. Treatment include multiple Z-plasty or skin graft or flap. - **Wide scar:** Caused by traumatic wounds that are not closed properly - **Hypertrophic scar:** This is an excessive scar tissue that **does not extend beyond the original wound boundaries.** It results from the prolongation of the inflammatory phase of wound healing and from inappropriate scar sitting such as wound across the lines of skin tension. - **Keloid scar:** Overproduction of collagen and fibroblasts, This is an excessive scar tissue that extends beyond the original wound boundaries. The etiology of keloid scar is unknown, but it is associated with deeply pigmented skin and inherited tendency. It is more common in the body triangle between the point of each shoulder tip, xiphisternum, ear, and chin of the tibia. Hypertrophic scars improve with time spontaneously, while keloid scars do not. All excisions have high rates of recurrence. - **Treatment :** • **Prevention** • **Medical:** steroids (first line), chemotherapy (5-FU) or radiation • **Non surgical:** pressure, silicone gels or sheets • **Surgical:** laser or surgery If failed in keloids the best treatment approach is a combination of intralesional excision followed immediately by low-dose radiotherapy

Features	Hypertrophic scar	Keloid scar
Genetic	Not familial	May be familial
Race	Not race related	Black > White
Sex	Female = Male	Female > Male
Age	Children	10-30 years
Borders	**Remains within wound (in the same area of scar)**	**Outgrows wound area**
Natural history	Subsides with time	Rarely subsides
Site	Flexor surfaces	Sternum, shoulder, face
Aetiology	Related to tension	Unknown

Not part of the objectives but very important	- **Clean (class I):** Nontraumatic, non infected wounds & no breach of Resp, GI, or GU tract. (No need for antibiotics) ex. Thyroid and breast surgeries. - **Clean-contaminated (class II):** Small breach in protocol; Resp/GI/GU tract are entered with minimal contamination. ex. Cholecystectomy, **Uncomplicated** appendicitis, Intestinal resection ONLY if there was **no spillage.** - **Contaminated/dirty (class III):** Fresh traumatic wounds; major break in sterile technique, **nonpurulent** inflammation; in or near contaminated skin. ex. Hemicolectomy, Resection of the intestine **with spillage.** - **Infected (class VI):** **Purulent** infection.ex. Traumatic open bone fracture, **Purulent** pyogenic perforated appendicitis.

Wound hematoma increases the risk of infection in surgical wounds.
To avoid contamination of the surgical wounds the sterile wound dressing pad should be applied after cleaning and drying the wound before removing the drape

Collagen:

- ★ Lysine and proline (purines) hydroxylation required for cross linkage (the main step in collagen synthesis) the role of Vit.C
- ★ **Normal skin ratio - Type I:Type III = 4:1,** Hypertrophic / immature scar 2:1 ratio
- ★ Formation of collagens is inhibited by: Colchicine, penicillamine, steroid, Vit.C deficiency and Fe deficiency. (They activate collagenase which degrades collagen synthesis and inhibits cross linkage hydroxylation of lysine and proline)

Approach to wound healing & Burn

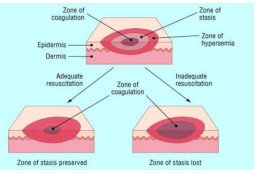
Burn

- Pathophysiology of burn: The local effects are the result of tissue destruction and inflammatory response. Insensible fluid loss can cause severe hypovolemia which might progress to hypovolemic shock (when > 15% of the body surface area is burned) and **decreased preload.**

Not part of the objectives but very important

- Burns have 3 zones :

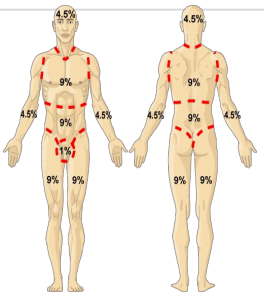
- **Zone of coagulations:** a central area of irreversible coagulative necrosis, its extent is determined by the duration of contact with the heat source and the degree of heat transfer to the skin.
- **Zone of stasis:** an area of potential reversible cell damage surrounding the central zone (tissue is damaged with decreased perfusion but still viable).
- **Zone of hyperemia:** an area characterized by inflammation and increased blood flow surrounding the stasis zone.



Obj 1: Calculation

- Rule of nines:

- Used to approximate burn size, Divides the body into areas that each represent approximately 9% of the total body surface area (TBSA) of an adult, less useful in children because of the relatively large head size (and the relatively small limbs.
- Palm without fingers = 1% (Palmer method “rule of palm”)
- Head in kids = 18% (9% in adults)
- Single leg in kids = 9% (18% in adults)
- **Add 25%** to the calculated TBSA if it’s an **inhalation injury**
- In summary: give 2L IV line → adjust according to your measurements → adjust according to urine output.



- Parkland formula:

- Fluid volume of crystalloid administration during the first 24 hours of admission
- **Volume of lactated Ringer's = 4 mL × weight (kg) × TBSA%**
- 50% given in the first 8 hours
- 50% given in the next 16 hours
- Start counting the hours from the start of injury not from the time patient reach the hospital

“4-2-1 rule” for maintaining fluid :It is the calculation of hourly fluid need according to weight . (4 ml/kg/ hour for the first 10 kg , 2 ml/kg/ hour for the next 10 kg and 1 ml/kg/ hour for every kilogram after that.)

-Mortality:(TBSA+age)/100

Not part of the objectives but very important

- Clinical classification of burns based on the depth:

- **First degree:** Epidermal injury only, Heal within 7 days, Clinically characterized by edema/erythema/pain
- **Second degree:** Injury to epidermal +/- dermal layers, Heal within 14 days, Clinically characterized by: painful, **blisters**, Skin is repopulated by viable germinal cells in follicles.
- **Third degree:** Entire dermal layer and subdermal fat injury, They form blisters and heal by hypertrophic scars, Clinically characterized by: **dry, inelastic and waxy appearing scar. The major systemic effect in second and third degree burns is decrease venous return.**
- **Fourth degree:** Dermis and deep tissue injury, Clinically characterized by: **injury to all skin layers, and injury to tendon, structures like nerve, bone and joint**

- Clinical classification of burns based on the cause:

- **Thermal:** Friction burn - **(Scald burn) the most common type of burns among children.**
- **Chemical:** Alkaline (more dangerous due to infiltration and liquefactive necrosis). - Acidic **(hydrofluoric acid can cause hypocalcemia) - If you don’t know the type of the chemical agent just dilute it with water to minimize its effect “The solution of pollution is dilution”**
- **Electrical:** **Systemic injury: cardiac arrhythmia** (if a current entered from one hand and exits from the other it means that it went through the heart), **sepsis, renal failure** (due to necrosis), **PNS.**
- **Sunburns**

- When to transfer to a burn center (transfer criteria) :

- >25% body surface area (BSA).
- >20% BSA in children.
- High voltage burns.
- Inhalation injuries.

Approach to wound healing & Burn

Obj 2: Surgical and Non surgical Management

- Surgical:

- **Escharotomy:** Indicated when there are: Poor tissue perfusion, Threat to perfusion after volume resuscitation, and Deep **Circumferential burns**.
- **Digital escharotomy:** Use mid-axial incisions: Index, long → ulnar incision. Ring → radial or ulnar incision. Little → radial incision.
- **Skin Grafting & Flap:** Skin grafting is a surgical procedure that involves removing skin from one area of the body and moving it, or transplanting it, to a different area of the body
  - **Grafts** are similar to first degree burns (First layer is removed and appendages are preserved in both of them so healing and epithelialization is possible) the difference is that grafts occur in a controlled environment unlike burns, used more with 3rd degree, do not contain blood vessels (the injury site needs to be well vascular e.g.above muscle or dermis).
  - types of grafts: ◦ **Split thickness graft:** epidermis and part of dermis. High risk of hypertrophic scar(contraction) ◦ **Full thickness graft:** epidermis and entire dermis. Less hypertrophic scar (less contraction) need to close it
  - **Flaps** is a bulky tissue (e.g. muscle flap, subcutaneous tissue flap), used when there's a deep burn (4th degree burns) or when a deep reconstruction following cancer ablation is needed. It Contain blood vessels (so they are used to cover deep structures like bones, tendon, or joints)

- Non-Surgical:

- The first step is removing the object especially in chemical burns.
- The first Priority would be maintaining an adequate airway and first Aid (ABCDE) especially in case of risk of inhalation injury, with continuous observation for signs of respiratory failure.
- **IV fluid resuscitation** if > 15% of TBSA is affected (Parkland formula).
- Normal Urine output: 1 cc/kg/hour. Pediatric, Electrical and inhalation aim for: 2cc/kg/hour
- **Prophylaxis against Tetanus** (eg: Clostridium Tetani) by Tetanus Immunoglobulins (TIG)
- **Analgesic** (eg: opioids).
- Placing the patient in a warm room (to reduce energy expenditure) and **enteral feeding using nasogastric tube** with **vitamin** supplements and **iron** (Better to eat normally after 48 hours).
- **Dressings**, essential to protect from contamination and for promotion of healing
- **Topical antibiotics**, like Silver Sulfadiazine (Flamazine) and Povidone Iodine (Betadine) (Not advised in the first 48 hours as they can make the determination of the depth more difficult). Topical application of (mafenide acetate) penetrates through eschar and may be effective against a wider variety of organisms.

- **Objective of treatment:** 1-prevent edema 2-prevent contractures 3- prevent infection 4-preserve viable tissue
- **First degree:** Mild analgesics/NSAIDs (nerve endings intact), daily cleansing and silver sulfadiazine (flamazine).
- **Second degree:** same as 1st degree and leave blisters intact, if debrided? Occlusive dressing.
- **Third degree:** surgically debride and remove the eschar then skin graft.
- **Fourth degree:** skin graft not adequate. Amputation or flap coverage.
- **Wound closure:** in deep second degree when burn didn't heal after 2 weeks
- **Chemical:** as GP dilute it with water to minimize its effect "The solution of pollution is dilution"
- **Electrical:** **Fasciotomy (compartment syndrome)**, Debridement of dead tissue, definitive: Amputation or flap coverage

Neck swelling

Lymphadenopathy

- **The most common sources of neck swelling is lymph node enlargement.**
- **Causes of cervical lymph gland enlargement** "MIAMI": Malignancy (e.g., lymphomas), Infection (e.g., TB), Autoimmune disease (e.g., SLE), Miscellaneous (e.g., sarcoidosis), and Iatrogenic (medications).
  - **Infection**
    - i. **Tonsillitis**
    - ii. **Tuberculous cervical <u>lymphadenitis</u>** ●pt. without a history of Vaccination (BCG), ●ESR (raised) ●NOT hot ●1 and 2 cm in diameter ●Indistinct, and (matted) **and <u>abscess</u>** ●The overlying skin turns reddish-purple ●Skin temperature is normal (cold abscess) ●Tender.

  - **Neoplasms**
    - i. **Primary** neoplasms of lymph nodes (lymphomas) divided into Hodgkin's and non-Hodgkin's lymphoma

    ● Common in children and young adults ● Painless lump that grows slowly ●Solid and rubbery ●Fever with rigors and weight loss (Constitutional symptoms)

    ● Hodgkin's lymphoma: Matted lymph in the posterior triangle.

    - ii. **Secondary** from head and neck, chest (breast) and abdomen

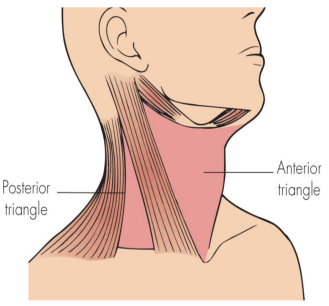
    ● Over the age of 50 years ● Common in males ● Painless lump in the neck.

    ● An enlarged supraclavicular lymph gland commonly indicates intra-abdominal or thoracic disease. This gland is called Virchow's gland; its presence is Troisier's sign.

The salivary glands

- Saliva is produced by the paired parotid, submandibular and sublingual glands and many other small, unnamed glands.
- The most common surgical conditions:
  - Infection (Acute bacterial parotitis)
  - **calculus formation in the submandibular gland**.
    - young to middle-aged adults.
    - Main symptoms are pain and swelling beneath the jaw.
    - Symptoms appear, or worsen, before and during eating
  - Tumours of the parotid gland.
    - The parotid gland is the most common site (The parotid gland lies in front of and below the lower half of the ear) of origin of salivary neoplasms, almost 80% of which are benign
    - patient presents with painful and rapid/sudden onset lump with skin changes and enlargement of lymph nodes and involvement of **facial nerves**

Anterior triangle differentials:	Posterior triangle differentials:
• Enlarged lymph nodes • **Thyroglossal cyst** • Thyroid pathology • **Carotid body tumour** • **Sternocleidomastoid tumor** • Submandibular salivary gland swelling	• Enlarged lymph nodes • **Cystic hygroma** • Zenker's diverticulum

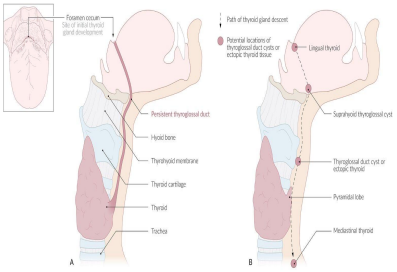


Anterior and posterior triangles are separated by sternocleidomastoid muscle

Neck swelling

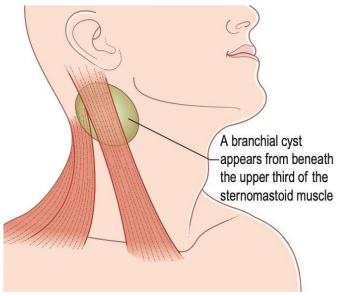
Thyroglossal cyst

- Portion of the thyroglossal duct remains patent
- Between the isthmus of the thyroid gland and the hyoid bone, and just above the hyoid bone. A midline swelling
- **History**
  - Age is Between 15 and 30 years old.
  - Common in females
  - Painless prominent lump
  - No systemic symptoms
- **Moves with protruding of the tongue.**
- Need surgery cause it's an important risk of cancer



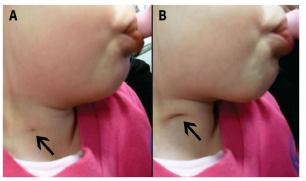
Branchial cyst

- Remnant of the second and third branchial cleft.
- Behind the anterior edge of the upper third of the sternomastoid muscle
- Between 5 and 10 cm long.
- Filled with golden yellow fluid and shimmers with fat globules and cholesterol crystals
- **History**
  - Mostly between the ages of 15 and 25 years



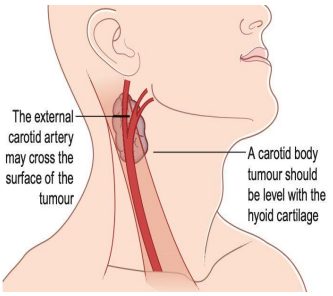
Branchial fistula and sinus

- Rare congenital abnormality.
- **Fistula:** An abnormal connection between two epithelium-lined surfaces.
- **Sinus tract:** an abnormal channel that originates or ends in one opening
- Characteristic finding: Tethering of the skin upon swallowing



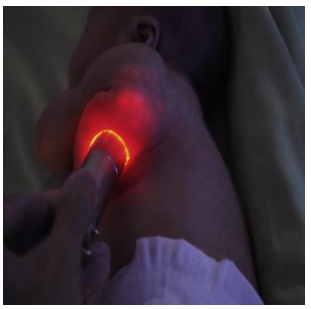
Carotid body tumour

- Rare tumour of the chemoreceptor tissue in the carotid body (chemodectoma)
- They are found in the upper part of the anterior triangle of the neck, level with the hyoid bone and beneath the anterior edge of the sternomastoid muscle. (Lateral neck swelling)
- Vary from 2–3 cm to 10 cm in diameter.
- Solid and hard, dull to percussion and do not fluctuate, Sometimes they pulsate.
- History
  - Between the ages of 40 and 60 years.
  - Benign, Painless & slowly growing lump.
  - Symptoms of **transient cerebral ischaemia**



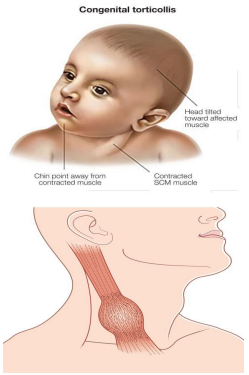
Neck swelling

Cystic hygroma



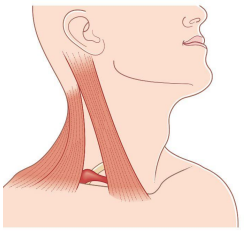
- Congenital <not familial> collection of lymphatic sacs in underdeveloped lymphatic channels that contain clear, colourless lymph.
- Their distinctive physical sign is a **brilliant translucence**
- Commonly occur near the root of the arm and the leg.
    - Pediatric (Cystic hygroma) Congenital
    - Adults (Lymph cyst and lymphocele) Iatrogenic
- Found around the base of the neck, usually in the posterior triangle.
- Diffuse swelling, not tender, not hot & normal skin.

Sternomastoid 'tumour'



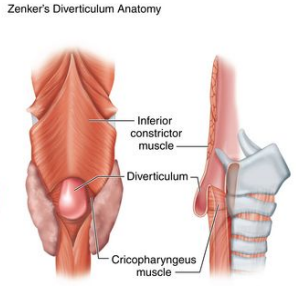
- Swelling of the middle third of the sternomastoid muscle due to ischemic attack
- **History**
    - Mother may notice the lump or that the child keeps their head turned to one side –**torticollis.**
- **Examination**
    - Tender Lump in the middle third of the neck on the anterolateral surface
    - Child will turn head to the other side but tilt head to same side.
    - Squint
    - An uncorrected torticollis may cause facial asymmetry.

Cervical Rib



- Basically it is additional rib from lower cervical vertebra adherent to the first rib by fibrous bands.
- It causes narrowing of the thoracic outlet, compressing a lot of structures like:
    - Subclavian artery
    - Brachial plexus.
- Although a cervical rib can cause serious **neurological and vascular** symptoms in the upper arm <Exacerbated by elevation of the hands>, clinical examination of the neck does not usually reveal any abnormalities (Discovered by x-ray)

Pharyngeal pouch (Zenker's diverticulum)



- 'Pulsion' diverticulum of the pharynx between the cricopharyngeus muscle below and the lowermost fibres of the inferior constrictor muscle above.
- **History**
    - Long history of **halitosis**
    - **regurgitation of froth and undigested food.**
    - It can be compressed and sometimes emptied with **gurgling sounds.**

Neck swelling

The thyroid gland

- Thyroid swelling characteristically moves upwards on **swallowing**
- **Lymphoma** and **papillary carcinoma** may occur in a thyroid that has been affected by **long-standing Hashimoto's disease.**

Diffuse, Eg:

- Graves
- Hashimotos
- Lymphoma
- Anaplastic tumours
- Diffuse hyperplastic (physiological: puberty & pregnancy)

Single, Eg:

- Cyst
- Adenoma
- Cancer:
  Medullary tumour
  Follicular tumour

Multinodular, Eg:

- Iodine deficiency (Most common cause).
- Toxic multinodular goiter (Plummer's disease)

- History
  - Compression symptoms: Dysphagia, odynophagia, Dyspnea, changes in voice.
  - Hyper/Hypo-thyroidism symptoms?
  - Family history of thyroid cancer?
  - Radiation exposure (especially in childhood)?

Tumors of the Thyroid

Benign

Follicular Adenoma

Malignant

Causes

Secondary

Primary

Differentiated

- Papillary Carcinoma (most common) (slow-growing) and Long time Asymptomatic tumor
- Follicular Carcinoma

Undifferentiated

- Anaplastic Carcinoma

Local invasion may involve the **recurrent laryngeal nerve**(s) and cause hoarseness, **trachea** causing dyspnoea and stridor, and the **oesophagus** causing dysphagia.

Medullary Carcinoma

- (originate from c cells)
- Amyloid deposit
- Calcitonin levels are elevated

The parathyroid glands

- Hypoparathyroidism
  - Usually seen post-operatively

- **hyperparathyroidism**
  - **Primary;** is due to an adenoma, (broken bones, **renal stones**, abdominal groans and psychic moans)
  - **Secondary;** there is over-secretion of PTH in response to low plasma levels of ionised calcium, usually because of renal disease, Vit. D deficiency or malabsorption
  - **Tertiary;** Excessive PTH secretion in secondary hyperparathyroidism may become autonomous, (This may occur after renal transplantation)
  - **Management** of primary and tertiary hyperparathyroidism: Remove all overactive parathyroid tissue, If two or more glands are enlarged, they should be removed. If all four glands are thought to be hyperplastic, then all but a portion of the smallest gland should be removed, then transplanted in other regions

Breast Diseases

(Based on 439 doctor's summary)

Common breast disease symptoms :

Breast Pain — Cyclic or Non-Cyclic

- **The cyclin pain** → (comes in relation to period), associated w/ benign diseases
- **The non cyclic pain** → is more pathological especially if it is associated with mass or nipple discharge or skin changes
  - **Investigations:** after clinical examination either by breast ultrasound or mammogram if patient age > 40 **OR** MRI if patients younger < 40 with strong family history.

Breast Mass

- **Indicates investigation:**
  - Radiologically by breast ultrasound and mammogram
  - Depending on age it'll indicate pathological mass , investigate by ultrasound guided trucut biopsy
    - BI-RADS 4 score indicates a suspicious finding or abnormality and require biopsy for further management
- Most common presentation of breast mass is painless mass

Final Assessment Categories

Category	Management	Likelihood of cancer
0 — Need additional imaging or prior examinations	Recall for additional imaging and/or await prior examinations	n/a
1 — Negative	Routine screening	Essentially 0%
2 — Benign	Routine screening	Essentially 0%
3 — Probably Benign	Short interval-follow-up (6 month) or continued	>0 % but ≤ 2%
4 — Suspicious	Tissue diagnosis (biopsy) True core biopsy	4a. low suspicion for malignancy (>2% to ≤ 10%) 4b. moderate suspicion for malignancy (>10% to ≤ 50%) 4c. high suspicion for malignancy (>50% to <95%)
5 — Highly suggestive of malignancy	Tissue diagnosis	≥95%
6 — Known biopsy-proven	Surgical excision when clinical appropriate	n/a

Nipple Discharge

- Considered pathological if → unilateral , spontaneous (coming by it self without squeezing the nipple) , from single duct or associated with pain or mass.
- **Investigation**: by taking discharge for culture , cytology , Radiology by breast ultrasound or mammogram or MRI depending on age if associated with mass the biopsy is indicated.

Abnormal Appearance

- Nipple retraction or deviation , skin tethering , retraction, Peau d'orange appearance visible mass or ulcer etc.
  - **When is nipple inversion worrisome?** If the onset is recent, if the inversion is unilateral and if upon examination you can't evert the nipple.
- Can be associated with infection , inflammatory disease or commonly associated malignant breast disease
- Should be evaluated clinically , Radiologically & pathologically by biopsy if there is mass

Benign breast diseases:

More common than malignant diseases

- ➔ Fibrocystic changes Pain **(main presentation)**
- ➔ Fibroadenoma mass **(main presentation)**
- ★ Duct Ectasia → presented with Variable color nipple discharge (green, yellow), transverse slit appearance
- ➔ Breast infection abscess ,mastitis
- ➔ Galactocele (lactating women with a lump)
- ★ Intraductal Papilloma → most common cause bloody nipple discharge
- ➔ Fat necrosis
- ➔ Breast cyst simple or complicated or complex cyst
- ➔ Phyllodes tumor
- ➔ Adenoma , Lipoma

Breast Diseases

Benign breast diseases:

fibroadenoma — 15-25 years

- ➔ **Location:** unilateral or bilateral,single or multiple of variables size
- ➔ Common presentation is breast mass
- ★ No risks of malignant transformation
- ➔ **Dx:** Is by history and examination, breast ultrasound and ultrasound guided trucut biopsy
- ★ 50% of fibroadenoma resolves by itself , other 50% can remain the same or become smaller or become bigger
- ★ **Surgical excision indication:**
  1- painful fibroadenoma regardless of the size
  2- size > 4 cm
  3- unclear pathology e.g. fibroadenoma with phyllodes variations , hypercellular with atypia
  4- rapid growth increases in size during follow up
  5- unusual age (> 40-50 years)
  6- family history of malignancy (tho no chance of malignant transformation) just to relieve pt anxiety
- Once a diagnosis of fibroadenoma has been established, options for management are reassurance with no follow-up or surgical excision.

Phyllodes tumor — 30 and above

- ➔ it is a variant of fibroadenoma but with more fibrous components
- ➔ 1% chance of recurrence if not completely excised
- ➔ about 1% chance of malignant transformation (sarcoma) which increase with increase in size
- ➔ **Tx:**
  1- if benign → complete excision
  2- intermediated type → excision with 1cm free margin
  3- if malignant → excision with 1cm free margin + Radiation therapy , it's not respondent to chemotherapy

Fibrocystic changes — 30-49 years

- ➔ **Common Sx:** pain in the form of fibrocystic changes , cyclic pain
  Pain maximizing before period then improves after that , some time associated with nipple discharge
- ➔ **Dx:** history and examination , lumpy bumpy nodular breast , tenderness
- Breast ultrasound is diagnostic , multiple bilateral variable size cysts in absence of other pathology.
- Pt = or > 40 years → mammogram is indicated to rule out malignancy
- ➔ **Tx:** reassure patient , and give her the advice in the from of:
  1- pain control by using simple analgesia (NSAID)
  2- Good supportive bra
  3- Exercise to activate pectoral muscle which helps in-moving fluids out of the breast by stretching rubber band vertical and horizontal
  4- Use vitamin E capsules for a month which my help in reducing symptoms if no improvement she can stop using it **Or** she can use Evening primrose oil capsules (nocturnal) for one month if no improvement she can stop using it.

Breast Diseases

Benign breast diseases:

★ Breast Cyst — Simple or Complex

- Pathological cyst → constant in one location and not getting smaller
- Simple **or** complex **or** complicated (indicates further investigation)
  - **Simple cyst** → 1- aspirated once → follow up patient with ultrasound
    2- cyst refill again → aspirated second time , fluids should be sent for cytology each time
    3- cyst refilled a third time → should be removed to rule out malignancy
  - **Complicated cyst** → aspiration and biopsy
  - **Complex cyst** → contains solid components , trucut biopsy should be done
- **Managements:** depends on the type of pathology results

★ US Can differentiate between solid and cystic lesions

Fat Necrosis

- Present later after 3-4 months from the injury as irregular, hard, non tender mass of dead or damaged breast tissue.
- **Investigation:** trucut biopsy

★ **Tx:** Reassurance (resolves with time).

Epithelial hyperplasia

★ If the hyperplastic cells revealed cellular atypical this condition called atypical hyperplasia 4-5 times increase in risk of developing Breast Cancer , after biopsy results you should perform frequent screening

Breast Infection

Lactating

- **Most common pathogen:** Staphylococcus aureus.
- How to differentiate between galactocele and abscess?
- Galactocele mass is associated with slight tenderness with no fever or rigors.

★ Abscess presentation is more acute, with way more tender breasts, sky high fever and rigors

★ Mastitis: antibiotics and continue lactating

★ Management in mastitis : Abx and continue breastfeeding

Non-lactating

- **Central (periareolar) infection** → Smoking is an important etiological factor
- **Peripheral non-lactating abscesses.**
- **Tubercular mastitis.**
- **Skin-associated infection.** (Intertrigo, Epidermoid (sebaceous cysts), Hidradenitis suppurativa)

Breast Diseases

Malignant breast diseases :

★ Most associated risk factor for breast cancer is previous history of benign masses

- Invasive ductal carcinoma (single most imp prognostic factor is the number of involved axillary lymph nodes)
- Lobular carcinoma
- Malignant phyllodes → sarcoma
- Paget's disease of the nipple

Screening:

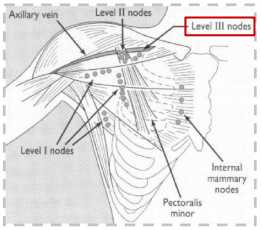
- Start at age of 40 years **unless** patient had potential risks of genetic disease the screening should start 10 years younger

➔ screening is by mammogram annually , Young patient can be screened initially by ultrasound or MRI

➔ **The risk of genetic disease:**

- History of breast cancer in the family at age less than 40 years
- Father had history of ca breast
- Bilateral breast malignant disease
- Uterine or ovarian malignancy
- More the 3 members of the family with history of malignancy

★ Clinical staging:



➔ < 2 cm no axillary lymph nodes **stage 1**
➔ 2-5 cm with mobile axillary lymph node **stage 2**
➔ > 5 cm , skin changes or fixed axillary lymph node **Stage 3**
➔ Any size with metastasis **stage 4**
➔ There are pathological staging and TNM staging.
➔ Level 3 lymph nodes has the worst prognosis

Investigation:

➔ Breast ultrasound , mammogram and MRI as indicated
➔ Tissue biopsy by ultrasound guided trucut biopsy including receptors status (ER ,PR ,HER2 ,Ki 67)
➔ Metastatic screening by CT scan CAP (chest abdomen and pelvic)
➔ Bone scan

Management:

The Tx depend on the stage of the disease,size of the tumor , receptors status

Multiple modalities

➔ **Surgery**
- conservative (**lumpectomy** + Should be combined **with sentinel node biopsy** or axillary clearance) with Radiation therapy as must Rx
- mastectomy + sentinel lymph node or axillary lymph dissection as indicated

➔ **Chemotherapy** as stage indicates
➔ **Hormonal therapy**
➔ **Monoclonal antibody**

Breast Diseases

Malignant breast diseases cont. :

Contraindications for conservatives surgery:

After patient wish

- ➔ Tumor size in relation to breast size if you can't get free tumor margins
- ➔ Multicentric disease
- ➔ Non availability of Radiation therapy or there is contraindications for Radiation therapy
- ➔ First trimester pregnancy
- ➔ Stage of the disease (stage 3)

Prognostic Factors:

- ➔ Stage of the tumour at diagnosis: its size and involvement of the axillary lymph nodes or the presence of any metastases.
- ➔ Biological factors: histological grade, histological type, presence of lymphatic and/or vascular invasion, hormone receptor and HER2 status.
- ➔ Genomic

Abdominal Wall, Umbilical & Other Hernias

Rectus muscle disorders

1- Desmoid Tumor

- Rare benign tumor. Can progress to be malignant fibrosarcoma
- Thought to arise from fibrous intramuscular septa in the lower rectus abdominis muscle.
- The tumors originate from fiber-producing connective tissue, are characterized by local and aggressive tumor growth, and do not metastasize.
- **Treatment involves radical surgical resection** because it's refractory to chemotherapy and radiotherapy.
- It does not change in size when the abdominal muscles are contracted.
- Associated with Gardner's syndrome.
- Most common in women of child-bearing age.

2- Rectus sheath Hematoma

- A painful swelling within the rectus sheath arises from the disruption of a branches of the superior or inferior epigastric arteries.
- This condition is rare, but may represent an unusual presentation of acute abdominal pain
- Trauma is the most common cause of hematoma

Risk factors

- Elderly patient
- Anticoagulants (How to correct it? Give fresh plasma and vitamin k which will reverse the anticoagulants effect)
- Excessive physical exertion
- Pregnancy
- Vigorous coughing (steroids)

Types

Acute
(in elderly after violent cough, especially in those on steroids)

A- Rupture of inferior epigastric artery:
- presents with tender mass in right or left iliac fossa and with overlying bruise.

B- Rupture of superior epigastric artery:
- presents with upper abdominal tenderness and a bruise below the costal margin.

Chronic

- Presents as non tender upper abdominal mass
- The swelling will be firm to hard, and without a definite edge because it is deep seated.

Clinical features

- **Carnett sign** (useful for differentiating abdominal wall pain from intra-abdominal pain).
  - The patient is in the supine position and the investigator identifies the point of maximal abdominal pain by deeply palpating with a finger; the patient is then asked to tense the abdominal muscles while the fingertip is released, followed again by deep palpation.
  - **Positive** if both stages (before and after muscle contraction) are **painful**. In contrast, pain originating from the abdominal organs is associated with just a pain in the first stage.
- **Fothergill sign** (useful for differentiating abdominal wall mass from intra-abdominal mass).
  - The patient is asked to lie in the supine position and contract the abdominal muscles, e.g., by lifting the head or lower limbs
  - **Positive** if the palpable abdominal **mass does not cross the midline** and remains palpable after contraction of the rectus abdominis muscles
- **Cullen sign** (periumbilical ecchymosis)
- **Grey Turner sign** (flank ecchymosis)
- **Signs of hypovolemic shock** (e.g., tachycardia, hypotension) due to Inability to tamponade the bleeding from the artery.

Diagnosis

- Serial measurement of **hematocrit and/or hemoglobin levels;** anemia and/or leukocytosis may be present.
- **CT scan (more accurate)**
- **Abdominal ultrasound**: can be used to confirm the diagnosis.

Treatment

Hemodynamically unstable patients with expanding hematoma

- Angiography with arterial embolization
- Surgical evacuation of the hematoma and ligation of the artery

Conservative

- Rest
- Analgesics (Morphine)
- Compression of the hematoma
- Correction of abnormal coagulation
- IV fluid resuscitation and/or blood transfusion

Abdominal Wall, Umbilical & Other Hernias

2- Hernias

- Hernia Derived from Latin word for rupture
- An abnormal protrusion of an organ or tissue through a defect in its surrounding walls.
- Hernias can be considered as a disease of collagen metabolism. Collagen I/III ratio is important.
- In summary: weak areas are caused by stretching or surgical incisions in association with a defect in collagen metabolism.

Hernia parts	• Hernia **neck** Located at the innermost musculoaponeurotic layer whereas the hernia **sac** is lined by peritoneum and protrudes from the neck. If a patient comes one week after surgery with a "hernia" and there is no sac. then this dehiscence (separation of surgical sutures) and it's not a hernia • No consistent relationship between the area of a hernia defect and the size of a hernia sac (In physical examination, we don't care how large the "sac" is. We must palpate the hernia and push it centrally to estimate its "defect" size.)
Hernia Contents	• A hernia may contain any intra abdominal structure, but most commonly contains omentum and/or small bowel. • A hernia is an abnormal protrusion of a cavity's contents through a weakness in the wall of the cavity, **but takes with it all the linings of the cavity** "To form its wall"
Terminology	• **Reducible Hernia :** when intraabdominal contents can easily move in and out the lump site to the abdominal wall • **Incarceration :** Irreducible Hernia when contents can not be returned to the abdominal wall cavity due to adhesions or edema. Its painless or slight discomfort, no tenderness nor skin changes • **Obstruction:** irreducible hernia when contents can not be returned to the abdominal wall cavity • **Strangulation :** Irreducible hernia with compromised blood supply "Gangrene", Usually due to occluded Vein then complicate to artery. Will be very painful, red and **skin changes** which differentiate between it and obstructed hernia. Strangulated hernia is a medical emergency needs immediate surgery.

Risk groups

- Intraabdominal high pressures from **constipation**, prostatic symptoms, excessive coughing & lifting, peritoneal dialysis.
  - However, it has been shown that hernia is no more common in Olympic **weightlifter** than in general population, suggesting that high pressure is not a major factor causing hernia.
  - Many patients will first notice hernia after excessive **straining**.
- **Pregnancy** due to hormonally induced laxity of pelvic ligaments.
- **Elderly** due to degenerative weakness of muscles and fibrous tissue.
- Hernia is more common in **smokers** because it affects collagen formation/degradation/modification.
- A recent Swedish report has shown that inguinal hernia is less common in obese patient with hernia risk being negatively related to BMI contrary to widespread belied.

Groin Hernias

- **Groin hernias are divided into (Inguinal & Femoral).**
- **"These are important facts you need to remember" you have to consider which type is more common in each gender.**
- (Femoral commonly occur in females more than males but still indirect inguinal hernia is considered the most common in both genders, Inguinal commonly occur in males "Indirect in children". Don't get confused when asked about this part)
- You can use these gender and age groups as tips if asked to determine the type of hernia in the MCQs.
  - ★ Indirect inguinal is the most common in both males and females.
  - ★ Direct hernias are very **uncommon** in women.

- Indirect inguinal and femoral hernias are more **common** on the **right side.**
- Attributed to a delay in atrophy of the processus vaginalis following the normal slower descent of the right testis to the scrotum during fetal growth; tamponading effect of the sigmoid colon on the left femoral canal

Inguinal Hernias

- **25M:1F For groin hernias**
- **can be painless swelling** in the groin or scrotum
- Prevalence of hernias increases with age, as does likelihood of **strangulation (**very painful and tender)
- Strangulation occurs in 1% to 3% of groin hernias
- 75% of all abdominal wall hernias occur in inguinal region
  - 2/3 (50%) are indirect, ⅓ (25%) direct

Femoral Hernias

- 5% of all hernias are femoral Other hernias represent 20%
- F:M is 10:1 for femoral hernias
- 10% of F and 50% of M who have a femoral hernias either have or will develop an inguinal hernia
- Femoral hernias have the **highest rate of strangulation** (15% to 20%) of all hernias

Abdominal Wall, Umbilical & Other Hernias

Groin Hernias (inguinal Hernia)

Inguinal canal

- The canal that contains spermatic cord (males) or round ligament (Females) & Processus Vaginalis
- Have deep (Internal) & Superficial (External) rings
- Passes **Above** inguinal ligament, **lateral** to inferior epigastric arteries
- 4 cm oblique passage in the lower anterior abdominal wall, lies just above inguinal ligament
- Bounded by the deep and superficial inguinal rings, and the four walls
- The internal (deep) inguinal ring is an opening in the transversalis fascia
- Bounded medially by **Inferior epigastric** artery Important landmark to differentiate between direct and indirect hernia
- The inguinal canal ends at the external (superficial) inguinal ring, which is an opening in the aponeurosis of the external oblique muscle

Content of the canal

Male

Three Nerves :
- Ilioinguinal
- Genital branch of genitofemoral
- Sympathetic fibers to vas and testes

Three veins:
- Pampiniform plexus.
- External spermatic vein.
- Deferential vein.

Three arteries:
- Cremasteric artery.
- Testicular artery.
- Artery of the vas deferens.

Three other things:
- Vas deferens
- Lymphatics of the testis & vas
- Cremaster Muscle

Three layers of fascia:
- External spermatic fascia.
- Middle spermatic fascia (Cremasteric)
- Internal spermatic fascia.

Female

Two main things:
- Fat pad
- Round ligament

Two vessels:
- Artery to round ligament
- Vein of the round ligament

Two nerves:
- Ilioinguinal
- Genital branch of genitofemoral

Indirect Inguinal Hernia

- The **most common** Hernia for both sexes
- **Predisposing factors** are: Persistence of a patent processus vaginalis which is the primary causative factor **in pediatric population**; in adults, the case is likely multifactorial.
- An indirect inguinal hernia enters the internal (deep) inguinal ring and descends **within the coverings of the spermatic cord** so that it can pass into the scrotum, the so-called inguino-scrotal hernia.
- It enters the inguinal canal. So, it will protrude above inguinal ligament, and lateral to the inferior epigastric artery, this protrusion may remain in the inguinal region, or extend by passing above the inguinal ligament to the scrotum.

Clinical Features

- May remain asymptomatic with some dragging or discomfort in the groin during lifting or straining or at the end of the day.
- Relieved by rest.
- It is unusual for a patient to present with a lump without a pain.
- In adults it Develops over months to years.
- More common in children, **spontaneous resolution during the first year of life.**
- Once the child begins to walk its resolution less likely.

Signs of the hernia

- Figure (A), Picture (A) Bubonocele:
- Hernia forms a swelling in the inguinal region (Appearing as a bulge)
- Figure (A), Picture (C) Hernia extended into the scrotum
- passes **above and medial** to the pubic tubercle (Landmark inguinal ligament), in contrast to a **femoral** hernia which bulge **and lateral** to the tubercle (Figure B)
- Clearly visible when patient stands, or asked to cough and reduces lying down
- **Cough impulse:** palpate the patient and ask the patient to cough, you feel something hitting your hand then its positive and highly of hernias
- If you couldn't find a protrusion, look for asymmetry (Bilateral hernia uncommon, and if present they wouldn't be symmetrical)
- If you also couldn't (Like in obese patients), try to palpate cough impulse

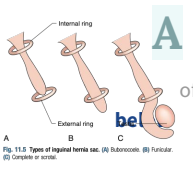
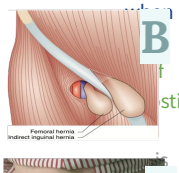


Fig. 11.5 Types of inguinal hernia sac. (A) Bubonocele. (B) Funicular. (C) Complete or scrotal.



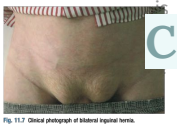


Fig. 11.7 Clinical photograph of bilateral inguinal hernia.

Abdominal Wall, Umbilical & Other Hernias

Groin Hernias (inguinal Hernia)

Direct Inguinal Hernia

Predisposing factors

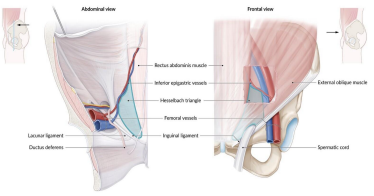
- Due to **weakness of the abdominal wall** (hesselbach's triangle).
- precipitated by increases in intra-abdominal pressure
- Other common causes of hernia
- Weakness is usually bilateral

Association with indirect hernia

- ⋆ The neck of the sac of a direct inguinal hernia lies medial to the inferior epigastric vessels, whereas that of an indirect hernia lies lateral to them. (Very important MCQ)
- A **combined indirect and direct** hernia may occur on the same side (**pantaloon (بنطلوني) or saddle-bag hernia**), with sacs straddling the inferior epigastric vessels.

Hesselbach's triangle

- A direct hernia is a weakness of the inguinal **floor**.
- Pushes the **transversalis fascia** -which is void of muscle- to protrude.
- It lies between **Inferior epigastric vessels** (Superolateral), **Lateral border of rectus abdominis muscle** (Medial) and **inguinal ligament** (Inferolateral)
- These boundaries mark the area known as Hesselbach's triangle.



Clinical features

- The hernia forms a bulge in the medial side of inguinal canal.
- **reduced by backward pressure,** and the edges of the defect may then be palpable.
- it is often difficult to distinguish between the direct and indirect hernias
- "deep ring occlusion test" may aid in differentiating, but it is unreliable test. Often the final differentiation made on the operating table.
- The hernia occasionally bulges through the external (superficial) inguinal ring, but the transversalis fascia cannot stretch sufficiently to allow it to descend down into the scrotum (Direct hernias never extend to scrotum) Does not go through with the spermatic cord
- The sac has a **wide neck,** so that the hernia **rarely** becomes irreducible, obstructs or strangulates.

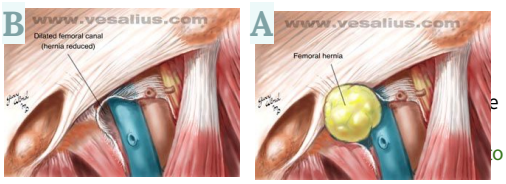
	Indirect hernia	Direct hernia
Prevalence	• Most common hernia in all genders. • Most commonly occur in **pediatrics**	• Most commonly occurs in males (**Adults**) • Rare in children
Predisposing factors	• Persistence of a **patent processus vaginalis**	• weakness of the **abdominal wall** (hesselbach's triangle).
Site	• **above** inguinal ligament, and **lateral** to the inferior epigastric artery. • **The defect (Neck) is not palpable**, as it is behind the fibres of the external oblique muscle	• **above** inguinal ligament, and **medial** to the inferior epigastric artery. • The defect (neck) **may be felt** in the abdominal wall above the pubic tubercle
Clinical Features	• Lump is Clearly visible when patient stands, or asked to cough, and reduces when lying down • Resolve **spontaneously** in children within 1st year of life	• Lump is Clearly visible when patient stands, or asked to cough, and reduces when lying down • **Bilateral** hernia is usually direct
Signs	• Lump **can** present either at groin or **scrotum** • Reduces upwards, then **laterally (**to pass inguinal ligament) and backwards • **Controlled** after reduction by pressure at internal inguinal ring	• **Does not** (hardly ever) go down into the **scrotum** • Reduces upwards and then straight backwards • **Not Controlled** after reduction
Complications	• **Occasionally** irreducible, rarely strangulated • testicular infarction is more common than bowel infarction.	• **rarely** becomes irreducible, obstructs or strangulates.

Abdominal Wall, Umbilical & Other Hernias

Groin Hernias (Femoral Hernia)

General info

- passes Through the femoral canal
- Small, easy to miss on examination (delayed diagnosis)
- Picture (A): This is a bowel passing through the femoral canal forming femoral hernia.
- Picture (B): And this is when the hernia reduced "void of bowel".
- As the hernia enlarges, it passes through the saphenous opening thigh and then turns upwards to lie in front of the inguinal (Forms J-shaped course). This will lead to frequently difficult or reduce the hernia



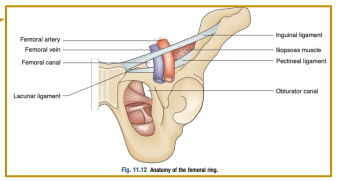
Femoral Canal Boundaries

What is in the femoral canal ?

- The small space medial to the vein within the femoral sheath is the femoral canal through which lymphatics pass from the thigh into the abdomen.

Mnemonic from Dr.bishr: CLIF

- Anterior: Inguinal (Poupart's) ligament
- Posterior: Pectineal (Cooper's) Ligament
- Lateral: Femoral vein
- Medial: Edge of the Lacunar (Gimbernat's) ligament
- In 10-20% of cases, the aberrant obturator artery passes adjacent to the lacunar ligament



Femoral Sheath

- surrounds the femoral vessels forming the femoral sheath.
- Femoral vein Medially, and Femoral artery laterally and Femoral **branch** nerve (a branch of genitofemoral nerve) are inside the sheath
- Femoral nerve is outside the lateral sheath

Clinical features

- Predisposing factors are similar to the direct hernia
- Femoral hernias are often small (Because it has many coverings) and easy to miss on clinical examination
- They are prone to obstruction and strangulation (Because it has a small (tight) neck) ~40% presents this way
- Patient presents either by a lump in the upper medial thigh may be the presenting symptom Or by exercise induced pain (groin pain related to exercise is also a common presentation
- frequently difficult or impossible to reduce because of its J-shaped course and the tight neck of the sac.
- There is cough impulse

Differential diagnosis

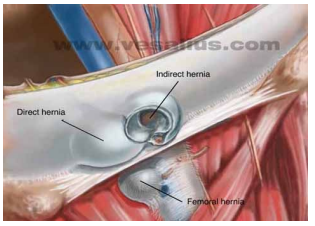
- Inguinal lymph node (No cough impulse, irreducible)
- Saphenous varix: (positive cough impulse or 'saphenous thrill', which is prominent on standing but disappears on elevating the leg)
- Ectopic testis, psoas abscess, hydrocele of spermatic cord
  - Needle aspiration is not advisable for any such swelling (only after clearly defining diagnosis or after removal of the mass)
- Psoas abscess

Sportsman hernia

- Groin injury leading to chronic groin pain is often referred to as the sportsman's hernia
- Mostly, there is no clinical signs (No lumps), despite the pain symptoms.
- If you perform dynamic U/S (rest and straining), you may find "impalpable hernia" in some patients which causes the pain, and you will find nothing in others.
- **MRI is gold standard** for diagnosis
- Treatment is controversial, some can be treated as hernia, and other will be treated with NSAIDs.
- A deficiency of the posterior inguinal wall is the most common operative finding in patients with chronic groin pain.
- The differential diagnosis includes: musculotendinous injuries, urological pathology, or bone and joint disease.

What's the simplest anatomical difference between inguinal and femoral hernias?

- **Inguinal hernias** in relation to:
  - Pubic tubercle: Above and medial
  - Inguinal ligament: Above
- **Femoral hernia** in relation to:
  - Pubic tubercle: Below and lateral
  - Inguinal ligament: Below



What is the easiest way to locate pubic tubercle (the landmark of inguinal ligament) to locate inguinal ligament?
By tracing the tendon of adductor longus upward

Abdominal Wall, Umbilical & Other Hernias

Treatment of inguinal & Femoral hernias

Management is not part of our objectives, but Dr. Yasser gave an example about mesh and non-mesh

Normally: we make an opening in the sac and reduce the contents back to the abdomen, then remove the sac if **it's ALL PERITONEUM**

All hernias should be treated **even if asymptomatic** to prevent symptoms and complications especially if they are elderly

Surgical repair of the hernial defect is the only definitive treatment of inguinal hernia

The management of **direct** and **indirect inguinal** hernia does not differ

- **Complicated inguinal hernia**
  - ➔ **Strangulated hernia and / or sign of bowel obstruction:** emergency surgery (within hours) to minimize the likelihood of gangrene.
  - ➔ **Incarcerated hernia without strangulation:** manual reduction
    - Successful manual reduction: close monitoring + schedule an elective hernia repair as early as possible.
    - Unsuccessful manual reduction: urgent surgery.
- **Uncomplicated inguinal hernia**
  - **Reducible hernia:** elective surgery consider watchful waiting in select patients.

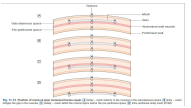
Treatment of **femoral** hernia: Surgery
- **Complicated femoral hernia:** Herniorrhaphy (Non-mesh)
- **Uncomplicated femoral hernia:** Hernioplasty (mesh repair)

Surgeries:
- **Hernioplasty (mesh repair) tension free repair** (زي الرقعة)
  - Definition: repair of a hernial defect using a synthetic mesh
  - Indication: **gold standard** for **inguinal** hernia repair

Open surgery: Preferred in patients with complicated inguinal hernia or contraindications for laparoscopic repair (the main contraindication for laparoscopic surgery is high anesthesia risk),

Laparoscopic surgery: preferred in patients with bilateral or recurrent inguinal hernia

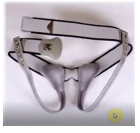
- When there is an opening in the abdominal wall. If you pull the two sides of the opening and suture them together, the tension on the sutures will be very high and chances of recurrence are high
- When there is a hole in your tire, you don't stitch the hole up because that will leave the tire with a weak point. What you do is you cover the hole with something. In our case, It's a mesh
- Position of mesh:
  - A is very bad since the chance of infection is high because it is relatively superficial
  - B is the worst since the foreign body (Mesh) will shrink and the chance of recurrence is very high
  - **C is the best**
  - D is okay but but the mesh causes adhesions with the bowel loops (it's done now with special double layered meshes that prevent adhesions)



- **Herniorrhaphy (non-mesh repair)**
  - Definition: open surgical repair of a hernial defect using autologous tissue
  - Indication: Consider in patients at high risk of surgical site infection.

Unilateral truss

Trusses
- They apply pressure to the hernia and reduce it backward, to keep the protruding tissue in place and relieve discomfort.
- Trusses can be used to provide symptomatic relief of hernias
- Hernia control (partial or total symptom relief) has been reported in 31-70% of patients with trusses.
  - Trusses should be used only for low-risk, completely reducible groin inguinal, **not femoral hernias**
  - Trusses should be preserved and offered to those in whom the surgical option is contraindicated, like those who cannot tolerate anaesthesia (severe CVD or respiratory disease)



Bilateral truss

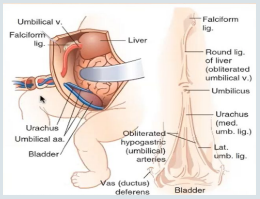
Abdominal Wall, Umbilical & Other Hernias

Other hernias

type	General info	Predisposing factors	Clinical features	Complications
Divarication (rectus diastasis)	Divarication of the rectus is a condition where the rectus abdominis muscles are no longer located next to each other as they run up and down the abdomen from the breastbone (xiphoid) to the pubic bone (symphysis pubis). The muscles separate from each other and is easiest to see when the patient does sit-ups and notices a linear bulge running up the centre of the abdomen. -it is considered as rectus muscle disease, it is **not true hernia**	• It is an important risk factor for developing ventral hernia • The most common cause in women is pregnancy. • Heavier men can develop this condition in their upper abdomen as well.	• Pregnancy induced rectus divarication can cause a significant shape change to the abdominal wall, even for very slender patients. Males have their own unique pattern of rectus divarication noticed as a midline bulge located between the xiphoid and the umbilicus. When patients have significant pain or associated hernias, a repair can be performed.	• The condition itself has no risk, because the inner aspect of the abdominal wall is smooth. This means that bowel cannot find its way into a hernia and potentially strangulate. However, the divarication can be associated with umbilical or epigastric hernias.
Lumbar	• **Grynfeltt's**: through superior lumbar triangle: 12th rib, paraspinal muscles, and internal oblique. (More common) • **Petit's**: through inferior lumbar triangle : iliac crest, latissimus dorsi muscle, and external oblique • Both may occur primary due to increased intra-abdominal pressure, or secondary complication to spinal surgery	• The overlapping nature of bulky muscles prevent the usual occurrence of hernias in these locations but acquired weakness after surgery, especially muscle cutting incisions or nerve damage leads to protrusion of lumbar fascia with extraperitoneal fat and an occasional hernial sac	• patient may complain of back pain, cosmesis, or weakness of activities associated with use of these muscles, in addition to the presence of a visible lump	
Spigelian	Hernia through the **Linea semilunaris** the lateral border of the rectus sheath between rectus abdominis and lateral abdomen • This area is Lack of posterior rectus fascia -> inherent weakness. • Nearly all occur at or below the arcuate line ○ The conjunction of arcuate line and **linea semilunaris** is the **weakest** area. • often interparietal - hernia sac dissects posterior to the external oblique aponeurosis. (at the outer border of the rectus abdominis muscle) • Treatment is surgical, as the hernia is liable to strangulate • absence of a posterior rectus sheath is a contributing factor at this location and therefore mostly occurs below the arcuate line	• may be related to stretching in the abdominal wall caused by obesity, multiple pregnancies, previous surgery or scarring	• Symptoms can vary from abdominal pain, lump in the anterior abdominal wall or patient may have history of incarceration with or without intestinal obstruction. Pain varies in type, severity, and location and depends upon contents of hernia. Pain often can be provoked or aggravated by maneuvers that increase the intra abdominal pressure and is relieved by rest.	• The risk of incarceration and strangulation is high due to the small neck and lack of clinical features to suspect as such
Perineal	• Hernia sac protrudes through the pelvic diaphragm	• **Older**, multiparous women , Multiple vaginal deliveries especially with difficult , prolonged labor • iatrogenic in those who underwent rectal surgery • surgeries associated include abdominoperineal resection , vaginal hysterectomy , and perineal prostatectomy	• The most common presenting signs of perineal hernia include tenesmus or constipation and perineal swelling. Perineal hernia is **diagnosed by digital rectal examination**	
Obturator	Through the obturator canal • The **diagnosis** is frequently made only when the hernia has **strangulated** and is **discovered at laparotomy.**	• 9 times more common in females due to their wider pelvis • Most patients **over 50 years old** • The other risk factors include chronic obstructive pulmonary disease, chronic constipation and ascites	• **knee pain** owing to pressure on the obturator nerve • pain on the medial aspect of thigh due to compression of obturator nerve, or palpable mass on the medial aspect of thigh. however, the diagnosis is frequently made only when the hernia has strangulated and is discovered at laparotomy.	• rare pelvic hernia with incidence of 1% and most commonly presents as acute Intestinal Obstruction It can also contain Appendix, Meckel's Diverticulum or omentum

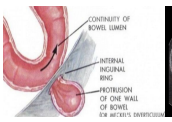
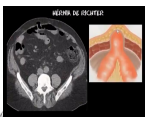
Abdominal Wall, Umbilical & Other Hernias

Other hernias

Hernia	Predisposing factors	Clinical features	Complications
Epigastric	• Common in **thin** individuals • Especially in children and associated with divarication of Rectus abdominis • Common in individuals with a single aponeurotic decussation	• It is a hernia of **linea alba**, above umbilicus and below xiphoid process • Patient complain of a local discomfort at that site Especially during ingestion • Rarely visible on inspection, but is palpable as a firm midline lump	• The herniation may consist of extraperitoneal fat or may be a protrusion of peritoneum containing omentum.
Umbilical **(infantile umbilical hernia)** 	• Age group: Occur in **infants** • Protrude through Umbilical ring • it is caused by a weakness in the adhesion between the scarred remnants of the umbilical cord and umbilical ring	• These small hernias occur in the superior margin of the umbilical ring • Easily reducible and become prominent when the infant cries • Most of these hernias **resolve** within the **first 24 months of life** (Strangulation is rare) • Over 95% of these hernias close spontaneously in the first 3 years.. Persistence after the 3rd birthday is an indication for elective repair.	
Paraumbilical **(Acquired umbilical hernia)**	• Age group: occur in **Adults** (Obese and Multiparous women, Ascites) • Protrusion is part of linea alba (Just above or below the Umbilicus): Above is more common • Caused by gradual weakening of tissues around the umbilicus (This weakening can be caused by excessive stretching).	• Do **not spontaneously resolve** but gradually increase in size • The dense **fibrous ring** at the neck of this hernia makes **strangulation** of herniated intestine or omentum an **important complication**	
Incisional	• Poor surgical technique. • wound infection after surgery • Obesity • Especially Elderly, diabetic patients, Immunocompromised patients or patients on steroids (All have poor healing)	• Hernia through an incision site • >50% of incisional hernias occur in the first 5 years after the original surgery. • Midline vertical incisions are most often affected. • The Bulge is better seen when patient contract his abdomen muscles “Coughing or raising from the bed”	• Strangulation is rare, but surgical repair is usually advised.

★ **Special Hernias**"يحبون يسألون عنها بالاختبارات مع انها نادرة جدا"

Know the rare hernias especially for the OSCE. Richter and sliding are more common than Littre and Amyand

Littre’s hernia	• hernia content include **Meckel's** diverticulum
Petersen hernia	• Seen after **bariatric** gastric bypass
Petit’s, Gyrnfeltt’s & Pantaloon hernias	• Peter’s & Gyrnfeltt’s had Mentioned Earlier in Lumbar hernia • Pantaloon: had mentioned in Inguinal hernias
Richter hernia 	• Incarcerated/strangulated hernia involving one sidewall of the bowel only (bowel wall only)  
Amyand’s hernia	• Hernia sac containing a ruptured appendix
Sliding hernia	• It is a sort of **chronic** groin hernias in which the protruded organ is “fused” with the sac wall • Is very important since retroperitoneal structures can form the wall • **Removing the sac is contraindicated** in this case
Maydl’s Hernia 	• hernial sac contains **two loops** of bowel with another loop of bowel being intra-abdominal • Small, seen in males, predominantly in the right side. • Postural or manual reduction of the hernia is contraindicated as it may result in non-viable bowel being missed

Abdominal Wall, Umbilical & Other Hernias

hernia complication

Irreducibility/Incarcerated

- **Reducible** hernia is when the intraperitoneal organs can move freely in and out of the hernia
- An **irreducible** hernia is one in which the contents cannot be manipulated back into the abdominal cavity.
- This may be due to narrowing of the neck of the sac by fibrosis, distension of the contained bowel, or adhesions to the walls of the sac.
- Irreducible hernia isn't an indication for emergent surgical intervention
- Can be detected by loss of cough impulse

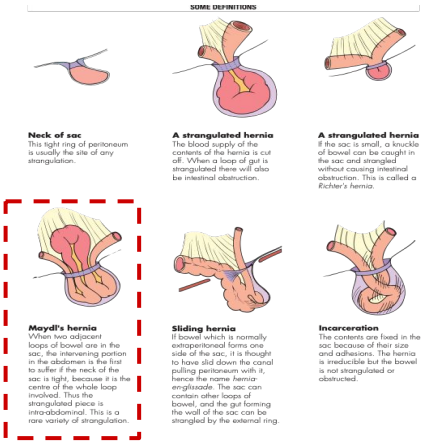
Obstruction

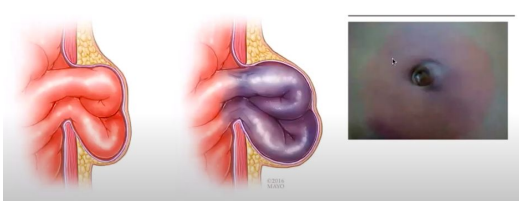
- Obstruction of the intestinal lumen (Precede the strangulation)
- There is no Ischemia
- colicky abdominal pain, vomiting, constipation and distension (signal the need for urgent operation before strangulation supervenes.)
- Obstruction is an indication of emergent surgery
- Painful, tenderness and no skin changes

Strangulation

- The worst possible complication. occur after obstruction
- The vessels supplying the bowel within a hernia may be compressed by the neck of the sac or by the constricting ring through which the hernia passes.
- The contents initially become swollen as a result of venous congestion (Veins occlude first) & Exudation
- The arterial supply is subsequently compromised and gangrene follows
- Very painful, skin changes which differentiate between it and obstructed hernia.
- The skin overlying the hernia is red, warm to touch and tender, cough impulse is lost, and there may be increasing evidence of circulatory collapse and sepsis.
- In richter's hernia, there may be strangulation w/o obstruction signs (Make it harder to diagnosis clinically, this non-diagnosed strangulation increases the mortality risk in these patients)
- In Maydl's hernia (Hernia-in-W shape) Postural or manual reduction of the hernia is **contraindicated** as it may result in non-viable bowel being missed (Look at the picture)

- What is the main clinical feature to differentiate between Obstructed and strangulation hernia ?
  - The color of the lump (strangulated hernia will be darkened, reddish or bluish). Pain may present in both.
- Complete and incomplete hernia (occur in indirect hernia)
  - **Complete** extend to scrotum while **incomplete** remain in the inguinal canal
- If the lump **isn't** associated with cough impulse, you have to think of other differentialis depending of the site of that hernia (Lymph nodes, mass)

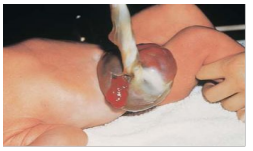




Abdominal Wall, Umbilical & Other Hernias

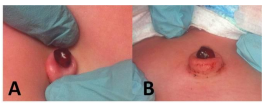
Umbilical Diseases

Exomphalos (Omphalocele)



- The intestines protrude through a central defect of all layers of the abdominal wall.
- Void of skin (this is the difference from Umbilical hernia)
- Their only covering is a thin, transparent membrane formed from the remnant of the coverings of the yolk sac
- If this membrane ruptures, death from peritonitis may follow.
- Present at birth
- Represents an intrauterine failure of the intestines to return to the abdomen
- combined with a failure of the two sides of the laterally developing abdominal wall to unite to cover the embryonic defect

Umbilical Granuloma



- It is a mass that occur in the umbilical ring, as result of inflammation, Granuloma and excessive granulation tissue
- (You need to send a specimen for pathology to confirm it is a granulation tissue).
- Not present at birth. It occur gradually after the umbilical cord has been tied
- Usually regress spontaneously in the first month, if not the possibility of a patent vitello-intestinal duct or an umbilical adenoma should be considered.
- The baby presents with an inflated umbilicus overlied by a bright-red, moist, friable, sometimes hemispherical mass of bleeding granulation tissue

Duct cyst

- Both these tracts may partially close, leaving a patent segment that becomes a cyst.
- **Vitello intestinal tract leaves a small mobile cyst**
- **Urachus tract cyst is immobile and large situated below the umbilicus**

Umbilical Adenoma

- The mother complains that the baby has a lump (**Raspberry like**) at the umbilicus and a **mucous discharge**.
- Pathology specimen for confirmation
- An umbilical adenoma is a patch of intestinal epithelium left behind when the vitello-intestinal duct closes.

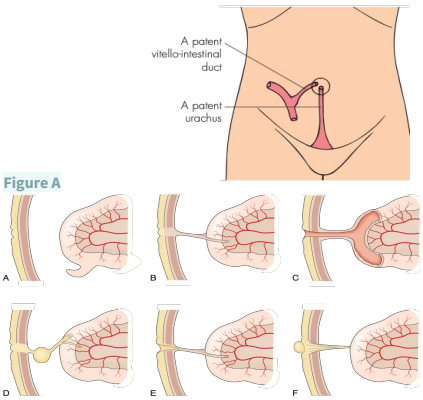
Endometrioma

- If in a female patient, the umbilicus enlarges, becomes painful and discharges blood during menstruation, it may contain a patch of ectopic endometrial tissue.

Umbilical Fistula

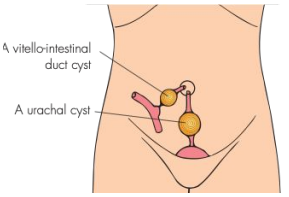
- Four structures pass through the umbilicus during fetal growth: the umbilical vein, the umbilical arteries, **the vitello-intestinal duct and the urachus**. If either of the last two tubes fails to close properly, there will be an intestinal or a urinary fistula.

Patent Vitello-intestinal tract



- The vitello-intestinal duct runs in intrauterine life from the apex of the midgut loop to the yolk sac. It is normally obliterated **long** before birth
- If not, it may persist as meckel's diverticulum, or rarely develop into another complication (Figure A)
  - (A) A Meckel's diverticulum.
  - (B) A fibrous cord to the ileum.
  - (C) An umbilical intestinal fistula.
  - (D) An enterocystoma.
  - (E) An umbilical sinus
  - (F) An enteroteratoma.
- Can Present either as **a complicated Intestinal fistula** in which food leak from the umbilicus**,** or resembles umbilical granuloma

Patent Urachus

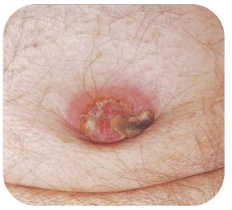


- A patent urachus can become a track through which urine can leak through umbilicus
- Rare in children, more common in adults with chronic retention
- The patient complains of a watery discharge from the umbilicus. (this discharge usually caused by umbilical infection, bladder fistula in which urine leak through the umbilicus is a complication)

Abdominal Wall, Umbilical & Other Hernias

Umbilical Diseases (Cont.)

Omphalitis

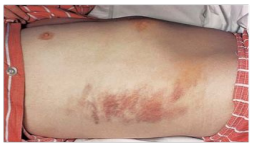


- Infection of the umbilicus (Dermatitis)
- True omphalitis is infection of the stump of the umbilical cord **following inadequate postnatal care and cleanliness.**

Predisposing Factors:

- **Common in adults**
- Risk factors include poor hygiene and sunken umbilicus caused by obesity
- The commonest cause of umbilical discharge. although, you need to exclude other causes.

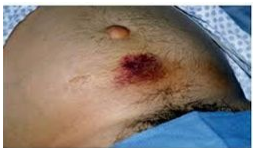
Discoloration of the umbilicus:



(Grey Turner's sign)

Clinical Features:

- The patient complains of umbilical discharge, pain and soreness.
- On examination, the skin within and around the umbilicus is red and tender (**cellulitis**) , and exuding a seropurulent discharge with a characteristic foul smell.
- **Blue Tinge (**around the umbilicus, caused by dilated, tortuous, sometimes visible veins, is called a **caput medusae)**
- **Yellow-blue bruising**
- Due to digested subcutaneous tissues following an attack of severe acute pancreatitis (E.g past history of gallstones) occur after few days of the acute attack.
- around the umbilicus **(Cullen's sign) =** tracked by falciform ligament to the umbilicus
- and in the flank **(Grey Turner's sign) =** tracked by retroperitoneal space
- **Bruising**
  - can also be associated with intra-abdominal bleeding, particularly when it is extraperitoneal.
  - Causes include ruptured ectopic pregnancy and accidental peri-uterine bleeding in pregnancy.



(Cullen's sign)

Secondary deposits

1- Ompholith (Umbilical stones)

- A stone form from accumulation of sebum with broken hair (or clothes fluff) and remain as a lump.
- **Risk factor is poor hygiene** "especially in those with deep and narrow umbilicus i.e obese"
- Patient present with pain, or as umbilical infection.

2- Sister Joseph's nodules

- These are A firm or hard nodule bulging into the umbilicus, underneath the skin or eroding through it, present in malignant cancer
- Primary malignant tumors are rare. Nearly all umbilical malignant tumors are metastatic through lymphatics from intra-abdominal cancer
- patient who is losing weight and looks unwell must attract your suspicion

causes of a discharge from the umbilicus

Acquired

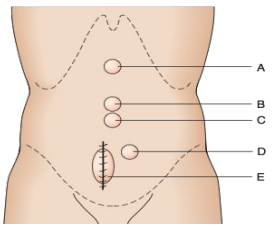
- Umbilical granuloma
- Dermatitis (intertrigo)
- Ompholith (umbilical concretion)
- Fistula (intestinal)
- Secondary carcinoma
- Endometriosis

Congenital

- Intestinal fistula
- Patent urachus
- Umbilical adenoma

imp: examples of cases from the Dr

- you have 55 y.o male came with groin swelling that he had since the last year, on and off. What's your best diagnostic tool?
  - Most hernias are diagnosed clinically, You need to check reducibility and cough impulse to check the complications.
  - If the diagnosis was hard (As in sportsman's hernia, pain w/o a visible mass) you can use MRI or CT
- pt came with nausea, vomiting and abdominal distension, what's the Dx? "they may add picture"
  - Depending on the picture :) but generally abdominal distension is caused by intestinal **obstruction**, if the hernia was red or blue colored then it is **strangulation**
- they may show picture with A B C label of bowel ischemia and ask you how you are gonna treat it? which type of hernia caused this?
  - I suppose the picture will come like this



Jaundice

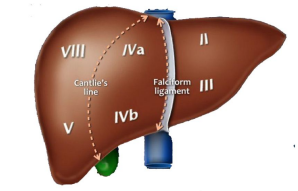
Definition:

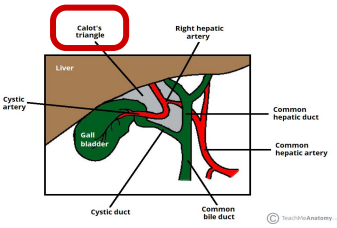
- **Jaundice** is the Yellowish discoloration of skin, mucous membranes, sclera first to be seen in the sclera, because it's white thus any change can be seen early and body fluids (in advance cases) due to hyperbilirubinemia Usually clinically apparent when **serum level of bilirubin (Dr: >** 40-**50 mmol/l) (Davidson: >50 µmol/l)**
- **500-1000 ml of bile is produced daily**
- **Charcot's triad** in ascending cholangitis consists of; **RUQ Pain**, **fever**, and **jaundice**. Highly sensitive symptoms that could even diagnose w/o imaging (start treatment right away).
- Charcot's triad + leukocytosis = Surgical emergency that need ERCP.
- Charcot's triad + hypotension and confusion = End organ damage (failure), the sepsis is getting worse (**Reynolds pentad**)

Surgical Anatomy of the biliary system:

- Gallbladder lies beneath segments IV and V of the liver

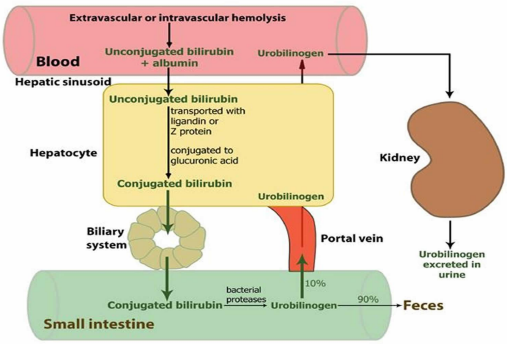
- Triangle of **C**alot boundaries (hepatobiliary triangle) **3Cs**:
  - Lateral: **C**ystic duct
  - Medial: **C**ommon hepatic duct
  - Superior: **C**ystic Artery, Liver





Excretion of bilirubin:

Bilirubin comes out in the form of **unconjugated bilirubin**, and it's not water soluble so it binds to albumin to be able to get transported to the blood gets picked up by the liver and gets conjugated **via glucuronyl transferase** to **glucuronic acid and becomes water soluble (conjugated bilirubin)**. Once it gets secreted to the GI tract it helps with the digestion of fatty food, it turns into **urobilinogen** (by bacterial proteases) which most of it 90% gets excreted in the feces. Some of it 10% gets reabsorbed in the portal venous circulation, goes back to the liver, then gets excreted by the urine



Jaundice

Classification:

Very helpful to the point that you don't even need to look at this page until the exam:)

Pre-hepatic jaundice:

- The liver conjugation is NOT compromised
- The liver excretion is not affected
- The capacity of the liver is overwhelmed The liver has a limited number of receptors when they are all occupied the rest will circulate freely
- Total bilirubin increased and UNCONJUGATED (Indirect) (Fat soluble)
  - also increased in:
    - **Hemolytic Anemia** E.g. sickle cell anemia, G6PD deficiency, thalassemia, hemophilia, spherocytosis, malaria, drugs (Rifampin) , sepsis
    - Transfusion reaction. specifically Hemolytic reaction
    - Hematoma secondary to trauma or surgery.
- Total bilirubin (>50%unconjugated) → prehepatic cause

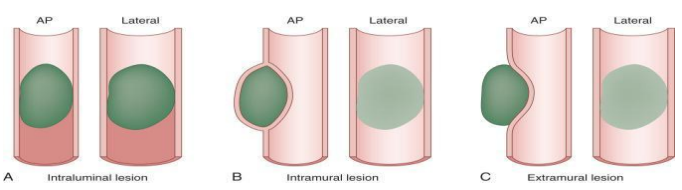
Hepatic jaundice:

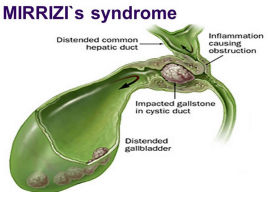
- Liver dysfunctional metabolism leads to increased bilirubin level and failure to remove bilirubin, may be due to storage problems, intrahepatic obstruction, or extrinsic insults.
- Results in Mixture of conjugated and unconjugated bilirubin.
  - **Infectious hepatitis**
    - Viral hepatitis (A-B-C) or parasitic increase unconjugated mostly
  - **Cirrhosis:** the most common cause of hepatic jaundice.
    - End stage liver disease that cause intrahepatic biliary duct obstruction e.g. PBC (affects bile ducts in the liver with stricture & fibrosis not hepatocytes). Not considered extrahepatic as it's still inside the liver. Anything beyond this area is extrahepatic. causing conjugated hyperbilirubinemia
    - Decompensated ones regardless of etiology
  - **Alcohol**
  - **Drugs:** paracetamol toxicity
  - Other causes: shock, liver cysts, Crigler-Najjar syndrome (Absent UDP-glucuronosyltransferase) and Gilbert syndrome (Low UDP- glucuronosyltransferase activity) (Mostly Unconjugated) and liver abscess.

Post-hepatic jaundice:

Obstructive jaundice gives dark urine and pale stools

- The two most common causes of obstructive jaundice are gallstones and cancer of the head of the pancreas.
- It is due to obstruction of the bile duct (extrahepatic ducts) after liver secretion. **Direct** hyperbilirubinemia. (Water soluble).
- Depending on the location of the duct obstruction can be classified into:
  - **Intraluminal (Intrinsic):** inside the **lumen** of the duct (most commonly gallstones) Caused by bilirubin stones or sludge (pre-stones stage, more friable) and polyps (Gallbladder polyps – if > 1 cm, need to worry about malignancy).Polyps in patients > 60 years more likely malignant. Tx: cholecystectomy.
  - **Intramural:** Inside the **wall** of the duct, Mass (cholangiocarcinoma, Ampullary carcinoma), Fibrosis, Benign Biliary Strictures (Congenital, Traumatic, Ischemic, Inflammatory e.g.Primary sclerosing cholangitis and infection of schistosoma) and choledcocal cyst (intramural and intraluminal)
  - **Extramural:** Outside the duct (extrinsic). Caused by lymph node metastasis, gastric, duodenal, hepatic, and pancreatic cancer and pancreatitis. Investigated by MRI and CT
  - Painless jaundice is cancer until proven otherwise.
  - Mirizzi syndrome: is defined as common hepatic duct obstruction caused by extrinsic compression from an impacted stone in the cystic duct. The bile duct is normal won't be dilated distally.





Jaundice

Difference between types of jaundice:

	Hemolytic (Prehepatic)	Hepatic	Obstructive (Posthepatic)
Age	Young	Young /middle age	Older age group
Abdominal pain (RUQ)	No	+/-	+
Color of urine	Normal	Yellow	Dark yellow
Color of stool	Normal	Normal	Clay (pale) colour Very specific
Pruritus [1]	--	--	+ Characteristic
Icterus	Lemon yellow	Yellow	Greenish / dark yellow
Liver	--	+	+
Gallbladder	--	--	+
Serum bilirubin Partial obstruction: (might) result in mild elevation of LFTs with normal bilirubin	4-5 mg/dl (indirect)	Up to 10-12 mg/dl (indirect/direct)	15-20 mg/dl (50% direct)
SGOT (AST)/SGPT (ALT)	Elevated	Markedly elevated	Normal / elevated
Alkaline phosphatase	Normal	Normal / elevated	Elevated Very specific
Serum proteins	Normal	Decrease	Normal

- ALT-AST-ALP-GGT are not actually functional so it's a misnomer, the only functional part is **bilirubin, coagulation factors, and albumin.**

Investigations:

- CBC, LFTs, coagulation profile, INR, hepatitis panel, serum protein and creatinine.
- **Ultrasonography**; best initial imaging test
- **MRCP**; Most accurate investigation but not therapeutic
- **ERCP**; diagnostic and therapeutic; insert stent (plastic and metal) when there is thickening , brushing for cytology (FOR CANCER)
    - complications: Bleeding, **Pancreatitis**, Infection and Inflammation.

Intraluminal cause:

Obstructive Jaundice

Gallstones (Cholelithiasis):

Definition	• Cholelithiasis: A stone in the gallbladder. • Presented as RUQ pain radiating to right shoulder. • Stones found in the gallbladder never causes obstructive jaundice unless ★ Stones pass through the common bile duct, At the level of the ampulla of vater → pancreatitis along with obstructive jaundice. ○ 80% of pancreatitis is caused by biliary stone ○ Gallstones is the most common cause of acute pancreatitis
Risk factors	• Age is important. 4 F's (all increase the risk of gallstones) ○ 40'S ○ Female ○ Fat (diet) ○ Fertile Meanwhile if the patient is 75 with obstructive jaundice then most likely it's cancer
Diagnosis	• **Laboratory tests:** CBC and LFTs are normal, elevated serum conjugated bilirubin. • **Ultrasound:** reveals gallstones with acoustic shadowing.
Treatment	Only in high risk group patient otherwise no need

Acute calculous cholecystitis:

Definition	Acute calculous cholecystitis: acute inflammation of the gallbladder, which is typically due to cystic duct obstruction by a gallstone. Presents with **+ve Murphy's sign** and signs of inflammation (**Fever** and **leukocytosis**) and **RUQ pain.**
Diagnosis	• **Laboratory tests:** CBC shows leukocytosis,↑CRP, normal level of bilirubin (no jaundice because biliary tract still intact). • **Ultrasound:** shows distended gallbladder with thickened wall and pericholecystic fluid. • **CT to confirm**

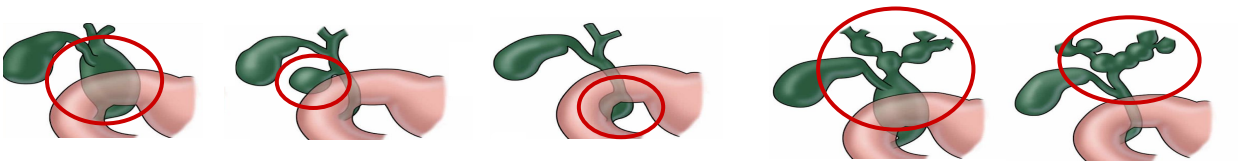
Cholangitis (surgical management):

Clinical features	★ **Charcot triad (Fever, jaundice and RUQ pain)** seen in less than 50% of the patients. (must give prophylaxis antibiotics) • Reynolds Pentad (Fever, jaundice, RUQ pain, Hypotension and Altered mental status) seen in toxic cholangitis with septic shock.
Diagnosis	• Diagnosed based on systemic signs of inflammation (fever, leukocytosis, ↑ CRP) in combination with signs of cholestasis (jaundice, ↑GGT, ↑ALP) and/or characteristic imaging (RUQ Ultrasound) findings such as dilated CBD
Management	• Cholangitis is an **acute surgical emergency** managed with ERCP to remove the stone.

Obstructive Jaundice

Intramural causes:

Fibrosis	
Definition	Fibrosis in the wall that causes obstruction and can transform to cholangiocarcinoma.
Etiology	• Can happen due to extrinsic compression, stones, previous infection/multiple cholangitis, iatrogenic, multiple ERCP, or primary biliary cholangitis (PBC) and primary sclerosing cholangitis (PSC) • Reaction to chronic pancreatitis that can cause obstruction in 2 forms: 1- Extrinsic 2- Secondary to reactive wall scarring and fibrosis
Investigation	• MRCP, ERCP, and Choledocalscopes to inspect the mucosa. • In any intramural thickening we assume it's cancer until proven otherwise as well as painless jaundice (esp. elderly)

Choledocal cyst	
Definition	• Congenital malformation with abnormal bile duct wall leading to accumulation of bile, obstruction, and jaundice. In choledochal cysts the common bile duct is dilated like the gallbladder → stasis → stones • Types 1-4: can present with extra hepatic jaundice. • Type 5: Intrahepatic cystic dilation, don't cause extra hepatic jaundice
Types	 **Type I**: complete fusiform dilation of the bile duct **Type II:** cystic dilation **Type III:** intrapancreatic portion dilation **Type IVA:** both bile duct and hepatic ducts dilated Type IVB: limited to extrahepatic duct. **Type V:** intrahepatic only
Management	• **Type I**: Remove all the bile duct, connect hepatic duct to jejunum • **Type II**: ;secondary gallbladder that can be removed without bile duct. • **Type III**: ERCP and open it. • **Type 4A and 5:** Need transplant due to intrahepatic biliary duct dilation.

Extramural cause:

- Metastasis to lymph nodes of porta hepatis, gastric cancer, duodenal cancer, pancreatic cancer, chronic pancreatitis
- External compression caused by a mass in any of the adjacent organs (duodenal cancer, pancreatic cancer..etc)
- Obstruction by impinging bile duct while in situ (in gallbladder not bile duct) May form extramural fistula, heals in pouch and obstructs bile duct.
- Mirizzi syndrome: CBD Obstruction and jaundice due to stone in gallbladder.
- Confirmed by CT or MRI

Obstructive Jaundice

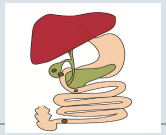
Stone

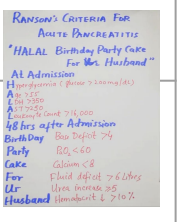
- Acute, sudden, healthy young patient, Painful (severe symptoms but not for long time , and on/off pain after meals)
- most common cause of post-hepatic jaundice is gallstones but if the patient is elderly we should consider malignancy
- obstructive jaundice caused by stone → shrink gallbladder

Mass

- Chronic, gradual, most common in elderly, loss of weight and appetite, night sweat, Painless so the patient presents late
- E.g. pancreatic head and ampullary masses and cholangiocarcinoma
- Obstructive jaundice caused by malignancy → distended (palpable) gallbladder

Complication of obstruction:

Gallstones ileus	
Definition	Mechanical bowel obstruction due to obstructive gallstones (rare complication of gallstone)
Pathophysiology 	Gallbladder perforation or Mirizzi syndrome → biliary-enteric fistula formation (most commonly cholecystoenteric fistula) between the inflamed gallbladder and bowel → gallstones passing down into bowel lumen **Sites of obstruction :** Terminal ileum
Clinical features	Features of mechanical bowel obstruction (abdominal pain and distention, nausea, vomiting) with history of gallstone

Acute pancreatitis	
Definition	Inflammatory condition of the pancreas most commonly caused by gallstones and alcohol use
Clinical features	Typical manifestation include sudden, severe epigastric pain that radiates to the back, fever, jaundice (rare). • **Splenic vein thrombosis** (Isolated) is the most common vascular complication of acute pancreatitis.
Investigation	Elevation of serum lipase or amylase **3 times higher than normal** 
Treatment	This condition should be managed as a **medical emergency** as it is a potentially fatal condition. Initiate fluid resuscitation as soon as this diagnosis is suspected

Obstructive Jaundice

What if the patient had previous bariatric surgery and developed cholangitis?

- The last option is bile duct exploration (laparoscopic surgery)
- Less two invasive solutions:

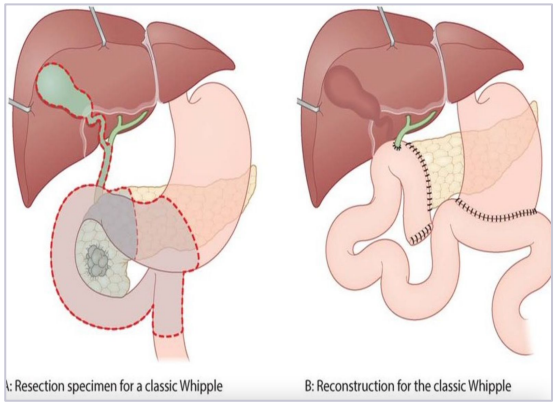
1. The least invasive: Through the **liver**, by
   a. antegrade percutaneous transhepatic cholangiogram (**PTC**)
   b. **Rendezvous procedure** where we go through PTC + ERCP
2. Laparoscopic with gastroscope still the original route

Cholangiocarcinoma (surgical management):

★ **Risk factors include:** Primary sclerosing cholangitis, Hepatitis C, Parasitic biliary infestation, and choledocal cyst.

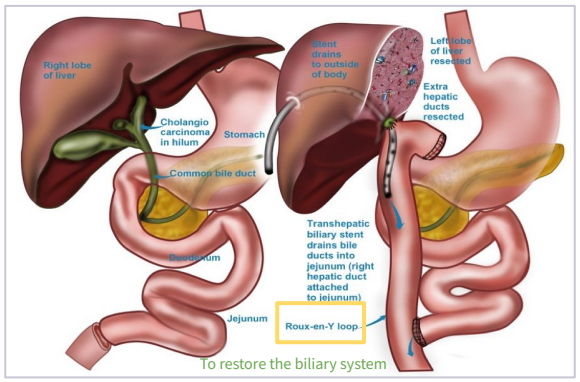
★ **Clinical features:** Abdominal pain, Anorexia, weight loss, Jaundice ,dark urine and pale stool, pruritus, and in patients with distal mass it presents with palpable gallbladder (Courvoisier's law)

Middle to Distal (Intrapancreatic portion) Bile duct cancer: → Whipple procedure



- **Why remove all of this?**
  - they share the same blood supply
  - they share the same lymph nodes
  - close proximity to each other, so there's a risk of direct invasion
  - Risk of ischemia to pancreas and duodenum.
- **Complications of Whipple procedure?**
  - Leakage from pancreas, Leakage from stomach, Leakage from bile duct

Middle - proximal bile duct cancer



- Middle - proximal bile duct cancer will spread to the liver either to the left or right side thus hepatectomy of the side of the involved duct, bile duct and gallbladder removal. For example, If we know left is positive for cancer we do left hepatectomy, bile duct resection, cystectomy and then roux-en-Y hepaticojejunostomy

Perforation and infarction of viscus (etiology, clinical features and complications)

Acute Abdomen:

Is any abdominal condition (With main symptoms being the pain) that mandate emergent intervention

1- Medical Acute abdomen

2- Surgical Acute abdomen, **such as:**

- Inflammatory (Acute pancreatitis, cholecystitis)
- Obstruction (Intestinal, biliary and ureteric)
- **Perforation (highest mortality) especially the colon**
- **Ischemia**
- Intra Abdominal bleeding (Ruptured aneurysm, ruptured ectopic pregnancy)

Types of pain

	Visceral (Autonomic)	Somatic
General info	Poorly localized ❖ From Viscera ❖ Supplied by **autonomic** fibers	Localized, presented with guarding and rigidity From body surfaces e.g abdominal wall ❖ Supplied by **somatic** fibers
Cause	Stretch/distention → act as receptor of pain Ischemia → w/ forceful contraction the vessels get compressed	Cutting Burning
Nature	Colicky (fluctuates in relation to contraction and relaxation)	Continuous
Intensity	Mild to moderate	Severe
Localization	Generalized Poorly localized	Well localized
Radiation	Radiates and Refer	Does not refer
peritonitis phases	**Phase 1** **(tenderness ONLY)**	**Phase 2** **(Rigidity & Rebound tenderness)**

Referred Pain

- **Definition:**
  - Defined as pain perceived at a location away from the site of the painful stimulus
- **Examples:**
  - Biliary colic → Right shoulder pain
  - Ureteric colic → Upper thigh
  - Cardiac pain → Left jaw
  - **Ice cream headaches**
- **Cause:**
  - Multiple inputs from sensory fibers

Pain Examples:

1- Appendicitis:

with time the pain pattern can change:

First 4-6 hours (phase 1) tenderness

Nerve impulses will go from the appendix to T10 (which also receives impulses from paraumbilical area), there will be REFERRED pain around the umbilicus initially.

After 4-6 hours (phase 2) rebound

Abdominal wall inflammation → pain shift into the right iliac fossa (somatic overrides autonomic, so it becomes more severe and localized)

2- **Gallbladder :**

Pain in the right hypochondria. Can extend from the midline along the right side and radiate to the tip of the scapula or the right shoulder (referred biliary colic)

3-**Pancreas:**

Start in the upper epigastrium radiates to the back, gradual and progressive.

Perforation and infarction of viscus
(etiology, clinical features and complications)

Perforation and Infarction

Etiology

1 Perforation:

1. Inflammation of wall
2. Ulceration of wall
   - PUD
   - Duodenal ulcer
3. Ischemia
4. Infarction & Gangrene of wall
5. Luminal Obstruction
6. Benign or Malignant tumor of wall
7. Trauma (RTA)
8. Post operative leak
9. Instrumental trauma

2 Infarction:

1. Arterial or Venous occlusion
2. Internal or external Strangulated Hernia most common (paraumbilical)
3. Obstruction of lumen
4. Benign or Malignant tumor of wall
5. Postoperative
6. Prolonged hypotension
7. Aggressive Inotropic support
8. Chemotherapy
9. Medical Disease
10. Trauma (Injury to the mesentery → Ischemia)

3 Clinical Causes:

1. Perforated Appendix
   Most common cause due to appendicitis
2. Strangulated hernia
3. Trauma
4. Anastomotic leak post-surgical
5. Perforated Diverticulitis
6. Bowel Ischemia
7. **Primary Peritonitis** → Spontaneous bacterial peritonitis **(SBP)** may occur in patients with **nephrotic syndrome,** but is more frequently seen in those with liver cirrhosis or chronic renal failure (particularly in patients on peritoneal dialysis).

Vascular Compromise
Decrease blood supply (Arterial/venous) e.g. PUH

Luminal obstruction
- If the lumen is obstructed→obstructed hernia
- If the blood vessels are obstructed → strangulated hernia

Wall infiltration
- Growth or mass causes luminal obstruction e.g. colon cancer
- Inflammation In the wall e.g. colitis which will make it thick but weak leading to perforation (crohn's)

Mechanism

Infarction: Depend on the size of the blood vessel
Short segment infarction, Segmental infarction and Massive infarction

Perforation:

Micro perforation
- Peritonitis → CT showing gas in the abdomen → no perforation found on surgery. We close the abdomen and watch the patient for improvement.

Sealed/concealed perforation
- Perforation sealed by the omentum. Initially fluid will go into the peritoneal cavity but later on it will stop.

Contained perforation
- Around the perforation, coils of the intestine separate the area (enclosed area).

Free peroration
- full blown peritonitis

Peritoneal Irritation
Normally: there is sterile peritoneal fluid in the peritoneum.
Irritation is caused by abnormal contents in the peritoneum, like:
- Blood (trauma) , Bile, Pus and Urine
- **Stool** contains highly virulent microorganisms e.g. clostridium spp and bacteroides causing severe infection and high grade peritonitis.
- **Gastric juice** (HCL → severe chemical burn of peritoneum → peritonitis). MOST LETHAL
- **Pancreatic Juice** pancreatic enzymes → digestion of any tissue in contact.
- **Chyme** partially digested food originating from the small intestine.

Peritonitis
Two Phase: Compensatory Mech Symptoms & Signs

Function of the Peritoneal fluids? lubrication.
Normally it's sterile. When the intestines perforates, its content will leak into the peritoneal cavity.
Collection of foreign material → peritoneal irritation → peritonitis.

Perforation and infarction of viscus (etiology, clinical features and complications)

Peritonitis

Phases of Peritonitis

Phase 1	Phase 2
Visceral peritonitis → Inflammation limited to the organ • **Tenderness ONLY (Autonomic) - Medical diseases**	Parietal peritonitis →Inflammation of parities of the abdominal wall • **Rebound tenderness (somatic) - Surgical diseases**

1. Peritonitis presents initially in Phase 1 with visceral symptoms and progresses to phase 2 gradually and both have different clinical picture.
2. Time interval between these phases differs,
    a. for the appendix it's 4 hrs, for gallbladder 10-14 hrs. But that doesn't mean the first will stop and second will start, they happen simultaneously.
3. You can only tell what is the inflammation in the second phase and where is it localized

Compensatory Mechanism

Mechanism	Purpose	Clinical Manifestation
Inflammation	As part of the immune response	1- Fever 2- Activation of other mechanisms
Pylorospasm Contraction of pyloric sphincter → gastric content accumulate → stomach distention → feel full	NPO	1- Anorexia 2- Nausea 3- Vomiting [1]
Paralytic Ileus [2] (**Intestinal paralysis** leading to losing intestinal tone)	Rest to abdominal organs	1- Abdominal distension 2- Decreased or absent bowel sounds 3- Constipation
CNS activation (Plasma volume decrease due to loss of fluid → blood shrinks) May **not** happen if **mild**	To support insult to cardiovascular system e.g. in **severe** perforated duodenum	1- Tachycardia (Help in assessing the extent of the inflammation) 2- Hypotension

Perforation and infarction of viscus
(etiology, clinical features and complications)

Peritonitis

Clinical Features

Symptoms

- **Abdominal Pain**
  - Due to inflammation
- **Anorexia | Nausea | Vomiting**
  - Due to pyloric spasm
- **Decreased urine output**
  - Due to CVS activation and ↓ plasma volume
- **Obstipation | Constipation**
  - Due to paralytic ileus
- **Fever | Tachycardia**
  - Due to inflammation and CVS activation

Signs

- **Abdominal Tenderness (PHASE ONE)**
- **Distension**
- **Rebound tenderness (PHASE TWO)**
  - The patient must be conscious
  - MCQ! Somatic nerves damage (paralytic – cerebral palsy) ⟶ loss of rebound tenderness
  - Under general anesthesia ⟶ no muscle tone ⟶ no rebound tenderness
  - **IT'S A TEST TO DIFFERENTIATE MEDICAL FROM SURGICAL CAUSES OF ABDOMINAL PAIN**
- **Guarding | Rigidity (PHASE TWO)**
- **Decreased bowel sounds** Due to paralytic ileus (other Ddx: post op, neurological, low Na)
- **DRE** Dilated rectum

Complication

Local

- **Local Peritonitis**
- **Collection** of remnant fluid (sterile , purulent)
- **Abscess formation**
- **Fistula formation**
- **Stricture formation**
- **Adhesion**

General

- **Generalized Peritonitis**
- **Septic shock** If not treated within **4-6 hrs** the pt will develop DIC
- **ARDS**
- **Multi Organ Failure**
- **Mortality (Death)**

Golden notes found in 438 team

Acute Diverticulitis

★ **Sigmoid colon** is the most common site

★ **Hinchey Classification or grading: (A-D or 1-4)**

- Grade 1- Contained **small** abscess
- Grade 2- One **large** abscess extending in the abdomen
- Grade 3- **Multiple abscess** with small air leak
- Grade 4- Fecal **peritonitis**

★ **Complication** ⟶ Rupture of a pericolic abscess ⇝ purulent peritonitis , fistula formation (To bladder)

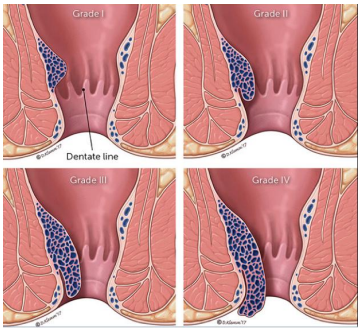
★ **Treatment** ⟶ **Grade 1 and 2** can be managed nonoperatively , **For Grade 3 and 4** require surgery (**Hartmann's surgery**)

Upper GI bleeding (this summary only include the objectives)				
	Etiology	**Pathogenesis**	**Clinical feature**	**Complications**
Peptic ulcer	• Helicobacter pylori • NSAIDs • Stress • Smoking • Increased acid secretion (e.g. Zollinger-Ellison Syndrome) • Genetic factors	-	★ **Acute** • Recurrent well-localised epigastric pain. • Heartburn, Anorexia, Waterbrash • Intolerance of certain foods • Intermittent vomiting may occur **Chronic** • Less severe and more intermittent pain that may radiate into either the back or the right hypochondrium • Night pain • Chronic blood loss	• The most significant hemorrhage occurs when duodenal or gastric ulcers penetrate into branches of the **gastroduodenal artery (GDA).**
Gastroesophageal varices **How to treat Variceal bleeding in sequence? (possible MCQ)** ABC → PPI → octreotide, non selective BB, antibiotics → endoscopy. **Rebleeding?** Consider TIPS and surgery	• Related to infection or alcohol history. Caused by liver fibrosis/cirrhosis and has clinical signs of liver disease such as jaundice. • The most common cause of portal hypertension is cirrhosis. • **Most common in the distal lower third of esophagus.**	• When portal vein pressure increase, veins will develop collaterals "Shunts" which will cause the Varices, Caput medusa & Hemorrhoids depending on the site of shunts. • Most commonly the result of bleeding from varices veins. As they enlarge, the overlying mucosa becomes increasingly tenuous, excoriating with minimal trauma.	• Acute upper gastrointestinal bleeding are examined for evidence of chronic liver disease. • The key investigation during an episode of **active bleeding is endoscopy**.	• Variceal hemorrhage is associated with an increased risk of rebleeding, increased need for transfusions, longer hospital stays, and increased mortality • 6-week mortality rate after the first bleeding episode is almost 20%.
Gastroduodenal Tumors (Neoplasms) Curative surgery requires more radical resection If not resectable we administer tyrosine kinase inhibitor like Imatinib (Gleevec)	• Neoplasms such as gastrointestinal stromal tumors (GIST) and adenocarcinomas rarely cause UGIB. • Most characteristic of the **GI stromal tumor (GIST)**	• it may occur with other tumors, including adenocarcinoma and lymphomas particularly after initiation of chemotherapy	• Melena, if massive bleeding then it is severe. +ve occult blood. • Associated with chronic anemia (Microcytic Hypochromic anemia) or hemoccult-positive stool (blood that is invisible to the naked eye, sensitive but not specific).	-
Portal hypertensive gastropathy	• Condition caused by cirrhosis (leading to portal hypertension) or portal vein thrombosis..	• A diffuse dilation of the mucosal and submucosal venous plexus with overlying gastritis and may exude blood	• Rarely causes major bleeding even in the absence of well-developed visible varices	• Can cause gastrointestinal ulcers or diffuse lower gastrointestinal bleeding.
Dieulafoy's lesion	• Not understood	• Dieulafoy's lesions are vascular **arterial** malformations characterized by **abnormally large diameter submucosal arterioles** which do not decrease in size as they approach the mucosa, if it gets irritated by acidity of the stomach it ruptures causing a massive bleeding.	• High pressure arterial bleeding. • Healthy, then sudden massive fresh bleeding.	• Shock status that responds to resuscitation (need massive blood transfusion)
Mallory Weiss syndrome	★ Classically, these lesions develop in alcoholic patients after a period of intense retching and vomiting. (Retching = forceful projectile vomiting due to strong contraction of the diaphragm)	• History of heavy drinking (alcoholics), also happens in pregnant women and cancer patients on chemo. ★ Longitudinal **Mucosal and submucosal tears that occur near the gastroesophageal junction**. (Not full thickness tear. Full thickness tear is classified as perforation)	• 90% of bleeding episodes are self-limited. • Acute presentation	• Hypovolemic shock if severe
Boerhaave Syndrome	• Intake of large amounts of **alcohol** or food in the recent past. • Repeated episodes of **vomiting** • Prolonged coughing • Childbirth • Seizures • Weightlifting	• Severe vomiting/increased intrathoracic pressure → rupture of **all layers of the esophageal wall (transmural perforation)** • In > 90% of cases, the rupture occurs in the distal third of the esophagus on the left dorsolateral wall surface. resistance.	• Severe retrosternal chest pain and mediastinal or subcutaneous emphysema. • Mackler's Triad: mid-epigastric or lower chest pain that often radiates to the back, vomiting and subcutaneous emphysema. mediastinal crunch (Hamman's sign) heard with stethoscope • Pleural pain worsen by neck flexion and swallowing.	• Septic shock very rapidly

Upper GI bleeding (this summary only include the objectives)				
	Etiology	**Pathogenesis**	**Clinical feature**	**Complications**
Hemobilia	● Liver biopsy ● Post-cholecystectomy ● Biliary instrumentation ● Injury due to ERCP ● Hepatocellular carcinoma ● Trauma ● Cholangiocarcinoma ● Coagulopathies in elderly post simple procedures.	-	● Jaundice, Right upper quadrant pain and GI bleeding.	-
Aortoenteric Fistula	● Primary: ○ Erosion of the aneurysm into the adjacent bowel. ○ Infection ○ Neoplasm (most commonly pancreatic cancer) ○ Radiation therapy ● Secondary: ○ Previous endovascular stent or graft aneurysm repair. (Most common cause)	-	● Associated with bacteremia and sepsis.	-
Esophagitis	● Severe GERD ● Irritating agents (drugs, radiation) ● Candidiasis	● Esophagitis is the inflammation of the esophageal mucosa secondary to direct mucosal injury ● It can also occur secondary to local infection	● Heartburn, which is a severe burning discomfort felt in the centre of the chest behind the heart ● Bitter taste developing in the mouth	-
Gastric antral vascular ectasia (GAVE)	-	● Dilation of small blood vessel in the antrum of the stomach. ● Common causes include collagen vascular disease (especially systemic sclerosis) and portal hypertension. The dilated vessels appear on endoscopy as long streaks resembling those on the surface of a watermelon.	● Most patients present with persistent iron deficiency anemia due to continued occult blood loss as a result of chronic bleeding from these vessels.	● Rarely causes acute severe bleeding
Iatrogenic bleeding	● This bleeding may follow a therapeutic or diagnostic procedure such as: ○ Percutaneous transhepatic procedures ○ Endoscopic sphincterotomy ○ Percutaneous endoscopic gastrostomy (PEG) placement	● perforation by a scope or ERCP.	-	-
Pancreatitis	-	● Due to the erosion of the pancreatic pseudocyst into the blood vessels (most commonly a splenic artery pseudoaneurysm) or pancreatic pseudocyst rupture.	-	● Can cause hemorrhage

Intra GI bleeding		
	Etiology	**Clinical feature**
Ruptured visceral aneurysm (splenic artery aneurysm)	● This may occur primarily as a complication of atherosclerosis in elderly patients where the calcified wall of the aneurysm may be visible on x-ray or secondary to intra-abdominal sepsis and acute or chronic pancreatitis. ● Rupture occurs more commonly during pregnancy (3rd trimester) or at labor.	● It ruptures into the peritoneal cavity with similar symptoms to those of splenic rupture: ◦ Abdominal pain ◦ Distention ◦ Drowsiness ◦ Pain in the tip of the left shoulder ◦ Hypovolemic shock.
Ruptured visceral aneurysm (hepatic artery aneurysm)	● **Intrahepatic (20%)** ◦ In the past, infections associated with IVDU were the most common cause. Nowadays, the majority are due to trauma or interventional biliary and hepatic procedures. ● **Extrahepatic (80%)** ◦ Due to degenerative and atherosclerotic changes ◦ Aneurysms due to liver transplant are usually extrahepatic and associated with infections.	● **Asymptomatic** (Found incidentally). ● If **symptomatic**, it usually presents with RUQ or Epigastric pain. Jaundice can also occur if the aneurysm compresses the bile duct. ● **Rupture**: Patients present with abdominal pain and shock. ◦ **Intrahepatic:** Rupture into biliary tree causing **Quincke triad** (Abdominal pain, Hemobilia, Obstructive jaundice). ◦ **Extrahepatic:** Intraperitoneal rupture.
Ruptured visceral aneurysm (mesenteric artery aneurysm)	● The primary etiology now is atherosclerotic. ● When excluding pseudoaneurysms, infection is currently an etiologic factor in less than 5% of cases. ● Medial degeneration, also seen in splenic and hepatic aneurysms, often with secondary atherosclerosis, accounts for 25% of SMA aneurysms. ● Other reported causes are inflammatory processes in the abdomen or retroperitoneum (cholecystitis, pancreatitis) and trauma.	● Most patients (90%) are symptomatic with abdominal pain and a pulsatile mass, may be notably mobile, differentiating it from an abdominal aortic aneurysm. ● Frank intraperitoneal hemorrhage ● Symptoms and signs of mesenteric ischemia. ● 50% of patients present with rupture, and the mortality rate is 30%.
Retroperitoneal hemorrhage (over anticoagulation)	● Retroperitoneal bleeding has been shown in patients on systemic anticoagulation with warfarin, unfractionated heparin, or low-molecular-weight heparin.	● Pain in the lower abdominal, flank, or inguinal area with radiation to the thigh and lumbar region. ● Hypotension ● Nerve-compression effects characterized by motor or sensory deficits in the groin and thigh may be additional suggestive signs.
Ruptured Abdominal Aortic Aneurysm (AAA)	● Smokers and may have a family history of atherosclerotic aneurysms.	● It's a surgical emergency; it should be suspected in a patient with the triad of severe abdominal or back pain, hypotension and a pulsatile abdominal mass. ● Pain begins in the centre of the abdomen but commonly radiates to the back and may radiate to the groin along the course of the genitofemoral nerve. ● **Cullen's sign** and **Grey Turner's sign** – bruising around the umbilicus and in the flank respectively are late (3–4 days) indicators of a long standing rupture. ● diminished bowel sounds caused by intraperitoneal blood.
Ruptured spleen	● The spleen may be ruptured by blunt or penetrating injuries. ● Delayed presentation, due to an unusual mechanism (e.g., postcolonoscopy).	● If the ribs are broken, there will be local pain, tenderness and sharp pain on inspiration. ● Splenic haemorrhage usually causes pain in the left hypochondrium and upper abdomen. It may be associated with left shoulder-tip pain ● Shifting dullness and flank dullness may be detected. ● Spleen is palpable in the right hypochondrium.
Ruptured hepatocellular carcinoma	● Patients have preexisting cirrhosis and many others have evidence of hepatitis B or C infection. ● 'aflatoxin' (derived from the fungus, Aspergillus flavus, which contaminates maize and nuts) is an important hepatocarcinogen.	● Progression of the existing liver disease symptoms. ● Abdominal pain and distention. ● Hepatosplenomegaly or palpable abdominal mass. ● Loss of appetite and weight loss, Fever. ● Spontaneous rupture with intraperitoneal hemorrhage.
Ruptured Ectopic Pregnancy	A fertilised ovum implants at an abnormal site; usually the fallopian tube. The erosive trophoblast may penetrate the wall of the tube, and often ruptures after about 6 weeks.	● The patient experiences bouts of cramping iliac fossa pain that may be associated with fainting and vaginal bleeding. ● Rupture produces sudden severe pain, bleeding and circulatory collapse, with the abdominal pain often becoming generalised.
Ruptured Ovarian Cyst	Benign ovarian cysts are a common cause of torsion, rupture and bleeding.	● Patients may collapse from the associated hypovolaemic shock or present with sudden and severe lower abdominal pain and then develop the signs of internal bleeding.
Ruptured Liver Adenoma	Liver cell adenoma can develop in young women taking the contraceptive pill containing high levels of estrogen and may rupture spontaneously.	● They may be asymptomatic but generally present with right hypochondrial pain as a result of haemorrhage within the tumor. ● Superficial tumors may bleed spontaneously and present with symptoms of hemoperitoneum. ● Hypovolemic collapse is common.

Lower Gastrointestinal Bleeding

	Characteristics	Diagnosis & Management	Complications
Diverticular Disease	• **Most common cause of significant lower GI bleeding** • Right-sided diverticular disease is responsible for more than 50% of the bleeding. • Etiology: low fiber & high fat diet, obesity & low physical activity • Usually asymptomatic, may manifest with abdominal discomfort or pain, especially if associated with chronic constipation • **Chronic constipation (most common risk factor)**→ ↑ pressure inside the lumen→ outpouching in the intestinal wall→ rupture→ bleeding	• Best **Initial**, **diagnostic** test and **treatment** is **Colonoscopy** • Mainly self limiting • If bleeding is massive or cannot be controlled by endoscopy treatment: Angioembolization or Surgery	• Diverticular bleeding • Acute inflammation; diverticulitis
Angiodysplasia **Both Diverticular disease and Angiodysplasia are in older ages (above 50 years because they require 10 years of constipation)**	• **Acquired** arteriovenous malformations, Secondary to progressive dilation of normal blood vessels within the submucosa of the intestine (5-10 years of chronic constipation, the blood vessel will stretch and dilate) • Rare disease, and if present rarely causes major bleeding • Almost uniformly found in patients > 50, 65 years. • Hemorrhage tends to arise from the right side of the colon, most common location is the cecum. • Manifests with episodic bleeding (hematochezia), fatigue, weakness, dizziness, shortness of breath, and, potentially, hematemesis • Associated with aortic stenosis and renal failure	• Colonoscopy or angiography can diagnose these lesions and treatment through. • Bleeding stops spontaneously in most cases	Anemia and/or life-threatening bleeding
Neoplasia Lower GI malignancies are common to bleed, unlike lower GI malignancies	• Polyps: mostly bleeds after polypectomy • Juvenile polyps: 2nd most common cause of spontaneous bleeding in patients < 20 years (1st most common is Meckel's diverticulum) • Colorectal carcinoma: uncommon cause of significant lower GIT bleeding but is the **most important diagnosis to rule out.** Slow, painless bleeding • Gastrointestinal stromal tumor: refer to upper GI bleeding	• Colonoscopy is the best diagnostic tool • If the polyp is the source of bleeding, it can be treated with by burning or clipping it (90 YO male presenting with loss of weight, constipation and blood per rectum, top differential? Cancer, but rule out hemorrhoids too)	
Internal hemorrhoids	• **Painless,** often accompanied by prolapsing tissue that reduces spontaneously or has to be manually reduced by the patient. • Presents with Low-volume bleeding, bright red blood per rectum seen in the toilet bowl and on the toilet paper 	• Best next step when finding an internal hemorrhoids is colonoscopy. • **Management is based on grading:** Grade 1: Conservative therapy with supplements, dietary & lifestyle changes (By decreasing the risk factors such as as losing weight, stopping smoking and treat straining and constipation by fibers or sitting in warm water with salt to relax the sphincter and release the pressure) Grade 2 & 3: rubber band ligation Grade 4: resection • If **constipation** is a feature, bulk laxatives or stool softeners may be indicated. (20 YO male presenting with hematochezia, top differential? Hemorrhoids, but rule out cancer even if it's less likely)	

Lower Gastrointestinal Bleeding

Causes of Colitis:

1. Inflammatory Bowel Disease

	Ulcerative colitis	Crohn's disease
Location	• Starts distally in the rectum (proctitis). • Progresses proximally to occasionally involve the entire colon.	• Can affect the entire GI tract. Most common site for crohn disease is the terminal ileum.
Pattern	• Superficial diffuse continuous ulcerative lesions. • Mucosal disease and sometimes submucosal (Not full thickness inflammation)	• Skip lesions • Transmural thickening • Granuloma formation
Bleeding	• **More likely** • Can present with up to 20 bloody bowel movements / day.	• Very rare • Positive fecal-occult blood • Not with bright red blood.
Associated Symptoms	• Crampy abdominal pain in LLQ • Tenesmus. • All extraintestinal manifestations are improved by control of colitis or colectomy except hepatobiliary manifestations (primary sclerosing cholangitis)	• Distinguishing Sx: occurrence of repeated episodes of diarrhea in the weeks before the attack. • Runs a chronic course: long Hx of colicky central/lower and pain coming on every 15-20 min associated with diarrhea.
Diagnosis	• Careful history • Flexible endoscopy with biopsy. • Colposcopy finding: superficial continuous ulcer	• Endoscopy • Biopsy • Contrast studies. MR or CT enterography

2. Infectious Causes:

Any GI infection can lead to bleeding so you treat them like any other infection based on the cause.

	C. difficile (Opportunistic)	Cytomegalovirus (CMV)
Risk factors	• **Prior antibiotic use** IV Abx • Hospitalization	• **Immunocompromised** patient

Symptoms	• Abdominal pain
	• Fever
	• Diarrhea:
	  ○ Explosive (≥ 3 stools per day)
	  ○ Foul-smelling
	  ○ May develop into toxic megacolon
		• Bloody Diarrhea

Bleeding	• Uncommon but can be present • Indicating severe mucosal slough	
Diagnosis	• History • Stool culture • Lower gastrointestinal endoscopy will show scattered inflammatory plaques between edematous mucosa (pseudomembranous colitis).	• History • Stool culture • Endoscopy with biopsy confirms the diagnosis.

Lower Gastrointestinal Bleeding

Causes of Colitis contd..

3. Radiation proctitis:

- **Proctitis:** An inflammation of the lining of the rectum caused by sexually transmitted infections (e.g., gonorrhea, genital herpes, candidiasis, chlamydia), radiation therapy, and inflammatory bowel disease.
- **Radiation proctitis** complication of radiation therapy.
- Patients with proctitis usually pass fresh blood or bloodstained mucus either mixed with stool or streaked onto the surface of normal or hard stool
- **Treatment:** Resection

4. Ischemia:

Ischemic Colitis (Colonic ischemia)	• Part of the colon with suboptimal blood supply, or atherosclerotic disease in elderly will lead to bleeding (It is a diagnosis of an older patient)
	• Sites of compromise/ ischemia: **Watershed areas** (These areas receive dual blood supply from the most distal branches of two large arteries (i.e., SMA and IMA)
	  ○ Splenic flexure
	  ○ Rectosigmoid colon
	  ○ Cecum
	• Symptoms: Bloody loose stool (60% of patients), Pain more diffuse
	• Diagnosis:
	  ○ **Colonoscopy:** Procedure of choice in mild to moderate cases of ischemic colitis. Findings include edema, and/or ulceration of mucosa
	  ○ **Angiography** is not indicated. If it is performed it is often normal.
	• Treatment: resection

Mesenteric ischemia	Risk factors:
	• Hypercoagulable states. E.g. malignancy, esp. Adenocarcinoma, autoimmune, pregnancy and trauma patients.
	• Vasculitis
	• Cardiovascular disease: Atrial fibrillation, Congestive heart failure, Acute myocardial infarction.
	• Recent abdominal vascular surgery
	• Medications: Vasopressors, Digoxin
	Classified into:
	• **Arterial insufficiency:**
	  ○ Occlusive: Embolic (most common cause), Thrombotic
	  ○ Nonocclusive: Low flow state (AMI / Shock)
	• **Venous insufficiency / venous thrombosis**
	Clinical features: Abdominal pain that is sudden, severe, diffused. No rebound tenderness, Diarrhea: bloody in later stages

DRS NOTES:

- **Post-intervention lower GI bleed:** Following a screening colonoscopy and polypectomy, patients may present with bleeding from the site of polypectomy. Treatment is burning or clipping.
- **Colonoscopy**:
  - Stable patients: Give bowel prep to clean the colon from stool and then scope them.
    Most of the time when the bleeding is proximal the blood acts as a strong laxative taking everything out, so you give them short/fast prep and then scope them.
  - Unstable patients: Get a CT angiogram and based on the location of bleeding you can perform surgery and resect the exact section, but it's very rare we go to this extent
  - Unstable patient with unknown source of bleeding: perform a total colectomy (very rare), some doctors clamp the colon to see which segment is the source.
- **Maroon stool** (not black nor red) or **anemia** and an unknown source of bleeding:
  Capsule or angiography, usually the etiology turns out to be AVM or GIST in the small bowel.

Gastric outlet, small and large bowel obstruction

- Defined as **mechanical** or **functional (non-mechanical)** obstruction of intestine by causes in the lumen/ wall/ outside wall resulting in stoppage in anal ward movement of bowel content, proximal dilation and distal collapse and associated with complex of symptoms and signs

Classification

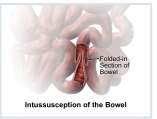
Location	• **Gastric outlet obstruction (GOO)**: obstruction at the level of the pyloric channel or duodenum. • **Small bowel obstruction (SBO):** obstruction at the level of the duodenum, jejunum, or ileum. According to the ligament of treitz, it's divided into **High or Low** • **Large bowel obstruction (LBO):** obstruction at the level of the cecum, colon, or rectum. (Most common site of obstruction is sigmoid and rectum because the sigmoid is the narrowest part of the colon)
Progress	• **Acute** ex: 2 weeks presentation • **Subacute** • **Chronic** ex: 6 months presentation that comes and goes

Severity	• **Partial**
	  ○ **High grade**
	  ○ **Low garde**
	• **Complete**

Anatomy	• **Open loop obstruction (Simple linear obstruction):** When there is one point of obstruction in intestine
	• **Closed loop obstruction (Rotational obstruction):** When there are two points of obstruction in intestines. more likely to strangulate and perforate. May present in those who have competent ileocecal valve (one way valve that allows the food to go from small bowel to large bowel 13% has competent valve and the rest has incompetent valve)
	Detection of closed loop:
	  ○ Colon: always presume that its closed
	  ○ Small bowel: detected by CT
	• Closed loop obstruction can occur in small bowel due to adhesions small bowel twisting on itself (volvulus) or malignant involvement of two parts of the small bowel.

Outcome	• **Simple bowel obstruction:** bowel obstruction with no evidence of complications • **Strangulated (complicated) bowel obstruction:** bowel obstruction associated with strangulation, ischemic necrosis, or perforation. A portion of bowel is dead.

Site	Intraluminal	○ **Impacted faeces:** common in elderly due to inability to pass stool.
		  ■ **Management**? if they fail laxative, fecal disimpaction via hand, sometimes done under GA.
		○ **Foreign bodies**
		○ **Gallstone ileus:** gallstone goes through the fistula into duodenum and erodes into ileocecal valve.
		  ■ **Management**? Surgical resection as it's stuck in the ileocecal valve, but if it's impacted with edema due to the obstruction we'd have to milk it backwards to a portion of normal bowel, open there and stitch the normal valve.
		○ **Bezoars:** solid mass of indigestible (high fiber) material "hair, food, nails or فصفص"
		○ **Parasitic infections:** Helminth e.g. ascaris

	Intramural (intrinsic)	○ **Tumors:** because they may origin from the layers of GI tract.
		○ **Inflammatory strictures:** IBD (CD → stricture)
		○ **Intussusception:** when one portion of the gut invaginates into an immediately adjacent segment due to peristalsis
		  ■ **Management**? Either spontaneously reduced, or needs to be resected.
		○ **Other causes include:**
		  ■ TB
		  ■ Diverticulitis (if inflammation is significant → edema and obstruction)
		 

	Extramural	○ **Adhesions**
		  ■ Post-op (but can rarely happen congenitally), more common in open surgery due to bowel injury and manipulation.
		  ■ **Management**? going into surgery causes more adhesions, but the aim is to do so in a way that heals without obstruction. It's mostly managed conservatively
		○ **Hernias**
		  ■ **Management**? You have to reduce the bowel back into the abdominal cavity thus removing obstruction
		○ **Tumors:** by compressing the bowel
		○ **Volvulus:** twisting or axial rotation of a portion of bowel about its mesentery, It has a characteristic radiological sign called (omega sign Ω / coffee bean sign)

Gastric outlet, small and large bowel obstruction

Etiology

SBO
Small bowel obstruction

- **Adhesions (Post-operative)** 60%
- **Tumor:** 20%
- **Incarcerated hernia** 10%
- **Crohn's disease** 5%
- **Gallstone ileus:** 3%

LBO
Large bowel obstruction

- **Colon cancer (90%)**
- **Volvulus (5%)**
- **Diverticular disease (3%**
- **Hernia**
- **Adhesions**

GOO
Gastric outlet obstruction

- **PUD (most common)**
- **Cancers**
- Gastric, duodenal polyps
- Pyloric stenosis
- Gallstone
- **Bezoars: Trichobezoars (Hairballs):** Usually in psychiatric patients eating hair
- Pancreatic pseudocysts
- **SMA (Superior mesenteric artery) syndrome:** acute angulation of the SMA causes compression

Paralytic ileus

The 5 **P**s:

- **P**eritonitis
- **P**ostoperative
- low **P**otassium
- o**P**ioids
- **P**elvic/spinal fractures are among the most common causes of paralytic ileus.

Clinical Features

Red flags:

- Pain out of proportion
- Peritoneal signs: tenderness with rigidity, guarding and peritonism/rebound tenderness
- Pyrexia (indicate ischaemia, perforation and inflammation), tachycardia and dehydration (hypovolemia)
- Hemodynamic instability & signs of systemic toxicity, e.g., SIRS
- Laboratory abnormalities: e.g., significant leukocytosis, leucopenia, metabolic acidosis, ↑ lactate dehydrogenase and potassium amylase
- Completely absent bowel sounds
- The risk of perforation increases as the cecal diameter exceeds 12 cm, so take immediately to the OR

Exclude **strangulation**, **perforation** and **peritonitis** symptoms. Then try to differentiate based on cardinal symptoms:

As we move distally; abdominal pain↓ vomiting↓ distention↑ constipation↑

	GOO	SBO	LBO	Paralytic ileus
Pain	• Epigastric pain	• Colicky, periumbilical	• Colicky or constant • Left lower abdomen	• Continuous (noncolicky) abdominal pain or discomfort
Vomiting/ nausea	• Postprandial, nonbilious projectile vomiting	• Early-onset • Large volume • Bilious • No vomiting in closed loop obstruction	• Late-onset • Little vomiting • Initially bilious Progresses to fecal vomiting	• Effortless vomiting
Constipation	-	• Late-onset	• Early-onset • Pronounced	-
Distention	• Upper abdominal distention	• Minimal or absent	• Early • Pronounced	• Marked
Other	• Dehydration • Early satiety • Alkalosis • Epigastric ,left or right hypochondrium tenderness • Succussion splash: sloshing sound heard during sudden movement of the patient • Sister mary joseph sign	• Dehydration. • Tympanic percussion. • Increased high-pitched, tinkling bowel sounds (early) or absent bowel sounds (late) • Generalized tenderness • Collapsed, empty rectum on digital rectal examination		• Tympanic percussion • Bowel sounds usually absent • No tenderness unless there is peritonitis

Gastric outlet, small and large bowel obstruction

Investigation:

In the workup of suspected mechanical bowel obstruction, **imaging** allows for quick confirmation of the diagnosis as well as detection of conditions requiring immediate surgery (e.g., perforation). **Laboratory tests** may further help to assess the severity of the condition (e.g., electrolyte imbalance due to vomiting).

Imaging

- Erect and supine abdominal x-rays (best initial):
  - SBO → central dilated loops
  - LBO → peripheral dilated loops
  - GOO → enlarged stomach
  - Paralytic ileus → generalized small and large bowel gaseous distention
- Erect chest x-ray: air under the diaphragm indicates perforation
- Abdominal CT with contrast: **Most commonly** widely used now most accurate determine the cause and assess the bowel
  - Mechanical obstruction → transition point: distended bowel followed by collapsed bowel.
  - Paralytic ileus → only to rule out suspected mechanical bowel obstruction
  - Strangulation: pneumatosis (air within the **wall** of bowel NOT lumen)
  - Masses
  - Hernias
- Contrast study: has a therapeutic role
  - **Contrast follow through**
    - Indicated in patient SBO with failure to resolve (enteroclysis) → to determine site & degree of obstruction
    - Contrast passed → Continue conservative management
    - Delayed passage & distended proximal → OR
    - Study of choice when investigating possible malrotation and contraindicated in the presence of acute obstruction and may be life-threatening.
  - **Contrast enema**
    - used to assess the level and the degree of obstruction.
    - Water-soluble enema used to differentiate large bowel obstruction from pseudo-obstruction.
    - Contraindicated when complete obstruction is suspected.
    - Has a therapeutic role.

Laboratory

- Blood work and electrolyte:
  - Vomiting → Hypochloremic **hypokalemic metabolic alkalosis** and Hyponatremia
  - Strangulation → neutrophilia, hyperkalemia, hyperamylasemia & raised LDH

Endoscopy

- Sigmoidoscopy → in carcinoma, volvulus
- **Gastroscopy → to determine pathology in GOO (confirmatory) and biopsy if malignancy is suspected**

Management

Management in general is conservative by **Fluid resuscitation, correction of electrolyte imbalance, Intestinal decompression(nasogastric tube), Bowel rest (NPO)** and **Administration of IV analgesics and antiemetics if needed**. Procedure:

	Mechanical	Adhesive	GOO	Pseudoobstruction
Procedure	**Exploratory laparotomy**	**Laparoscopic adhesiolysis**	Vagotomy and antrectomy with Billroth II reconstruction **Gastrojejunostomy**	**Colectomy**
Indication	• Virgin abdomen (No previous surgery) • Failure of conservative management • Tender, irreducible hernia • Strangulation or perforation • Complete or closed-loop obstruction	• Failure of conservative treatment > 72 hours • Evidence of strangulation or peritonitis • Free intraperitoneal gas in the abdominal imaging • Complete intestinal obstruction in the abdominal imaging	• If medical therapy failed	Failure of conservative treatment which is: • Stimulant enemas • Colonoscopic deflation • IV erythromycin • IV neostigmine

Gastric outlet, small and large bowel obstruction

Management	

Other	• **Sigmoid volvulus management:** rehydration and NGT insertion then colonoscopic detorsion
	• **Management of gastric volvulus:** surgical repair
	• **dark colored segment above the level of relieved obstruction?** resect it
	• **management of paralytic ileus:**
	  ○ **conservative management**
	  ○ **Nutritional management:** Gradual increase in enteral feeding as tolerated by the patient. Parenteral nutrition may be considered if ileus persists for ≥ 7 days. (e.g TPN)
	  ○ **Surgery:**
	  ■ If the treatment of the underlying cause requires surgery
	  ■ Treatment of complications (e.g. intestinal ischemia) or perforation

Pseudo Obstruction		
Etiology	• Metabolic: (DM, **hypokalemia**, uremia, myxedema) • Severe trauma (lumbar spine and pelvis) • Shock • Major burns • Extensive myocardial infarction • Septicemia	• Stroke • Idiopathic • **Postoperative** • Drugs (Tricyclic antidepressants, Phenothiazine, Laxatives) • Secondary GIT involvement (scleroderma Chagas diseases)
Pathophysiology	Etiological factors → impairment/destruction of the autonomic nervous system → imbalance between sympathetic and parasympathetic control of intestinal motility → accumulation of feces, air, and intestinal secretions in the intestine → Intestinal dilation	

Small Bowel Pseudo Obstruction	
Clinical features	Recurrent subacute intestinal obstruction
Diagnosis Not part of the objs	Exclusion of mechanical causes is required before the diagnosis of small intestine pseudo obstruction which is performed by: • Gastrografin follow through • CT scan
Management Not part of the objs	• Correction of any underlying cause • Metoclopramide • Erythromycin

Large Bowel Pseudo Obstruction		
Types	Acute (Ogilvie's Syndrome)	Chronic
Diagnosis Not part of the objs	• **Laboratory tests:** May show signs of the underlying cause • **Abdominal x-ray** • **Contrast enhanced CT:** confirms x-ray findings and rule out mechanical obstruction (It's more with elderly bed ridden patients (comorbid in the geriatrics ward) that's why we need to rule mechanical obstruction out because it affects the same group that sigmoid volvulus effects) • **Colonoscopy:** hemodynamically stable patients who can't perform contrast enhanced CT. Shows distended colon without any level of obstruction.	
Management Not part of the objs	Treat the underlying cause.	

	• Supportive when the patient has mild symptoms and cecal dilatation is **<12 cm**
	• Neostigmine: No improvement > 24 - 48 hours and/or cecal dilatation is **> 12 cm** with no signs of colonic ischemia, perforation or peritonitis
	• Neostigmine is **contraindicated**: colonoscopies bowel decompression
	• **Surgery** indications:
	  ○ Signs of colonic ischemia, perforation or peritonitis
	  ○ Failure of conservative therapy
		• **Conservative** management
		  ○ Dietary modifications
		  ○ Osmotic laxatives and enema
		  ○ Prokinetic drugs: **Erythromycin** (stimulate motility by binding to colonic motilin receptors), metoclopramide, neostigmine
		• **Surgery** indications:
		  ○ Patients who do not improve on conservative therapy