1. Etiology and pathogenesis of acute appendicitis, classification, clinical manifestations, treatment.

Acute appendicitis is an inflammation of the inner lining of vermiform appendix.

ETIOLOGY

- Obstruction (calcium phosphates, bacteria and epithelial debris)
- Fecolith or a stricture, are the most common cause of appendiceal obstruction.
- Obstruction of the appendiceal orifice by tumor, carcinoma of the cecum
- hypertrophy of lymphoid tissue narrows the lumen
- Decreased dietary fibers and increased use of carbohydrates
- aerobic and anaerobic bacterial proliferation within the appendix
- Intestinal parasites, particularly Oxyuris vermicularis (pinworm), can proliferate in the appendix

PATHOGENESIS

Once obstruction occurs, continued mucus secretion and **inflammatory exudation increase intraluminal pressure**(from 0.1ml to 0.5ml), and make obstructing lymphatic drainage. Edema and mucosal ulceration develop with bacterial translocation to the submucosa. (Resolution in this case antibiotic therapy.)
If the condition develops, distension of the appendix may stimulates the nerve endings (vagus) and venous obstruction and ischaemia of the appendix wall so acute appendicitis occure. Finally, ischemic necrosis produces gangrenous appendicitis, which reach to serosa of the appendix and in turn parietal peritoneum in the region, which produces pain in the right lower quadrant.

CLINICALI MANIFESTATION

- Periumbilical pain
- Pain shifting to the right iliac fossa
- Anorexia
- Nausea, vomiting
- Diarrhea or constipation
- Cutaneous hyperesthesia in the area supplied by the spinal nerves
- Muscular resistance to palpation of the abdominal wall
- muscle spasm increases

- **CLINICAL SIGNES** :

1. **Aure-Rozanova sign**: Increased pain on palpation with finger in right Petit triangle
2. **Bartomier-Michelson's sign**: Increased pain on palpation at the right iliac region when lies on his/her left side
3. **Dunphy's sign**: Increased pain in the right lower quadrant with coughing.
4. **Hamburger sign**: The patient refuses to eat
5. **Kocher's (Kosher's) sign**- the start of pain in the umbilical region with a subsequent shift to the right iliac region.
6. **Psoas sign**: Also known as the "Obraztsova's sign" is right lower-quadrant pain that is produced with either the passive extension of the right hip or by the active flexion of the person's right hip while supine
7. **Rovsing's sign**: Pain in the lower right abdominal quadrant with continuous deep palpation starting from the left iliac fossa upwards
8. **Sitkovskiy (Rosenstein)'s sign:** Increased pain in the right iliac region as the person is being examined lies on his/her left side.
9. **Obturator sign-** The person being evaluated lies on her/his back with the hip and knee both flexed at ninety degrees.

- **Postoperative Complications of Acute Appendicitis**
  - ✓ Postoperative wound infection risk
  - ✓ Intra-abdominal and pelvic abscesses
  - ✓ Small-bowel obstruction

TREATMENT

A. Preoperative preparation
   - Intravenous isotonic fluid replacement should be initiated to achieve a brisk urinary output and to correct electrolyte abnormalities.
   - Nasogastric suction is helpful, especially in patients with peritonitis.
   - Temperature elevations are treated with acetaminophen and a cooling blanket.
   - Anesthesia should not be induced in patients with a temperature higher than 39°C.

B. Antibiotic therapy (second-generation cephalosporin) 3-5 days **.**
   penicillin-allergic patients, carbapenems are a good option.
   Antibiotic prophylaxis is generally effective in the prevention of postoperative infectious complications.

C. APPENDECTOMY :
   - **Conventional appendectomy**(The incision that is widely used for appendectomy is the so-called gridiron incision , is made at right angles to a line joining the anterior superior iliac spine to the umbilicus, its center being along the line at McBurney's point)
   - **Retrograde appendectomy**(When the appendix is retrocecal and adherent)
   - **Laparoscopic appendectomy** (less postoperative pain and to be discharged from hospital and return to activities of daily living sooner than those who have undergone open appendectomy)

2. Clinical symptoms of acute appendicitis of common and abnormal localization,differential diagnosis.

CLINICALI MANIFESTATION
- Periumbilical pain
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- Anorexia
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  - ✓ Postoperative wound infection risk
  - ✓ Intra-abdominal and pelvic abscesses

- ✓ Small-bowel obstr

ABNORMAL LOCALIZATION

- Absent of apependix
- Ectopic appendix
  - ✓ Fawcitt found an appendix in the thorax
  - ✓ Babcock reported the removal of an appendix in the lumbar area
  - ✓ located within the posterior cecal wall, and which did not have a serous coat
- left side appendix
- duplication of appendix

(normal position :retrocecal, pelvic, subcecal, preileal, or right pericolic position)

DIFFERENTIAL DIAGNOSIS

Children	**Adult**	**Adult female**	**Elderly**
Gastroenteritis	Regional enteritis	Mittelschmerz	Diverticulitis
Mesenteric adenitis	Ureteric colic	Pelvic inflammatory disease	Intestinal obstruction
Meckel's diverticulitis	Perforated peptic ulcer	Pyelonephritis	Colonic carcinoma
Intussusception	Torsion of testis	Ectopic pregnancy	Torsion appendix epiploicae
Henoch–Schönlein purpura	Pancreatitis	Torsion/rupture of ovarian cyst	Mesenteric infarction
Lobar pneumonia	Rectus sheath hematoma	Endometriosis	Leaking aortic aneurysm

3. Clinical features of acute appendicitis in children, aged people, pregnant women.

1. **CHILDREN :**

- diagnosis of acute appendicitis is more difficult in young children than in the adult.
- physical examination findings of maximal tenderness in the right lower quadrant,the inability to walk or walking with a limp, and pain with percussion, coughing.
- Progression to rupture and the inability of the underdeveloped greater omentum to contain a rupture lead to significant morbidity rates.
- The wound infection rate after the treatment of nonperforated appendicitis in children is 2.8% compared with a rate of 11% after the treatment of perforated appendicitis.
- The incidence of intra-abdominal abscess also is higher after the treatment of perforated appendicitis than after nonperforated appendicitis.
- Laparoscopic appendectomy is safe for children.

2. **AGED PEOPLE :**

- Compared with younger patients, elderly patients more difficult diagnostic problem because of :
- ✓ the atypical presentation,
- ✓ expanded differential diagnosis,
- ✓ and communication difficulty.
- lower abdominal pain, and present tenderness on the right lower quadrant
- The usefulness of the Alvarado score appears to decline in the elderly.
- Although currently there are no criteria that definitively identify elderly patients with acute appendicitis who are at risk of rupture, prioritization should be given to patients :
- ✓ with a temperature of >38°C (100.4°F) and
- ✓ a shift to the left in leukocyte count of >76%,

- ✓ especially if they are male,
- ✓ are anorectic,
- ✓ or have had pain of long duration before admission.

3. **PREGNANCY :**

- Appendicitis is the most common extrauterine acute abdominal condition in pregnancy with very low frequency.
- Reduce in 3th trimester
- Suggested relation with female sex hormones
- Separation of the visceral and parietal peritoneum due to the enlarging uterus limits localization of the pain by decreasing the somatic component of the pain.
- nausea and vomiting
- The normal and postpartum positions of the base of the appendix are medial to McBurney's point.
- At the fifth month, the appendix is at the level of the umbilicus and iliac

4. Acute appendicitis complications. Classification, principles of diagnostics and treatment.

COMPLICATION

- Periumbilical pain
- Pain shifting to the right iliac fossa
- Anorexia
- Nausea, vomiting
- Diarrhea or constipation
- Cutaneous hyperesthesia in the area supplied by the spinal nerves
- Muscular resistance to palpation of the abdominal wall
- muscle spasm increases

- **CLINICAL SIGNES** :

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9. **Obturator sign-** The person being evaluated lies on her/his back with the hip and knee both flexed at ninety degrees.

- **Postoperative Complications of Acute Appendicitis**
  - ✓ Postoperative wound infection risk
  - ✓ Intra-abdominal and pelvic abscesses
  - ✓ Small-bowel obstruction

DIAGNOSIS

1. **LABORATORY FINDINGS :**
   - CBC - Mild leucocytosis
   - increased C-reactive protein (CRP)
   - urinary tract as the source of infection

- Liver and pancreatic test

2. **Imaging test**
   - CT- the inflamed appendix appears dilated >5cm
   - Sonograghy - the appendix is identified as a blind-ending, nonperistaltic bowel loop originating from the cecum.
   - MRI-is useful in pregnancy
   - Radionuclide – has not been enough experience
3. **Barium enema**

TREATMENT

A. Preoperative preparation
   - Intravenous isotonic fluid replacement should be initiated to achieve a brisk urinary output and to correct electrolyte abnormalities.
   - Nasogastric suction is helpful, especially in patients with peritonitis.
   - Temperature elevations are treated with acetaminophen and a cooling blanket.
   - Anesthesia should not be induced in patients with a temperature higher than 39°C.

B. Antibiotic therapy (second-generation cephalosporin) 3-5 days **.**
penicillin-allergic patients, carbapenems are a good option.
Antibiotic prophylaxis is generally effective in the prevention of postoperative infectious complications.

C. APPENDECTOMY :
   - **Conventional appendectomy**(The incision that is widely used for appendectomy is the so-called gridiron incision , is made at right angles to a line joining the anterior superior iliac spine to the umbilicus, its center being along the line at McBurney's point)
   - **Retrograde appendectomy**(When the appendix is retrocecal and adherent)
   - **Laparoscopic appendectomy** (less postoperative pain and to be discharged from hospital and return to activities of daily living sooner than those who have undergone open appendectomy)

5. Peritonitis due to acute appendicitis. Medical and surgical treatment, complications.
6. Retrocaecal acute appendicitis. Clinical manifestations, diagnosis, treatment.

CLINICAL MANIFESTATION
- Rigidity is often absent,
- Application of deep pressure may fail to elicit tenderness (silent appendix), the reason being that the cecum, distended with gas, prevents the pressure exerted by the hand from reaching the inflamed structure. However, deep tenderness is often present in the loin, and rigidity of the quadratus lumborum may be in evidence.
- Psoas spasm, due to the inflamed appendix being in contact with that muscle, may be sufficient to cause flexion of the hip joint.
- Hyperextension of the hip joint may induce abdominal pain when the degree of psoas spasm is insufficient to cause flexion of the hip.

DIAGNOSIS
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7. Acute appendicitis complications of other organs and systems. Clinical manifestations, diagnosis, treatment.

8. Chronic appendicitis. Clinical manifestations, diagnostics, differential diagnosis, treatment.
chronic appendicitis mainly presents as a less severe, continuous abdominal pain lasting longer than 1–2-period per day, and often extending to weeks, months, or even years.

CLINICALI MANIFESTATION

A. symptoms of chronic appendicitis may be mild may come and go . In some cases, abdominal dull pain is the only symptom with chronic appendicitis.

B. Other symptom :
   - abdominal pain
   - fever
   - abdominal swelling and tenderness
   - fatigue or lethargy, which is a lack of energy
   - malaise, which is a general feeling of discomfort or illness

C. The most **common complications** include:
   - acute appendicitis
   - ruptured appendix
   - abscess, which is a pocket of infection
   - sepsis, which is your body's serious response to infection
   - peritonitis, which is inflammation of the abdomen's lining

DIAGNOSIS

1. **LABORATORY FINDINGS :**
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3. **Barium enema**

DIFFERENTIAL DIAGNOSIS

- gastrointestinal disorders
- Crohn's disease
- ulcerative colitis
- urinary tract infection
- kidney infection
- irritable bowel syndrome (IBS)
- ovarian cysts
- pelvic inflammatory disease (PID)

TREATMENT

1. **Antibiotic therapy** (second-generation cephalosporin) 3-5 days **.**
   penicillin-allergic patients, carbapenems are a good option
2. Doctor may also **drain** the pus that forms in your appendix.
3. Most common treatment **APPENDECTOMY** :
   - **Conventional appendectomy**(The incision that is widely used for appendectomy is the so-called gridiron incision , is made at right angles to a line joining the anterior superior iliac spine to the umbilicus, its center being along the line at McBurney's point)
   - **Retrograde appendectomy**(When the appendix is retrocecal and adherent)
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9. Definition, classification, clinical manifestations, diagnosis and treatment of chronic appendicitis.

chronic appendicitis mainly presents as a less severe, continuous abdominal pain lasting longer than 1–2-period per day, and often extending to weeks, months, or even years.

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- acute appendicitis
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- abscess, which is a pocket of infection
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(DIFFERENTIAL DIAGNOSIS

- gastrointestinal disorders
- Crohn's disease
- ulcerative colitis
- urinary tract infection
- kidney infection
- irritable bowel syndrome (IBS)
- ovarian cysts
- pelvic inflammatory disease (PID))

10. Hernias. Etiology and pathogenesis of hernia formation, classification.

- hernia is the protrusion of an organ or part of an organ in its coverings or tissue (peritoneum or fat) through a congenital or acquired defect in the wall of the cavity into an abnormal position.

- A hernia consists of:
  - ✓ **sac** (mouth , neck , body , fundus)
  - ✓ **coverings** (skin ,subcutaneous fat , aponeurosis , muscle , endo-cavity fascia , endothelial lining - peritoneum in the abdomen.)
  - ✓ **its contents**(small bowel , greater omentum , meckel's diverticulum , bladder , ovary with or without fallopian tube , ascetic fluid)

ETIOLOGY

The two main factors : **increased intracavity pressure and a weakened abdominal wall**

1. **increased intracavity pressure due to:**
   - chronic cough
   - straining to pass urine (problems with obstruction of the bladder outlet and urethra)
   - straining to pass faeces(constipation)
   - abdominal distension- presence of an intra-abdominal disorder
   - change in abdominal contents - encysted fluid, benign or malignant tumour, pregnancy, fat.
   - Ascites – fluid in abdominal cavity
2. **weakened abdominal wall due to :**
   - advancing age
   - malnutrition
   - damage to, or paralysis of, motor nerves
   - abnormal collagen metabolism.
   - the presence of a patent, congenitally formed sac
   - multiple pregnancy
   - previous abdominal surgery

PATHOGENESIS OF HERNIA FOEMATION

A. A normal abdominal wall has sufficient strength to resist high abdominal pressure and prevent herniation of content.
B. Herniation happen due to increased intracavity pressure and/or two essential components : a defect in the **wall** and **content**, that tissue has been forced outwards through the defect which may small or very large in size. the weekness may be entirely muscle (incisional hernia) or fascia (epigastric hernia through linea alba). it may have bony component (femoral hernia)
C. A small defect with rigid walls traps the content and prevents it from freely moving in and out of the defect, increasing the risk of complications. if such a hernia enlarges then peritoneum may also be pulled into the hernia secondarily along with intraperitoneal structures such as bowel or omentum; example :'sliding type' of inguinal hernia.
D. when peritoneum is lying immediately deep to the abdominal wall weakness, pressure forces the peritoneum through the defect and into the subcutaneous tissues. This 'sac' of peritoneum allows bowel and omentum to pass through the defect too. In most cases, the intraperitoneal organs can move freely in and out of the hernia, a 'reducible' hernia, but if adhesions form or the defect is small, bowel can become trapped and unable to return to the main peritoneal cavity, an 'irreducible' hernia, with high risk of further complications.

CLASSIFICATION

According to pathogenesis

1. Occult – not detectable clinically; may cause severe pain
2. Reducible – a swelling which appears and disappears
3. Irreducible – a swelling which cannot be replaced in the abdomen, high risk of complications
4. Obstructed – disturbances in bowel contest passing through the hernia
5. Strangulated – painful swelling with vascular compromise, requires urgent surgery
6. Infarcted – when contents of the hernia have become gangrenous, high mortality

according to their localization

1. Inguinal (indirect or direct)
2. Femoral
3. Umbilical
4. Linea alba
5. Linea semilunaris (Spieghel's line hernia)
6. Lumbar (Petit's triangle hernia)

according the time of occurrence

1. Congenital

2. Acquired

in relation to the skin

1. External
2. Internal

according the current of disease

1. Uncomplicated (reducible, irreducible, sliding)
2. Complicated (strangulated)

11. Selection of a surgical procedure in hernia treatment. Tension and tension-free hernioplasty methods.

- "Tension" repair methods:

1. **Girard-Spasokukotcki's method**:
   upper leaf of aponeurosis and abdominal muscles (internal oblique and transversus abdominis) are sewed to the inguinal ligament above the spermatic cord. Then sew lower leaf of aponeurosis to the upper leaf and form the duplication.
2. **Martynov's method:**
   upper leaf of aponeurosis is sewed to the inguinal ligament above the spermatic cord. Then sew lower leaf of aponeurosis to the upper leaf and form the duplication.
3. **Bassini's method:**
   internal oblique and transversus abdominis are sewed to the inguinal ligament behind the spermatic cord. Then leaves of the external oblique aponeurosisis are sewed together without forming the duplication.

- "Tension free" repair methods:

1. **Lichtenstain's method:**

a mesh patch is sutured over the defect with a slit to allow passage of the spermatic cord

12. Strangulated and obstructed hernia. Definition, causes, clinical picture, diagnosis, treatment.

STRANGULATED

When strangulation occurs, the contents of the hernia are constricted by the neck of the sac to such a degree that their circulation is cut off. Example : femoral, indirect inguinal and umbilica .

OBSTRUCTED

The obstructed hernia contains intestine in which The lumen has become occluded. Obstruction is usually at the neck of the sac but may be caused by adhesions within it. If the obstruction is at both ends of the loop, fluid accumulates within it and distension occurs.

CAUSE

The two main factors : **increased intracavity pressure and a weakened abdominal wall**

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   - chronic cough
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   - change in abdominal contents - encysted fluid, benign or malignant tumour, pregnancy, fat.
   -
2. **weakened abdominal wall due to :**
   - advancing age
   - malnutrition
   - damage to, or paralysis of, motor nerves
   - abnormal collagen metabolism.
   - the presence of a patent, congenitally formed sac

CLINICAL PICTURE

- ✓ Severe pain
- ✓ Vomiting

- ✓ constipation
- ✓ Examination reveals a tender, tense hernia that cannot be reduced and has no cough impulse
- ✓ skin becomes inflamed
- ✓ oedematous
- ✓ noisy bowel sounds

DIAGNOSIS
- ✓ CBC
- ✓ Stain or culture of nodal tissue
- ✓ Electrolytes ,blood urea nitrogen , creatinine
- ✓ Urinalysis
- ✓ Lactate
- ✓ Ultrasound
- ✓ CT

TREATMENT
All surgical repairs follow the same basic principles:

- reduction of the hernia content into the abdominal cavity with removal of any non-viable tissue and bowel repair if necessary;
- excision and closure of a peritoneal sac if present or replacing it deep to the muscles;
- reapproximation of the walls of the neck of the hernia if possible;
- permanent reinforcement of the abdominal wall defect with sutures or mesh.

Mesh can be used:

- to bridge a defect: the mesh is simply fixed over the defect as a tension-free patch;
- to plug a defect: a plug of mesh is pushed into the defect;
- to augment a repair: the defect is closed with sutures and the mesh added for reinforcement.

Principles of management:
- Not all hernias require surgical repair
- Small hernias can be more dangerous than large
- Pain, tenderness and skin color changes imply high risk of strangulation
- Irreducible hernia should be offered repair
- Femoral hernia should always be repaired
- Surgery should be offered to younger adults

Limitations to the use of mesh
- The presence of infection particularly heavyweight types. If a mesh becomes infected then it often needs to be removed.
- Some infected meshes can be salvaged using a combination of debridement of non-incorporated mesh, appropriate antibiotics and modern vacuum-assisted dressings.
- meshes are expensive, especially those for intraperitoneal use, but prices are falling.

13. Inflamed hernia. Definition, causes, clinical picture, treatment.
The contents are inflamed by any process that causes this in the tissue or organ that is not normally herniated, e.g.: (acute appendicitis , Meckel's diverticulitis , acute salpingitis.)

CAUSE
- Obstruction (calcium phosphates, bacteria and epithelial debris)
- hypertrophy of lymphoid tissue narrows the lumen
- Decreased dietary fibers and increased use of carbohydrates
- aerobic and anaerobic bacterial proliferation
- inflammation disease
- increased intracavity pressure
- weakened abdominal wall

CLINICAL PICTURE

- ✓ Severe pain
- ✓ Vomiting
- ✓ constipation
- ✓ Examination reveals a tender, tense hernia that cannot be reduced and has no cough impulse
- ✓ skin becomes inflamed
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- excision and closure of a peritoneal sac if present or replacing it deep to the muscles;
- reapproximation of the walls of the neck of the hernia if possible;
- permanent reinforcement of the abdominal wall defect with sutures or mesh.
- IN CASE OF **appendicitis , and diverticulum** :
- appendectomy
- Patients are frequently recommended to take a high-fibre diet(avoid nut and popcorn) and bulk-forming laxatives .
- **Acute diverticulitis** is treated by intravenous antibiotics

14. Reducible, irreducible and sliding hernia. Definition, causes, diagnosis, treatment.

Reducible

Reducible hernia The contents of a reducible hernia can be replaced completely into the peritoneal cavity.

- ✓ usually not painful, although it may be accompanied by some discomfort.
- ✓ Examination reveals a reducible lump with a cough impulse.

irreducible

A hernia becomes irreducible usually because of adhesions of its contents to the inner wall of the sac, or sometimes as a result of adhesions of its contents to each other to form a mass greater in size than the neck of the sac.

- ✓ If the hernia will not reduce but is painless and there are no other symptoms, irreducibility is diagnosed.
- ✓ The absence of a cough impulse alone does not indicate strangulation, because in an irreducible femoral hernia, for example, the neck is often plugged by omentum, which prevents the cough impulse from being felt.

sliding hernia

A sliding hernia exists when a retroperitoneal organ, usually the sigmoid colon, cecum, bladder, or ureter, forms part of the wall of the sac; these organs may be injured during hernia repair.

CAUSE

The two main factors : **increased intracavity pressure and a weakened abdominal wall**

3. **increased intracavity pressure due to:**
   - chronic cough
   - straining to pass urine (problems with obstruction of the bladder outlet and urethra)
   - straining to pass faeces(constipation)
   - abdominal distension- presence of an intra-abdominal disorder
   - change in abdominal contents - encysted fluid, benign or malignant tumour, pregnancy, fat.
   -
4. **weakened abdominal wall due to :**
   - advancing age
   - malnutrition
   - damage to, or paralysis of, motor nerves
   - abnormal collagen metabolism.
   - the presence of a patent, congenitally formed sac

DIAGNOSIS

- ✓ CBC

- ✓ Stain or culture of nodal tissue
- ✓ Electrolytes ,blood urea nitrogen , creatinine
- ✓ Urinalysis
- ✓ Lactate
- ✓ Ultrasound
- ✓ CT

TREATMENT
All surgical repairs follow the same basic principles:

- reduction of the hernia content into the abdominal cavity with removal of any non-viable tissue and bowel repair if necessary;
- excision and closure of a peritoneal sac if present or replacing it deep to the muscles;
- reapproximation of the walls of the neck of the hernia if possible;
- permanent reinforcement of the abdominal wall defect with sutures or mesh.

Mesh can be used:

- to bridge a defect: the mesh is simply fixed over the defect as a tension-free patch;
- to plug a defect: a plug of mesh is pushed into the defect;
- to augment a repair: the defect is closed with sutures and the mesh added for reinforcement.

Principles of management:
- Not all hernias require surgical repair
- Small hernias can be more dangerous than large
- Pain, tenderness and skin color changes imply high risk of strangulation
- Irreducible hernia should be offered repair
- Femoral hernia should always be repaired
- Surgery should be offered to younger adults

Limitations to the use of mesh
- The presence of infection particularly heavyweight types. If a mesh becomes infected then it often needs to be removed.
- Some infected meshes can be salvaged using a combination of debridement of non-incorporated mesh, appropriate antibiotics and modern vacuum-assisted dressings.
- meshes are expensive, especially those for intraperitoneal use, but prices are falling.

15. Umbilical hernias. Anatomy, classification, clinical picture, diagnosis, treatment.
ANATOMY
Umbilical hernia is protrusion of the visceral organs covered by parietal peritoneum through the umbilical ring. The layers of umbilical ring are:
• skin; • scar tissue; • umbilical fascia; • peritoneum.
CLINICAL PICTURE
- Pain due to tissue tension
- Bowel obstruction
- Vomiting
- Abdominal discomfort
- In large hernias, the overlying skin may become thinned, stretched and develop dermatitis.
- Has tenderness, swelling or discoloration at the site of the hernia
- In baby bulge may be visible when baby cry,
- cough black infants is up to eight times higher than in white

DIAGNISIS

- Clinical feature
- radiological
- ultrasound
- CT

TREATMENT

1. Open umbilical hernia repair

   Very small defects less than 1 cm in size may be closed with a simple figure-of-eight suture, or repaired by a darn technique where a non-absorbable, monofilament suture is criss-crossed across the defect and anchored firmly to the fascia all around. By **Mayo's method**(an umbilical ring is cut at the transversal direction and lower leaf of aponeurosis is sewed to the upper leaf by U-shaped sutures, upper leaf is sewed over the lower leaf forming the duplication.)

✓ The **mesh** may be placed in one of several anatomical planes:

- **Within the peritoneal cavity** – (a tissue separating mesh is placed through the defect and spread out on the underside of the abdominal wall and fixed to it)
- **In the retromuscular space_**(the linea alba is opened both vertically and both left and right posterior rectus sheaths are incised 1 cm to the side of the midline exposing the rectus muscle.)
- **In the extraperitoneal space –** (it is difficult, but possible, to develop the plane below the posterior rectus sheath, just outside the peritoneum.)
- **In the subcutaneous plane –(** this is the simplest technique, called an onlay mesh. The peritoneal sac and contents are dealt with as above.)

2. Laparoscopic umbilical hernia repair

3. When the sac is massive, it is protected with a dressing soaked in mild antiseptic. Gradual epithelialization takes place and later repair may then be undertaken

16. Inguinal hernias. Anatomy, classification, clinical picture, diagnosis, treatment.

ANATOM _ inguinal canal and 2 rings

✓ Anteriorly: skin, superficial fascia and external oblique aponeurosis cover the full length of the canal; the internal oblique covers its lateral third.
✓ Posteriorly: the conjoint tendon forms the posterior wall of the canal medially; the transversalis fascia lies laterally.
✓ Above: the lowest fibres of the internal oblique and transversus abdominis transversalis fascia
✓ Below: lies the inguinal ligament.

- The internal(deep) ring represents the point at which the spermatic cord pushes through the transversalis fascia; it is demarcated medially by the inferior epigastric vessels as they pass upwards from the external iliac artery and vein.
- The external(superficial) ring is an inverted V - shaped defect in the external oblique aponeurosis and lies immediately above and medial to the pubic tubercle

CLASSIFICATION

according to position

- Direct(midle)-hernial sac enter through the posterior wall of the inguinal canal medial to the inferior epigastric vessels.
- Indirect(lateral/obliq)- hernial sac enters the inguinal canal through the deep inguinal ring lateral to the inferior epigastric vessels, and exits the canal through the superficial inguinal ring, most common than direct
- Other points that differentiate a direct from an indirect hernia are that the **direct is always acquired** and is therefore extremely rare in infancy or adolescence

according to European society

- primary or recurrent (P or R);
- lateral, medial or femoral (L, M or F);
- defect size in finger breadths assumed to be 1.5 cm.

other classification by surgen

including : Casten, Halverson and McVay, Zollinger, Ponka, Gilbert and Nyhus.

CLINICAL PICTURE

- Mostly in men
- Weakness or pressure in your groin
- A burning or aching sensation at the bulge
- Pain or discomfort in your groin, especially when bending over, coughing or lifting
- A heavy or dragging sensation in your groin
- Fever , nausea , vomiting
- Sudden pain
- Red , purple , and dark in skin

DIAGNISIS

Clinical feature of physical examination
radiological
ultrasound
CT

TREATMENT

- **Elective surgery** for inguinal hernia is a common and simple operation
- **Herniotomy**
- **Open suture repair** :
  - ✓ Bassini
  - ✓ Shouldice
  - ✓ Desarda
  - ✓ McVay

  (The surgeon enters the inguinal canal by opening its anterior wall, the external oblique aponeurosis . commonly performed either by plicating the transversalis fascia in the posterior wall with a nylon suture (Shouldice repair) or by reinforcing the posterior wall with a nylon or polypropylene mesh (Lichtenstein repair).)
- **Open mesh repair by laparoscopy:**
  - ✓ Lichtenstein
  - ✓ Plugs
  - ✓ Hernia systems
  - ✓ A truss is only prescribed in patients who are of very poor general condition
- **Open preperitoneal repair :**
  - ✓ stoppa

  (It is useful when multiple attempts at open standard surgery have failed and the hernia(s) keeps recurring.)
- **Laparoscopic inguinal hernia repair :**
  - ✓ The totally extraperitoneal (TEP)
  - ✓ transabdominal preperitoneal (TAPP)

  (In both, the aim of surgery is to reduce the hernia and hernia sac within the abdomen and then place a 10 × 15 cm mesh just deep to the abdominal wall)

17. Femoral hernia. Anatomy, clinical picture, diagnosis, treatment.
ANATOMY

Femoral herniae are below and lateral to the pubic tubercle
The walls of a femoral canal :
femoral vein laterally, the inguinal ligament anteriorly, ileopectineal ligament (Cooper's) posteriorly and the lacunar ligament (Gimbernat's) medially.
CLINICAL PICTURE

- Mostly in women
- Easily missed on examination
- Pain, nausea , vomiting
- Mostly no symptom but if trapped it caused :
- Obstructed hernia
- Strangulated hernia which stop blood flow and cause :

✓ sudden, worsening pain and extreme tenderness around a hernia
✓ fever
✓ nausea
✓ rapid heart rate
✓ skin redness around the bulge
✓ vomiting

DIAGNISIS

✓ Clinical feature of physical examination
✓ radiological
✓ ultrasound
✓ CT

TREATMANT

✓ There is no alternative to surgery for femoral hernia
✓ There are three open approaches and appropriate cases can be managed laparoscopically:
✓ **Low approach (Lockwood-Bassini)**

(This is the simplest operation for femoral hernia but only suitable when there is no risk of bowel resection. It can easily be performed under local anesthesia. A transverse incision is made over the hernia. The sac of the hernia is opened and its contents reduced)

✓ **The inguinal approach (Lotheissen)**

(The initial incision is identical to that of a Bassini or Lichtenstein operation into the inguinal canal , A femoral hernia lies immediately below this incision and can be reduced. Once reduced, the neck of the hernia is closed with sutures or a mesh plug.)

✓ **High approach (McEvedy)**

(This more complex operation is ideal in the emergency situation where the risk of bowel strangulation is high.)

✓ **Laparoscopic approach**- The totally extraperitoneal (TEP) , transabdominal preperitoneal (TAPP)

(This is ideal for reducible femoral hernias presenting electively but not in emergency cases nor for irreducible hernia)

18. Rare types of hernias (Richter's, W-Maydl's, spigelian, lumbar, obturator, internal hernias).

RICHTER'S
In a special circumstance (Richter's hernia) only part of the bowel wall enters the hernia. It may be small and difficult or even impossible to detect clinically. Bowel obstruction may not be present but the bowel

wall may still become necrotic and perforate with life-threatening consequences. Femoral hernia may present in this way often with diagnostic delay and high risk to the patient .

SPIGELIAN HERNIA
These hernias are uncommon although are probably underdiagnosed. They affect men and women equally and can occur at any age, but are most common in the elderly. They arise through a defect in the Spigelian fascia which is the aponeurosis of the transversus abdominis muscle. Often these hernias advance through the internal oblique as well and spread out deep to the external oblique aponeurosis. The Spigelian fascia extends between the transversus muscle and the lateral border of the rectus sheath from the costal margin to the groin where it blends into the conjoint tendon. Most Spigelian hernias appear below the level of the umbilicus near the edge of the rectus sheath but they can be found anywhere along the 'Spigelian line'.

Surgery is recommended as the narrow and fibrous neck predisposes to strangulation. Surgery can be open or laparoscopic.

LUMBAR HERNIA
Most primary lumbar hernias occur through the inferior lumbar triangle of Petit bounded below by the crest of the ilium, laterally by the external oblique muscle and medially by the latissimus dorsi
A lumbar hernia must be distinguished from:

- a lipoma;
- a cold (tuberculous) abscess pointing to this position;
- pseudo-hernia due to local muscular paralysis. Lumbar pseudo-hernia can result from any interference with the nerve supply of the affected muscles, the most common cause being injury to the subcostal nerve during a renal operation.

treatment
Lumbar hernias can be approached by open or laparoscopic surgery. The defects can be difficult to close with sutures and mesh is recommended.

OBTURATOR HERNIA, which passes through the obturator canal , mostly in women. The swelling is liable to be overlooked because it is covered by the pectineus. It seldom causes a definite swelling in Scarpa's triangle, but if the limb is flexed, abducted and rotated outwards, the hernia sometimes becomes apparent. In more than 50 per cent of cases of strangulated obturator hernia, pain is referred along the obturator nerve by its geniculate branch to the knee.
Operation is indicated. The diagnosis is rarely made preoperatively and so it is often approached through a laparotomy incision

INTERNAL HERNIA
Internal herniation occurs when a portion of the small intestine becomes entrapped in one of the retroperitoneal fossae or in a congenital mesenteric defect.
Internal herniation in the absence of adhesions is rare.The standard treatment of an obstructed hernia is to release the constricting agent by step but shouldn't be done cases of herniation involving the foramen of Winslow, mesenteric defects , The distended loop in such circumstances must first be decompressed (minimising contamination) and then reduced.
The following are potential sites of internal herniation (all are rare):

- the foramen of Winslow;
- a defect in the mesentery;
- a defect in the transverse mesocolon;
- defects in the broad ligament;
- congenital or acquired diaphragmatic hernia;
- duodenal retroperitoneal fossae – left paraduodenal and right duodenojejunal;
- caecal/appendiceal retroperitoneal fossae – superior, inferior and retrocaecal;
- intersigmoid fossa.

19. Recurrent and incisional hernias. Clinical picture, diagnosis, treatment, prophylaxis.

INCISIONAL HERNIA

- After 10–50 % of laparotomy incisions and 1–5 per cent of laparoscopic incisions.

- Factors predisposing to their development are **patient factors** (obesity, general poor healing due to malnutrition, immunosuppression or steroid therapy, chronic cough, cancer), **wound factors** (poor quality tissues, wound infection) and **surgical factors** (inappropriate suture material, incorrect suture placement).
- These start as disruption of the musculofascial layers of the abdominal wall in the region of a postoperative scar. The classic sign of wound disruption is a serosanguinous discharge.

CLINICAL PICTURE

- localized swelling
- Incisional hernias tend to increase steadily in size with time
- here may be several discrete hernias along the length
- Attacks of partial intestinal obstruction are common as there are usually coexisting internal adhesions.
- Strangulation is less frequent because most incisional hernias are broad-necked and carry a low risk of strangulation.

TREATMENT

Open and laparoscopic repairs possible. (The repair should cover the whole length of the previous incision. Approximation of the musculofascial layers should be done with minimal tension and prosthetic mesh should be used to reduce the risk of recurrence.)

PROPHYLAXY

improving the patients' general condition e.g. weight loss for obesity, or improving nutritional state for malnutrition.

Closing the fascial layers with non-absorbable, or very slowly absorbable, sutures of adequate gauge is important.

20. Etiology and pathogenesis of acute pancreatitis.

Definition:

Acute pancreatitis is an inflammatory disease of a complex etiology that is associated with the intrapancreatic enzymes activation and can result in the gland and regional tissues necrosis and development of systemic complications.

ETIOLOGY

1. Increasing of pancreatic duct pressure (obstructive causes)
2. Direct damaging influence (toxins, traumas, etc.)
3. Alcohol
   - "Secretion with blockage" mechanism – alcohol causes hypertension in the pancreatic duct system: *Increasing production of more viscous juice *Spasm of the sphincter of Oddi
   - Ethanol is a metabolic toxin to the pancreatic cells
4. Biliary tract disease (Biliary AP)
   - Common channel hypothesis (of Opie) – reflux of bile into the pancreatic duct and enzymes activation in the pancreas
   - Ductal hypertension hypothesis – gallstone block of the common channel or pancreatic duct leads to ductal pressure increase. It is not important of there is reflux of bile into the pancreatic duct or not
5. Trauma – blunt and open
6. Iatrogenic pancreatitis *Surgical trauma *Endoscopic retrograde cholangiopancreatography
7. Idiopathic
8. Rare causes of acute pancreatitis
   - Obstructive causes:
     *Ampullary and duodenal lesions
     *Choledochal cyst/choledochocele
     *Parasites (Ascaris, Clonorchis)
     *Foreign bodies (Latin: corpus alienum)
9. Non-obstructive causes of acute pancreatitis:
   *Toxins (Venoms, drugs)
   *Metabolic diseases (hypertriglyceridemia, hypercalcemia, uraemia)
   *Autoimmune pancreatitis
   *Ischemia (Hypoperfusion, embolia, vasculitis)
   *Infectious agents

*Viral infections (Hepatitis viruses, Paramyxovirus, Cytomegalovirus, HIV)
*Bacterial infections (Yersinia enterocolitica and Yersinia pseudotuberculosis, Coxsackievirus, Campylobacter, Mycoplasma)

PATHOGENESIS

*Activation of the pancreatic enzymes happens inside an acinar pancreatic cell
*These active digestive enzymes begin autodigestion that leads to cell necrosis
*It releases the enzymes and necrotic products into the pancreatic intercellular space (interstitial space)
*Inflammatory process begins in the pancreas and surrounding tissues
*Formation of edema, infiltration, pancreatic fluid collections and occasionally necrosis

21. Classification of acute pancreatitis. Laboratory and instrumental diagnostics.

CLASSIFICATION

Atlanta classification

1. Acute interstitial pancreatitis – 85-90%
2. Acute necrotic pancreatitis – 10-15%
3. Infected necrosis

Local complications

*Acute pancreatic fluid collection
*Acute necrotic collection
*Acute pseudocyst
*Walled-off necrosis

Systemic complications

*Organ failure
*SIRS(Systemic inflammatory response syndrome)

Phases

*Early (1-2 weeks) *Late (>2 weeks)

Severity

*Mild *Moderately mild *Severe

DIAGNOSIS

1. Complete Blood Count –
   - Leukocytosis with neutrophilia and "left shift" (leukopenia in severe AP may occurs) Elevated hematocrit (packed cell volume, PCV) in the early phase of AP because of dehydration
     Anemia due to ineffective hematopoiesis (or hemorrhage) - Toxic thrombocytopenia
   - Urinalysis - Increased Urine specific gravity (dehydration) - Toxic proteinuria - Hyaline casts
   - Biochemical analysis
     - Serum Amylase or Lipase activity greater than 3 times normal Elevation of the other pancreatic enzymes (Pancreatic Isoamylase, Trypsinogen-2, Phospholipase A2, Pancreatic Elastase-1 – highly specific but expensive methods)
     - Marks of SIRS: C-Reactive Protein, Procalcitonin, hypocalciumemia
     - Metabolic acidosis
     - Coagulopathy (hypercoagulation, Disseminated Intravascular Coagulation syndrome)
     - Renal damage – elevation of serum Urea and Creatinine
     - Liver damage – elevated Transaminases, decreased Albumin
2. X-ray- decrease position , Gobiet's sign (gastric and colonic distention)
3. Ultrasound
4. CT with intravenous contrast enhancement
5. Laparoscopy diagnosis _ *Peritoneal exudate with high amylase level ,
   *Hemorrhagic or glass , *Spots of fat necrosis

22. Clinical manifestations of acute pancreatitis according to the disease form. Assessment of acute pancreatitis severity.

CLINICAL MANIFESTATION

Pain:

*Dull, boring, and steady
*Intensity from mild to severe and pain shock
*Located in the upper abdomen
*May irradiate to the back
*Encircling pain

Dyspepsia

* Nausea (80-90%)
* Repeated vomiting without relief (40-60%)

Peritoneal syndrome

*Peritonitis (enzymatic or purulent) *Palpatory defined painful mass in the abdomen, right

Specific signs

*Körte's sign – painful resistance of anterior abdominal wall in the upper abdomen where the pancreas is located (60-80%)
*Voskresenskiy's sign – impossibility to identify the abdominal aorta pulsation due to pancreatic edema, gastric and colonic paresis (60-80%)
*Mayo-Robson's sign – pain while pressing at the left costovertebral angle (40-50%)
*Kamenchik's sign – pain with pressure under the xiphoid process

Skin signs in severe pancreatitis

*Mondor's sign (purple spots on a face and body)
*Lagerlof's sign (cyanotic color of a face and extremities)
*Halsted's sign (cyanotic and mottle abdominal skin)
*Grey-Turner's sign (hemorrhagic discoloration of the flanks)
*Cullen's sign (hemorrhagic discoloration of the umbilicus)
*Grünwald sign (appearance of ecchymosis, large bruise around the umbilicus and on the buttocks)

ASSESSMENT OF SEVERITY

- ✓ When assessing prognosis, we have to take into account:
  *Patient's age
  *Patient's weight (more severe course in obese patients)
  *Score systems (Ranson, APACHE II, Glasgow etc.)
  *Expression of systemic inflammatory response syndrome
  *Expression of clinical picture (intensity of pain, abdominal distension, repeated vomiting, skin signs, Ultrasound and CT data)
  *Laboratory findings:
  C-reactive protein level (CRP)
  Procalcitonin level (PCT)
  IL-6 (InterLeukin)

- ✓ Manifestations of severe pancreatitis
  *Severe pain
  *High or low body temperature (SIRS)
  *Tachypnea (pulmonary failure)
  *Low urine amount, anuria (renal failure)
  *Tachycardia, low blood pressure (SIRS, cardiovascular failure)
  *Acute encephalopathy, unconsciousness (central neural system damage)
  *"Silent" abdomen, peritonitis

23. Treatment of acute pancreatitis. Conservative treatment, indications for surgery, types of operations.

The choice of a treatment method depends on:
*Severity (mild, moderately mild, severe)
*Phase (early, late)
*AP etiology (alcoholic, biliary etc.)
*Morphology (interstitial, necrotizing)

Treatment of Mild AP
*Pain control by nonsteroidal anti-inflammatory drugs
*Antispasmodics (papaverin, drotaverin)

*Optimal fluid balance and replacement
*Histamine H2 -blockers or proton pump inhibitors
*Resting the pancreas through restriction of oral food and fluids (regimen of "nil-per-mouth") for few days
*Local hypothermia (ice on upper abdomen, cold water through nasoduodenal tube)

Treatment of Severe AP
*Treatment in Intensive Care Unit
*Continuous epidural anesthesia in case of severe pain
*Placement of central venous and urinary catheter and massive fluid and electrolytes replacement
*Intravenous wide spectrum antibiotics (imipinem)
*Early enteral feeding via a transduodenal tube
*Decreasing pancreatic secretion (atropine, somatostatine analogues)
*Protease-inhibiting drugs (aprotinin, gabexate mesylate, camostate, and phospholipase A2 inhibitors)
*Continuous arterial infusion via superior mesenteric artery or celiac trunk

Surgical treatment
Influence to etiologic factors of AP: In the course of biliary AP, when common bile duct stone is present, decompression of biliary system is needed by :

1. Drainage of biliary tract system
2. Endoscopic sphincterotomy

✓ Drainage of the toxic products
* Acute pancreatic fluids collections
* Large pseudocysts,
* Abscesses Laparoscopic or Transcutaneous USguided drainage

✓ Evacuation of the necrotic and infected material (surgical debridment or necrosectomy)
Indications: *Infected pancreatic necrosis
*Extensive (>50%) necrosis with irreversible clinical deterioration despite maximum supportive care for at least 2 weeks from onset of symptoms even without infection

✓ Re-operations:
*Scheduled (for ex. re-debridement in 3-5 days)
*On-demand (when patient condition doesn't improve)

24. Gallstone disease, chronic calculous cholecystitis. Etiology and pathogenesis, classification, clinical manifestations, diagnostics, treatment.

✓ **Gallstone** disease is the presence of concrements (solid and firm) in the gallbladder, the biliary ducts, or both.
✓ **Chronic Calculous Cholecystitis** *Chronic inflammation and fibrosis *The gallbladder may be distended or shrunken with fibrous serosal adhesions around it *The gallbladder wall is usually thickened *Sometimes there is a calcification of the gallbladder wall (porcelain gallbladder)

ETIOLOGY
- Your genes
- Your weight(obesity)
- Problems with your gallbladder(bile contains too much; cholesterol , bilirubin)
- Diet
- Pregnancy
- Drug
- Hereditary

PATHOGENESIS
Formation of Cholesterol Stones :
*Cholesterol and other lipids in the bile are not water soluble but have to be kept solubilized
*The mechanism of solubilization depends on formation of micelles
*Lipophilic core that carries cholesterol is surrounded by bile salts and lecithin

*Increasing cholesterol concentration or decreasing amount of bile salt and lecitin cause precipitation of cholesterol molecules to crystals

CLASSIFICATION

By size

*Big
*Small
*Microlithiasis
*Sludge :

- ✓ *Biliary sludge is a viscous gel (mucin, microscopic precipitates of multilamellar vesicles, cholesterol monohydrate, and calcium bilirubinate)
- ✓ *The formation of biliary sludge precedes the formation of macroscopic cholesterol gallstones

By type

1. Cholesterol (in 75-90% of patients) (Pale yellow, round to ovoid)
2. Pigment stones:

*Black :

- Contain 20-30% of cholesterol + calcium bilirubinate, carbonate, and phosphate
- secondary haemolytic disorder
- Form in the gallbladder

*Brown

- Secondary to bacterial infection
- Foreign bodies within the bile ducts (stents, sutures, parasites)
- (*Bacterial degradation products *Bacterial cell bodies *Calcium salts of fatty acids *Unconjugated bilirubin *Cholesterol)
- May form in the gallbladder or in the bile ducts

CLINICAL MANIFESTATION

***Biliary colic (**Steady, severe, right upper quadrant or epigastric abdominal pain that may radiate into the back)
***Dyspepsia and chronic pain** (belching, bloating, abdominal discomfort, heartburn, and food intolerances)
***Cardiac form** (thoracic retrosternal pain as in stenocardia)

DIAGNOSIS

1. *Ultrasonography
2. *Endoscopic Ultrasound (EUS)
3. *Computed Tomography
4. *Magnetic Resonance Imaging
5. *3D and Virtual Navigation Endoscopy
6. *Endoscopic Retrograde Cholangiography
7. *Cholangiography

TREATMENT

Asymptomatic patient :

1. Cholecystectomy is not routinely recommended
2. Prophylactic cholecystectomy is reasonable in:
   *children (long term physical presence of stones)
   *increased risk of gallbladder cancer (“porcelain” gallbladder , gallbladder polyps >1)
   *sickle cell anemia
   *simultaneously, during elective colonic operations, laparoscopic antireflux operations

Symptomatic patient :

1. Non surgery therapy (not use now a day due to cost and recurrence rate)
   *Extracorporeal shock wave lithotripsy
   *Dissolution of cholesterol gallstones by oral bile acids
2. Surgery
   ***Laparoscopic Cholecystectomy**
   - Grasping the Hartmann’s pouch and retracts it laterally to open the triangle of Calot
   - Clipping and dividing the cystic artery and duct
   - GB is dissected off from the liver bed

- Hospital stay 2-4 days

***Open Cholecystectomy**
***Minilaparotomy Cholecystectomy**
***SILS Cholecystectomy** (SILS - Single incision laparoscopic surgery)
***N.O.T.E.S. Cholecystectomy** (*N.O.T.E.S. – Natural orifice transluminal endoscopic surgery
*Absent of any incisions over abdominal wall)

25. Complications of gallstone disease. Clinical manifestations, diagnosis, treatment.

Complications (choledocholithiasis, cholangitis, biliary acute pancreatitis, etc.)
Choledocholithiasis

- ✓ Common bile duct stones (in 6 to 12% of patients with stones in the gallbladder)
- ✓ Secondary stones - form within the gallbladder and migrate to the CBD (Western countries)
- ✓ Primary stones - form straight within CBD, brown pigment type (Asian populations)

Complications
*Biliary colic (with spontaneous resolution) *Obstructive jaundice (progressive or intermittent)
*Cholangitis *Gallstone (biliary) pancreatitis

TREATMENT

1. Endoscopic Sphincterotomy and Stones Removal
2. Operative Stone Extraction (*If endoscopic treatment has failed)
3. Operative Stone Extraction (*Then, CBD is drained (most commony by the T-tube) for 1-2 weeks By 4 ways: 1. Vishnevskiy 2. T-tube 3. Pikovskiy 4. Halsted)
4. Choledochal Drainage Procedures Choledochoduodenostomy
   Indications:
   *Impossibility of the stone removal removal
   *Very dilated duct (> 1.5 cm)
   *Bile duct strictures : Choledochojejunostomy , Choledochoduodenostomy

Cholangitis

- ✓ Acute cholangitis is an ascending bacterial infection of the bile ducts with partial or complete obstruction of the bile ducts

Complications
Charcot's triad :
*fever
*epigastric or right upper quadrant pain
*jaundice
Reynolds pentad:
*fever, jaundice, right upper quadrant pain
*septic shock
*mental status changes

TREATMENT

- includes intravenous antibiotics and fluid resuscitation
- if first step not work emergency biliary decompression may be required endoscopically (sphincterotomy, drainage, stent) via the percutaneous transhepatic route, or surgically (decompression of the common bile duct with a tube).

DIAGNOSIS OF BOTH

1. Lab test- elevation of WBC, bilirubin, alkaline phosphatase, and aminotransferase
2. Ultrasonography
3. Endoscopic Ultrasound (EUS)

4. Computed Tomography
5. Magnetic Resonance Imaging
6. 3D and Virtual Navigation Endoscopy
7. Endoscopic Retrograde Cholangiography
8. Cholangiography

26. Acute calculous cholecystitis. Etiology and pathogenesis, clinical manifestations,diagnostics, treatment.

*Impacted gallstone obstruct the neck of the gallbladder or cystic duct for more than 6 hours
*Distension of the gallbladder
*Edema and thickening of the gallbladder wall
*Secondary bacterial infection

ETIOLOGY

- Your genes
- Your weight(obesity)
- Problems with your gallbladder(bile contains too much; cholesterol , bilirubin)
- Diet
- Pregnancy
- Drug
- Hereditary

PATHOGENESIS

Formation of Cholesterol Stones :
*Cholesterol and other lipids in the bile are not water soluble but have to be kept solubilized
*The mechanism of solubilization depends on formation of micelles
*Lipophilic core that carries cholesterol is surrounded by bile salts and lecithin
*Increasing cholesterol concentration or decreasing amount of bile salt and lecitin cause precipitation of cholesterol molecules to crystals

CLINICAL MANIFESTATION

*Right upper quadrant or epigastrium pain
*Abdominal guarding and local tenderness
*Anorexia, nausea and vomiting
*Inflammatory syndrome *Palpation of the enlarged gallbladder
*Murphy's sign - pain upon palpation of the right upper quadrant when the patient inhales deeply
*Grekov's sign – pain while hitting right low ribs
*Mussie's sign – irradiation of pain through diaphragmatic nerve

complication :

- ✓ Perforation of the Gallbladder - peritonitis
- ✓ Pericholecystic abscess (Sealed by the omentum)
- ✓ Empyema of the Gallbladder
- ✓ Mucocele of the Gallbladder (Lumen becomes filled with a clear or slightly milky fluid due to the mucous secretion of the epithelium)

DIAGNOSIS

1. Lab test- elevation of WBC, bilirubin, alkaline phosphatase, and aminotransferase
2. physical palpation (enlargement of gallbladder)
3. Ultrasonography
4. Endoscopic Ultrasound (EUS)
5. Computed Tomography
6. Magnetic Resonance Imaging
7. 3D and Virtual Navigation Endoscopy
8. Cholangiography

TREATMENT

1. Intravenous fluids, Antibiotics, Analgesia
2. Early cholecystectomy in 2 to 3 days
   ✓ Laparoscopic
   ✓ Open
3. Cholecystostomy when:
   patient unfit to cholecystectomy (severe comorbidities)

27. Choledocholithiasis. Etiology, clinical manifestations, diagnostics, differential diagnosis, treatment.

Choledocholithiasis

✓ Common bile duct stones (in 6 to 12% of patients with stones in the gallbladder)
✓ Secondary stones - form within the gallbladder and migrate to the CBD (Western countries)
✓ Primary stones - form straight within CBD, brown pigment type (Asian populations)

ETIOLOGY

1. **Stones**
   - Primary stone (brown pigmented , May form in the gallbladder or in the bile ducts)
   - Secondary stone (cholesterol)
   - Residual stones (which are missed at the time of cholecystectomy)
2. **Duodenal diverticulum**

CLINICAL MANIFESTATION

*Biliary colic (with spontaneous resolution)
*Obstructive jaundice (progressive or intermittent)
*Cholangitis
*Gallstone (biliary) pancreatitis

DIFFERENTIAL DIAGNOSIS

- Appendicitis
- Bile Duct Strictures
- Bile Duct Tumors
- Cholangiocarcinoma
- Cholecystitis
- Emergent Treatment of Gastroenteritis
- Gallbladder Cancer
- Pancreatic Cancer
- Peptic Ulcer Disease

DIAGNOSIS

1. Lab test- elevation of WBC, bilirubin, alkaline phosphatase, and aminotransferase
2. physical palpation (enlargement of gallbladder)
3. Ultrasonography
4. Endoscopic Ultrasound (EUS)
5. Computed Tomography
6. Magnetic Resonance Imaging
7. 3D and Virtual Navigation Endoscopy
8. Cholangiography

TREATMENT

1. Endoscopic Sphincterotomy and Stones Removal
2. Operative Stone Extraction (*If endoscopic treatment has failed)
3. Operative Stone Extraction (*Then, CBD is drained (most commony by the T-tube) for 1-2 weeks
   By 4 ways: 1. Vishnevskiy 2. T-tube 3. Pikovskiy 4. Halsted)
4. Choledochal Drainage Procedures Choledochoduodenostomy
   Indications:
   *Impossibility of the stone removal removal
   *Very dilated duct (> 1.5 cm)
   *Bile duct strictures : Choledochojejunostomy , Choledochoduodenostomy

28. Cholangitis. Etiology, clinical manifestation, diagnostics, treatment.

Cholangitis

✓ Acute cholangitis is an ascending bacterial infection of the bile ducts

Complications
Charcot's triad :
*fever
*epigastric or right upper quadrant pain
*jaundice
Reynolds pentad:
*fever, jaundice, right upper quadrant pain
*septic shock
*mental status changes

DIAGNOSIS

1. Lab test- elevation of WBC, bilirubin, alkaline phosphatase, and aminotransferase
2. Ultrasonography
3. Endoscopic Ultrasound (EUS)
4. Computed Tomography
5. Magnetic Resonance Imaging
6. 3D and Virtual Navigation Endoscopy
7. Endoscopic Retrograde Cholangiography
8. Cholangiography

TREATMENT

- includes intravenous antibiotics and fluid resuscitation
- if first step not work emergency biliary decompression may be required endoscopically (sphincterotomy, drainage, stent) via the percutaneous transhepatic route, or surgically (decompression of the common bile duct with a tube).

29. Stenosis of the major duodenal papilla. Etiology, classification, clinical manifestations, diagnosis, treatment.

common bile duct (**stenosis of the sphincter of Oddi**) is usually associated with inflammation, fibrosis, or muscular hypertrophy. The pathogenesis is unclear.

CLASSIFICATION

functional gallbladder disorder

functional biliary sphincter of Oddi disorder

ETIOLOGY

- trauma from the passage of stones
- sphincter motility disorders
- scarring
- congenital anomalies have been suggested.
- Episodic pain of the biliary type with abnormal liver function tests is a common presentation
- recurrent jaundice or pancreatitis also may play a role.

CLINICAL MANIFESTATION

- pain which may last 30 min
- vomiting , nausea
- feve
- common bile duct dilation
- pancreatitis
-

DIAGNOSIS

- endoscopic retrograde cholangiopancreatography examination
- ultrasonography
- CT

- MRI

TREATMENT
Endoscopicor operative sphincterotomy will yield good results.

30. Mirizzi syndrome. Etiology, clinical manifestation, diagnostics, treatment.
The impaction of a stone in the infundibulum of the gallbladder or cystic duct which causes obstruction of bile duct and common hepatic duct
ETIOLOGY
CLINICAL MANIFESTATION
- jaundice,
- fever,
- right upper quadrant pain
- vomiting , nausea

DIAGNOSIS

1. Lab test
2. Ultrasonography
3. Endoscopic Ultrasound (EUS)
4. Computed Tomography
5. Magnetic Resonance Imaging
6. 3D and Virtual Navigation Endoscopy
7. Endoscopic Retrograde Cholangiography
8. Cholangiography

TREATMENT
*Treatment - choledochal drainage procedures

31. Biliary fistulas and acute gallstone ileus. Etiology, clinical manifestation, diagnostics, treatment.
*The gallstone may erode into adjacent bowel, usually the duodenum, to cause cholecystoenteric fistula and obstruction in the small intestine
ETIOLOGY
- Stone of gallbladder
- Trauma
- Iatrogenic
- Penetrating injury

CLINICAL MANIFESTATION
- Pain
- Muscle spasm
- Vomiting
- Infection
- Peritonitis
- Ascites (accumulation of fluid in abdominal cavity)

DIAGNOSIS
1. Lab test
2. Ultrasonography
3. Endoscopic Ultrasound (EUS)
4. Computed Tomography
5. Magnetic Resonance Imaging
6. 3D and Virtual Navigation Endoscopy
7. Endoscopic Retrograde Cholangiography
8. Cholangiography

TREATMENT
*Treatment – stone removal and cholecystectomy

32. Small intestine diverticula. Definition, etiology, clinical manifestations. Principles of treatment.

are astructural abnormality that can occur from the esophagus to the rectosigmoid junction characterized by the presence of multiple saclike mucosal herniations through weak points in the intestinal wall.Small intestinal diverticula are far less than colonic diverticula.
Congenita (true). All three coats of the bowel are present in the wall of the diverticulum, e.g. Meckel's diverticulum.
Acquired (false). There is no muscularis layer present in the diverticulum, e.g. sigmoid diverticula.

ETIOLOGY :
Low-fiber diet
High-fat diet
Advancing age Heredity: No evidence indicates that heredity plays a role in the development of small bowel diverticula.
Systemic sclerosis
Visceral myopathy
Visceral neuropathy

CLINICAL MANIFESTATION
Diverticular pain
Bleeding
Diverticulitis – fever and localized tenderness
Intestinal obstruction (constipation , nausea , vomiting)
Perforation and localized absecessb
Malabsorbtion (diarrhea ,weight loss)
Anemia
Volvulus
Biliary colic
Flatulence (bacterial overgrowth)

TREATMENT
- Patients are frequently recommended to take a high-fibre diet and bulk-forming laxatives .
- **Acute diverticulitis** is treated by intravenous antibiotics
- An abscess can be drained percutaneously, 5 cm is frequently regarded as a cut off between an abscess likely to settle with antibiotics and one likely to require intervention.
- The aim of **emergency surgery** is to control peritoneal sepsis , neoplasm, perforation ,abscess, Laparotomy and thorough washout of contamination are performed and then a choice has to be made between a Hartmann's procedure (sigmoid resection with formation of left iliac fossa colostomy and closure of the rectal stump) and resection with colonic washout and anastomosis
- **Diverticular fistulae** can only be cured by resection of the affected bowel
- **Hemorrhage** from diverticular disease should be distinguished from angiodysplasia. It usually responds to conservative management and only occasionally requires resection

33. Intestine diverticula. Etiology and pathogenesis, incidence, clinical manifestations, diagnosis, treatment.

are astructural abnormality that can occur from the esophagus to the rectosigmoid junction haracterized by the presence of multiple saclike mucosal herniations through weak points in the intestinal wall.Small intestinal diverticula are far less than colonic diverticula

ETIOLOGY :
Low-fiber diet
High-fat diet
Advancing age Heredity: No evidence indicates that heredity plays a role in the development of small bowel diverticula.
Systemic sclerosis
Visceral myopathy
Visceral neuropathy

PATHOGENESIS
The cause of this condition is not known. It is believed to develop as the result of abnormalities in peristalsis, intestinal dyskinesis, and high segmental intraluminal pressures. The resulting diverticula emerge on the mesenteric border Diverticula are classified as true and false. True diverticula are composed of all layers of the intestinal wall, whereas false diverticula are formed from the herniation of the mucosal and submucosal layers the muscle layer is removed . Meckel diverticulum is a true diverticulum. Diverticula can be classified as intraluminal or extraluminal. Intraluminal diverticula and Meckel diverticulum are congenital. Extraluminal diverticula may be found in various anatomic locations and are referred to as duodenal, jejunal, ileal, or jejunoileal diverticula.

INCIDENCE
Low-fiber diet
High-fat diet
Advancing age Heredity: No evidence indicates that heredity plays a role in the development of small bowel diverticula.
Systemic sclerosis
Visceral myopathy
Visceral neuropathy

CLINICAL MANIFESTATION
Diverticular pain
Bleeding
Diverticulitis – fever and localized tenderness
Intestinal obstruction (constipation , nausea , vomiting)
Perforation and localized absecessb
Malabsorbtion (diarrhea ,weight loss)
Anemia
Volvulus
Biliary colic
Flatulence (bacterial overgrowth)

DIAGNOSIS

CT
Barium enemas (and colonoscopy/flexible sigmoidoscopy)
Ultrasonography
Nuclear imaging - Scanning with technetium-99m (99m Tc) – labeled red blood cells often is performed to locate the site of active gastrointestinal tract bleeding, and it may be helpful in evaluating bleeding due to diverticulosis
Angiograpy - Angiography may demonstrate diverticular hemorrhage as puddling or staining that persists beyond the capillary and venous phases. Embolization or the intra-arterial infusion of vasopressin may be used to treat gastrointestinal bleeding.

TREATMENT
- Patients are frequently recommended to take a high-fibre diet(avoid nut and popcorn) and bulk-forming laxatives .
- ***Acute diverticulitis*** is treated by intravenous antibiotics
- An abscess can be drained percutaneously, 5 cm is frequently regarded as a cut off between an abscess likely to settle with antibiotics and one likely to require intervention.
- The aim of ***emergency surgery*** is to control peritoneal sepsis , neoplasm, perforation ,abscess, Laparotomy and thorough washout of contamination are performed and then a choice has to be made between a *Hartmann's procedure* (sigmoid resection with formation of left iliac fossa colostomy and closure of the rectal stump) and *resection with* colonic washout *and anastomosis*
- ***Diverticular fistulae*** can only be cured by resection of the affected bowel
- ***Hemorrhage*** from diverticular disease should be distinguished from angiodysplasia. It usually responds to conservative management and only occasionally requires resection

34. Intestinal fistulas. Etiology, classifications, clinical manifestations, diagnosis, treatment.

A fistula is defined as an abnormal communication or opening between two epithelialized surfaces. The communication occurs between two parts of the GI tract or adjacent organs in an internal fistula (enterocolonic fistula or colovesicular fistula) or external fistula (enterocutaneous fistula or rectovaginal fistula) involves the skin or another external surface epithelium

ETIOLOGY

- Cancer
- Inflammatory bowel disease (crohn , ulcerative colitis, appendicitis)
- Bowel obstruction
- Diverticular disease
- Perforatin of duodenal ulcer
- surgical suture problems
- incision site problems
- an abscess
- an infection
- a hematoma, or blood clot under your skin
- a tumor
- malnutrition
- aortic aneurism or previous aortic surgery
- trouma (gun shot , accident)

CLASSIFICATION

1. Intestinal fistula (internal) In an intestinal fistula, gastric fluid leaks from one part of the intestine to the other where the folds touch. This is also known as a “gut-to-gut” fistula. E.g. enterocolonic fistula or colovesicular fistula

2. External fistula In an external fistula, gastric fluid leaks through the skin. It’s also known as a “cutaneous fistula.”

3. Extraintestinal fistula This type of fistula occurs when gastric fluid leaks from your intestine to your other organs, such as your bladder, lungs, or vascular system.

4. Complex fistula A complex fistula is one that occurs in more than one organ.

CLINICAL MANIFESTATION

- The most serious complication of GIF is sepsis
- abdominal
- pain painful

- bowel obstruction
- fever elevated
- white blood cell count
- diarrhea
- rectal bleeding
- a bloodstream infection or sepsis
- poor absorption of nutrients and weight loss
- dehydration worsening of the underlying disease

DIAGNOSIS

- serum test (leukositosis , obtain of albumin ,creatinine , electrolyte concentration which show nutrition status)
- Microbiology (Abscess culture)
- Urinalysis or urine culture(Urine culture findings may help to direct antibiotic therapy)
- Procedurse :
- Endoscopy or colonoscopy
- Fistuloscopy
- Cystoscopy
- Dye injection (Instilling methylene blue into the rectum)
- Imaging studying :
- CT scanning
- MRI
- Fistulography
- Ultrasonography

- Barium enema and small bowel series

TREATMENT

Medication therapy :

- Somatostatin Analogs : These agents inhibit the release of serotonin and the secretion of many hormones involved in GI function. Octreotide (Sandostatin)

- Immunosuppressant : Azathioprine (Imuran, Azasan) Infliximab (Remicade)

Surgery :

The surgical procedure for intestinal fistula treatment depends on the structures involved.

The basic surgical principles for treatment of all intestinal fistulas include the following:

The procedure involves resection of the intestinal segment, fistula tract, and the adjacent part of the involved.

In the absence of extensive infection or inflammation, primary anastomosis of the divided intestinal segments is done to reestablish GI continuity and repair of the involved structure to maintain function

In the presence of extensive infection or inflammation, the divided intestinal segments are exteriorized and the surgical procedure is modulated to allow replacement or maximal preservation of function

Depend on type of surgery ; end to end , bypass in case of dense adhesion , proximal and distal ends in case of sepsis , en-bloc resection in case of entroenteric fistula , in case of nephroentric fistula we choice nephrectomy and en-bloc resection fistula.

35. Crohn's disease. Clinical manifestations, diagnostics, differential diagnosis.Principles of conservative treatment, indications for surgery, types of operations.

chronic inflammatory process that can affect any part of the gastrointestinal tract from the mouth to the anus, mostly ileum . it cause by foods , diet , smoking , genetic factor .

CLINICAL MANIFESTATIONS

- *mild diarrhea* extending over many months
- vomiting , nausea
- rectal bleeding
- fever
- weight loos , malnutrition , bone loss , vit. Deficiency
- *pain* (particularly in the right iliac fossa)
- tender *mass* may be palpable
- Intermittent *fevers*
- secondary *anemia* and *weight loss* are common.
- After months of repeated attacks with acute inflammation the affected area of intestine begins to narrow with fibrosis , causing *obstructive symptoms*.

- With progression of the disease, **adhesions** and **transmural fissuring**, ***intra-abdominal abscesses and fistula tracts*** may develop
- Colonic CD presents with symptoms of *colitis* and *proctitis*
- ***perianal problems***. In the presence of active disease, the perianal skin appears bluish. Superficial ulcers with undermined edges are relatively painless and can heal with bridging of epithelium. Deep cavitating ulcers are usually found in the upper anal canal; they can be painful and cause perianal abscesses and fistulae, discharging around the anus and sometimes forwards into the genitalia
- gallston
- amyloidosis

DIAGNOSIS

Vital signs normal but tachycardia in anemic or dehydration
Laboratory : anemia , fall in serum albumin, magnesium, zinc and selenium , serology test , stool study
Endoscopy :colonoscopy , enteroscopy , ileocolonoscopy , laparoscopy
Image : x-ray
CT
Barium contrast

DIFFERENTIOAL DIAGNOSIS
- ulcerative colitis
- acute appendicitis

CONSERVATIVE TREATMENT
Medical therapy should always be considered as an alternative to surgery, although surgery should not be delayed when a clear indication for surgery exists
Patients must be optimised as far as possible prior to surgery, and this may require preoperative total parenteral nutrition

- steroid
- aminosalicylate
- antibiotic
- Monoclonal antibody
- Nutritional support

INDICATIONS FOR SURGERY

- recurrent intestinal obstruction
- bleeding
- perforation
- failure of medical therapy
- intestinal fistula
- fulminant colitis
- malignant change
- perianal disease.

TYPE OF SURGERY
- **Ileocaecal resection** is the usual procedure for terminal ileal Crohn's with a primary anastomosis between the ileum and the ascending or transverse colon, depending on the extent of the disease.

- **Segmental resection** of short segments of small or large bowel strictures can be performed.
- **Colectomy and ileorectal** anastomosis is commonly performed for colonic CD with rectal sparing and a normal anus.
- **Subtotal colectomy** and ileostomy for Crohn's. The indications are similar to those for UC.
- **Temporary loop ileostomy**. This can be used either in patients with acute distal CD, allowing remission and later restoration of continuity, or in patients with severe perianal or rectal disease.
- **Proctocolectomy**. Patients with colonic and anal disease failing to respond to medical treatment will eventually require a permanent ileostomy.
- **Strictureplasty**. Multiple strictured areas of CD can be treated by a local widening procedure, strictureplasty , to avoid small bowel resection.
- Anal disease should be treated conservatively to avoid sphincter injury by simple drainage of abscesses and the use of setons through fistulae.

36 . Upper and low gastrointestinal hemorrhage. Differential diagnosis.

37. Acute intestinal obstruction. Definition, etiology, pathogenesis, classification

Intestinal obstruction occurs when the normal forwarding and passage of intestinal contents does not occur. This obstruction can involve only the small intestine , the large intestine or both (generalized ileus). The "obstruction" can involve a mechanical obstruction or, in contrast, may be related to ineffective motility without any physical obstruction, so-called functional obstruction, "pseudo-obstruction," or ileus. The obstruction may be either 'open loop'- i.e. bowel content can escape proximally or 'closed loop', in which a segment of gut is obstructed at both ends.

Intestinal obstruction can also be **classified** according to :
etiopathogenesis (mechanical or functional obstruction),
time of presentation, and duration of obstruction (acute , subacute or chronic obstruction),
the extent of obstruction (patial or complete),
type of obstruction (simple, closed-loop, or strangulation obstruction).

Etiology :
Mechanical :
Luminal--- Gallstone (gallstone ileus) -Food bolus -Meconium ileus – faeces - faeces –gas – fluid filled .
Mural (wall) --- Stricture – Congenital – Inflammatory – Ischaemic – Neoplastic – Intussusception -diverticular
Extramural--- Adhesions – Congenital – Inflammatory – Malignant – Ischaemic – Hernia -Volvulus ((twisting) ---– Congenital – Acquired) – lymphoma

Pathogenesis
Proximal to an obstruction, intestinal contractions are increased in both magnitude and frequency. The bowel diameter increases and, because of this, contractions may eventually fail. The intestinal wall becomes oedematous and this, with reduced reabsorption of secretions, may cause extracellular volume deficiency. Strangulation causes blood loss into the affected loops and may produce hypovolaemia and, because of underperfusion of the affected segment, lactic acidosis occure .

Eventually a strangulated loop will die (usually within 4-6 hours) and rupture, with the production of a severe bacterial peritonitis which is often fatal.
Distension of the abdomen by the dilated loops may restrict diaphragmatic movement and so interfere with respiratory function. Vomiting may result in inhalation.

38. Functional intestinal obstructions. Etiology, clinical manifestations, diagnostics, differential diagnosis, treatment.

Etiology

Luminal--- Gallstone (gallstone ileus) -Food bolus -Meconium ileus – faeces –gas – fluid filled .
Mural (wall) --- Stricture – Congenital – Inflammatory – Ischaemic – Neoplastic – Intussusception -diverticular
Extramural--- Adhesions – Congenital – Inflammatory – Malignant – Ischaemic – Hernia -Volvulus ((twisting) ---- Congenital – Acquired) – lymphoma

Clinical manifestation

Pain- lower abdominal colic and localised, often severe pain because strangulated loop is in contact with the inner aspect of the abdominal wall.

Vomiting –initially upper-gastrointestinal obstruction contents of vomiting are food residue and dark greenish fluid but, unless the obstruction is in the upper jejunum, dark brown, bitter, foul smelling , In obstruction distal to the ileocaecal valve, vomiting may be absent because the small bowel can continue to propel its content into the distensible colon above the obstruction.

Distension – mostly in distal part.

Constipation,

Dehydration.

Diagnostic

1. General Physical findings :
    - Loss of water and electrolyte .
    - Temperature may be raised in a strangulating obstruction, in simple obstruction it is usually normal.
    - Distension- approximately related to the level of obstruction - the lower ,the obstruction in the small bowel; the greater , In large-bowel obstruction.
    - Palpation-show tenderness and guarding also feel mass in any part of abdomen , Auscultation- increase frequency of segmenting tikling and pitch sound , percussion -produce tympanic sound
    - Rectal and (in females) pelvic examination reveal pelvic mass .
2. Plain abdominal X-rays
3. Contrast X-rays
4. CT
5. endoscopy
6. Ultrasound
7. Air or barium enema: the doctor will insert air or liquid barium into the colon through the rectum. For intussusception in children, an air or barium enema can actually fix the problem most of the time

8. Haematological and biochemical test - A raised white cell count suggests either an active inflammatory cause or strangulation, as does a metabolic acidosis. The disturbance of water and electrolyte metabolism.

Treatment

1. non-operative (conservative)
2. operative

non- operative :
indication;

- strangulation or perforation as suggested by signs of hypovolaemia, systemic inflammatory response and peritoneal irritation
- incomplete obstruction which suggest non-progression, e.g. Crohn's disease in the small bowel
- some instances of complete small-bowel obstruction - the usual most suitable example is an adhesive obstruction.

Treatment ;

- proximal decompression by a nasogastric tube with aspiration either continuously or on a regular intermittent basis
- water and electrolyte replacement
- medication: anti nausea , pain killer , antibiotic (cephalosporin)
- therapeutic enema(push medication or tap water into bowel through rectum)
- repeated (4-6-hourly) evaluation of the clinical state - abdominal girth, development of tenderness, changes in bowel sounds and in cardiovascular status
- repeated plain X-rays or contrast studies and haematological and biochemical reassessment of the features of strangulation

operation :
indication ;

- established or suspected strangulation, including those with irreducible external hernia
- complete large-bowel obstruction with tenderness in the right iliac fossa - indicative of closed-loop obstruction with possible perforation of the caecum
- failure of resolution after a period of nonoperative management
- a cause (e.g. carcinoma) requiring surgical removal.
- Emergent surgery is indicated in incarcerated external hernia and when there is clinical and radiologic evidence of strangulation, gangrene, or perforation.

Treatment ;

At operation, the obstruction is relieved and, if possible, the underlying cause removed. Dead or damaged intestine must be excised. Occasionally an irremovable obstruction (e.g. a fixed neoplasm) is bypassed.

- In Gallstone 'ileus' :

1. **enterolithotomy** alone in unstable patients , **Interval cholecystectomy** with fistula repair can be performed in these patients once they have recovered from the acute episode .
2. The other surgical option in young and stable patient is to perform **enterolithotomy** , cholecystectomy, and fistula repair in one stage.

- Food bolus
- ilus
- Adhesions
- Volvulus - Operation is undertaken usually for small-bowel obstruction of unknown cause. **laparotomy**

- Sigmoid volvulus - A sigmoidoscope or colonoscope is introduced, a wide-bore flatus tube is passed along it and the sigmoid loop decompressed by careful negotiation of the obstructed loop.

- If the caecum is viable and the volvulus reduces, there are a number of options: • reduction alone - but this is associated with the highest risk of recurrence • **right hemicolectomy • caecostomy • caecopexy**

39. Treatment of acute intestinal obstruction. Treatment-and-diagnostics complex, indications for surgery, preoperative preparation, types of operations

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

Treatment-and-diagnostics complex :
DIAGNOSIS:

1. General Physical findings :

    - Loss of water and electrolyte .
    - Temperature may be raised in a strangulating obstruction, in simple obstruction it is usually normal.
    - Distension- approximately related to the level of obstruction - the lower ,the obstruction in the small bowel; the greater , In large-bowel obstruction.
    - Palpation-show tenderness and guarding also feel mass in any part of abdomen , Auscultation- increase frequency of segmenting tikling and pitch sound , percussion -produce tympanic sound
    - Rectal and (in females) pelvic examination reveal pelvic mass .

2. Plain abdominal X-rays
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7. Air or barium enema: the doctor will insert air or liquid barium into the colon through the rectum. For intussusception in children, an air or barium enema can actually fix the problem most of the time
8. Haematological and biochemical test - A raised white cell count suggests either an active inflammatory cause or strangulation, as does a metabolic acidosis. The disturbance of water and electrolyte met

TREATMENT :
Emergent surgery is indicated in incarcerated external hernia and when there is clinical and radiologic evidence of strangulation, gangrene, or perforation.

- In case of complete doctor may recommend surgery the obstruction with a self-expanding metal stent. The wire mesh tube is inserted into your colon via an endoscope passed through your mouth or colon. It forces open the colon so that the obstruction can clear. Stents are generally used to

treat people with colon cancer or to provide temporary relief in people for whom emergency surgery is too risky

- In case of pseudo-obstruction prescribe medication that causes muscle contractions, if not effective use surgery , n cases where the colon is enlarged, a treatment called decompression may provide relief. Decompression can be done with colonoscopy, a procedure in which a thin tube is inserted into your anus and guided into the colon.

INDICATION FOR SURGERY ;

- established or suspected strangulation, including those with irreducible external hernia
- complete large-bowel obstruction with tenderness in the right iliac fossa - indicative of closed-loop obstruction with possible perforation of the caecum
- failure of resolution after a period of nonoperative management
- a cause (e.g. carcinoma) requiring surgical removal.
- Emergent surgery is indicated in incarcerated external hernia and when there is clinical and radiologic evidence of strangulation, gangrene, or perforation.

PREOPERATIVE PREPARATI ;
CT
ultrasound These tests may or may not include a barium enema,
(which involves inserting a small amount of contrast material into your rectum to help better visualize the structures).
sigmoidoscopy or a colonoscopy

type of operation :

- Surgical resection: Removal of the obstruction is necessary when there is a mass, such as a tumor.
- Removal of adhesions: If you have scar tissue squeezing your intestines from the outside, this often requires careful incisions to cut them away, although scar tissue can return again.
- Stent placement: A stent, which is a tube that holds the intestine open, may be placed inside the intestine to allow passage of food and stool and to prevent another blockage. This may be necessary when a bowel obstruction is recurrent or when the intestines are severely damaged.
- Colostomy/ ileostomy: If your intestines are damaged or inflamed, a permanent or temporary ileostomy or colostomy, which is an artificial opening in your abdomen for waste or stool evacuation, may be needed. Sometimes, these are temporary structures needed to prevent a severe gastrointestinal infection from spreading throughout the body. However, it is possible that the ends of the intestines cannot be reconnected, and these openings may be needed for the long term.
- Revascularization: Ischemic colitis may require revascularization, which is repair of the blocked blood vessels that supply blood to the intestines.

40. Differential diagnosis of gastroduodenal and large intestine hemorrhage.
41. Etiology and pathogenesis of perforated peptic ulcer. Clinical manifestations, diagnostics, treatment.

Ulcer perforations due to peptic ulcer disease are located mostly in the stomach or duodenum. This is imbalance between the protective and the ulcerogenic factors is obvious in ulcer formation

ETIOLOGY

- H. pylori
- NSAIDs (non-steroidal Anti-inflammatory Drugs)
- Genetic factor
- Lifestyle factors
- Severe physiological stress (emotional ,stress, CNS trauma, hypotension , respiratory failure , systemic illness)
- Smoking

PATHOGENESIS
Mechanisms and factors in pathogenesis of perforated peptic ulcer :
(A) an imbalance between between hostile and protective factors start the ulcerogenic process, and
(B) although many contributors are known, helicobacter infection and use of non-steroidal anti-inflammatory drugs appear of importance in disturbing the protective mucosal layer and
(C) expose the gastric epithelium to acid. Several additional factors :

(D) may augment the ulcerogenic process (such as smoking, alcohol and several drugs) that lead to erosion
(E). Eventually, the serosal lining is breached
(F), and when perforated, the stomach content, including acidic fluid reach to muscularis mucosa, then will enter the abdominal cavity giving rise to intense pain, local peritonitis that may become generalized and eventually lead to a systemic inflammatory response syndrome and sepsis with the risk of multiorgan failure and mortality.

CLINICAL MANIFESTATION

Primary phase

- **0-2 h after oncet**
- suden severe abdominal pain (stab pain) sometimes syncope in epigastric region Pain may radiate to the top of the right or both shoulders
- The abdomen exhibits a board-like rigidity
- The abdomen does not move with respiration
- Acid fluid in the peritoneal cavity releases vasoactive mediators causing : tachycardia, cool extremities, and a low temperature.
-
- last only a few minutes up to 2 h

Second phase

- abdominal pain is better getting only due to worse with movement
- Rectal examination is often tender as is palpation
- duration of the second phase is usually 2–12 h after primary onset.
-

Third phase

- The third phase usually begins more than 12 h after onse
- increasing abdominal distension is noted, but abdominal pain, tenderness, and rigidity may be less evident than in phase one.
- Temperature and hypovolemia due to third-spacing develop, tachycardia worsens, and hypovolemic shock may occur

DIAGNOSIS

- Detailed history (Epigastric abdominal pain? Drugs (NSAIDs, Aspirin)
- Physical examination (Abdominal rigidity? Bowel movements? Tenderness ?)
- Blood sample Leucocytes, CRP
- erect plain chest or abdomen radiograph (abdomen X-ray ; free air?)
- In case of the absence of the air, the endoscopy with following x-ray film should be performed
- All patients should have serum amylase performed, as distinguishing between peptic ulcer, perforation and pancreatitis can be difficult.
- CT
- MRI

TREATMENT

- Intravenous fluids, stabilization of hemodynamic instability, and nasogastric suction are the first steps of the treatment strategy, which have to be applied early on.
- Nonoperative management, including parenteral nutrition and antibiotics, may be successful in well-selected patients.

the treatment is principally surgical:

- Gastrectomy (Billroth II)
- gastroenterostomy
- Alternatively, laparoscopy may be used.
- truncal vagotomy and pyloroplasty
- The most important component of the operation is a thorough peritoneal cavity to remove all of the fluid and food debris.
- All patients should be treated with systemic antibiotics in addition to a thorough peritoneal lavage

- (Operative Treatment :The objective of therapy for perforation is prompt closure of the perforation in the duodenum or stomach. In the stomach, ulcers should be excised to rule out a malignancy, while in the duodenum, closure without excision is the usual practice. Usually, sutures placed in seromuscular fashion across the site of perforation are sufficient for secure closure. Some groups tie in a tag of omentum with these sutures to prevent a leakage of the suture line, but there is little evidence for this. Thorough lavage of the peritoneal cavity with 10–20 l fluid is an essential part of the operation. Laparoscopic repair appears to be a reasonable option for patients with a history less than 24 h, with no hypovolemic shock, and with a perforation not more than 6 mm. In up to 25%, conversion to an open operation is necessary. Morbidity and mortality seem to be comparable in published series, but larger randomized studies are still lacking.)

42. Sealed perforated ulcer. Clinical manifestations, diagnosis, therapy, indications for surgery, types of operations.

Sometimes perforations ulcer will **seal owing to the inflammatory response and adhesion within the abdominal cavity** (sealed perforation), and so the perforation may be self-limiting

ETIOLOGY

- H. pylori
- NSAIDs (non-steroidal Anti-inflammatory Drugs)
- Genetic factor
- Lifestyle factors
- Severe physiological stress (emotional ,stress, CNS trauma, hypotension , respiratory failure , systemic illness)
- Smoking

PATHOGENESIS

Primary phase

- **0-2 h after oncet**
- suden severe abdominal pain (stab pain) sometimes syncope in epigastric region Pain may radiate to the top of the right or both shoulders
- The abdomen exhibits a board-like rigidity
- The abdomen does not move with respiration
- Acid fluid in the peritoneal cavity releases vasoactive mediators causing : tachycardia, cool extremities, and a low temperature.
-
- last only a few minutes up to 2 h

Second phase

- abdominal pain is better getting only due to worse with movement
- Rectal examination is often tender as is palpation
- duration of the second phase is usually 2–12 h after primary onset.
-

Third phase

- The third phase usually begins more than 12 h after onse
- increasing abdominal distension is noted, but abdominal pain, tenderness, and rigidity may be less evident than in phase one.
- Temperature and hypovolemia due to third-spacing develop, tachycardia worsens, and hypovolemic shock may occur

Mechanisms and factors in pathogenesis of perforated peptic ulcer :
(A) an imbalance between between hostile and protective factors start the ulcerogenic process, and
(B) although many contributors are known, helicobacter infection and use of non-steroidal anti-inflammatory drugs appear of importance in disturbing the protective mucosal layer and
(C) expose the gastric epithelium to acid. Several additional factors :
(D) may augment the ulcerogenic process (such as smoking, alcohol and several drugs) that lead to erosion
(E). Eventually, the serosal lining is breached
(F) perforations ulcer will **seal owing to the inflammatory response and adhesion within the abdominal cavity** (sealed perforation), and so the perforation may be self-limiting

CLINICAL MANIFESTATION

Primary phase

- **0-2 h after oncet**
- suden severe abdominal pain (stab pain) sometimes syncope in epigastric region Pain may radiate to the top of the right or both shoulders
- The abdomen exhibits a board-like rigidity
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Second phase

- abdominal pain is better getting only due to worse with movement
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Third phase

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- increasing abdominal distension is noted, but abdominal pain, tenderness, and rigidity may be less evident than in phase one.
- Temperature and hypovolemia due to third-spacing develop, tachycardia worsens, and hypovolemic shock may occur

✓ abdom

DIAGNOSIS

- Detailed history (Epigastric abdominal pain? Drugs (NSAIDs, Aspirin)
- Physical examination (Abdominal rigidity? Bowel movements? Tenderness ?)
- Blood sample Leucocytes, CRP
- erect plain chest or abdomen radiograph (abdomen X-ray ; free air?)
- In case of the absence of the air, the endoscopy with following x-ray film should be performed
- All patients should have serum amylase performed, as distinguishing between peptic ulcer, perforation and pancreatitis can be difficult.
- CT
- MRI

TREATMENT

- Intravenous fluids, stabilization of hemodynamic instability, and nasogastric suction are the first steps of the treatment strategy, which have to be applied early on.
- Nonoperative management, including parenteral nutrition and antibiotics, may be successful in well-selected patients.

the treatment is principally surgical:

- Gastrectomy (Billroth II)
- gastroenterostomy
- Alternatively, laparoscopy may be used.
- truncal vagotomy and pyloroplasty
- The most important component of the operation is a thorough peritoneal cavity to remove all of the fluid and food debris.

43. Atypical perforated peptic ulcer. Clinical manifestations, diagnosis, differential diagnosis, surgery.

the most common site of perforation is the anterior aspect of the duodenum. However, the anterior or incisural gastric ulcer may perforate and, in addition, gastric ulcers may perforate into the **lesser sac or to retroperitoneal space** (atypical perforation), which can be particularly difficult to diagnose

ETIOLOGY

- pylori

- NSAIDs (non-steroidal Anti-inflammatory Drugs)
- Genetic factor
- Lifestyle factors
- Severe physiological stress (emotional ,stress, CNS trauma, hypotension , respiratory failure , systemic illness)
- Smoking

PATHOGENESIS

Mechanisms and factors in pathogenesis of perforated peptic ulcer :
(A) an imbalance between between hostile and protective factors start the ulcerogenic process, and
(B) although many contributors are known, helicobacter infection and use of non-steroidal anti-inflammatory drugs appear of importance in disturbing the protective mucosal layer and
(C) expose the gastric epithelium to acid. Several additional factors :
(D) may augment the ulcerogenic process (such as smoking, alcohol and several drugs) that lead to erosion
(E) Eventually, the serosal lining is breached
(F) gastric ulcers may perforate into the **lesser sac or to retroperitoneal space** (atypical perforation), which can be particularly difficult to diagnose

CLINICAL MANIFESTATION

DIAGNOSIS

- Detailed history (Epigastric abdominal pain? Drugs (NSAIDs, Aspirin)
- Physical examination (Abdominal rigidity? Bowel movements? Tenderness ?)
- Blood sample Leucocytes, CRP
- erect plain chest or abdomen radiograph (abdomen X-ray ; free air?)
- In case of the absence of the air, the endoscopy with following x-ray film should be performed
- All patients should have serum amylase performed, as distinguishing between peptic ulcer, perforation and pancreatitis can be difficult.
- CT
- MRI

TREATMENT

- Intravenous fluids, stabilization of hemodynamic instability, and nasogastric suction are the first steps of the treatment strategy, which have to be applied early on.
- Nonoperative management, including parenteral nutrition and antibiotics, may be successful in well-selected patients.

the treatment is principally surgical:

- Gastrectomy (Billroth II)
- gastroenterostomy
- Alternatively, laparoscopy may be used.
- truncal vagotomy and pyloroplasty
- The most important component of the operation is a thorough peritoneal cavity to remove all of the fluid and food debris.

44. Pathogenesis of peptic ulcer hemorrhage. Clinical manifestations, methods of examination and evaluation of blood loss volume.

PATHOGENESIS

Bleeding occurs more commonly from ulcers in the duodenum rather than the stomach, The two most common causes of PUD are: infection with Helicobacter pylori and the use of nonsteroidal anti-inflammatory drugs (NSAIDs

often due to posterior erosion into the gastroduodenal artery. Patients with bleeding peptic ulcers present commonly with hematemesis with or without melena. If the bleeding is massive, hematochezia may occur. Nasogastric lavage yielding blood or ‘‘coffee-ground’’ material confirms bleeding.

CLINICAL MANIFESTATION

- ✓ abdominal pain
- ✓ Patients with duodenal ulcer often experience pain 2 to 3 hours after a meal and at night
- ✓ nausea,

- ✓ bloating,
- ✓ weight loss,
- ✓ stool positive for occult blood,
- ✓ anemia.

METHOD OF EXAMINATION AND EVALUATION OF BLOOD LOSS VOLUME

- ✓ Initial assessment must include a medication history to determine the use of NSAIDs, anticoagulants, and anti-platelet agents.
- ✓ Vital signs are obtained to document signs of hypovolemia and shock.
- ✓ After vital signs have stabilized, the patient should undergo an **esophagogastroduodenoscopy (EGD)** as the diagnostic test of choice.
- ✓ Initial endoscopy should be performed emergently in high-risk patients
- **The ulcer can be graded by the Forrest classification**:
- ✓ Ia - Spurting active bleeding
- ✓ Ib - Nonspurting active bleeding
- ✓ IIa - Nonbleeding ‘‘visible vessel’’
- ✓ IIb - Nonbleeding ulcer with overlying clot
- ✓ IIc - Nonbleeding ulcer with hematin covered base
- ✓ III - Clean ulcer base with no signs of bleeding

- The risk of recurrent bleeding can be predicted based on ulcer size (>1 cm) as well as the endoscopic appearance of the ulcer bed

45. Principles of treatment of peptic ulcer hemorrhage. Medical, endoscopic and surgical treatment.

TREATMENT

- EGD is not only the diagnostic test of choice, but also acts as the potential first line therapeutic option.
- Medical treatment has limited efficacy. All patients are commonly started on either an **H2-receptor antagonist or a proton pump inhibitors .**
- Therapeutic **endoscopy** can achieve hemostasis in approximately 70 per cent of cases. The three major modalities used during endoscopic therapy are : **injection therapy, thermocoagulation, and mechanical therapy.**
  - ✓ Injection therapy acts primarily by tamponade due to the volume effect with a secondary pharmacologic effect dependent on the agent used (Epinephrine ,ethanol, Thrombin, fibrin)
  - ✓ Thermocoagulation :involves the use of a probe positioned on the bleeding site to provide local tamponade followed by application of heat or electrocoagulation to achieve coagulative coaptation.
  - ✓ Mechanical therapy : include endoclip placement or band ligation.

(Therapeutic endoscopy will probably never be effective in patients who are bleeding from large vessels and with which the majority of the mortality is associated. In patients where the source of bleeding cannot be identified or in those who rebleed after endoscopy, angiography with transcatheter embolization may offer a valuable alternative to surgery in expert centers.)

- it is often possible to close the mucosa over the ulcer. The pyloroplasty is then closed with interrupted sutures in a transverse direction as in the usual fashion. In a giant ulcer the first part of the duodenum may be destroyed making primary closure impossible. In this circumstance one should proceed to Billroth gastrectomy or Roux-en-Y reconstruction.
- Gastrectomy in some case is require

46. Etiology and pathogenesis of peptic ulcer penetration. Clinical manifestations, diagnostics, treatment.

Ulceration of stomach and duodenum ,imbalance between the protective and the ulcerogenic factors fainaly ulcer erodes into another organ such as the liver or the pancreas instead of into the peritoneal cavity. Penetration occurs in order of frequency

Ø into the pancreas, Ø gastrohepatic omentum, Ø biliary tract, Ø liver, Ø greater omentum, Ø mesocolon, Ø colon, and vascular structures.
ETIOLOGY

- H. pylori
- NSAIDs (non-steroidal Anti-inflammatory Drugs)
- Genetic factor
- Lifestyle factors
- Severe physiological stress (emotional ,stress, CNS trauma, hypotension , respiratory failure , systemic illness)
- Smoking
- perforated ulcer

PATHOGENESIS
(A) an imbalance between between hostile and protective factors start the ulcerogenic process, and
(B) although many contributors are known, helicobacter infection and use of non-steroidal anti-inflammatory drugs appear of importance in disturbing the protective mucosal layer and
(C) expose the gastric epithelium to acid. Several additional factors :
(D) may augment the ulcerogenic process (such as smoking, alcohol and several drugs) that lead to erosion
(E). Eventually, the serosal lining is breached
(F), and when perforated, the stomach content, including acidic fluid, will penetrate to near organs such as pancreas, liver , biliary tract

CLINICAL MANIFESTATION

- perivisceral abscess
- erosion into vascular structures leading to exsanguinating haemorrhag
- erosion into the cystic artery
- haematobilia
- Fistulization into the pancreatic duct has also been reported with penetrating duodenal ulcer.

DIAGNOSIS

- Oesophagogastroduodenoscop
- CT
- MRI
- Detailed history (Epigastric abdominal pain? Drugs (NSAIDs, Aspirin)
- Physical examination (Abdominal rigidity? Bowel movements? Tenderness ?)
- Blood sample Leucocytes, CRP
- erect plain chest or abdomen radiograph (abdomen X-ray ; free air?)
- In case of the absence of the air, the endoscopy with following x-ray film should be performed
- All patients should have serum amylase performed, as distinguishing between peptic ulcer, perforation and pancreatitis can be difficult

TREATMENT
the treatment is principally surgical:

- Gastrectomy (Billroth II)
- gastroenterostomy
- Alternatively, laparoscopy may be used.
- truncal vagotomy and pyloroplasty
- The most important component of the operation is a thorough peritoneal cavity to remove all of the fluid and food debris.
- All patients should be treated with systemic antibiotics in addition to a thorough peritoneal lavage

47. Etiology and pathogenesis of gastric outlet obstruction. Classification, clinical manifestations of the disease depending on its stage.
48. Diagnostics of gastric outlet obstruction. Differential diagnosis, indications for surgery, types of operations.
49. Peritonitis. Definition, classification, principles of diagnostics.
50. Treatment of peritonitis. Preoperative preparation, operative treatment, principles of

antimicrobial treatment.
51. Limited peritonitis (subhepatic, subphrenic, Douglas space, interintestinal abscesses). Causes, clinical presentation, diagnosis, treatment
52. Peritonitis due to acute appendicitis. Medical and surgical treatment, complications.
53. Aneurysms of the thoracic aorta. Classifications, hemodynamic disturbances, clinical manifestations, diagnosis, indications for surgery.
Aortic aneurysm is defined as a permanent, localized dilatation of the aorta to a diameter that is at least 50% greater than is normal at that anatomic level
The normal aorta derives its elasticity and tensile strength from the medial layer, which contains approximately 45 to 55 lamellae of elastin, collagen, smooth muscle cells, and ground substance
Any alteration in this delicate balance can lead to aortic disease

CAUSE :

1. Atherosclerosis
2. Nonspecific medial degeneration - is the most common cause
   •Weakening of the medial layer
   •The underlying causes of medial degenerative disease remain unknown (age???).
3. Aortic dissection
4. Poststenotic dilatation
5. Trauma
6. Genetic disorder :
   - Marfan syndrome - an autosomal dominant genetic disorder of connective tissue
   - Loeys-Dietz syndrome
   - Ehlers-Danlos syndrome - – Includes a spectrum of inherited connective tissue disorders of collagen synthesis.
   - Familial aortic aneurysms
   - Congenital bicuspid aortic valve

CLASSIFICATION

1. By type :
   “False" aneurysm (pseudoaneurysms) are leaks in the aortic wall that cause blood to collect in pouches of scar tissue on the exterior of the aorta
   “True“ aneurysm ;
2. By shape :
   Fusiform (symmetrical dilatations of the aorta)
   Saccular (localized outpunching of the aorta)
3. By anatomy :

- Root Aneurysms
- Ascending aorta
- Transverse aortic arch
- Descending thoracic aortic aneurysms
- Thoracoabdominal aneurysms

CLINICAL MANIFESTATIONS

1. Patients often are asymptomatic
2. Compression or erosion of adjacent structures
   chest discomfort and mild chronic precordial pain and; the pain radiate to the neck and jaw, mimicking angina.
3. compression of the superior vena cava, the pulmonary artery, the airway, or the sternum
4. stretching the recurrent laryngeal nerve, which results in left vocal cord paralysis and hoarseness
5. complication :

- Severe chest pain
- Collapse
- Cardiac tamponade
- Aortic valve regurgitation – progressive heart failure
- Distal embolism – occlusion and thrombosis of the visceral, **renal**, or lower-extremity branches
- Rupture – severe hemorrhagic shock

DIAGNOSIS

- Ultrasonography
- Angiography , Echocardiography
- Computed tomography (CT) and CT angiography (CTA)
- Magnetic resonance imaging (MRI)

INDICATIONS FOR OPERATION

- the diameter of an ascending AA is >5.5 cm
- the diameter of a descending AA is >6.5 cm
- the rate of dilatation is >1 cm/y
- In patients with connective tissue disorders, such as Marfan and Loeys-Dietz syndromes and rate of growth (5.0 cm for the ascending aorta and 6.0 cm for the descending thoracic aorta).
- valve regurgitation

Surgery :

1. cardiopulmonary bypass

2. Open Repair :

➢ Graft replacement of the tubular portion of the ascending aorta
➢ Graft replacement of the entire proximal aorta, including the aortic root, and reattachment of the coronary arteries and brachiocephalic branches

3. Endovascular Repair :
   *Balloon catheter technique
   *Expandable stent

4. Hybrid Repair : include the elimination of cardiopulmonary bypass, circulatory arrest, and cardiac ischemia

54. Aneurysms of abdominal aorta. Classifications, hemodynamic disturbances, clinical manifestations, diagnosis, indications for surgery.

The most common location of aortic aneurysms is the infrarenal aorta The natural history of an AAA is to **expand and rupture**

DIAGNOSIS

- history and physical examination
- Ultrasonography
- Angiography , Echocardiography
- Computed tomography (CT) and CT angiography (CTA)
- Magnetic resonance imaging (MRI)
- ECG , CBC

CLASSIFICATION

1. By type :
   “False" aneurysm (pseudoaneurysms) are leaks in the aortic wall that cause blood to collect in pouches of scar tissue on the exterior of the aorta
   “True“ aneurysm ;
2. By shape :

Fusiform (symmetrical dilatations of the aorta)
Saccular (localized outpunching of the aorta)

3. By location mostly infra-renal aorta

CLINICAL MANIFESTATIONS
*Most are asymptomatic, and they are usually found incidentally during work-up for chronic back pain
*Rarely, patients present with a tender pulsation mass in abdomen
*The patient should be immediately taken to the open repair
*Ruptured AAA should be suspected when a patient :

- is hemodynamically unstable
- has acute back pain and/or syncope,
- has history of AAA or a pulsation abdominal mass

INDICATION FOR SURGERY
*Infrarenal abdominal aortic aneurysms should be repaired in men when the diameter reaches 5.5 cm and in women when the diameter reaches 5.0 cm
*Rapid expansion of >0.5 cm within 6 months can be considered a relative indication for elective repair

TREATMENT :

- Infrarenal abdominal aortic aneurysms should be repaired in men when the diameter reaches 5.5 cm and in women when the diameter reaches 5.0 cm
- Rapid expansion of >0.5 cm within 6 months can be considered a relative indication for elective repair
- Prosthetic grafts –bifurcated graft for an aortobi-iliac or aortobifemoral bypass reconstruction
- Stent graft that is fixed proximally and distally to the nonaneurysmal aortoiliac segment
- Endovascular Repair :
  *Balloon catheter technique
  *Expandable stent

- in case of renal artery repair use : Aortorenal bypass , Hepatorenal and splenorenal bypass ,Endarterectom

55. Aortic dissection and rupture. Classifications, hemodynamic disturbances, clinical manifestations, diagnosis, indications for surgery.

AORTIC DISSECTION
Progressive separation of the aortic wall layers that usually occurs after a tear forms in the intima and inner media.
As the separation of the layers of the media propagates, at least two channels form:
the original lumen, which remains lined by the intima and which is called the true lumen, and the newly formed channel within the layers of the media, which is called the false lumen
The dissecting membrane separates the true and false lumens.
ETIOLOGY :
*Any condition that weakens the aortic wall increases the risk of aortic dissection (smoking, hypertension, atherosclerosis, and hypercholesterolemia, connective tissue disorders, aortitis, bicuspid aortic valve, preexisting medial degenerative disease)
*Aortic injury during cardiac catheterization or surgery
*The thoracic aortic aneurysm may cause dissection
*But in most cases, dissection occurs in patients without aneurysms.

CLASSIFICATION :
*Ascending aorta
*Descending aorta

*Entire aorta
- Acute < 14 days after the initial tear
- Chronic > 14 days

HEMODYNAMIC DISTURBANCES
Outer wall of the false lumen is extremely thin, inflamed, and fragile, which makes it prone to expansion (aneurysm) or rupture
The expanding false lumen can compress the true lumen and branch vessels (coronary, carotid, intercostal, visceral, renal, and iliac arteries) and cause **malperfusion syndrome**
When the separation of layers occurs within the aortic root, the aortic valve commissures can become unhinged, which results in acute **valvular regurgitation .**

CLINICAL MANIFESTATIONS
- Severe chest or back pain ("tearing“) that migrates distally as the dissection progresses
- Dyspnea
- Dysphagia
- Abdominal pain

***Malperfusion syndrome:**
*cardiac ischemia (coronary arteries),
*stroke (brachiocephalic arteries),
*paraplegia or paraparesis (intercostal arteries),
*mesenteric ischemia (superior mesenteric artery),
*kidney failure (renal arteries),
*limb ischemia or loss of motor function (brachial or femoral arteries)

DIAGNOSIS
- history and physical examination
- Ultrasonography
- Angiography , Echocardiography
- Computed tomography (CT) and CT angiography (CTA)
- Magnetic resonance imaging (MRI)
- ECG , CBC

INDICATION FOR SURGERY
*aortic rupture,
*rapidly expanding aortic diameter,
*uncontrolled hypertension,
*persistent pain despite adequate medical therapy
* pre-existing aneurysm
*Acute malperfusion (percutaneous interventions have largely replaced open surgery)

TREATMENT
- pharmacologic management to prevent rupture (anti hypertension: beta blocher , propranol and for pain management : narcotics and opiates) special for chronic aortic dissection
- pharmacologic management of acute descending aortic dissection results in lower morbidity and mortality rates than surgical treatment does
- open surgery :
  - ❖ Emergent graft replacement of the ascending aorta , Repairs performed in the chronic phase has better outcomes than in the acute phase
  - ❖ Placement of a stent graft in the true lumen

RUPTURE of AAA

Rupture risk appears to be directly related to aneurysm size , The rupture risk is quite low for aneurysms <5.5 cm and begin after this size

CLINICAL MANIFESTATIONS
*Most are asymptomatic, and they are usually found incidentally during work-up for chronic back pain
*Rarely, patients present with a tender pulsation mass in abdomen
*Ruptured AAA should be suspected when a patient :
• is hemodynamically unstable
• has acute back pain and/or syncope,
• has history of AAA or a pulsation abdominal mass
*The patient should be immediately taken to the open repair

treatment :
Prosthetic grafts –bifurcated graft for an aortobi-iliac or aortobifemoral bypass reconstruction
Stent graft that is fixed proximally and distally to the nonaneurysmal aortoiliac segment

56. Non-atherosclerotic arterial diseases (Takayasu's arteritis, thromboangiitis obliterans, fibromuscular dysplasia, congenital abnormalities, Raynaud's syndrome).
57. Acute and chronic mesenteric ischemia. Etiology, clinical manifestations, diagnostics, treatment.
58. Aortoiliac and lower extremity occlusive disease. Etiology, classification, clinical manifestations, diagnostics, treatment.
59. Acute arterial obstruction. Definition, etiology, pathogenesis, clinical manifestations, diagnostics, treatment.
60. Acute limb ischemia. Definition, incidence, etiology and pathogenesis, classification, clinical picture.
61. Principles of treatment of acute limb ischemia. Indications for surgery, types of operations, medical treatment.
62. Ulcerative colitis. Principles of diagnostics and medical treatment, indications for surgery, types of operations.

63. Hemorrhoids. Etiology, clinical manifestations, diagnostics, treatment.

Definition :
Hemorrhoids are swollen blood vessels in the lower rectum. They are among the most common causes of anal pathology Hemorrhoids are not varicosities; they are clusters of vascular tissue (eg, arterioles, venules, arteriolar-venular connections), smooth muscle (eg, Treitz muscle), and connective tissue lined by the normal epithelium of the anal canal.
We have external and internal :
Most external -are defined by their original location below the dentate line.
hemorrhoids are the external concomitants of internal hemorrhoids, so apart from thrombosis,
Internal hemorrhoids are the anal cushions defined by their original internal location—above the dentate line — that are prolapsing and/or bleeding which has 4 degrees :

DEGREE	DESCRIPTION
First	Hemorrhoids bleed but do not prolapse
Second	Hemorrhoids prolapse on straining but reduce spontaneously
Third	Hemorrhoids prolapse and require manual reduction
Fourth	Prolapsed hemorrhoids cannot be manually reduced

ETIOLOGY

Hemorrhoids develop when the supporting tissues of the anal cushion deteriorate, or as a result of excessive downward pressure, as in prolonged straining at stool or pregnancy.
Decreased venous return
Constipation
Portal hypertension and anorectal varices
Other risk factors:
Lack of erect posture
Familial tendency
Higher socioeconomic status
Chronic diarrhea
Colon malignancy
Hepatic disease
Obesity Loss of rectal muscle tone
Rectal surgery
Episiotomy
Anal intercourse Inflammatory bowel disease, including ulcerative colitis, and Crohn disease

CLINICAL MANIFESTATION

common complaints of burning, itching, swelling, and pain result from pruritus ani, anal abrasion, anal fissure, thrombosed external hemorrhoids, or prolapsed anal papilla.

The most common manifestations of internal haemorrhoids are painless, bright red rectal

bleeding associated with bowel movements

DIAGNOSIS

1. Physical examination to see :

Redundant tissue
Fissures
Fistula
Sign of infection or abscess formation
Rectal or hemorrhoids prolapse appearing as bluish , tender perianal mass

2. Hematological test
3. Anoscopy and flexible sigmoidoscopy

TREATMENT
Conservative treatment :
Medical management is the initial treatment of choice for grade I internal and nonthrombosed external hemorrhoids. It consists of warm baths , a high-fiber diet , adequate fluid intake , stool softeners , topical and systemic analgesics , proper anal hygiene , and in some cases, a short course of topical steroid cream.
Medical :
Stool softener :
Docusate sodium (Colace ,dulcolax , correctol)
Mild astringent :
Hamamelis water
Analgesic for pain control :
Acetaminophen

Nonsurgical treatment :

- Rubber band ligation is suitable for symptomatic first-, second-, and some third-degree internal hemorrhoids that do not respond to fiber supplementation.
- Sclerotherapy
- Infrared photocoagulation, laser coagulation, electrosurgery.
- Hemorrhoidectomy

Surgical treatment :

- Thrombosed External Hemorrhoids - Treatment is aimed at relief of pain, prevention of recurrent thrombosis
- hemorrhoidectomy.
- Stapled hemorrhoid surgery/procedure for prolapsing hemorrhoids (PPH)

- Doppler-guided transanal hemorrhoidal dearterialization
- Hemorrhoidal artery ligation and rectoanal repair

64. Hemorrhoids complications. Definition, etiology, classification, clinical manifestations, diagnostics, treatment.

Definition :
Hemorrhoids are swollen blood vessels in the lower rectum. They are among the most common causes of anal pathology Hemorrhoids are not varicosities; they are clusters of vascular tissue (eg, arterioles, venules, arteriolar-venular connections), smooth muscle (eg, Treitz muscle), and connective tissue lined by the normal epithelium of the anal canal.
We have external and internal :
Most external -are defined by their original location below the dentate line.
hemorrhoids are the external concomitants of internal hemorrhoids, so apart from thrombosis,
Internal hemorrhoids are the anal cushions defined by their original internal location—above the dentate line — that are prolapsing and/or bleeding which has 4 degrees :

DEGREE	DESCRIPTION
First	Hemorrhoids bleed but do not prolapse
Second	Hemorrhoids prolapse on straining but reduce spontaneously
Third	Hemorrhoids prolapse and require manual reduction
Fourth	Prolapsed hemorrhoids cannot be manually reduced

ETIOLOGY

Hemorrhoids develop when the supporting tissues of the anal cushion deteriorate, or as a result of excessive downward pressure, as in prolonged straining at stool or pregnancy.
Decreased venous return
Constipation
Portal hypertension and anorectal varices
Other risk factors:
Lack of erect posture
Familial tendency
Higher socioeconomic status
Chronic diarrhea
Colon malignancy
Hepatic disease
Obesity Loss of rectal muscle tone
Rectal surgery
Episiotomy
Anal intercourse Inflammatory bowel disease, including ulcerative colitis, and Crohn disease

CLINICAL MANIFESTATION

common complaints of burning, itching, swelling, and pain result from pruritus ani, anal abrasion, anal fissure, thrombosed external hemorrhoids, or prolapsed anal papilla.
The most common manifestations of internal haemorrhoids are painless, bright red rectal bleeding associated with bowel movements

DIAGNOSIS

1. Physical examination to see :

- Redundant tissue
- Fissures
- Fistula
- Sign of infection or abscess formation
- Rectal or hemorrhoids prolapse appearing as bluish , tender perianal mass

2. Hematological test
3. Anoscopy and flexible sigmoidoscopy

TREATMENT

Conservative treatment :

Medical management is the initial treatment of choice for grade I internal and nonthrombosed external hemorrhoids. It consists of warm baths , a high-fiber diet , adequate fluid intake , stool softeners , topical and systemic analgesics , proper anal hygiene , and in some cases, a short course of topical steroid cream.

Medical :

Stool softener :

Docusate sodium (Colace ,dulcolax , correctol)

Mild astringent :

Hamamelis water

Analgesic for pain control :

Acetaminophen

Nonsurgical treatment :

- Rubber band ligation is suitable for symptomatic first-, second-, and some third-degree internal hemorrhoids that do not respond to fiber supplementation.
- Sclerotherapy
- Infrared photocoagulation, laser coagulation, electrosurgery.
- Hemorrhoidectomy

Surgical treatment :

- Thrombosed External Hemorrhoids - Treatment is aimed at relief of pain, prevention of recurrent thrombosis
- hemorrhoidectomy.
- Stapled hemorrhoid surgery/procedure for prolapsing hemorrhoids (PPH)
- Doppler-guided transanal hemorrhoidal dearterialization
- Hemorrhoidal artery ligation and rectoanal repair

65. Anal fissure. Definition, etiology, classification, clinical manifestations, diagnostics, treatment.

DEFINITION

Anal fissure is an ulcer in the lower anal canal in the short term, usually involves only the epithelium and, in the long term, involves the full thickness of the anal mucosa. Fissures are acute or chronic, primary or secondary.

ETIOLOGY , classification :

A primary fissure arises without association to other local or systemic disease, whereas a secondary fissure may be due to Crohn disease or ulcerative colitis. Atypical anal ulcers, may be due to HIV, anal cancer, tuberculosis, syphilis, or hematologic malignancy

The exact etiology of anal fissures is unknown, but the initiating factor is thought to be trauma from the passage of a particularly hard or painful bowel movement. Low-fiber diets, inflammatory of bowel disease

CLINICAL MANIFESTATION

Anal pain, particularly during and after bowel movement, is the most prominent symptom, followed by bleeding with defecation.
Skin tag or small lump of skin
Itching
Visible tear around anus skin
Constipation increase the complain .

DIAGNOSIS
Anal examination
Anoscopy

TREATMENT
Initial treatment of acute anal fissure is **pain relief**, with warm sitz baths to relax the anal canal. **Application of topical anesthetic gel or ointment directly to the fissure** before bowel movement is sometimes helpful. **Bulking and softening the stool** with fiber supplementation and increased water intake (8–10 glasses/day) .

For chronic anal fissure, a reasonable first treatment is the application of 0.2% nitroglycerine or 2% diltiazem ointment directly to the fissure. Botulinum toxin A injection into the internal sphincter is another approach to reduce internal anal sphincter hypertonicity
Surgery :

Lateral internal sphincterotomy - The purpose of the operation is to cut the hypertrophied internal sphincter with or without fissurectomy , thereby releasing tension and allowing the fissure to heal .

Sphinger dilation : This procedure is a controlled anal stretch or dilatation under general anesthetic. It is performed because one of the causative factors for anal fissure is thought to be a tight internal anal sphincter. Stretching the tight sphincter helps correct the underlying abnormality, thus allowing the fissure to heal.

66. Surgical treatment of anal fissure.

Surgery :

Lateral internal sphincterotomy - The purpose of the operation is to cut the hypertrophied internal sphincter with or without fissurectomy , thereby releasing tension and allowing the fissure to heal .

Sphinger dilation : This procedure is a controlled anal stretch or dilatation under general anesthetic. It is performed because one of the causative factors for anal fissure is thought to be a tight internal anal sphincter. Stretching the tight sphincter helps correct the underlying abnormality, thus allowing the fissure to heal.

67. Perirectal abscess. Definition, etiology, classification, clinical manifestations, diagnostics, treatment.

In the wall of the anal canal, four to ten anal glands open directly into the anal crypts at the dentate line. Infection of the anal glands, blocked duct or orifice, is the origin of perianal abscesses. Because the anal glands lie between the internal and external sphincter muscles, an inter-sphincteric abscess forms first. The infection may then spread to the anal crypts at the dentate line
ETIOLOGY :

1. Both aerobic and anaerobic bacteria:
- The anaerobes most commonly implicated are Bacteroides fragilis, Peptostreptococcus, Prevotella, Fusobacterium, Porphyromonas, and Clostridium.
- The aerobes most commonly implicated are Staphylococcus aureus , Streptococcus, and Escherichia coli

2. Other cause including Crohn disease, trauma, immunodeficiency resulting from HIV infection or malignancy (both hematologic and anorectal cancer), sexually transmitted diseases, radiation therapy, foreign bodies, perforated diverticular disease, inflammatory bowel disease.

CLASSIFICATION :
Abscess classified according to their anatomy location , are:

- perianal (subcutaneous),
- ischioanal (ischiorectal),
- intersphincteric, submucous, and
- supralevator (pelviorectal)

CLINICAL MANIFESTATION

- severe anal pain, aggravated by activity.
- Swelling is usually prominent, and skin changes .
- Some patients develop urinary retention.
- Fistula formation
- Bacteremia and sepsis

DIAGNOSIS

- Lab. Test
- CT , Ultrasound , MRI
- Endoscopy

TREATMENT

- the anorectal abscess must be **drained** as soon as possible. In addition, **antibiotics** should be considered.
- **Perianal abscesses** are the most superficial and the easiest to treat. They are usually small and can be drained under local anesthesia. the incision should be made as close to the anus as is compatible with safety.Pus is collected and sent for culture. Hemostasis is achieved with manual pressure, and the wound is packed with iodophor gauze.
- **An ischioanal** abscess causes a diffuse swelling of the ischioanal fossa, and drainage can be done wherever the abscess is most superficial.
- **Supralevator** abscess is uncommon and difficult to diagnose. Because it is adjacent to the abdominal cavity,
  If the abscess is from upward extension of an intersphincteric abscess, it should be drained into the rectum.
  If the supralevator abscess arises from the upward extension of an ischioanal abscess, it should be drained through the ischioanal fossa**.**

68. Fistula-in-ano. Definition, etiology, classification, clinical manifestations,diagnostics, treatment.

Fistula-in-ano is a *chronic form of perianal abscess* that is spontaneously or surgically drained
Fistula - in - ano which form an abnormal hollow tract or cavity that is lined with granulation tissue and that connects a **primary opening** inside the anal canal to a **secondary opening** in the perianal skin

ETIOLOGY
•previous anorectal abscess
•secondary to trauma (eg, rectal foreign bodies), Crohn disease, anal fissures, carcinoma, radiation therapy, actinomycoses, tuberculosis, and lymphogranuloma venereum secondary to chlamydial infection.

CLASSIFICATION

1. Intersphincteric
2. Transsphincteric
3. Suprasphincteric
4. Extrasphincteric

CINICAL MANIFESTATION

- Perianal discharge
- Pain
- Swelling
- Bleeding
- Skin excoriation
- External opening
- Inflammatory bowel disease
- Abdominal pain
- Weight loss
- Sepsis
- Change in bowel habits

DIAGNOSIS

1. Imaging study :
- Fistulography
- Endoanal or endorectal ultrasonography
- MRI
- Computed tomography
2. Barium enema/small bowel series
3. Proctosigmoidoscopy/colonoscopy

TREATMENT

1. If patients are without symptoms and a fistula is found no therapy is required.
2. If fistula because of Acute perianal abscess requires incision and drainage.
3. **Panproctocolectomy**-Recurrent fistulous disease to the rectum and perineum with persistent anorectal sepsis .
4. **Fistulotomy** : A probe is passed into the tract through the external to internal openings Granulation tissues are curetted and the edges of the wound
5. **istulectomy**, the excision of the fistulous track, has no advantages over fistulotomy, which is lay open of the track.
6. **Seton placement** - require for : Complex fistulas Recurrent fistulas after previous fistulotomy Anterior fistulas in female patients Poor preoperative sphincter pressures Patients with Crohn disease or patients who are immunosuppresse Stages : 1. Cutting 2.draining / fibrosing
7. **Mucus advancement flap**: use in patients with chronic high fistula , A U-shaped flap of mucosa is created to eradicate the internal opening.
8. **Lift procedure** : It is performed by accessing the intersphincteric plane with the goal of performing a secure closure of the internal opening and by removing the infected cryptoglandular tissue

69. Pilonidal sinus. Definition, etiology, classification, clinical manifestations,diagnostics, treatment.

small groups of hairs and debris get trapped in the pores of the skin below the coccyx (natal cleft , 5 cm aboveanus) and form a "sinus," or cyst , that grows to become an abscess that have some tendency to recur.

ETIOLOGY

- Obesity
- Sedentary life style
- Mostly in main
- Thick body hair
- Family history
- Previous pilonidal cyst

CLASSIFICATION

- Acute
- Chronic
- Complex or recurrent pilonidal disease

CLINICAL MANIFESTATION

- fever
- pain to the top of the buttocks,
- swelling,
- redness,
- discharge of blood or pus (if abscess ruptures or "pops"), and
- foul-smelling odor.

DIAGNOSIS

- physical examination - A pilonidal cyst looks like a lump, swelling, or abscess
- in case of infection CBC
- imaging studies are not routinely obtained however, there have been reports of complicated disease progressing to osteomyelitis, necrotizing fasciitis, toxic shock syndrome

TREATMENT

- One of the simplest medical treatments of pilonidal sinuses in case of absent of abscess is to shave the sacral area free of hair and to pluck all visible embedded hair in the sinus.
- Fibrin glue and phenol injections- in case of chronic type ,Phenol sterilizes the sinus tract and removes embedded hair after that use fibrin glue for early healing .
- Antibiotics have a limited role in the treatment of either acute or chronic pilonidal disease, though oral or intravenous agents may be considered in patients with significant cellulitis, underlying immunosuppression, or concomitant systemic.
- Laser ablation of the pilonidal sinus is receiving interest.
- A pilonidal abscess is managed by incision, drainage, and curettage of the abscess cavity to remove hair nests and skin debris.
- Flap-based procedures may be performed, especially in the setting of complex and multiple-recurrent chronic pilonidal disease when other techniques have failed

70. Rectal prolapse. Definition, etiology, classification, clinical manifestations,diagnostics, treatment.

Rectal prolapse is when the rectum -- the bottom part of your large intestine -- drops down towards or outside your anus which usually starts in the anterior rectum.

ETIOLOGY

1. Paitent with history of: constipation and straining or, less commonly, chronic diarrhea.
2. Pregnancy
3. Previous surgery
4. Cystic fibrosis
5. Benign prostatic hypertrophy
6. Several abnormalities are consiste :

- Abnormally deep rectovaginal
- Unusually redundant sigmoid colon
- Lack of normal fixation of the rectum and an elongated mesorectum

CLASSIFICATION

Acute
Chronic

CLINICAL MANIFESTATION

- Transabdominal rectopexy involves a full mobilization of the rectum to the level of the pelvic floor musculature with suture fixation of the mesorectum to the presacral fascia below the sacral promontory.
- Perineal rectosigmoidectomy (Altemeier operation) is a transanal approach in which the prolapsed rectum and redundant sigmoid colon are excised endorectally
- The Delorme procedure
- two general approaches to surgery for rectal prolapse – abdominal operations (through the belly) and perineal operations (through "the bottom") **,**A reasonable approach to the problem of rectal prolapse is to use a perineal operation for older, sicker patients and an abdominal approach for younger, healthier ones.

71. Colon diverticular disease. Etiology, classification, clinical manifestations, diagnosis,treatment.

Diverticular disease of the colon begins as diverticulosis (colonic outpouchings), which may develop into diverticulitis (diverticular inflammation and perforation).
Diverticula usually occur adjacent to the vasa recta, (the small vessels that extend into the submucosa, because these are the weakest areas of the colonic wall.) Therefore, diverticula usually occur on the mesenteric side of the colon.

ETIOLOGY

- mechanical
- when naturally weak places in your colon give way under pressure.
- inflammatory disease
- Low-fiber diet
- High-fat diet
- Advancing age Heredity

CLASSIFICATION:
Diverticula involve the :

- sigmoid colon : 95% ,
- The cecum : 5% of patients
- Complex (rare)

DIAGNOSIS

- CT
- Barium enemas (and **colonoscopy**/flexible sigmoidoscopy)
- Ultrasonography
- Nuclear imaging - Scanning with technetium-99m (99m Tc) – labeled red blood cells often is performed to locate the site of active gastrointestinal tract bleeding, and it may be helpful in evaluating bleeding due to diverticulosis
- Angiograpy - Angiography may demonstrate diverticular hemorrhage as puddling or staining that persists beyond the capillary and venous phases. Embolization or the intra-arterial infusion of vasopressin may be used to treat gastrointestinal bleeding.

TREATMENT

- Nonsurgical treatment includes oral or intravenous antibiotics and diet modification
- Image-guided percutaneous drainage is usually the most appropriate treatment for stable patients with large diverticular abscesses.
- After resolution of the incident diverticulitis, colonoscopy should be performed
- In case of sigmoid diverticulum Urgent sigmoid colectomy is required for diffuse peritonitis or when nonsurgical management fails.
- **Hemorrhage** from diverticular disease should be distinguished from angiodysplasia. It usually responds to conservative management and only occasionally requires resection

72. Complicated diverticular disease. Clinical manifestations, diagnostics, differential diagnosis, treatment.

are astructural abnormality that can occur from the esophagus to the rectosigmoid junction haracterized by the presence of multiple saclike mucosal herniations through weak points in the intestinal wall.Small intestinal diverticula are far less than colonic diverticula.

CLINICAL MANIFESTATION
Diverticular pain
Bleeding
Diverticulitis – fever and localized tenderness
Intestinal obstruction (constipation , nausea , vomiting)
Perforation and localized absecessb
Malabsorbtion (diarrhea ,weight loss)
Anemia
Volvulus
Biliary colic
Flatulence (bacterial overgrowth)

DIAGNOSIS

CT
Barium enemas (and colonoscopy/flexible sigmoidoscopy)
Ultrasonography
Nuclear imaging - Scanning with technetium-99m (99m Tc) – labeled red blood cells often is performed to locate the site of active gastrointestinal tract bleeding, and it may be helpful in evaluating bleeding due to diverticulosis
Angiograpy - Angiography may demonstrate diverticular hemorrhage as puddling or staining that persists beyond the capillary and venous phases. Embolization or the intra-arterial infusion of vasopressin may be used to treat gastrointestinal bleeding.

DIFFERENTIAL DIAGNOSIS
- Acute gastritis
- Acute pancreatitis
- Acute pyelonephritis
- Cholangitis
- Cholecystitis
- Chronic mesenteric ischemia
- Inflammatory bowel disease
- Gynecologic pain
- Ovarian cyst
- Recto vaginal fistula
- Liver abscess
- Urinary tract infection
- Urinary tract obstraction
- Viral gastroenteritis

TREATMENT
- Patients are frequently recommended to take a high-fibre diet(avoid nut and popcorn) and bulk-forming laxatives .
- **Acute diverticulitis** is treated by intravenous antibiotics
- An abscess can be drained percutaneously, 5 cm is frequently regarded as a cut off between an abscess likely to settle with antibiotics and one likely to require intervention.
- The aim of **emergency surgery** is to control peritoneal sepsis , neoplasm, perforation ,abscess, Laparotomy and thorough washout of contamination are performed and then a choice has to be made between a Hartmann's procedure (sigmoid resection with formation of left iliac fossa colostomy and closure of the rectal stump) and resection with colonic washout and anastomosis
- **Diverticular fistulae** can only be cured by resection of the affected bowel
- **Hemorrhage** from diverticular disease should be distinguished from angiodysplasia. It usually responds to conservative management and only occasionally requires resection

73. Patent ductus arteriosus. Hemodynamic disturbances, clinical manifestations, diagnostics, treatment.

Patent ductus arteriosus (PDA), in which there is a persistent communication between the descending thoracic aorta and the pulmonary artery that results from failure of normal physiologic closure of the fetal ductus, is one of the more common congenital heart defects.

HEMODYNAMIC DISTURBANCE

- Left-to-right shun
- Right ventricle overload volum
- Low diastolic pressure
- Pulmonary hypertension may develop within the first year of life
- If untreated - irreversible pulmonary vascular disease (Eisenmenger's syndrome) with the ultimate result of pulmonary and right heart failure (only treatable by pulmonary-heart transplantation)

CLINICAL MANIFESTATION

- At first when they born they may have not any respiratory and heart problem
- Tachycardia,
- tachypnea,
- ventricular hypertrophy
- in case of untreated right heart failure (edema of internal organ)
- Poor eating, which leads to poor growth
- Sweating with crying or eating

DIAGNOSIS

- physical examination
- CBC(polycythemia ,high level of red blood cell)
- Pulse oximetry
- ECG(T- wave inversion and ST segment depression) ischemia
- Echo
- Chest radiography
- Cardiac catheterization and angiography

TREATMENT

- In premature infants, aggressive intervention with indomethacin or ibuprofen to achieve early closure of the PDA is beneficial
- mechanical closure (surgically, thoracoscopy, or catheterbased) .

74. Atrial and ventricular septal defect. Hemodynamic disturbances, clinical manifestations, diagnostics, treatment.

ATRIAL SEPTAL DEFECT
is characterized by a defect in the interatrial septum allowing pulmonary venous return from the left atrium to pass directly to the right atrium.

HEMODYNAMIC DISTURBANCE
*Left-to-right shunting through the defect.
*Increase in pulmonary blood flow
*Volume overload in the RA and RV
*RA and RV hypertrophy
*Irreversible pulmonary vascular disease (Eisenmenger's syndrome)

CLINICAL MANIFESTATION

- Dyspenia
- Tachycardia
- Fatigue
- arrhythmia

- pulmonary hypertention
- cyanosis
- cyncop
- stroke
- infected endocarditis

DIAGNOSIS
- CBC ,Biological test
- Chest radiography
- Transthoracic echocardiography
- MRI (identify the size and position)
- ECG(prolonged P-R interval , high monophasic R wave and deedly inverted T wave)
- Oxiometry

TREATMENT
- In general, surgical closure in 4 and 5 years of age (earlier in symptomatic patients, even in infancy)

VENTRICULAR SEPTAL DEFECT

Is Opening between the LVs and RVs
A.Type I (conal, infundibular, supracristal, subarterial)
B. Type II (perimembranous) (80%)
C. Type III (atrioventricular canal type or inlet septum type).
D. Type IV (single or multiple).

HEMODYNAMIC DISTURBANCE

*Left-to-right shunt from the LV to the RV
*Elevating RV pressure to the level of systemic
*Increase in PA blood flow
*If untreated, severe pulmonary hypertension
*Eisenmenger's syndrome
*Reversal of flow (right-to-left shunt)
*In small defect, RV pressure is either normal or minimally elevated (asymptomatic course)

CLINICAL MANIFESTATION
The symptoms and physical findings depend on the size of the defect and the magnitude of the left-to-right shunt.
- IF SMALL –may have mild or no symptom
- Tachypenia at rest or feeding
- Fatigue
- In exercise may have : cyanosis , cyncop , hemoptysis , chest pain , dyspenia

DIAGNOSIS
- CBC ,Biological test
- Chest radiography
- Transthoracic echocardiography
- MRI (identify the size and position)
- ECG(violation in QRS wave
- Oxiometry

TREATMENT
- Asymptomatic small or moderate-size VSD may be observed may not
- Small VSD may close or narrow spontaneously (1 month of age - 80% chance, 12 months of age 25%) CPB
- Medication therapy :
  diuretic (Furosemide) use to trat hypertention , hear failure

Angiotensin Converting Enzyme inhibitor (ACE)(CAPTOPRIL), vasoconstrictor, redusing systemic afterload
- Large defects or severe symptoms should be repaired Requirement in the use of CPB(cardiopulmonary bypass)
- Transcatheter closer

75. Abnormal pulmonary venous drainage. Hemodynamic disturbances, clinical manifestations,diagnostics, treatment.

HEMODYNAMIC DISTURBANCE
Abnormal drainage of the pulmonary veins into the right heart (instead of LA) and Mixing of venous and arterial blood right atrium it cause Cyanosis and severe hypoxemia (PO2 < 20mmHg) with metabolic acidosis . also Blood returns to the left heart through an Atrial Septal Defect(ASD), which is almost uniformly present

CLINICAL MANIFESTATION
- Tachypnea
- Tachycardia
- Cyanosis
- Pulmonary hypertension due to low blood flow and cynosis
- Present systolic murmur

DIAGNOSIS
- Lab test (impair oxygenated ,acid base status)
- ECG(violation of QG pattern in right lead ,enlargement of right atrium)
- Chest radiography
- ECHO
- MRI

TREATMENT
- Anastomosis of the common pulmonary venous channel to the left atrium
- Closure of the Atrial Septal Defect (ASD)

- Medication : Nitric oxide (usful as a pulmonary dilator) post operative

76. Disease of Fallot. Hemodynamic disturbances, clinical manifestations, diagnostics, treatment.
HEMODYNAMIC DISTURBANCE

Tetralogy of Fallot (TOF) is a congenital heart defect with four components :
1. Large (VSD) ventricular septal defect - a large perimembranous VSD adjacent to the tricuspid valve
2. an overriding aorta;
3. RV outflow obstruction (hypoplasia and dysplasia of the pulmonary valve, or obstruction at the subvalvular and PA level
4. and right ventricular hypertrophy.

CLINICAL MANIFESTATION
- Mild **cyanosis** at a birth, which then progresses (Cyanosis usually becomes significant within the first 6 to 12 months of life)
- **hypoxemia**, when pulmonary blood flow is decreased with elevation of systemic vascular resistance (triggered by fever or vigorous physical activity)
- Evaluation in the older patient with tetralogy of fallot may **demonstrate clubbing (drumstick fingers), polycythemia, hemoptysis, or brain abscesses**
- Heart failure

DIAGNOSIS

- Physical examination in the older patient with demonstrate clubbing, polycythemia, or brain abscesses.
- Chest radiography will demonstrate a boot-shaped heart, and ECG will show the normal pattern of right ventricular hypertrophy.
- Echocardiography confirms the diagnosis because it demonstrates the position and nature of the Ventricular Septal Defect , defines the character of the Right ventricular outflow tract (RVOT) obstruction, and often visualizes the branch pulmonary arteries and the proximal coronary arteries.
- Occasionally, aortography is necessary to delineate the coronary artery anatomy.
- ECG
- Cardiac catheterization

TREATMENT

- Medication therapy :

  Vasodilator(Alprostadil)- The use of a vasodilator will reduce systemic vascular resistance, allowing more forward flow, improving cardiac output.

  Diuretics (furosemide)- decrease plasma volume and edema by causing diuresis.

  B-blocker (metoprolol)- Beta-blocker therapy is used to reduce right ventricular infundibular spasm.
- Systemic-to-pulmonary shunts may be preferred with an unstable neonate younger than 6 months of age (palliation)
- The use of early shunt procedures provided blood to the lungs by using the higher pressure arterial supply (aorta) to the lower pressure lung arteries Treatment
- **Complete repair :**
- Systemic-to-pulmonary arterial shunts are ligated
- The pulmonary artery outflow obstruction is relieved
- Patch closure of the VSD

77. Aortic coarctation. Hemodynamic disturbances, clinical manifestations, diagnostics, treatment.

*Luminal narrowing in the aorta
*Obstruction to blood flow.
*Extensive collateral circulation
*Involvement of the intercostals and mammary arteries.

HEMODYNAMIC DISTURBANCE

*LV outflow obstruction,
*Pulmonary overcirculation
*Biventricular failure
*Proximal (upper body) systemic hypertension
*Hypoperfusion-induced activation of the renin– angiotensin–aldosterone system increases hypertension

CLINICAL MANIFESTATION

- Circle of Willis aneurysms,
- aortic dissection and rupture,
- myocardial infarction
- Early life: Congestive heart failure, severe acidosis, or poor perfusion to the lower body.
- Beyond infancy: Usually none; however, hypertension, headache, nosebleed, leg cramps, muscle weakness, cold feet, or neurologic changes may be seen

DIAGNOSIS

- Chest radiography
- Barium esophagography
- Echocardiography
- MRI
- ECG
- Cardiac catheterization

TREATMENT

*In hemodynamically significant COA the routine management is surgical (in all ages)
*The most common surgical techniques in current use are resection with end-to-end anastomosis
*The subclavian flap aortoplasty

*Prosthetic materials: patch aortoplasty, interposition tube graft, shunting
*Catheter-based intervention: balloon dilatation and primary stent implantation

78. Diagnostic methods of surgical cardiac diseases.

1. **Electrocardiogram**
   *The ECG is used to detect rhythm disturbances, heart block, atrial or ventricular hypertrophy, ventricular strain, myocardial ischemia, and MI.
2. **Chest X-ray**
   *Standard and lateral chest x-rays are excellent for determining cardiac enlargement and pulmonary hypertension and congestion
3. **Echocardiography**
   *Transthoracic Echocardiography (ultrasound of the heart) - the most widely used cardiac diagnostic study
   *It can evaluate cardiac size, wall motion, and valvular pathology and oth.
4. **Transesophageal Echocardiography**
   *Ultrasound transducer in a flexible endoscope
   *The image more precise that in a transthoracic study
   *It can assess the condition of the aorta and main vessels also (Atherosclerotic disease, coarctation, etc.)
5. **Radionuclide Studies**
   *Thallium scan
   *The study also gives excellent, specific information about the patient's cardiac functional status

6. **Radionuclide Studies**
   *Gated blood pool scan (equilibrium radionuclide angiocardiography) using technetium-99m
   *It can detect areas of hypokinesis and measure left ventricular ejection fraction (LVEF), endsystolic volume, and end-diastolic volume

7. **Positron Emission Tomography**
   *The positron emission tomography (PET) scan is a radionuclide imaging technique used to assess myocardial viability in underperfused areas of the heart
8. **Magnetic Resonance Imaging**
   *Magnetic resonance imaging (MRI) may be used to delineate the extent of MI and to distinguish between reversible and irreversible myocardial ischemic injury
9. **Cardiac Catheterization-**
   *Measurement of intracardiac pressures and cardiac output,
   *localization and quantification of intracardiac shunts,
   *determination of internal cardiac anatomy and ventricular wall motion by cineradiography, and
   *determination of coronary anatomy by coronary angiography.

10. **Computed Tomography Angiography**

79. Acquired mitral valve disease. Hemodynamic disturbances, clinical manifestations,diagnostics, treatment.

1) Mitral stenosis - rheumatic heart disease.

rheumatic fever occurs as a complication of group A streptococcal infection. Other rare causes of include carcinoid causes, systemic lupus erythematosus, rheumatoid arthritis, and some mucopolysaccharidoses.

HEMODYNAMIC DISTURBANCE
Pathology - Elevation in left atrial pressure
*Atrial fibrillation with clot formation and embolization
*The LV function usually remains normal
*Pulmonary venous congestion and hypertension

*Right-sided heart failure (jugular venous distention, hepatomegaly, ascites, or ankle edema)

CLINICAL MANIFESTATION

- Mild mitral stenosis does not usually cause symptoms.
- Eventually the disorder progresses and people develop symptoms such as:
- Shortness of breath, especially with exertion or when you lie down
- Fatigue, especially during increased physical activity
- Swollen feet or legs
- Heart palpitations — sensations of a rapid, fluttering heartbeat
- Dizziness or fainting
- Coughing up blood
- Chest discomfort or chest pain
- Those people with a low level of oxygen in the blood and high blood pressure in the lungs may have a plum-colored flush in the cheeks (called mitral facies).
- Pulmonary edema
- Pulmonary hypertension
- Myocardial infarction

TREATMENT

*Open mitral commissurotomy

*Percutaneous balloon valvuloplasty

2) Mitral insufficiency

(Degenerative diseases, rheumatic heart disease (15 to 20%), ischemic disease (15 to 20%), endocarditis, congenital abnormalities, and cardiomyopathy)
Regurgitation of a portion of the LV stroke volume into the left atrium
*↓ forward blood flow from LV
*Elevated pressure and dilation of the LA, atrial fibrillation
*Pulmonary congestion
*Volume overload of the LV
*Atrial fibrillation
*Dilation of the LV

TREATMENT OF MITRAL INSUFFICIENCY
*Surgery is recommended in any symptomatic patient
*Valve replacement is most likely in patients with longstanding rheumatic disease
*Mitral valve reconstruction include resection of the posterior leaflet, chordal shortening/transposition / artificial replacement, **annuloplasty**

DIAGNOSIS OF BOTH DISEASE

- complete blood cell (CBC) count,
- electrolyte status,

- renal and liver function tests. Chest radiography
- Echocardiography
- MRI
- ECG
- Cardiac catheterization

80. Acquired aortic valve disease. Hemodynamic disturbances, clinical manifestations, diagnostics, treatment.

Aortic stenosis – obstruction of blood flow across the aortic valve due to acquired calcific disease , bicuspid aortic valve ,rheumatic disease

HEMODYNAMIC DISTURBANCE
*1/3 of normal cross-sectional area (2.5 to 3.5 cm2) doesn't lead to significant hemodynamic changes
*The systolic function of the ventricle usually remains well preserved for many years
*Progressive concentric LV hypertrophy with little ventricular dilatation
*The LV mass and LV systolic wall tension demand more oxygen
*Dyspnea, low exercise capacity, heart failure, angina, and syncope.

CLINICAL MANIFESTATION
- Chest pain
- Heart failure
- Syncope
- Systolic hypertension
- Systolic murmur

TREATMENT OF AORTIC STENOSIS
*Once the patient becomes symptomatic, prompt operation is indicated (risk of death exceeds 30 to 50% over the next 5 years)
*Open aortic valve replacement
*Transcatheter aortic valve replacement

Aortic insufficiency
(*Etiology: degenerative diseases, inflammatory or infectious diseases (endocarditis, rheumatic fever), congenital diseases, aortoannular ectasia or aneurysm of the aortic root, and aortic dissection.)

HEMODYNAMIC DISTURBANCE

*Blood regurgitates into the left ventricle during diastole, producing LV volume overload (increased preload).
*The ventricle compensates and increases the LV stroke volume during systole.
*A widened pulse pressure
*Low diastolic pressure
*Low coronary perfusion
*LV dilation and hypertrophy

CLINICAL MANIFESTATION

TREATMENT
*The development of symptoms is an absolute indication for surgery
*Aortic valve repair (rarely)
*Aortic Valve Replacement
Ross Procedure:
*Replacement of the aortic valve with an autograft from the patient's native pulmonary valve
*The resected pulmonary valve is then replaced with a pulmonary homograft

DIAGNOSIS OF BOTH DISEASE
- Serum electrolyte levels

- Cardiac biomarkers
- Complete blood count
- ECG Chest radiography
- Echocardiography
- Coronary angiography
- Cardiac catheterization
- Exercise stress testing

81. Lymphedema (elephantiasis). Causes, clinical manifestations, diagnosis, treatment.

as abnormal limb swelling caused by the accumulation of increased amounts of high protein interstitial fluid secondary to defective lymphatic drainage in the presence of (near) normal net capillary filtration**.**

(The disease is associated with poor sanitation. The parasite enters lymphatics from the blood and lodges in lymph nodes, where it causes fibrosis and obstruction, due partly to direct physical damage and partly to the immune response of the host. Proximal lymphatics become grossly dilated with adult parasites. The degree of edema is often massive, in which case it is termed **elephantiasis .**)

CAUSES

Primary lymphedema	Congenital (onset <2 years old): sporadic; familial (Nonne–Milroy's disease)
	Praecox (onset 2–35 years old): sporadic; familial (Letessier–Meige's disease)
	Tarda (onset after 35 years old)
Secondary lymphedema	Parasitic infection (filariasis)
	Fungal infection (tinea pedis)
	Exposure to foreign body material (silica particles)
	Primary lymphatic malignancy
	Metastatic spread to lymph nodes
	Radiotherapy to lymph nodes
	Surgical excision of lymph nodes
	Trauma (particularly degloving injuries)
	Superficial thrombophlebitis
	Deep venous thrombosis

CLINICAL MANIFESTATION

- Swelling, clothing or jewellery becoming tighter
- Constant dull ache, even severe pain
- Burning and bursting sensations
- General tiredness and debility
- Sensitivity to heat
- 'Pins and needles'
- Cramp
- Skin problems, including flakiness, weeping, excoriation and breakdown
- Immobility, leading to obesity and muscle wasting
- Backache and joint problems
- Athlete's foot
- Acute infective episodes

DIAGNOSIS

- 'Routine' tests(lab test : include a full blood count, urea and electrolytes, creatinine, liver function tests, thyroid function tests, plasma total protein…)
- Lymphangiography (Direct lymphangiography involves the injection of contrast medium into a peripheral lymphatic vessel)
- Isotope lymphoscintigraphy
- Computed tomography
- Ultrasound
- MRI
- Limb volume measurement

TREATMENT

- Drugs **:**

1. Benzpyrones - flavonoids
2. Diuretics-furosemide , torsemide
3. Intravenous antibiotics
4. heparin prophylaxis will reduce the risk of deep vein thrombosis

✓ the **three goals** of treatment are to :

1. **relieve pain**: its severity and underlying cause(s) will vary depending on the etiology of the lymphedema. In **cancer related** disease treatment involves the considered use of non-opioid and opioid analgesics, corticosteroids, tricyclic antidepressants, muscle relaxants, anti-epileptics, nerve blocks, physiotherapy and adjuvant anticancer therapies (chemo-, radio- and hormonal therapy), as well as measures to reduce swelling if possible.
   In patients with **non-cancer-**related lymphedema, the best way to reduce pain is to control swelling and prevent the development of complications**.**

2. **reduce swelling :** preferred term is : decongestive lymphedema therapy (DLT), which comprises two phases. **The first** is a short intensive period of therapist-led care ,
   second is a maintenance phase in which the patient uses a self-care regimen with occasional professional intervention.
   The intensive phase comprises skin care, manual lymphatic drainage (MLD) and multi-layer lymphedema bandaging (MLLB), and exercises.

3. **prevent the development of complications**

✓ **surgery for:** bypass procedures, liposuction and reduction procedures**,** sistrunk ,homans, thompson

82. Lymphadenitis, lymphangitis. Causes, clinical manifestations, diagnosis, treatment.

Lymphadenitis is the inflammation or enlargement of a lymph node. Lymphadenitis may affect a single node or a group of nodes (regional adenopathy) and may be unilateral or bilateral.

CAUSES

- Bacteria: Streptococcus pyogenes or Staphylococcus aureus, rubella , salmonella, yersinia
- Mycobacteria
- Viruses: cytomegalovirus , viral pharyngitis
- Fungi
- Parasites
- commonly develops after cutaneous inoculation

CLINICAL MANIFESTATION

- Pain
- Redness (erythematous streak)
- warm place
- fever
- anorexia

- upper respiratory tract infection , sore throat, earache , coryza , impetigo

DIAGNOSIS
- physical examination
- laboratory study
- ultrasonography
- chest X-ray (in case of pulmonary involvement)
- lymph nod biopsy

TREATMENT
- The treatment depend on cause of disease
- reduce lymphatic drainage and elevated to reduce swelling
- treated with intravenous antibiotics based (trimethoprim-sulfamethoxazole)
- chemotherapy and radiation for malignancy

83. Varicose disease of lower extremities. Etiology and pathogenesis, classification, clinical manifestations, diagnostics.

Varicose veins (VVs) are dilated, tortuous, superficial veins (saphenous veins, saphenous tributaries, or nonsaphenous superficial leg veins.) ,the legs are the most common site.

ETIOLOGY
- Primary or familial varicose veins.
- Pregnancy
- Secondary to postphlebitic limb (perforator failure).
- Congenital:

Klippel–Trenaunay syndrome (port-wine stain, varicose veins, bony and soft tissue hypertrophy involving an extremity)
Parkes–Weber syndrome (cutaneous flush with underlying multiple microarteriovenous fistulas, in association with soft tissue and skeletal hypertrophy of the affected limb).
- Iatrogenic: following formation of an arteriovenous fistula.
- Deep venous thrombosis

PATHOGENESIS
Venous valve failure, usually at the saphenofemoral or saphenopopliteal junction (and sometimes in perforating veins), results in increased venous pressure in the great and small saphenous vein with progressive vein dilatation and further valve disruption.

CLASSIFICATION
Clinical classification
C0: No visible or palpable signs of venous disease.
C1: Telangiectasia or reticular veins.
C2: Varicose veins.
C3: Oedema.
C4a: Pigmentation.
C4b: Lipodermatosclerosis.
C5: Healed venous ulcer.
C6: Active venous ulcer.

aEtiological classification
- Ec: Congenital.
- Ep: Primary.
- Es: Secondary (postthrombotic).
- En: No venous cause found.

Anatomical classification
- As: Superficial veins.
- Ap: Perforator veins.
- Ad: Deep veins.
- An: No venous location identified.

Pathological classification

• Pr: Reflux.
• Po: Obstruction.
• Pr,o: Reflux and obstruction.
• Pn: No venous pathophysiology identified.

CLINICAL MANIFESTATION
- Leg heaviness
- Exercise intolerance
- Pain or tenderness
- Pruritus
- Burning sensations
- Edema
- Skin change
- Swelling
- Leg fatigue
- Cramping
- Ulceration
- Eczematous lesions
- Acrocyanosis

DIAGNOSIS
1. Ankle Brachial Pressure Index measurement

- highly sensitive and specific for the presence of peripheral vascular disease
- non-invasive
- quick, cheap and easy to perform
- reproducible

2. Color –flow Duplex ultrasound (of Deep venous thrombosis)
- A mobile, free-floating thrombus may be seen on B-mode imaging.
- Loss of the spontaneous phasic flow on colour flow Doppler.
- Loss of the spectral Doppler pattern on augmentation if the vein is occluded.

3. Examination
- A patient with varicose veins must be examined while standing
4. Inspection
   a) Presence distribution of varicose vein (short,or long saphenous or complex)
   b) Old scars
   c) Signs of chronic venous insufficiency
5. Palpation
- Tap test: Place one finger at the top of a varicose vein and the other at the bottom.
- Tourniquet test: Lie the patient down and raise the legs to empty the superficial veins. Place a tourniquet as high on the thigh as you can.
- Trendelenburg' s test detects reflux from deep into superficial veins, and when carefully performed can identify the site of the incompetent connections
- Perthes' test: Raise the leg to empty the veins. Place a tourniquet around the leg
6. Venography
7. MRI

TREATMENT
1. Compression Therapy(Stockings and Bandages)
- Indicated for minor varicosities, and for the elderly, the pregnant and the unfit.

2. Surgical treatment
- **Indications** :
  • haemorrhage occurring from a varicosity;
  • varicosities being grossly dilated or otherwise symptomatic;

• skin changes , typically in the medial gaiter area, which may suggest coincident deep venous insuffi ciency;
• incompetent perforator veins

- Surgery involves disconnecting the great saphenous vein from the femoral vein; the terminal branches of the great saphenous vein are individually ligated and divided. This may be combined with stripping of the great saphenous vein from groin to knee. If there are other incompetent communications (perforators), these need to be individually ligated or avulsed. Small varicose venules can be avulsed via a small skin incision.

3. Endovenous laser therapy_ thermal ablation technique that uses a laser fiber placed inside the vein.
4. Radiofrequency _ thermal ablation technique that uses a specially developed proprietary RF catheter placed inside the vein
5. ablation Stab-avulsion technique_(ambulatory phlebectomy) allows removal of short segments of varicose and reticular veins through tiny incisions

6. Sclerotherapy -Superficial varicosities that are cosmetically undesirable may be obliterated by injection of a small volume of sclerosant with the vein emptied.
7. Saphenectomy_ introduction of a special probe into the vascular lumen and mechanical removal of the pathologically changed vein. The removal of even both of the veins: the greater and the lesser one, does not cause any disturbances in the circulation.

84. Complications of varicose disease, clinical manifestations, diagnosis, treatment.

COMPLICATIONS OF VARICOSE

a) **Superficial thrombophlebitis** -This is acute thrombosis of the superficial venous system presenting as an intense inflammatory reaction (pain, swelling and erythema)
b) **Haemorrhage**- This is usually due to minor trauma to a dilated vein.
c) **Deep venous insufficiency**- Varicose veins appear when superficial veins are dilated by blood entering via incompetent perforating veins or incompetent superficial valves.
d) **Swelling** , particularly of the lower leg
e) **Pigmentation of skin** , particularly the medial gaiter area
f) **Eczema** , particularly over the pigmented area, causing **pruritus**
g) **Ulceration** occurs as a consequence of the poor skin nutrition.

CLINICAL MANIFESTATIONS

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- Exercise intolerance
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- Pruritus
- Burning sensations
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- Leg fatigue
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7. Saphenectomy_ introduction of a special probe into the vascular lumen and mechanical removal of the pathologically changed vein. The removal of even both of the veins: the greater and the lesser one, does not cause any disturbances in the circulation.

85. Treatment of varicose disease of lower extremities. Medical treatment, types of operations, sclerotherapy.

1. Compression Therapy(Stockings and Bandages)
   - Indicated for minor varicosities, and for the elderly, the pregnant and the unfit.

2. Surgical treatment
   - **Indications** :
     • haemorrhage occurring from a varicosity;
     • varicosities being grossly dilated or otherwise symptomatic;
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TYPE OF OPERATION

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MEDICAL THERAPY
sclerosing agents -indicated for uncomplicated spider veins

SCLEROTHERAPY
Superficial varicosities that are cosmetically undesirable may be obliterated by injection of a small volume of sclerosant with the vein emptied. The vein is kept compressed with firm pressure bandaging for a period of 2 weeks to enable fibrosis to take place. This outpatient treatment is used for small - or moderate - sized varices below the knee. Recurrences can be treated by further injections.

86. Acute deep vein thrombosis. Clinical manifestations, diagnostics, treatment.

DEFINITIONS
A thrombus is a solid mass of platelet, fibrin and other components of blood that forms locally in a vessel. A deep venous thrombosis (DVT) is a condition in which the blood in the deep veins of the legs or pelvis (rarely upper limbs) forms into a clot.

CLINICAL MANIFESTATION

- Pulmonary embolism
- Dyspnoea ± pleuritic chest pain.
- Tachycardia and tachypnoea.
- Cough up blood ± haemoptysis, fever.
- Massive Pulmonary embolism causes circulatory arrest

- Hypercoagulability
- Stasis
- Endothelial injury
- leg pain or swelling , red or discoloured
- dizzy

DIAGNOSIS

- D-dimers (byproduct of fibrinolysis – 95% sensitivity)
- Duplex ultrasound imaging: Excellent for femoral and popliteal DVT
- MRI
- CT venography may used if ilio-femoral DVT is suspected.
- Venography- A dye is injected into a large vein in your foot or ankle. An X-ray creates an image

TREATMENT

Essential management :

• Mechanical compression: stockings or intermittent pneumatic compression devices.
• Pharmacological:

- ✓ low dose unfractionated heparin (LDUH) – 5000 IU s.c., b.i.d.
- ✓ low molecular weight heparin (LMWH) –
- ✓ LDUH fondaparinux (Factor Xa inhibitor) – 2.5 mg/day warfarin and newer anticoagulants (hirudin, lepirudin).
- ✓ For reducing wall inflammation :
  - Regular cannula change
  - Reducing fracture
- ✓ For improving flow : passive and active exercise , pneumatic compression boots

definitive treatment

- ✓ Anticoagulation for 12 weeks: IV unfractionated heparin (check efficacy with APTT) or s.c. low molecular weight heparin (no APTT monitoring required)
- ✓ oral anticoagulation (warfarin) in nonpregnant patients (target INR 2.0–3.0).
- ✓ In pregnant use low molecular weight heparin (LMWH)
- ✓ Compression stockings: graduated-compression below knee elastic stockings × 2 years reduces risk of developing postphlebitic limb.
- ✓ (Thrombolysis: may be useful in selected patients with iliofemoral DVT – haemorrhage a major side-effect. Thrombectomy rarely performed.)

87. Occlusion of superior vena cava branches. Clinical manifestations, diagnostics,treatment.

88. Lung cyst. Clinical picture, diagnosis, indications for surgery, types of operations.

Cystic lung disease (CLD) is a group of lung disorders characterized by the presence of multiple cysts, defined as air-filled sac with thin wall or round parenchymal lucency .

CLINICAL PICTURE

- Chest pain
- Tiredness
- Coughing blood(may by blood)
- Shortness of breathing
- Wheezing
- Pleural effusion
- Collapsed lung

DIAGNOSIS

- CT
- bronchoscopic biops

89. Bronchiectasis. Clinical manifestations, diagnosis, therapy, indications for surgery, types of operations.

Bronchiectasis is defined as a **pathologic and permanent dilation of bronchi with bronchial wall thickening**. This condition may be localized to certain bronchial segments, or it may be diffuse throughout the bronchial tree, typically affecting the medium-sized airways

ETIOLOGY

- Bronchial obstruction
- Cystic fibrosis
- Allergic bronchopulmonary aspergillosis
- Immunodeficiency states
- Connective-tissue disorders
- Autoimmune diseases
- Toxic gas exposure

CLINICAL MANIFESTATIONS

- daily persistent **cough** and **purulent sputum** production
- **progressive symptoms** and **respiratory impairment**
- Dyspnea, pleuritic chest pain, wheezing, fever, weakness, fatigue, and weight loss
- bleeding may result from erosion of the hypertrophied bronchial arteries.
- Crackles, rhonchi, scattered wheezing, and inspiratory squeaks on auscultation
- Digital clubbing
- Nasal polyps and signs of chronic sinusitis

DIAGNOSIS

- Physical examination : crackles sound
- CT – scanning
- X-ray
- Sputum culture
- CBC
- Serum immiunoglobulin (IgG,IgA,IgM)
- Spirometry provides an assessment of the severity of airway obstruction and can be followed to track the course of disease.
- Autoimmune screening test

THERAPY

- Medicine :I/V in acute , inhaled or orally in chronic antibiotic according to culture sputum (amoxicillin , tetracycline, azithromycin or clarithromycine) , 2nd generation of cephalosporin
- Acute exacerbations should be treated with a 2- to 3-week course of broad-spectrum intravenous **antibiotics** tailored to culture and sensitivity profiles, followed by an oral regimen; this will result in a longer-lasting remission.
- Supportive treatment :avoid smoking ,oxygen therapy
- **Surgical resection** of a localized bronchiectatic segment or lobe which is indicated in patients with significant hemoptysis, although bronchial artery embolization is the preferred first option **Surgical is not required for older .**
- Patients with end-stage lung disease from bronchiectasis may be potential candidates for a **bilateral lung transplant**.

90. Pneumothorax and hemothorax . Etiology, classification, diagnostics, treatment.

Pneumothorax

Pneumothorax is defined as the presence of air or gas in the pleural cavity which can impair oxygenation and/or ventilation

ETIOLOGY

- Smoking
- Marfan syndrome
- Pregnancy
- Family history
- Disease and condition associated with secondary pneumothorax :
- COPD
- Asthma
- Tuberculosis
- Cystic fibrosis
- Sarcoidosis
- Bronchial carcinoma

CLASSIFICATION
according to aetiology :

- primary, Primary spontaneous pneumothoraces occur most commonly in young, tall, thin males with no predisposing lung disease or history.
- secondary, Secondary pneumothoraces occur when there is another lung disease or abnormality.
- iatrogenic , An iatrogenic pneumothorax is most commonly caused by central vein cannulation(mostly subclavian)
- Traumatic.

DIAGNOSIS

- Physical examination :
  Respiratory distress (considered a universal finding) or respiratory arrest
  Tachypnea (or bradypnea as a preterminal event)
  Decreased tactile fremitus
  Adventitious lung sounds (crackles, wheeze; an ipsilateral finding)
  Tachycardia

- Arterial blood gas analysis
- Chest radiography
- CT
- Contrast – enhanced esophagography
- Ultrasonography

TREATMENT

- Pharmacotherapy
- Supplemental oxygen
- Simple aspiration
- Chest tube placement
- Thoracostomy with continuous wall suction
- Thoracotomy

HEMOTHORAX

Hemothorax is the presence of blood in the pleural space. The source of blood may be the chest wall, lung parenchyma, heart, or great vessels, Blood in the pleural space can be associated with both hemorrhagic shock and respiratory compromise.

ETIOLOGY

- Trauma
- Penetrating injuries of the lungs, heart, great vessels, or chest wall
- Neoplasia Blood dyscrasias, including complications of anticoagulation
- Pulmonary embolism with infarction
- Bullous emphysema
- Necrotizing infections
- Hereditary hemorrhagic telangiectasia

DIAGNOSIS

- Laboratory test
- Chest radiography

- CT
- Ultrasonography

TREATMENT

- Blood should be drained by **tube thoracostomy**. In cases of hemopneumothorax, placement of two chest tubes may be preferred, with the tube draining the pneumothorax placed in a more superior and anterior position
- **Thoracotomy**
- **Intrapleural fibrinolysis** - Intrapleural instillation of fibrinolytic agents is advocated in some centers for evacuation of residual hemothorax in cases in which initial tube thoracostomy drainage is inadequate.

91. Pleural empyema. Etiology, pathogenesis, clinical features, diagnosis, treatment.

Empyema is defined as a collection of pus in the pleural cavity effusion Common associated conditions include **a pneumonic process** in patients with pulmonary disorders and neoplasms, cardiac problems, diabetes mellitus, drug and alcohol abuse, neurologic impairments, postthoracotomy problems, and immunologic impairments.

ETIOLOGY

- Bacterial infection more common gram positive mostly streptococcus species
- penetrating chest trauma,
- esophageal rupture,
- complication from lung surgery, or inoculation of the pleural cavity after thoracentesis or chest tube placement.
- An empyema can also occur from extension of a subdiaphragmatic or paravertebral abscess Contamination from a source contiguous to the pleural space (50%–60%) :
- lung
- Mediastinum
- Deep cervical area
- Chest wall and spine
- Subphrenic area

PATHOGENESIS

As organisms enter the pleural space, an influx of polymorphonuclear cells and fluid occurs, with subsequent release of inflammatory mediators and toxic oxygen radicals. These mechanisms lead to variable degrees of endothelial injury and capillary instability. This process overwhelms the normal pleural lymphatic drainage. This early effusion is watery and free-flowing in the pleural cavity.
this stage yields fluid with a pH typically above 7.3, a glucose level greater than 60 mg/dL, and a low LDH level (<500 U/L)

• Stage 1 Exudative with swelling of pleural membranes
• Stage 2 Fibrinopurulent with heavy fibrin deposits
• Stage 3 Organization with ingrowth of fibroblasts and deposition of collagen

CINICAL MANIFESTATION

• Shortness of breath, cough , chest pain - common to pneumonia.
• Aerobic empyema - acute febrile illness.
• Anaerobic empyema - more indolent, usually 10 days.(SLOWLY PROCESS)

DIAGNOSIS

• Chest x-ray - The posterior lateral diaphragmatic angle - The most dependent position - Most empyema are found. (Inverted D or pregnant lady sign).
• Sonography – guide thoracocentesis.
• Fluid analysis
• Aerobic pus - little odor.
• Anaerobic - foul smelling

TREATMENT

- Effective management require:

1) Control infection and sepsis by antibiotics.
2) Evacuation of pus from pleural space.
3) Obliteration the empyema cavity.

- Drainage :
    1. Early in the parapneumonic process, when the purulent fluid is relatively thin , complete drainage with simple large-bore thoracentesis is possible ,
    2. If pleural fluid may become thick and loculated over the course of hours to days and may be associated with fibrinous adhesions (the fibrinopurulent stage). At this stage, **chest tube** insertion with closed-system drainage or drainage with **thoracoscopy** may be necessary to remove the fluid,
    3. In Progressive stage _ complete lung decortication by either thoracoscopy or thoracotomy would then be necessary.
    4. Remove-- drainage less than 50 ml within 24 hour, lung re-expansion. Usually within 5-10 day. Antibiotics should continue 6 week.

92. Lung abscess. Etiology, pathogenesis, clinical features, diagnosis, treatment

A lung abscess is a localized area of pulmonary parenchymal necrosis formation of cavities containing necrotic debris or fluid caused by an infectious organism; Tissue destruction results in a solitary or dominant cavity measuring at least 2 cm in diameter ,the infection is typically referred to as a necrotizing pneumonia. An abscess that is present for more than 6 weeks is considered chronic. Both lung abscess and necrotizing pneumonia are manifestations of a similar pathologic proce

ETIOLOGY

I. Primary
A. Necrotizing pneumonia
1. Staphylococcus aureus, Klebsiella, Pseudomonas,Mycobacterium
2. Bacteroides, Fusobacterium, Actinomyces
3. Entamoeba, Echinococcus
B. Aspiration pneumonia
1. Anesthesia
2. Stroke
3. Drugs or alcohol
C. Esophageal disease
1.Achalasia,
2. Zenker's diverticulum,
3. Gastroesophageal reflux
D. Immunodeficiency
1. Cancer (and chemotherapy)
2. Diabetes
3. Organ transplantation
4. Steroid therapy
5. Malnutrition

II. Secondary
A. Bronchial obstruction
1. Neoplasm
2. Foreign body
B. Systemic sepsis
1. Septic pulmonary emboli
2. Seeding of pulmonary infarct
C. Complication of pulmonary trauma
1. Infection of hematoma or contusion
2. Contaminated foreign body or penetrating injury
D. Direct extension from extraparenchymal infection

1. Pleural empyema
2. Mediastinal, hepatic, subphrenic absces

PATHOGENESIS

Lung abscesses result when necrotizing microorganisms infect the lower respiratory tract via:
• inhalation of aerosolized particles,
• aspiration of oropharyngeal secretions,
• hematogenous spread from distant sites.
Direct extension from a contiguous site is less frequent. Most primary lung abscesses are suppurative bacterial infections , secondary to aspiration.
Risk factors for pulmonary aspiration:
• advanced age,
• conditions of impaired consciousness
• suppressed cough reflex,
• dysfunctional esophageal motility,
• laryngopharygeal reflux disease,
• and centrally acting neurologic diseases (e.g., stroke).

CINICAL MANIFESTATION

The **typical** presentation may include: productive cough, fever (>38.9°C), chills, leukocytosis (>15,000 cells/mm3), weight loss, fatigue, malaise, pleuritic chest pain, and dyspnea.
Lung abscesses may also present in slowly process , with weeks to months of cough, malaise, weight loss, low-grade fever, night sweats, leukocytosis, and anemia, foul-smelling sputum.
Severe complications:
• massive hemoptysis,
• endobronchial spread to other portions of the lungs,
• rupture into the pleural space and development of pyopneumothorax,
• septic shock and respiratory failure are rare in the modern antibiotic era.

DIAGNOSIS

- X-ray
- CT scan
- Contrast-enhanced CT
- Bronchoscopy -for obtaining uncontaminated cultures using bronchoalveolar lavage
- Routine sputum cultures are often of limited usefulness because of contamination with upper respiratory tract flora

TREATMENT

Medical therapy :

The duration of antimicrobial therapy varies from 3 to 12 weeks for necrotizing pneumonia and lung abscess.
For community-acquired infections secondary to aspiration, likely pathogens are oropharyngeal streptococci and anaerobes. Penicillin G, ampicillin, and amoxicillin are the main therapeutic agents, but a β-lactamase inhibitor or metronidazole should be added to cover the increasing prevalence of gramnegative anaerobes that produce β-lactamase. Clindamycin is also a primary therapeutic agent.
For hospital-acquired infections, Staphylococcus aureus and aerobic gramnegative bacilli are common organisms of the oropharyngeal flora. Piperacillin or ticarcillin with a β-lactamase inhibitor (or equivalent alternatives) provide better coverage of likely pathogens

Surgery : (drainage is uncommon)

Indications for surgical drainage procedures for lung abscesses
1. Failure of medical therapy
2. Abscess under tension
3. Abscess increasing in size during appropriate treatment
4. Contralateral lung contamination
5. Abscess >4–6 cm in diameter
6. Necrotizing infection with multiple abscesses, hemoptysis, abscess rupture, or pyopneumothorax
7. Inability to exclude a cavitating carcinoma

The choice between tube thoracostomy versus radiographically guided catheter placement depends on the treating physician's preference and the availability of interventional radiology. Surgical resection is required in fewer than 10% of lung abscess patients. Lobectomy is the preferred intervention for bleeding from a lung abscess or pyopneumothorax. An important intraoperative consideration is to protect the contralateral lung with a doublelumen tube, bronchial blocker, or contralateral main stem intubation. Surgical treatment has a 90% success rate, with an associated mortality of 1% to 13%.