

esophagus:

anatomy:

The esophagus is a 25 cm- long muscular tube (40 cm from the mouth) that begins at the pharynx (lower border of C6) -past- and ends at the opening of the stomach (cardia) The muscle type varies along

the esophagus:

1. Upper 1/3 → skeletal muscle.
2. Middle 1/3 → mixed (skeletal + smooth)
3. Lower 1/3 → smooth muscle.

➤ There are 3 areas of Narrowing:

1. At the beginning of the esophagus (caused by the cricopharyngeus muscle). (C6) --this is the narrowest part of the esophagus--.
2. Where the left main bronchus and aorta cross. (T4) -so it is posterior to the aorta in the mediastinum.
3. At the hiatus of diaphragm.

when it crosses the hiatus it angles or curves anteriorly and left through diaphragmatic hiatus (T10).

after the diaphragm it becomes abdominal and covered by peritoneum.

It has 2 sphincters:

1. Upper esophageal sphincter (UES): anatomical sphincter, caused by actual thickening of the muscular wall, its main function is swallowing.
2. Lower esophageal sphincter (LES): functional sphincter -not an anatomical sphincter-, so it's an area of high pressure, its main function is prevention of reflux.

Physiology:

Types of peristalsis:

Primary peristalsis: Progressive, contractions triggered by swallowing.

Secondary peristalsis: Involuntary, initiated by esophageal distension.-(e.g., food bolus stuck).-

Tertiary peristalsis: Non-propulsive, simultaneous contractions (abnormal motility). Simultaneous contractions (e.g., diffuse esophageal spasm). so these are pathologic.

Anti-reflux mechanism:

1. Lower esophageal sphincter (LES) tone –ideally around 6mmhg--
2. Crura of diaphragm.
3. Cardiac angle (angle of His) .
4. Peristaltic movement.
5. Saliva
6. Intraabdominal portion of the esophagus longer then 2cm.

Zenker's Diverticulum

Definition: is a diverticulum (outpouching) of the mucosa of the pharynx, just above the cricopharyngeal muscle (i.e. above the upper sphincter of the esophagus). It is a pseudo diverticulum (not involving all layers of the esophageal wall).

pathophysiology:

The upper esophageal sphincter has two parts, Upper Oblique (thyropharngueus) and lower transverse (cricopharyngeus), between these muscles, there's a weak area. through which herniation occurs

⚡ Signs and Symptoms:

1- Dysphagia:

- Transfer dysphagia (difficulty initiating the swallowing)
- For solids Only.

2- Halitosis (bad smell)

3- Food reaggregation.

4- Posterior neck mass.

Dx: barium swallow// we dont do endoscopy and NG tube due to risk of perforation

treatment: surgical resection.

Achalasia

⚡ Definition: a failure of smooth muscle fibers to relax, which can cause a sphincter to remain closed and fail to open when needed.

signs and Symptoms:

1- Dysphagia

- For solids and liquids.

➤ Progressive

2- Regurgitation of food

Complications:

➤ Aspiration pneumonia.

➤ Weight loss.

➤ Esophageal carcinoma--long standing cases are associated with adenocarcinoma even without smoking or alcohol.--

diagnosis: Imaging:

1. Barium swallow: (best initial test) → bird's beak appearance (narrow LES + dilated esophagus) --remember the best test--

2. Upper endoscopy+ biopsy: to confirm diagnosis +rule out CA

3. Esophageal manometry: (the definitive diagnosis) → absence of peristalsis+ non relaxing LES.

treatment:

pneumodilatation: (BEST initial therapy)

Botox (botulinum toxin injection)

➤ Effective in 65% of patients.

➤ Requires repeating therapy within 6-12 months.

Surgical myotomy.

Which of the following statements about achalasia is CORRECT?

- A. In most cases the cause is a parasitic infestation by *Trypanosoma cruzi*.
- B. Chest pain and regurgitation are the usual symptoms.
- C. Distal-third esophageal adenocarcinomas may occur in as many as 20% of patients within 10 years of diagnosis.
- D. Manometry demonstrates failure of LES relaxation on swallowing and absent or weak simultaneous contractions in the esophageal body after swallowing.
- E. Endoscopic botulinum toxin injection of the LES, pneumatic dilatation, and esophagomyotomy provide highly effective curative therapy for achalasia.

answer:D

Barrett esophagus

- It's an intestinal metaplasia of lower esophageal mucosa (change from stratified squamous epithelium into simple columnar epithelium with goblet cells).
- Risk factors are smoking and GERD, but many cases lack these risk factors.
- Diagnosed by endoscopy.
- Management is by PPI and follow up:
 - i. No dysplasia → 3-5 years
 - ii. Low-grade dysplasia → 6-12 months
 - iii. High-grade dysplasia → 3 months

know the follow up.

in high grade dysplasia actually we need immediate surgery because it has 10–30% risk of progression to adenocarcinoma.

Requires intervention, not just surveillance.

Esophagectomy offers the highest chance of cure for HGD, with 5-year survival >90%.

Follow up endoscopy was done for a patient with known Barrett's esophagitis, biopsy was taken and it showed metaplasia with high grade dysplasia, the best next step in management of this patient is:

- A. Esophagectomy
- B. Increase dose of PPI
- C. Increase frequency of follow up endoscopy
- D. Anti reflux surgical procedure (Nissen fundoplication)
- E. H pylori eradication therapy

answer: A

Which is false regarding Barrett's esophagus?

- A. Asymptomatic Barrett's requires annual follow up
- B. Symptomatic Barrett's requires medical therapy with PPI
- C. Anti-reflux surgery can reverse metaplasia in 35% and improve symptoms
- D. Bleeding, ulceration, and stricture can complicate the disease
- E. Patients with mild dysplasia need more frequent screening

answer: A not annual but 3-5 years

HY Point 1: Goblet cells = intestinal metaplasia (required for Barrett's diagnosis).

HY Point 2: No goblet cells = no Barrett's (e.g., gastric metaplasia alone is insufficient).

What is the initial test for evaluation of patients with gastroesophageal reflux disease?

- A. 24-pH manometry
- B. Upper endoscopy
- C. Esophageal manometry
- D. Barium swallow
- E. Gastric emptying study

Answer: B

Upper endoscopy (EGD): First-line if alarm symptoms (e.g., dysphagia) or screening for complications (e.g., Barrett's esophagus).

24-hour pH monitoring: Gold standard for reflux confirmation (if EGD negative or atypical symptoms).

Barium swallow: Poor for GERD; used for **structural issues** (e.g., hiatal hernia, strictures).

Critical Distinctions:

Esophageal manometry: Evaluates **motility disorders** (e.g., achalasia), not GERD.

Gastric emptying study: Rules out **gastroparesis** (not GERD)

esophageal CA:

in upper 2 thirds it is mostly squamous cell carcinoma

the main risk factors are mucosal irritation:

smoking and hot drinks / alcohol

Most common (90%) worldwide (and in endemic areas)

in the lower third the most common type is adenocarcinoma the risk factor is acid reflux -Barret's esophagus.-.

➤ The most common type in USA (& Jordan).

also "Poor nutrition" leads to Micronutrient deficiencies (e.g., zinc, selenium) and increase risk

fruits and vegetables are said to be protective.

HY Point 1: Alcohol + smoking = synergistic SCC risk (10–25x increased).

HY Point 2: Hot beverages (>65°C) = SCC risk (IARC Group 2A).

HY Point 3: Fruits/vegetables = protective

The most common risk factor for developing adenocarcinoma of the esophagus, is:

- A. Alcohol
- B. H pylori infection
- C. Obesity
- D. Barrit's metaplasia
- E. Smoking

answer:D

symptoms:

1- Dysphagia.

➤ The earliest sign

➤ For solids then liquids.

2- Loss of appetite and Weight loss, weakness and retrosternal discomfort.

3- Achalasia-like symptoms.

Mechanical complications (aspiration, obstruction) occur in advanced disease.

early satiety is not esophageal symptom it is a gastric symptom.

HY Point 1: Progressive dysphagia = red flag for esophageal cancer.

HY Point 2: Early satiety → think gastric, not esophageal, involvement.

A 72-year-old man, a long-standing smoker, complains of progressive dysphagia with food sticking behind his mid-sternum for 3 months. The problem started with solids, but now he has difficulty with liquids. He has lost 14 kg of weight during this time. The most likely diagnosis is:

A. Achalasia of cardia

B. Carcinoma of esophagus

C. Diffuse esophageal spasm

D. Gastro-esophageal reflux disease (GERD)

E. Pharyngeal diverticulum

answer:B

HY Point 1: Progressive dysphagia + weight loss = cancer until proven otherwise.

HY Point 2: Smoking + alcohol = squamous cell carcinoma; GERD/obesity = adenocarcinoma.

☰ The only way to cure esophageal CA is surgery:

- Stage 1 and 2 → surgery.
- Stage 3 → neoadjuvant chemotherapy/radiotherapy to shrink the tumor → then surgery.
- Stage 4/or patients is unfit → chemotherapy/palliative surgery.

weird questions

A patient has difficulty in swallowing solid food, what is the diagnostic test?

- A. Barium swallow - first step
- B. Manometry
- C. 24-h pH monitoring
- D. EGD with biopsy

answer: A.

Manometry can show all except:

- A. Length of intraabdominal esophagus - length of LES
- B. Pressure in the esophagus
- C. Peristaltic contractions
- D. Degree of gastric reflux

answer:D

Regarding esophageal cancer, which is wrong:

Answer: A

- A. Around 80% present with dysphagia
- B. Dysphagia causes weight loss
- C. All adult pts with dysphasia should undergo esophagoscopy to rule out malignancy
- D. Screening for esophageal cancer in Jordan, is not cost effective

answer:D

stomach:

PEPTIC ULCER DISEASE:

Definition: Peptic ulcer disease represents a spectrum of diseases characterized by ulceration of the stomach or proximal duodenum due to imbalance between acid secretion & mucosal defense mechanisms.

Epidemiology: It's a very common disease.

Classification:

- Duodenal: on the anterior wall (more common).
- Gastric: (less, common, has 5 categories):

Type I → lesser curvature (near incisura angularis), it's associated with decreased mucosal production.

Type II → lesser curvature + duodenal, it's associated with increased acid production.

Type III → Prepyloric, it's associated with increased acid production.

Type IV → proximal stomach/ cardia, it's associated with decreased mucosal production.

Type V → anywhere in the stomach, it's medication induced.

there is past questions about the types

etiology:

1-Helicobacter pylori (the most common cause)

➤ It causes both duodenal ulcers (90%) and Gastric ulcers (70-80%)

2. NSAIDs (2nd most common cause)

➤ It causes both duodenal ulcers (8%) and Gastric ulcers (40%).

3. Acid hypersecretion

➤ Associated with duodenal ulcers.

➤ EX: Zollinger Ellison syndrome

we suspect Zollinger ellison when we have recurrent PUD and diarrhea. or ulcers in unusual site like the jejunum.

4. smoking

⚙️ Investigations: Flexible upper endoscopy to confirm the diagnoses, once the diagnosis is confirmed, we have to look for the cause:

1. H. Pylori infection tests:

➤ Non-invasive: Serological antibody tests, Urea breath test, Fecal antigen test.

➤ Invasive: Biopsy (gold standard) → to rule out malignancy, Culture + Urase test.

2. Fasting serum Gastrin levels: to rule out ZES it is the best test.

3. Endoscopic biopsy to rule out Gastric Ulcer.

treatment:

Medical:

- H. pylori eradication: Triple therapy for 10-14 days
- NSAIDs -associated PUD → Stop the drug, then initiate anti-secretory Treatment.
- Smoking cessation.
- Follow up with endoscopy

we rarely need surgery:

- Indications For

surgery:

1. Non-healing ulcers.
2. Perforated Ulcers.
3. Bleeding Ulcers.
4. Gastric outlet obstruction.
5. Malignant Ulcers.

Complications:

- Bleeding.
- Perforation.

➤ Obstruction.

1. Bleeding PUD

It's the leading cause of death due to PUD.

➤ The most common cause of UGI bleeding.

☰ The most common site of bleeding duodenal ulcer is the posterior wall, typically eroding the gastroduodenal artery.

treatment: aggressive fluids and correction of coagulopathy.

then we do endoscopy.

treatment Requires oversewing + gastroduodenal artery ligation

2. Perforated PUD

☰ Most commonly seen in the anterior duodenal wall

also the most frequent indication for emergency surgery in patients with peptic ulcer disease.

symptoms:

1. Sudden onset of severe abdominal pain (less dramatic in elderly/ hospitalized and immunocompromised)

2. Peritonism (Fever/tachycardia and guarding).

☰ investigations:

1. CBC → leukocytosis

2. Abdominal/chest X-ray → air under diaphragm.-also called pneumoperitoneum seen in 70% of cases

treatment:

☸ Stabilize the patient: Aggressive fluid resuscitation, analgesia, broad spectrum antibiotics.

Omental patching (Graham patch): Gold standard for sealing perforations.

3. Gastric outlet obstruction

☸ It occurs due to:

1. Healing of a circumferential ulcer & fibrosis by scar tissue.
2. Edema & spasm
3. Antral tumors.

1. History:

- Recurrent vomiting of poorly digested food.
- Dehydration.
- Hypochloremic hypokalemic metabolic alkalosis.

2. Physical examination:

- Dilated full stomach.
- Visible peristaltic waves.
- +ve succussion splash.

3. NG tube will expel a muddy fluid in large quantities

☸ Stabilize the patient → insert NG tube, start IV hydration

☸ When the patient is stable → Do endoscopy (to rule out CA

we treat with surgery.

In upper GI bleeding due to gastric ulcer, it is not recommended to do which of the following?

- A. History and physical exam
- B. IV fluids resuscitation
- C. IV Proton pump inhibitor
- D. Gastric lavage with Nasogastric tube
- E. Serial Hb monitoring

answer:D

First-line management:

Hemodynamic stabilization (IV fluids, blood transfusion if Hb <7 g/dL).

PPI infusion (reduces rebleeding risk post-endoscopy).

Diagnostics:

EGD (gold standard for diagnosis + therapy)

NG tube has no role.

Concerning perforated peptic ulcer (PPU), all of the following statements are true EXCEPT:

- A. Perforation represents the most frequent indication for emergency surgery in patients with peptic ulcer disease.
- B. In patients with perforated peptic ulcer, peritonitis resulting from acid exposure may present as abdominal "board-like rigidity".

- C. Only one third of patients with PPU have a previous history of or current known ulcer at time of diagnosis of perforation.
- D. Compared to open surgery, laparoscopic repair of PPU is associated with lower rates in mortality and in clinically relevant postoperative complications.
- E. In patients with PPU, sepsis is frequently present on arrival to the operating theatre and is the leading cause of death.

answer:D.

weird question

memorize the answer.

For a patient with PUD one of the following manifestations increase the possibility of gastrinoma:

- A. jaundice
- B. constipation
- C. abdominal distention
- D. flatulence
- E. diarrhea

answer:E

Best test for diagnosis of gastrinoma is:

- A. 24 hour urine gastrin
- B. Acidity of the stomach
- C. fasting gastrin *

answer:C

Not a manifestation of Zollinger-Ellison syndrome:

- A. Migratory rash
- B. Diarrhea
- C. Multiple peptic ulcerations
- D. Vomiting

answer:A

Concerning management of upper gastrointestinal bleeding (UGIB), all of the following statements are true EXCEPT:

- A. There is a need to insert bilateral, 16-gauge (minimum), upper extremity peripheral intravenous lines.
- B. Once the maneuvers to resuscitate are underway, a nasogastric tube should be inserted, and then aspiration and lavage performed.
- C. If the nasogastric aspirate reveals clear gastric fluid and contains no bile, the gastrointestinal bleeding is emanating from below the ligament of Treitz.
- D. Upper gastrointestinal endoscopy should be performed initially after endotracheal intubation (if indicated), hemodynamic stabilization, and adequate monitoring.
- E. The indication for patients in patients with bleeding peptic ulcer includes failure of medical therapy and endoscopic hemostasis with persistent recurrent bleeding.

answer:C --memorize it---

Bariatric surgery:

indications for bariatric surgery:

- Morbid obesity (BMI>40)
- Severe obesity (BMI>35) + comorbidities.
- Severe comorbidities.
- Social and psychological implications.

Types of bariatric surgeries:

- Restrictive: decreases the size of the stomach.
- Malabsorptive: it's better to be called maldigestive, in these surgeries, we are diverting the food away from the duodenum → poor digestion → poor absorption, such surgeries are not done anymore nowadays.
- Combined.

restrictive surgeries:

1-Laparoscopic adjustable gastric band:

Laparoscopic procedure, done by placing a silicone band with an inflatable balloon around the proximal part of the stomach, at the angle of His
the band creates a proximal small pouch.

50-60% weight loss,

low mortality.

More bands are removed now than placed.

Complications: slipped band, obstruction, band erosion -Band may erode through the wall-, adjusting device malfunction, pouch/esophageal dilation.

- Band slippage → it's an emergency.
- Not good for sweet eaters (sweet dissolves)
- The least effective In terms of EWL

Reoperation rate in band surgery is more than other bariatric procedures-- past → 30–50% need band removal/revision.

Dumping syndrome is not a significant concern after LAGB --past

Leak rate after LAGB is less than other procedures --past

2- laparoscopic Sleeve gastrectomy:

Resection of part of stomach, leaves antrum and pylorus intact, -85% of the stomach is resected, from the pylorus to the angle of His along the greater curvature.

- By cutting the stomach, the size that's available for food is reduced, moreover, the Ghrelin hormone secretion is decreases → Less hunger. -

(80%) weight loss which is a good number.

low mortality.

Complications: leak --➤ Fever is an ominous sign → could indicate leakage.--, stricture.

it could be used as a Bridge procedure for Biliopancreatic procedure.

- A bridge procedure can be done before the “real” surgery to lose weight & make the “real” surgery easier.
- Another bridging procedure is placing an intragastric balloon laparoscopically for very obese patients for 6 weeks preoperatively.

combined: In these procedures, the stomach is converted to a small pouch (restrictive), then the stomach is anastomosed to more distal part of the small intestine to by-pass it (malabsorptive). All these procedures have failure due to:

- Dilatation of the pouch.
- Vitamin for life.

1- Roux-en-y gastric by-pass.-also called Laparoscopic gastric bypass (LGBP)-

It's the most popular surgery in U.S.A

70% success rate

☰ Procedure:

- 1) The stomach is cut into small pouch (connected to the esophagus).

2) A 75-150 cm of the small intestine (that's connected to the remainder of the stomach) is cut. -this is the Bilopancreatic limb it is made of rest of stomach and duodenum and some jejunum.

3) The remaining small intestine is called Roux limb, the roux limb is anastomosed with the gastric pouch.

4) The cut small intestine - bilopancreatic limb-(that's connected to the stomach) is anastomosed after 75 cm of the Roux limb. -jejuno-jejunal anastomosis-

the other combined procedure is:

2-Biliopancreatic Diversion (\pm Duodenal switch): more Malabsorptive.

☹️ It has many complications (severe vitamin deficiency, anemia)

Which of the following bariatric procedures is known to reduce appetite and weight by affecting the hunger hormone:

- A. laparoscopic roux – en – y gastric bypass
- B. laparoscopic sleeve gastrectomy
- C. biliopancreatic diversion
- D. laparoscopic adjustable gastric banding
- E. intragastric balloon

answer:B

The least effective bariatric procedure in term of excess weight loss (EWL) is:

- A. Laparoscopic sleeve gastrectomy

- B. Laparoscopic adjustable gastric band
- C. Laparoscopic gastric bypass
- D. Laparoscopic minigastric bypass
- E. Laparoscopic biliopancreatic diversion with duodenal switch

answer:B

gastric tumors:

Types:

- Adenocarcinoma: The most common type (95%), .
- GIST -GI stromal tumors-
- Lymphoma.
- Carcinoid. (Very rare)

adenocarcinoma:

low incidence worldwide// but very high incidence in Japan

Subtypes of Gastric

Adenocarcinoma:

1. Diffuse type: (30%)

2. Intestinal type: (70%)

Lauren classification

- 1965
- **The intestinal type:**
 - typically arises in the presence of a precancerous condition
 - gastric atrophy or intestinal metaplasia,
 - more common in men
 - incidence increases with age
 - dominant type in areas in which gastric cancer is epidemic
 - well differentiated
 - spread haematogenously to distant organs.
- **The diffuse type:**
 - poorly differentiated
 - lacks gland formation
 - composed of signet ring cells.
 - Clusters of small uniform cells, tends to spread submucosally and metastasizes early by transmurial extension and via lymphatics.
 - Poor prognosis
 - More common in women and younger age groups
 - Associated with blood type A and familial cases suggesting genetic aetiology.

etiology of adenocarcinoma:

Dietary risk factors:

- Smoked meat.
- High nitrates contents.
- Low fruits and vegetables.
- Smoking.

Demographic risk factors:

- Male gender.
- Low socioeconomic state.
- Blood Group type A.

Medical risk factors:

- H. pylori infection.
- Atrophic gastritis.
- Previous partial gastrectomy.
- Ménétrier's disease.
- P53 mutation is found in 50% of cases

the most important risk factor to know is H.pylori.

The most important risk factor for developing gastric adenocarcinoma is: ****

A. Smoking

B. Prior gastric surgery

C. Alcohol

D. Obesity

E. Helicobacter pylori infection

answer:E.

Signs of distant metastases:

- Virchow's node: enlarged supraclavicular lymph node.
- Sister Mary Joseph's node: infiltration of the Umbilicus.
- Blumer's shelf: fullness in the pelvic Cul-De-Sac (solid peritoneal deposit anterior to the rectum, forming a shelf palpated on PR).
- Krukneberg's tumor: enlarged ovaries on pelvic exam (metastases to the ovaries). -- it is caused by Transcoelomic spread-- paaaast
- Hepatosplenomegaly with ascites and jaundice.
- Irish's node: left axillary lymphadenopathy.
- Cachexia

diagnosis:

in high risk patients we can do screening with endoscopy or contrast studies

Investigations:

1. Flexible upper endoscopy + Biopsy:

- It's the investigation of choice.

➤ Take at least 7 biopsies from the edges of the ulcer → to increase sensitivity.

2. Double contrast barium enema → not used anymore.

☰ Staging: (TNM)

1. Endoscopic Ultrasound:

➤ Used for T staging → Can't detect T2.

➤ For N staging → regional lymph nodes.

➤ Can't differentiate the tumor cells from fibrosis after neoadjuvant chemotherapy.

2. CT-Scan :

➤ It's complimentary to EUS in T staging.

➤ Can't differentiate between T1 and T2.

➤ Used also for Distant metastasis and lymph nodes.

these investigations

both for diagnosis and staging are for all the gastric cancer.

The modality of choice to diagnose a patient with gastric cancer is:

A. Flexible endoscopy with multiple biopsy

B. Diagnostic laparoscopy

C. Double contrast barium swallow

D. Ct

E. Endoscopic ultra sound

answer:A

Flexible endoscopy with biopsy is the only modality that provides definitive diagnosis of gastric cancer by combining direct visualization and histopathology. While EUS/CT are critical for staging, they cannot replace tissue confirmation

HY Point 1: Endoscopy + biopsy = gold standard for diagnosis.

HY Point 2: EUS for T/N staging; CT for M-staging.

other tumors:

Gastrointestinal stromal tumors (GIST) -most common mesenchymal tumor of the GI tract

☰ Gastrointestinal stromal tumors (GIST), previously known as leiomyosarcomas are rare GI tumors arising from mesenchymal component (interstitial cells of Cajal)-paasttt-, these are only 3% of gastric tumors.

☰ sites: GI tract, from esophagus to rectum:

- Most common site → The stomach (60%) in the fundus
- The second most common sites → the small intestine (30%)

Gastric GISTs have a better prognosis than small bowel tumors. Resection is advised for tumors ≥ 2 cm.

--Usually C-KIT (CD 117) +ve

- So it's the tumor marker for GIST.
- It's a target for chemotherapy.

LYMPHOMA:

due to H.pylori infection which will cause MALTOMA

low grade is treated by eradicating the infection

High grade MALToma → Chemotherapy/radiotherapy.

surgery is for complicated cases.

All are true about GIT lymphoma except:

- A. Gastric lymphoma is the most common extra nodal site
- B. Burkitt's presentation is usually bleeding from proximal jejunum
- C. H.pylori associated with MALT
- D. Celiac associated with T lymphoma
- E. Surgical excision of stomach is reserved for those with perforation and bleeding

answer:B

Burkitt's lymphoma: Typically involves distal ileum/cecum (not proximal jejunum).

other Qs:

What is the metabolic change associated with excessive vomiting? ****

- A. Hypochloremic hypokalemic metabolic alkalosis
- B. Hyperchloremic hyperkalemic metabolic alkalosis
- C. Hyperchloremic hyperkalemic metabolic acidosis
- D. Hypochloremic hypokalemic metabolic acidosis

answer:A

One of the following can't be candidate for fundoplication

- A. Young patient
- B. Patient with paraesophageal hernia
- C. Patient with esophageal dysmotility
- D. Patient with less pressure 8mmhg
- E. Patient with lateral sliding hernia

answer:C

fundoplication is the surgery to treat GERD.

Regarding GERD, all of the following are true except:

- A. Triad of heartburn, regurgitation and dysphagia are the usual presentation
- B. Improvement on PPI is one of the diagnostic criteria
- C. Ambulatory pH monitoring is used to assess GERD in patients with persistent symptoms
- D. Esophageal manometry is used to evaluate esophageal peristalsis before anti-reflux surgery
- E. Lap. nissen fundoplication is indicated for patients with normal length esophagus

ANSWER:A

Which of the following isn't considered a cause of stress ulcer:

- A. Steroid

- B. Burn
- C. Head injury
- D. IV antibiotics

answer:D

All of the following are on the transpyloric plane except:

- A. Fundus of the gallbladder
- B. Termination of the spinal cord
- C. Dudeno-juenal flexture
- D. Neck of the pancreas
- E. Origin of inferior mesenteric artery

answer:E

Regarding Gastrin all are true except:

- A. Secreted by G cells in antrum
- B. Decreased by PPI (PPI results in hypogastrenemia)
- C. Responsible for gastric phase of acid secretion
- D. Increased in zollinger ellison
- E. When elevated causes gastric carcinoid

answer:B

PPI use: Increases gastrin (due to reduced acid feedback inhibition).

Chronic hypergastrinemia: Causes ECL cell hyperplasia → gastric carcinoid tumors (type I).

All increase gastrin secretion except:

- A. Antrectomy
- B. Vagotomy
- C. Z-E syndrome
- D. Atrophic gastritis
- E. Achlorhydria

answer:A

Metastatic disease to the stomach can occur with the following tumors. Which one is the most common?

- A. Melanoma
- B. Breast cancer
- C. Testicularcancer
- D. Colon cancer
- E. Prostate cancer memorize

answer:B

Sequence of return of gastrointestinal motility after abdominal surgery is: ***

- A. Intestine, stomach, colon
- B. Stomach, intestine, colon
- C. Colon, intestine, stomach

D. Colon, stomach, intestine

E. Stomach, colon, intestine

answer:A

All of the followings are true about bariatric operations, EXCEPT:

A. Laparoscopic gastric bypass (LGBP) is a good option for sweet eater patients.

B. The main factor for weight reduction in standard LGBP is restrictive not malabsorptive.

C. The ideal procedure for pediatric age group is laparoscopic adjustable gastric band (LAGB).

D. Laparoscopic sleeve gastrectomy (LSG) is associated with decrease in hunger hormone.

E. Laparoscopic gastric plication is associated with high failure rate.

ANSWER: B

The diffuse type of gastric cancer:

A. Is well differentiated

B. Has Good prognosis

C. Is more common in men

D. Its incidence increases with age

E. Is associated with blood type A and familial cases suggesting genetic etiology

Answer:E

Staging of gastric cancer involves all of the following methods EXCEPT:

- A. CT scanning of chest and abdomen
- B. Endoscopic ultrasonography
- C. PET scanning
- D. Laparoscopy
- E. Exploratory laparotomy

answer:E

Regarding the intestinal type of gastric cancer according to Lauren, all the following statements are true EXCEPT:

- A. Dominant type in areas in which gastric cancer is epidemic.
- B. Associated with blood type A and familial cases suggesting genetic etiology
- C. More common in men
- D. Typically arises in the presence of a precancerous condition gastric atrophy or intestinal metaplasia.
- E. Usually well differentiated and spread haematogenously to distant organs

answer:B

What's the best bariatric intervention for a patient with BMI 50, sweet eater, diabetic, hypertensive, with reflux:

- A. laparoscopic gastric bypass
- B. jejunoileal bypass
- C. gastric band
- D. sleeve

answer:A

Not a complication of sleeve?

- A. anastomosis leak
- B. stenosis
- C. nutritional imbalances

answer:A

Wrong about bariatric surgery:

- A. Gastric bypass is restrictive not malabsorptive
- B. bypass is good for sweat eaters
- C. banding is number one in children

answer:A

Bleeding artery in duodenal ulcer is:

- A. Gastroduodenal artery
- B. Right gastroepiploic artery
- C. Hepatic artery
- D. Right gastric artery
- E. Splenic artery

answer:A

In the stomach, which of the following substances is released from the D cells:

- A. Pepsin – chief
- B. Gastrin – g cells

C. Histamine – enterchromaffin like cell

D. Ghrelin – p/d1

E. Somatostatin – d cells

Answer:E

Which of the following statements is FALSE of gastrointestinal (GI) secretions?

A. Pancreatic fluid is alkaline.

B. The chloride content of gastric fluid is around 110 mmol/L. memorize

C. Gastric fluid has a high concentration of potassium.

D. Bile has a pH of 7.2.

E. Most losses can be replaced with normal saline with or without potassium
explained in genereal surgery:

answer:C

Absorption of the majority of nutrients takes place in which part of the
gastrointestinal tract? (general?)

A. Stomach

B. Duodenum

C. Jejunum

D. Ileum

E. Colon

answer:C

Wrong about GERD:

A. 90% will have esophagitis on endoscopy due to reflux (60% will show normal mucosa on endoscopy).

B. Not all types of reflux are diagnosed by PH monitoring

C. Barium swallow diagnoses hiatal hernia

Answer:A

pancreas:

it is composed of 2 components:

endocrine (islets of langerhans): which secretes hormones: insulin and glucagon - tumors arising from this part are neuroendocrine tumors-

Exocrine (Acinar, centroacinar, and ductal cells):

Acinar cells: Secrete digestive enzymes:

Trypsin

Chymotrypsin

Amylase

Lipase

Carboxypeptidase

Centroacinar and ductal cells: Secrete water and electrolytes (Na^+ , K^+ , HCO_3^- , Cl^-) in response to secretin stimulation.

acute pancreatitis:

EZ subject

the causes of acute pancreatitis can be remembered with the mnemonic

I GET SMASHED:

- I Idiopathic
- G Gallstones most common cause
- E Ethanol (Alcohol)
- T Trauma
- S Steroids
- M Mumps/Infection Viral
- A Autoimmune
- S Scorpion Sting
- H Hypertriglyceridemia TG > 1000 mg/dL
- E ERCP only in 3-10% of cases of ERCP
- D Drugs Top culprits:(Didanosine), AZA (Azathioprine), Valproate, Furosemide.

Biliary and alcoholic causes account for 90% of the cases

alcoholic accounts for 30% in western countries in Jordan it is much less

pathophys:

In acute pancreatitis, enterokinase (from duodenal mucosa) activates trypsinogen to trypsin in the pancreas. Excess trypsin then triggers a self-digesting cascade,

activating other enzymes (e.g., elastase, phospholipase A2), and it autoactivates itself leading to autodigestion, inflammation, and tissue damage.

A 25 y/o female patient presented with right iliac fossa pain, which of the following is the least possible differential diagnosis? ***

- A. Ovarian cyst
- B. Crohn's disease
- C. Ectopic pregnancy
- D. Mid-menstrual cycle pain
- E. Pancreatitis

answer:E

Which of the following enzymes has been implicated in the etiology of pancreatitis: ***

- A. Gastrin
- B. Pepsin
- C. Trypsin
- D. Lipase
- E. Amylase

answer:C

Activation of trypsinogen as an initial step in acute pancreatitis is conducted by:

- A. Lipase

- B. Amylase
- C. Enteropeptidase
- D. Phospholipase
- E. Elastase

answer:C

The enzyme that's auto activated to its active form is: **

- A. Enterokinase
- B. CCK
- C. Chemotrypsin
- D. Gastrin
- E. Trypsin

answer:E

A 45 year old male patient arrived to the Accident and Emergency department at Jordan University Hospital complaining of upper abdominal pain that radiate straight to the back with vomiting. He had unremarkable past medical illness; his vital signs were stable, examination of the abdomen showed only tenderness at the epigastrium. The most likely cause of his illness is:

- A. Perforated peptic ulcer disease
- B. Acute pyelonephritis
- C. Acute diverticulitis

D. Early acute pancreatitis

E. Viral hepatitis

answer:D

All of the following can be considered as investigations for suspected pancreatitis except:

A. abdominal U/S

B. abdominal lavage

C. abdominal CT

D. neck U/S

answer:D

For suspected pancreatitis, lipase + abdominal U/S (for gallstones) are first-line. CT is reserved for severe cases. Neck U/S has no role, while abdominal lavage is trauma-specific.

now for the diagnosis of pancreatitis we have criteria:

atlanta criteria:

we need 2 out of 3 of the following:

(1) abdominal pain consistent with pancreatitis,

(2) serum lipase or amylase levels at least three times above the upper limit of normal,

(3) characteristic findings on imaging (CT or MRI)

so if we have the first 2 we can diagnose without imaging

notes: Amylase is more sensitive.

Lipase is more specific.

The increase in amylase level is not proportional to the severity of the pancreatitis.

amylase level increases then decreases after a few days (So if the patient presented after a few days and amylase level where normal, check for lipase.

In order to diagnose acute pancreatitis, 2 out of three which of the following criteria are required? **

- A. Epigastric pain, radiological evidence of pancreatitis, serum lipase at least 2 times normal
- B. Epigastric pain, radiological evidence of pancreatitis, serum amylase at least 3 times normal
- C. Cholelithiasis, radiological evidence of pancreatitis, serum amylase at least 3 times normal
- D. Epigastric pain, cholelithiasis, serum lipase at least 2 times normal
- E. Choledolithiasis, R/E of pancreatitis, serum lipase at least times normal

answer:B

we have a score that predicts mortality in acute pancreatitis patients:

RANSEN score:

****Within 24 hours (GA LAW): [Point for each]** -here there is a difference in the ranges if it is caused by gallstones or non gallstones

Glu >200 mg, Age >55, LDH >350 U/L, AST>250 U/L, WBC>16,000

**** After 48 hours (C HOBBS): [Point for each]**

Ca²⁺ <8mg/dl, Hct decreased > 10%, O₂ (Arterial PO₂)< 60mmHg,

Base deficit>4meq/L, BUN increased> 5mg/dl, Sequestered fluid >6 L

treatment:

- Conservative management: (90% of cases resolve spontaneously)
 - NPO
 - NG suction may be needed.
 - IV hydration -the most common cause of death is hypovolemia-
 - Analgesia and antiemetic are the only drugs needed,
 - Broad spectrum antibiotics are used ONLY if infection is established

Wrong about ERCP:

- A. 1/3 of patients get pancreatitis
- B. It's a diagnostic and therapeutic procedure
- C. Could be used to perform stone extractions
- D. Can be used for biliary stenting.

answer: A only 3 -10%

pancreatic pseudo cyst:

Definition:

Encapsulated collection of pancreatic fluid in the lesser sac.

Pseudocyst's wall consists only of the inflammatory response of the neighboring organs (granulation tissue or fibrosis) and not epithelium (That's why it's called PSEUDOCyst).

most common cause is acute pancreatitis:

however By definition, a fluid collection appearing in the first 4 weeks after the onset of pancreatitis is an acute fluid collection; after 4 weeks, it becomes an acute pseudocyst.

so a fluid collection needs to be present for 4 weeks to be called pseudocyst.

Small pseudocysts may resolve on their own with out treatment; large pseudocysts with mature organized walls generally do not resolve.

Imaging:

➤ Ultrasound: Fluid-filled mass

➤ CT scan: It's the diagnostic imaging of choice; it gives information about the wall thickness, calcifications and number of pseudocysts which affect the prognosis.

treatment:

we wait 6 weeks (50% of them will resolve on their own) those that do not resolve require drainage.

-You wait 6 weeks for the pseudocyst's wall becomes mature and firm enough to hold sutures-

About acute pancreatitis what is wrong:

- A. Gall bladder stones including microlithiasis is MCC worldwide
- B. Alcohol is responsible for 30% of cases in Jordan
- C. Incidence following ERCP is 6%

answer: B

pancreatic CA:

in general tumors can arise from the endocrine part: neuroendocrine tumors
or from the exocrine part:-majority arise from the ductal system.

types

--Mature cell teratoma (dermoid cyst) is a benign, well-differentiated, extremely rare germ cell neoplasm.

❖ Solid pseudopapillary tumor is a low-grade malignant neoplasm of the pancreas of papillary architecture with special histopathological (part-solid, part-cystic) features that typically affect young women.

❖ Cystic tumors of the pancreas may be serous or mucinous.

❖ Serous cystadenomas are typically found in older women, and are large aggregations of multiple small cysts, almost like bubblewrap. They are benign.

❖ Mucinous tumors have the potential for malignant transformation. They include mucinous cystic neoplasms (MCNs) and intraductal papillary mucinous neoplasms (IPMNs). MCNs are seen in perimenopausal women, show up as thick-walled cysts in the pancreatic body or tail and they can be confused with pseudocysts. IPMNs are more common in the pancreatic head and in older men.

these have a new lecture -cystic tumors of the pancreas added this year--

All true about pancreatic cysts except;

- A. Solid pseudopapillary occurs in middle aged men and is aggressive
- B. Pancreatic pseudocysts are distinguished from other pancreatic cysts by lack of epithelial lining
- C. Ct scan is the investigation of choice for pancreatic pseudocysts
- D. Pancreatic pseudocysts don't require treatment, in most cases they resolve on their own.

answer:A

adenocarcinoma: comprises 85% of total pancreatic tumors

most common risk factor is smoking

The initial presentation of pancreatic cancer varies according to tumor location.

Approximately 60 to 70% are localized to the head of the pancreas.

The most common presenting symptoms are pain, jaundice, and weight loss.

know Courvoisier sign: which is obstructive jaundice with painless palpable gallbladder.

and Trousseau's syndrome which is unexplained migratory superficial thrombophlebitis which reflects hypercoagulable state.

metastases affect the liver/ peritoneum and lung and less commonly bone.

one of the tumor markers associated with it is (CA19.9)

Endoscopic retrograde cholangiopancreatography

- ERCP provides an opportunity to collect tissue samples
- while on MRCP it will show the double duct sign

Not an indicative symptom of pancreatic head cancer:

- A. Weight loss
- B. Clay-colored stool
- C. Dark urine
- D. Abdominal pain
- E. Diabetes insipidus

answer:E

the talk about treatment of adenocarcinoma is very long Dr.khaled focused on these points

SURGICAL (if resectable):

➤ Periampullary or pancreatic head CA → Whipple procedure (pylorus-preserving).

Body or tail CA → Distal resection (Near-total pancreatectomy)

we dont resect if

1. Liver metastasis.

2. Celiac or hepatic hilar lymph nodes involvement (outside of resection area)
3. Peritoneal implants.
4. Invasion of major vessels (Portal, celiac and SMA).

only 20% of patients present as respectable cases.

Prognosis:

- Unresectable tumor → 5-year survival is < 5% (they live about 4-6 months).
- After successful resection → 5-year survival 15-20% (they live about 12-19 months). so they even consider the surgery as a palliative surgery

weird question didn't find it in slides or dossier.

Pancreatic adeno carcinoma, which is false:

- A. 70% in the head
- B. 90% ductal
- C. In resectable, 20% 5-yr survival
- D. P16 mutation is found in more than 90% (this is true)
- E. Papillary and mucinous cystadenocarcinoma are worse prognosis

the file says it is E

DeepSeek says it is D : the P16 is found in 50% of patients.

KRAS mutations (>90%) are far more common.

neuroendocrine tumors:

most common is insulinoma: but the thing about it; 90% are benign while other neuroendocrine tumors most of them are malignant.

Gastrinoma is the second most common
but the most common in MEN1-from endo surgery--

acute abdomen:

Acute abdomen presentations vary by etiology: colicky pain signals obstruction, while continuous pain suggests inflammation. Board-like rigidity indicates perforation, and vomiting is nonspecific but common. Bowel sounds are not universally silent—they evolve from hyperactive to absent as pathology progresses.

Early acute abdomen: Bowel sounds may be hyperactive (e.g., intestinal obstruction).

Late peritonitis: Sounds become absent (but not "characteristically silent" in all cases).

small intestines:

small intestinal obstruction:

most common cause for obstruction is adhesions

A 35 year old male patient, admitted with abdominal pain, distension and excessive vomiting. He had previous history of appendectomy at the age of 18.

The most likely cause for this illness is:

A. Internal hernia

- B. Right colon cancer
- C. Volvulus
- D. Adhesions
- E. Acute mesenteric ischaemia

answer: D as it is the most common cause overall of SBO.

Crampy pain is the only non-life-threatening symptom in intestinal obstruction, reflecting visceral distension. Fever, rigidity, or silent abdomen signal strangulation/perforation, requiring emergent surgery.

Simple intestinal obstruction may be associated with all the following EXCEPT:

- A. Oliguria.
- B. Edema of the intestinal wall.
- C. Bacterial translocation.
- D. Abdominal rigidity.
- E. Leukocytosis.

answer: D

Colicky pain, distension, vomiting, obstipation (no peritonitis).

Oliguria: Due to dehydration/third-spacing (common).

Bowel wall edema: From venous/lymphatic congestion.

Bacterial translocation: Occurs with prolonged stasis.

Red Flags for Strangulation/Complicated Obstruction:

Rigidity: Indicates peritonitis/ischemia.

Rigidity = peritoneal irritation → strangulation/perforation (not simple obstruction).

(Leukocytosis): Mild elevation from stress/dehydration (not always present).

tumors of small intestine:

the most common tumor is carcinoid

Carcinoid tumors are neuroendocrine neoplasms with variable behavior based on location.

most common site is ileum

ileal carcinoids are multicentric in 30-40% of patients.

also ileal carcinoids are more malignant.-more mets-

Up to 30% have synchronous GI tumors (e.g., colon cancer).

they secrete serotonin and are of enterochromaffin origin

Carcinoid syndrome, what is wrong:

A. Comes with neuroendocrine tumors

B. Can be with MEN1

C. The syndrome is associated with 5-HIAA

D. Tumors originate from fibrous cells

answer: D

originate from enterochromaffin.

High-Risk Conditions for Small Bowel Cancer:

Crohn's disease: 10-30x increased risk of adenocarcinoma (due to chronic inflammation).

FAP (Familial Adenomatous Polyposis): 100% risk of duodenal/ampullary adenomas → cancer.

Peutz-Jeghers syndrome (PJS): Hamartomatous polyps → 15% risk of small bowel adenocarcinoma.

Scleroderma (Systemic Sclerosis):

Causes intestinal dysmotility/pseudo-obstruction but no increased cancer risk.

HY Point 1: Crohn's = most common acquired risk for small bowel adenocarcinoma.

HY Point 2: FAP/PJS require duodenal surveillance (EGD/MRI enterography).

mesenteric ischemia:

decreased blood supply to the intestine

2 main types

1-occlusive :

of the occlusive there is 2 types

embolic -a fib or infective endocarditis etc-and thrombotic-atherosclerosis-.

2- and non occlusive : hypoperfusion

Patient presented with cardiogenic shock due to MI , resuscitated, admitted to the ICU and he was having sinus rhythm then, on the second day, he had abdominal pain, dx:

- A. Mesentric artery thrombosis
- B. Non-occlusive mesentric ischemia
- C. Occlusive mesentric ischemia
- D. Mesentric embolus
- E. Venous mesentric obstruction.

answer is B

Cardiogenic shock → systemic hypoperfusion → splanchnic vasoconstriction (even without vessel occlusion).

A 60 y/o female patient with known history of HTN and A.fib presented to the ED with acute generalized abdominal pain that isn't backed up by the physical findings on her abdomen, what would be top on your Ddx list?

- A. Acute embolic mesenteric Ischemia (emboli are migrating thrombi)
- B. Chronic mesenteric Ischemia
- C. Acute thrombotic mesenteric Ischemia
- D. Acute pancreatitis

answer:A

small intestinal venous obstruction:

typically caused by thrombophilia or portal HTN

spleen:

it has the odd numbers rule

1 inch thick

3 wide

5 long

7 ounces in weight

occupies the ribs from 9 to 11

the tail of the pancreas touches the hilum of the spleen. past idea.

Splenic Ligaments:

Gastrosplenic ligament: Contains short gastric arteries and left gastroepiploic vessels.

Splenorenal (lienorenal) ligament: Houses the splenic artery/vein

Phrenosplenic, splenocolic, pancreaticosplenic ligaments: Avascular peritoneal folds.

Surgical Relevance:

The gastrosplenic ligament is high-risk for bleeding during splenectomy if not properly ligated.

The vascularized ligament of the spleen is:

- A. Lienorenal.
- B. Phrenosplenic.
- C. Splenocolic.
- D. Gastrosplenic.
- E. Pancreaticosplenic

answer in the file was A

but DeepSeek says it is D:

Critical Distinction:

"Vascularized" means vessels pass through the ligament (not just adjacent to it).

The short gastric arteries and left gastroepiploic vessels traverse this ligament, making it the only vascularized one.

(Lienorenal/Splenorenal): Incorrect → Contains splenic vessels but is not vascularized (vessels are within the ligament, not supplying it)

HY Point 1: Gastrosplenic ligament = only vascularized one (short gastrics + gastroepiploics).

HY Point 2: Ligate gastrosplenic ligament first in splenectomy to avoid hemorrhage.

HY Point 3: Splenorenal ligament contains splenic vessels but is avascular.

Context: The spleen is the most commonly injured organ in blunt abdominal trauma. Management balances preservation vs. splenectomy risks.

so we can manage splenic trauma either conservatively to preserve the spleen or we can remove it.

so U need to know that splenic traumas are graded from 1 to 5// 1 being the least trauma and 5 most. dont need to memorize their specific characteristics.

1- Non-Operative Management (NOM)

When?

Hemodynamically stable (no shock, normal BP).

No peritonitis or other operative abdominal injuries.

Any injury grade (1-5)—yes, even high-grade with contrast blush. blush is extravasation of blood into the splenic parenchyma seen on CT.

How?

Admit to ICU for 24-48h.

Serial exams + Hb checks .

along side NOM we can do Angioembolization if:

Contrast extravasation on CT ("blush").

Grade 3-5 injuries (higher rebleed risk).

Success Rate:

90% for adults, >95% for kids.- kids are higher because they have a thicker spleen capsule.

Failure signs: ↓Hb, tachycardia, ↑fluid requirement.

2. Operative Management (When to Cut)

Indications:

Unstable (persistent hypotension despite resuscitation).

Peritonitis (hollow viscus injury suspected).

Failed NOM (rising lactate, dropping Hb).

Options:

Splenorrhaphy (repair) if partial injury.

Splenectomy if shattered spleen or bleeding.

after splenectomy we must give vaccines against encapsulated bacteria.

complication of non operative management:

Delayed rupture (rare, <1%; peaks at 5-10d post-trauma).

complications of operative:

OPSS (Overwhelming Post-Splenectomy Sepsis):

Risk: 1% lifetime (50% mortality if it occurs).

Pathogens: *S. pneumoniae* (#1), *H. influenzae*, *N. meningitidis*.

another weird complication is thrombocytosis which is common we don't treat it unless the platelets reach more than 1 million

if it doesn't we don't do anything it will go back to normal with time.

preoperative considerations:

Pre-Splenectomy Essentials (High-Yield)

1. Vaccinations: Non-Negotiable

Why?

No spleen = No filtering of encapsulated bacteria → OPSS risk (1% lifetime, 50% mortality if sepsis occurs).

What to Give? *(Minimum 2 weeks before elective splenectomy, or ASAP post-emergency splenectomy)*

okay so when to give the vaccine after the surgery or before it???

good question

depends if it is elective surgery give it minimum 2 weeks before

If emergency splenectomy: Vaccinate post-op before discharge.

2- stabilize the patient

Optimize Blood Counts

Hemoglobin: Transfuse if Hb <7-8 g/dL (or higher if cardiac disease).

Platelets: >50K/ μ L for surgery (transfuse if lower).

3-VTE Prophylaxis: Aggressive

Why?

Splenectomy has ~10% VTE risk (vs. ~2-5% for other abdominal surgeries).

Mechanism: Loss of spleen \rightarrow \uparrow platelets + hypercoagulability.

What to Do?

LMWH (e.g., enoxaparin) starting pre-op or post-op.

Extended prophylaxis (2-4 weeks post-op) for high-risk patients.

above i said we deal with platelets if they are only more than 1million

and this is correct but VTE prophylaxis is different

we give VTE prophylaxis for every patient post splenectomy

so we give every pateint LMWH but if the platalets rise to more than 1 million after 1 week for example we add aspirin to the LMWH.

so U can think of VTE as immediate complication while thrombocytosis takes time to develope.

and VTE can happen even with low platelet counts.

All are true about spleen except:

- A. It spans intercostals 9-10-11
- B. Palpation started in the right iliac fossa
- C. CML is an established cause of splenomegaly
- D. Most important risk post splenectomy is hemorrhage

Answer: D

OPSI (infection) is the #1 lifelong risk (1% lifetime incidence, 50% mortality if sepsis occurs).

Hemorrhage is an acute post-op complication (not the most important long-term concern).

also VTE is more common than hemorrhage but still hemorrhage can happen??????????

The Full Truth About Post-Splenectomy Risks:

Immediate (24-48h) Risks:

Hemorrhage (from missed short gastrics/splenic bed)

Pancreatic injury (tail of pancreas near splenic hilum) also gastric injury.

Early (1-4 weeks) Risks:

VTE (#1 surgical complication, ~10% risk)

Due to thrombocytosis (platelets often >500K, may exceed 1M)

Higher than hemorrhage risk in modern surgery

Subphrenic abscess

Lifelong Risks:

OPSI (#1 long-term mortality risk)

B (Palpation starts in RIF): True. "Traps" the spleen by rolling patient + palpating from RLQ → LUQ.

C (CML causes splenomegaly): True. Myeloproliferation → massive spleen (can extend to pelvis).

patient underwent splenectomy, all are possible complications post splenectomy except:

- A. Postoperative bleeding
- B. Gastric perforation
- C. Pancreatic fistula
- D. Jaundice
- E. Pulmonary infection

answer D

pulmonary infection can happen early due to atelectasis

or late due to encapsulated bacteria if the patient is not vaccinated.

Splenic trauma lecture for Dr Khaled is so amazing and highly recommended.

LIVER:

anatomically the liver is divided by the falciform ligament into the left lobe and the right lobe. the most common lobe to be affected by abscesses is the right lobe-

E. coli is the leading cause of pyogenic liver abscesses globally, particularly in biliary-obstructed patients. *Klebsiella* is emerging (especially in Asia) but remains secondary in most settings.

if a question comes what is the most common it is *E. coli*.

while functionally the liver has 8 segments

they are divided by the 3 hepatic veins

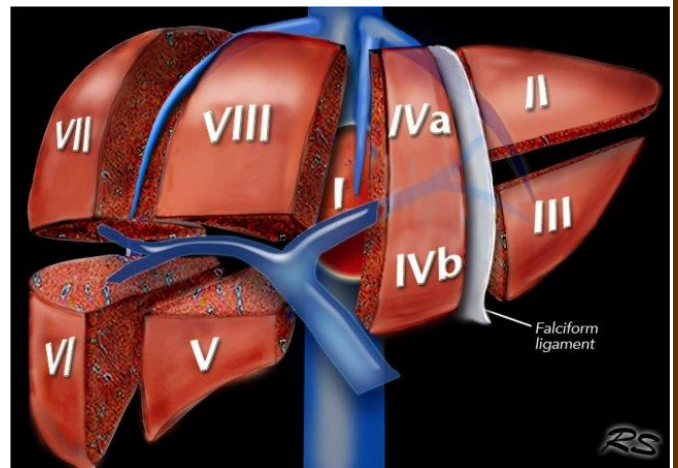
right middle and left

into 4 longitudinal segments

then the portal vein also divides those segments into those that are superior or inferior

so the end result is 8 segments

we count the segments clockwise.



to the left the falciform ligament is segments 2 and 3

while 1 (the caudate segment) is posterior to it and to the right.

the rest of the segments are to the right of falciform.

now there is a weird segment which is 4 which has 4a and 4b and they make the quadrate lobe.

Surgeon resects a portion of the liver to the left of the attachment of the falciform ligament. The segments that have been resected are:

- A. segment two and three
- B. segment one and 4b
- C. seg 4a and 4b
- D. segment one and three
- E. segment one and four

answer: A

the most common lobe to be effected by abscesses is the right lobe-

there are 3 types of abscesses

1-pyogenic (bacterial)

2- parasitic

3- fungal

E. coli is the leading cause of pyogenic liver abscesses globally, particularly in biliary-obstructed patients. Klebsiella is emerging (especially in Asia) but remains secondary in most settings.

if a question comes what is the most common it is E coli.

Biliary tract infections (e.g., cholangitis) are the leading cause of pyogenic liver abscess, especially in settings like choledocholithiasis or post-ERCP

so the source of the bacteria is usually from a biliary tract infection.

the hepatoduodenal ligament contains the 3 structures:

portal vein/ hepatic artery proper/ bile duct.

the portal vein is posterior

the artery is medial -and to the left-.

the duct is lateral -and to the right-

The hepatoduodenal ligament contain the common bile duct (CBD), hepatic artery and the portal vein in which of the following arrangement:**

- A. Artery right of CBD and vein posterior.
- B. Artery right of vein and CBD posterior.
- C. CBD right of artery and vein posterior.
- D. CBD right of vein and artery posterior.
- E. Portal vein right of artery and CBD posterior.

answer:C

the liver and gallbladder do not cover the first part of the duodenum

The 1st part of the duodenum (duodenal bulb) is not covered by the liver or gallbladder.

It lies posterior to the liver and gallbladder, but they do not "cover" it.

The quadrate lobe of the liver is anterior to the pylorus of the stomach, not the duodenum.

HYDATID CYST:

Hydatid Cyst Rupture Complications:

Anaphylactic shock: Most life-threatening (massive IgE-mediated reaction to cyst fluid).

Biliary rupture: Common (cyst fluid leaks into bile ducts → obstructive jaundice, cholangitis).

Intraperitoneal rupture: Risk of disseminated cysts ("secondary echinococcosis").

Thoracic rupture (bronchial/pericardial): Rare but severe (pneumonitis, cardiac tamponade).

Gastric rupture: Extremely rare.

Critical Distinction:

Anaphylaxis is the most acute and lethal complication.

Biliary rupture is the most common but less immediately fatal.

mechanism:

The cyst wall erodes into adjacent bile ducts in 25–50% of cases, causing bile leakage, obstruction, or infection.

Classic presentation: jaundice, biliary colic, or cholangitis.

extra: there is a sign for this complication on imaging called "Water lily sign".

The most common complication of hepatic hydatid disease is:

- A. Fever and urticaria.
- B. Rupture into biliary channel.
- C. Rupture into peritoneal cavity.

D. Anaphylactic shock.

E. Suppuration

the answer in the file was A

while DeepSeek says it is B.

Why Biliary Rupture (B) is Typically #1:

Higher reported incidence (25–50% in studies) due to anatomic proximity of cysts to bile ducts.

Leads to obstructive jaundice/cholangitis, requiring urgent intervention.

Possible Reasons for Your Doctor's Perspective(A):

Clinical experience: Fever/urticaria may be more commonly observed in early stages, while biliary rupture is diagnosed later.

Regional variations: In some settings, asymptomatic biliary communications may be underreported.

So choose what U like.

it can be treated surgically or medically with albendazole

Hydatid Cyst: Surgical Indications

Operate if:

Superficial cyst (high rupture risk).

Size >10 cm (especially with daughter cysts).

Cystobiliary communication (fistula → cholangitis/jaundice). symptomatic or non symptomatic

Mass effect (compresses vessels/bile ducts).

Infected cyst (abscess formation).

Extrahepatic location (e.g., lung, spleen). extra hepatic localized*** not multiple

Use Albendazole if:

Patient is inoperable/unfit for surgery.

Multiple cysts in ≥ 2 organs (disseminated disease).

Small/deep liver cysts (high surgical risk).

Peritoneal cysts (difficult to resect).

Incomplete surgery or relapse.

Post-rupture/aspiration (prevent secondary infection).

HY Point 1: Albendazole = #1 choice (WHO guidelines).-albendazole is the best drug by faaar.-

HY Point 2: Combine with praziquantel for tough cases (synergy).

HY Point 3: Steroids only for anaphylaxis (e.g., rupture-induced shock).

portal HTN:

The portal vein forms posterior to the pancreatic neck by the union of the splenic vein and superior mesenteric vein.

Most lethal acute complication is esophageal varices rupture: 30% mortality with first bleed and can reach up to 50%; rebleeding risk is 70% without intervention.

Pathophysiology: Varices form at gastroesophageal junction (high pressure + thin walls) → rupture → hematemesis/melena.

Management Emergencies:

IV octreotide + endoscopic band ligation (gold standard).

splenomegaly is the most common physical sign in patients with portal HTN.

another finding is caput medusa.

treatment by shunt has complications:

the most common cause of death perioperatively is hepatic failure secondary to reduced blood flow

most common cause of morbidity post op is hepatic encephalopathy.

the child pugh score is used to assess the severity of chronic liver disease

it is composed of 5 components :

albumin/ PT (INR) not PTT/ encephalopathy/ ascites/ and total bilirubin.

the previous note is for IM a little but there is a question on it.

jaundice:

3 types:

prehepatic

intrahepatic

post hepatic

revise them if U feel like U cannot differentiate them.

remember there is 2 types of hyperbilirubinemia

conjugated or direct/ and unconjugated or indirect also revise them if U like.

Conjugated hyperbilirubinemia results from impaired bile excretion (obstruction or hepatocyte dysfunction), while hemolysis exclusively elevates unconjugated bilirubin. Key clinical clues:

Obstructive causes (e.g., stones, tumors) present with high ALP + bilirubinuria.

Hemolysis shows elevated LDH, reticulocytosis, and absent bilirubinuria.-but it will have urobilinogen in the urine.-

only conjugated bilirubin is water soluble and appears in the urine.

HY Point 1: Hemolysis = unconjugated bilirubin (no bile duct involvement).

HY Point 2: CBD stone/tumor = conjugated bilirubin + ALP $\uparrow\uparrow$.

HY Point 3: Bilirubinuria confirms conjugated hyperbilirubinemia.

Obstructive jaundice is characterized by conjugated hyperbilirubinemia, bilirubinuria, and absent urobilinogen due to blocked bile flow.

the patient will have high ALP/ GGT and normal ALT/AST (early).

HY Point 1: ALP \uparrow + bilirubinuria + no urobilinogen = obstructive jaundice.

HY Point 2: AST/ALT normal unless concurrent hepatitis (e.g., gallstone pancreatitis).

HY Point 3: Urobilinogen absent in obstruction (key difference from hemolysis).

U/S is always first to confirm obstruction and assess for gallstones/masses.

MRCP is second line. it help to precisely locate the obstruction site.

ERCP is therapeutic (e.g., stent placement, stone extraction) but not first-line for diagnosis alone.

In obstructive jaundice, U/S is the initial test to identify biliary dilation, gallstones, or masses. If U/S is inconclusive, MRCP provides detailed duct imaging without invasiveness. ERCP/PTC are reserved for therapeutic interventions or complex cases

HY Point 1: U/S first in obstructive jaundice—fast, cheap, and effective.

HY Point 2: MRCP > ERCP for diagnosis (ERCP is for therapy).

In obstructive jaundice, U/S is the initial test to identify biliary dilation, gallstones, or masses. If U/S is inconclusive, MRCP provides detailed duct imaging without invasiveness. ERCP/PTC are reserved for therapeutic interventions or complex cases.

in MRCP we inject a dye into the biliary tree to visualize it.

ERCP we go with an upper endoscope to reach the duodenum then we go medially through the second part to reach the ampulla of vater to enter the bile duct

using this technique we can remove the stone

or put a stent

it was used for diagnosis in the past so it is diagnostic and therapeutic

so ERCP is a therapeutic cornerstone for biliary/pancreatic diseases (e.g., stone removal, stenting), but its diagnostic use has declined due to MRCP/EUS. The most feared complication is pancreatitis (5–10%), the percentage had a question on it :)

tumors:

Metastatic deposits are the most common malignant liver tumors (up to 20x more frequent than primary liver cancers).

Common primaries -places where it could get mets from-: Colon, stomach, breast, lung, pancreas.

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy (associated with cirrhosis, HBV/HCV).

HCC also called hepatoma.

the most useful marker for HCC is alpha fetoprotein.

HCC arises most often in cirrhotic livers, with HBV, HCV, and alcohol as key etiologies. AFP supports diagnosis, but imaging (arterial washout on CT/MRI) is definitive. Biopsy is avoided in surgical candidates to prevent seeding. Metastases typically target lungs, bones, or peritoneum. Early detection via ultrasound + AFP surveillance in high-risk patients improves outcomes

It follows a Geographical distribution of HBV which is endemic in Asia/Africa, where HCC incidence is highest.

HY factoids to know :

Alcohol accelerates cirrhosis → HCC.

Metastasis to lung/bone/peritoneum: These are the most common sites.

AFP is main tumor marker: AFP >200 ng/mL + mass = HCC until proven otherwise.

EXTRA from slides:

IF CIRRHOSIS IS PRESENT, OR HBV OR HCV ARE PRESENT, WITH ELEVATED AFP THE DIAGNOSIS IS MADE WITHOUT BIOPSY.

- IF NO CIRRHOSIS, AFP IS NOT HIGH, HBV, HCV ARE NEGATIVE , PROCEED TO BIOPSY
- PORTAL VEIN THROMBOSIS WITH THE PRESENCE OF LIVER LESION: HCC

guidelines state biopsy is contraindicated if surgical resection/transplant is planned due to risk of tumor seeding.

Diagnosis is typically made via multiphase CT/MRI (showing arterial hyperenhancement + washout)

we do biopsy if we wont be doing surgery.

there is a subtype of HCC

called Fibrolamellar hepatocellular carcinoma (FL-HCC) or FLC.

Demographics & Epidemiology: FL-HCC occurs in younger patients (15-35 years) - with no gender predominance (vs. HCC: older, male-predominant).-

without underlying cirrhosis (unlike classic HCC). also no association with HBV or HCV.

Biomarkers: AFP is typically normal (vs. elevated in conventional HCC).

Prognosis & Management: Better prognosis than HCC, but resection is often challenging due to large size and central location.

Pathology & Imaging:

Histology mimics FNH -folliculo nodular hyperplasia- (fibrous bands, large eosinophilic hepatocytes) but FLC is malignant.

Extra info not in the past:

CT/MRI:

Hypoattenuating solitary mass with central scar (scar does not enhance, unlike FNH). so looks like FNH on imaging as well but there is difference in the color of the scar.

Vitamin B12-binding protein (haptocorrin) may be elevated (investigational).

Wrong about fibrolamellar liver CA :

- A. elevates alpha feto protein in 90% of cases
- B. Doesn't have male predominance
- C. Happens in ages 5-35
- D. Cirrhosis isn't an identified risk factor

answer: A

Hepatocellular Adenoma (HCA) is a rare, benign liver tumor strongly linked to estrogen (OCPs, pregnancy).

Risks: Bleeding (most common complication)

malignant transformation (<10%, higher if >5 cm or in men).

OCPs are the #1 risk factor (relative risk ~25x with long-term use).

Size ≥ 5 cm increases rupture/malignancy risk;

Diagnosis: MRI with hepatobiliary contrast -- not CT--

management:

here is the debate for a question.

the guidelines have different approaches for different genders.

if male resect

if female:

if it is smaller than 5cm stop the OCP and look for a decrease in size after 6 months using MRI.

if it is larger than 5cm then resect immediately

in the slides the treatment is not focused on.

32 y/o Female patient was evaluated was found to have hepatic adenoma > 7cm, she is on OCPs, what to do:

- A. Stop OCPs and reevaluate in six months
- B. Observe and follow up in the next month
- C. Excision of adenoma
- D. Start her on chemotherapy

file says it is A

DeepSeek says it is C.

A 34 years old lady presented with RUQ pain. She has been on oral contraceptive pills for 10 years. CT abdomen shows a 4 cm hypervascular lesion in

the right lobe of the liver. The most likely diagnosis is:

- A. Hepatocellular carcinoma
- B. Focal nodular hyperplasia
- C. Cholangiocarcinoma
- D. Adenoma
- E. Metastatic breast carcinoma

answer: D.

gallbladder:

Biliary Colic vs. Cholecystitis:

Biliary colic: Transient gallstone obstruction → intermittent pain, normal labs.

Cholecystitis: Persistent obstruction → fever, leukocytosis, RUQ tenderness (Murphy's sign).

a key distinguishing feature is the timing of pain

in biliary colic it lasts less than 6 hours while in acute cholecystitis it will last more than 6h.

RUQ ultrasound is first-line for gallstones (95% sensitive).

HIDA scan is for acalculous cholecystitis (if US negative but high suspicion). it is called CCK induced HIDA

U inject a dye into the gallbladder.-not that important just know that U use it if the ultrasound is negative but U still have high suspicion of cholecystitis it can diagnose acalculous choly which is inflammation caused by stasis not obstruction.

Acalculous cholecystitis (10-15% of cases): Gallbladder inflammation without stones. the pathophysiology is ischemia to the gallbladder due to sepsis or shock causing stasis.-past idea-.

Caused by bile stasis (critically ill patients, TPN, burns, trauma, sepsis).

Ischemic injury → gallbladder wall necrosis.

Calculous cholecystitis (85-90% of cases): Caused by gallstone obstruction of the cystic duct.

Acalculous has worse outcome the mortality can reach 30%-50% or higher.

X ray: Only 15% of gallstones are radiopaque so X ray is not useful for biliary diagnosis.

CT: Less sensitive for gallstones; used for complications (perforation, abscess).

HY Factoid: CT misses 20% of gallstones.

HY Point 1: RUQ ultrasound is first-line for biliary pain (even if labs normal).

HY Point 2: Normal amylase + no fever = likely biliary colic, not pancreatitis/cholecystitis.

HY Point 3: HIDA scan is only if US negative but high suspicion (e.g., acalculous cholecystitis).

weird types of cholecystitis:

Xanthogranulomatous Cholecystitis (XGC) - High-Yield Facts

What it is:

A rare, aggressive inflammatory condition of the gallbladder, mimicking cancer on imaging.

Caused by chronic obstruction (usually stones) → bile leaks into gallbladder wall → foamy macrophages (xanthoma cells) + necrosis.

Key Features:

Mimics Gallbladder Cancer

Irregular wall thickening, hypodense nodules on CT, lymphadenopathy.

Often requires frozen section biopsy during surgery to rule out malignancy.

Symptoms:

Similar to chronic cholecystitis (RUQ pain, nausea) but more severe/prolonged.

Treatment:

Cholecystectomy (often difficult due to adhesions/inflammation).

May need partial liver resection if adjacent tissue is involved.

Why it's tested:

Classic "great mimicker" of cancer. If a question describes mass-like gallbladder thickening, think XGC vs. adenocarcinoma.

so basically it is inflammation of the gallbladder with characteristic cell type which is xanthomas these are lipid laden macrophages and there is destructive granuloma.

it Mimics cancer due to irregular wall thickening.

ez right?

Emphysematous Cholecystitis - High-Yield Facts

What it is:

Life-threatening infection with gas-forming bacteria (e.g., Clostridium, E. coli) in the gallbladder wall.

Seen in diabetics or immunocompromised patients.-typically seen in males / elderly and / diabetics.

Key Features:

Gas in the Gallbladder Wall

"Air crescent sign" on X-ray or CT (pathognomonic).

Symptoms:

Severe RUQ pain + septic shock (fever, hypotension).

Treatment:

Emergency cholecystectomy (open preferred due to inflammation).

IV antibiotics (broad-spectrum: piperacillin-tazobactam + vancomycin).

so U treat with emergent surgery as soon as possible if untreated mortality is up to 25%

again to make it EZ bacteria infecting the gallbladder causing gas in the wall -Gas bubbles in the wall are pathognomonic; no cancer mimicry.-

test Yourself

Which one of the following types of cholecystitis may mimic gall bladder adenocarcinoma?

- A. Emphysematous cholecystitis
- B. Xanthogranulomatous cholecystitis
- C. Acute calculous cholecystitis
- D. Acute acalculous cholecystitis
- E. Chronic cholecystitis

answer: B

Which of the following is false regarding emphysematous cholecystitis?

- A. Typical age 50 – 70 years old
- B. It is caused most commonly by *klebsiella pneumoniae*
- C. More common in diabetics
- D. More common in males

answer is B

now the treatment of cholecystitis is cholecystectomy

either gold period cholecystectomy

Definition: Performing cholecystectomy within 72 hours of symptom onset.

Advantages:

Lower risk of conversion to open surgery (due to less inflammation/fibrosis).

Shorter hospital stay and fewer postoperative complications (e.g., bile duct injury).

Prevents recurrent biliary events (e.g., pancreatitis, cholangitis).

Interval Cholecystectomy (Delayed, >6 weeks):

Used historically for severe inflammation (thought to reduce surgical difficulty).

Disadvantages:

Higher risk of recurrent gallstone complications while waiting.

More fibrosis/adhesions → technically harder surgery.

gold period is now the standard of care for stable patients.

what if a patient cannot handle surgery?

we can do Cholecystostomy

Definition: Percutaneous drainage of the gallbladder (tube placement).

Indications:

High-risk surgical patients (severe sepsis, ICU patients, major comorbidities).

Bridge to surgery (stabilize infection before cholecystectomy).

Acalculous cholecystitis in critically ill patients.

Advantages:

Less invasive than surgery (can be done under ultrasound/CT guidance).

Mortality benefit in unstable patients (avoids emergency surgery).

Limitations:

Not definitive treatment (30-40% need later cholecystectomy).

Risk of tube dislodgement/infection.

test Urself:

All of the following statements are true about cholecystitis, except: **

- A. Emphysematous cholecystitis should be treated conservatively
- B. Acalculuscholecystitis is associated with poor outcome comparing to calculus cholecystitis
- C. Golden period cholecystectomy is superior to interval cholecystectomy
- D. Xanthogranulomatouscholecystitis is difficult to differentiate from gall bladder carcinoma
- E. Cholecystostomy is an option for treatment in some cases

Correct about gas used in lap cholecectectomy?

- A. Low water content
- B. It's mainly composed of O₂
- C. It has high nitrogen content
- D. It's loaded with topical antibiotics

answer: A

Standard Gas in laparoscopy is:

CO₂ (Carbon Dioxide) is used universally due to:

High solubility in blood → reduces embolism risk.

Non-flammable (critical for electrocautery).

Rapid absorption/excretion by lungs.

Properties of CO₂:

Low water content (dry gas) → can cause peritoneal irritation.

No nitrogen (unlike room air, which is 78% N₂).

No oxygen (O₂ supports combustion → avoided).

No Adjuncts:

Antibiotics/heparin are not mixed with insufflation gas.

Gallbladder function all true except:

- A. Absorption of water
- B. Absorption of H
- C. Absorption of Na
- D. Absorption of Cl
- E. Secretion of glycoprotein

answer: B

gallstone ileus:

it is a an obstruction caused by a gallstone reaching to the small intestine and causing obstruction

now the gallstone needs a fistula to reach the small intestine -biliary-enteric fistula- usually in the duodenum.(cholecystoduodenal).-Most frequent fistula (75-85% of cases) due to anatomic proximity of gallbladder to duodenum-

now the gallstone will move through the intestine until it reaches the narrowest point which is the ileocecal valve.-if it was very big it can dislodge in other sites-

Classic Triad (Rigler's Triad):

Pneumobilia (air in biliary tree).

Small bowel obstruction.

Ectopic gallstone (usually at ileocecal valve).

The clinical picture of gallstone ileus includes all of the following EXCEPT:

- A. Air in the biliary tree.
- B. Small bowel obstruction.
- C. A stone at the terminal ileum.
- D. Acholic stools.
- E. Bouts of cholangitis.

the file says it is D.

DeepSeek says it is E.it says D is also correct but E is more correct-

Gallstone ileus most commonly caused by:

- A. Gallbladder to second part of duodenum
- B. CBD to duodenum
- C. CBD to gastric
- D. Gallbladder to gastric
- E. CBD to jejunal

Answer: A

Wrong about cholecystitis:

- A. U/S is the practical diagnostic test
- B. U/S can't differentiate between calculus and acalculus

- C. Ischemia is the cause of calculus cholecystitis
- D. We use antibiotics in all patients
- E. Emphysematous cholecystitis needs emergent intervention

the answer is C

but B is also not that correct because in calculus U will see stone while on acalculus U wont

D as well, we use only if we suspect infection. but it has been repeated and the choice was correct so if U see it in the exam it is correct.

Wrong about acute cholecystitis:

- A. Open cholecystectomy is the 1st line of treatment
- B. Physical examination shows positive murphy's sign
- C. U/S is the diagnostic tool of choice
- D. IV antibiotics are given to all patients

answer A.

Laparoscopic cholecystectomy is first-line for uncomplicated acute cholecystitis (less morbidity, faster recovery).

Open cholecystectomy is reserved for:

Complications (gangrene, perforation, emphysematous cholecystitis).or failed laproscopic .

45-year-old male, non diabetic scheduled for laparoscopic cholecystectomy, the best antimicrobial prophylaxis that have significantly lower overall infectious complications is:

- A. Cephazolin
- B. Cefuroximesodium
- C. Ceftriaxone
- D. Gentamycin
- E. Metronidazole

this question is repeated in general surgery

the answer is A

but a high yield factoid is that the goal is to Prevent surgical site infections (SSIs), not treat existing infection.

we need to Cover skin flora (Staph, Strep) and enteric bacteria (E. coli, Klebsiella).

gallstones:

types:

black seen in hemolysis and cirrhosis made purely of calcium bilirubinate make 10% of all stones "black pigmented stones occur mostly in the gallbladder" this statement is correct. Past—from slides -they might also contain calcium carbonate and small amounts of calcium palmitate.

brown from bacterial infection -infection of the bile duct by enteric bacteria e.g Ecoli. so they will be inside the bile duct -past idea-.

it is made of mainly calcium palmitate but it contains a little of calcium bilirubinate.

yellow is the cholesterol stones

risk factors for cholesterol stones can be remembered 4Fs:

female/ fertile/ forty/ fat.

other risk factors are rapid weight loss.

-Female hormones (Estrogen, pregnancy, OCPs) → ↑ cholesterol saturation.-

pure cholesterol stones made 100% of cholesterol are usually solitary meaning a single large stone not multiple stones. 10% of all stones

Small stones threaten pancreatitis via migration-they can dislodge easily and obstruct the pancreatic duct.

large stones risk Mirizzi syndrome by cystic duct compression.-it is a condition caused by obstruction in the fundus of the GB or a stone in the cystic duct which will cause inflammation and enlargement eventually obstructing the common hepatic duct leading to signs of obstructive jaundice.

now if U remove the GB there is still some risk for stones to develop.

Pathophysiology: Primary bile duct stones form due to chronic infection/stasis (e.g., biliary strictures) and require years to develop.

Guidelines: Post cholecystectomy stones within 1 year are retained stones (missed during surgery), not primary.

there is a past idea "Primary gallstones occur in one year postcholecystectomy"

this is wrong it takes years to develop

About black pigmented gall bladder stones, which is WRONG:

- A. Associated with hemolysis
- B. Associated with cirrhosis
- C. Associated with infected bile
- D. Contain mainly calcium carbonate and less calcium palmitate.

the answer in the file was C which is definitely wrong infection causes brown stones
about choice D in the slides it is written to be made of these so memorize it if U like.

All of the following are associated with increased formation of cholesterol gallstones except: *

- A. Exogenous estrogen
- B. High calorie diet
- C. Obesity
- D. Prolonged TPN
- E. Rapid weight loss.

the answer in the file was B.

TPN causes cholesterol stones- from slides-

With regard to gall bladder stones (GBS), all of the following statements are true, EXCEPT:

- A. Pure cholesterol stones are usually solitary
- B. Black pigmented stones occurs mostly in the gall bladder

- C. The main component of brown pigmented stones is calcium bilirubinate
- D. Black pigmented stones is associated with hemolysis
- E. Brown pigmented stones is related to biliary tract infection

answer:C

All are true regarding the pathogenesis of brown stones except?

- A. They are formed mainly in the bile ducts.
- B. They are related to infections.
- C. Their content of cholesterol is less than 20%.
- D. They contain little amount of calcium palmitate.
- E. The calcium bilirubinate they contain is a monomer form

answer D

Gallbladder cancer:

Gallbladder carcinoma is rare but linked to chronic inflammation (PSC, choledochal cysts), structural abnormalities (porcelain gallbladder, large polyps), and large gallstones

Large gallstones (>3 cm) increase GBC risk due to chronic mucosal irritation.

Multiple small stones are common in cholelithiasis but not strongly linked to malignancy.

Polyps ≥ 1 cm warrant cholecystectomy due to malignant potential.

Risk Stratification by Size:

<1 cm: Low malignant potential (<5% risk).

≥1 cm: Higher risk (10-50% malignancy risk, especially if >1.5 cm).

why size matter?

cholesterol polyps (85% of cases) are typically <1 cm, multiple, and benign.

Adenomas/adenocarcinomas tend to be larger (>1 cm), solitary, and sessile.

Management Guidelines:

<0.5 cm: No follow-up needed (benign, likely cholesterol polyps).

0.5–1 cm:

Ultrasound surveillance every 6–12 months.

Consider cholecystectomy if:

Growth observed (>2 mm/year).

Symptomatic (biliary pain, cholecystitis).

Risk factors present (PSC, Indian ethnicity, age >50).

≥1 cm: Prophylactic cholecystectomy (NCCN/ACG guidelines).

now an infection in the bile duct is called acute cholangitis or ascending cholangitis.

usually due to obstruction that leads to stasis/bacterial overgrowth.

Pathophysiology:

Biliary obstruction + infection (e.g., gallstones, strictures) → bacterial proliferation in bile.

Common pathogens: E. coli, Klebsiella, Enterococcus.

so the Bacteria ascend from the duodenum into the biliary tract (not hematogenous).-thus the name ascending cholangitis.-

Evidence: Cultures show enteric bacteria (e.g., E. coli), not bloodborne pathogens.

Classic Triad (Charcot's Triad):

RUQ pain, fever, jaundice (present in ~50-70% of cases).

Severe Cases (Reynolds' Pentad):

Charcot's triad + hypotension + AMS -altered mental status- (usually due to suppurative cholangitis).-has worse prognosis-.

Treatment:

Antibiotics + biliary drainage (ERCP/PTBD).-ERCP is definitive (Tokyo Guidelines).-

HY Point 1: Charcot's triad = Pain + fever + jaundice (50-70% sensitive).

HY Point 2: Reynolds' pentad = Charcot's + hypotension + AMS (suppurative).

HY Point 3: ERCP > surgery for drainage (unless unstable). we don't use surgery unless we have to.

Case of acute cholangitis, which is wrong:

- A. Hematogenous spread of the organism via portal vein
- B. Classical presentation is abdominal pain, fever and jaundice
- C. Treat by antibiotics, monitoring sepsis, and biliary drainage
- D. If suppurative could also present with hypotension and altered mental status
- E. Most commonly caused by biliary stasis and obstruction

Answer: A .

cholangiocarcinoma : which is cancer of bile ducts:

Cholangiocarcinoma (CCA) Risk Factors

Memorize these:

Primary sclerosing cholangitis (PSC) – *Strongest risk in Western countries (10-20% lifetime risk)*.

Liver fluke infection (Opisthorchis/Clonorchis) – #1 risk in Asia (up to 25% risk).

Choledochal cysts – 15% risk of CCA if not resected.

Hepatolithiasis (recurrent bile duct stones) – Common in Asia.

Chronic HCV/HBV – Less common than HCC, but still a risk.

Ulcerative colitis – Even without PSC, slight increased risk.

Toxin exposure .

Radiation exposure .

past: Factor that increases the risk of cholangiocarcinoma the most is:

PSC (primary sclerosing cholangitis).

appendix:

acute appendicitis is caused by obstruction
in children due to lymphoid hyperplasia
in adults due to fecalith
it consists of 3 parts:
base/ body/ tip.

Blood Supply:

Appendicular artery arises from the ileocolic artery (branch of SMA).

Position:

Most common position: Retrocecal (74%), not pelvic (21%).

Base of the appendix is fixed to the cecum.

Histology:

Submucosa contains abundant lymphoid follicles (peak in teens, regresses with age).

Argentaffin (neuroendocrine) cells are found at the base of crypts.

Laparoscopic appendectomy is the gold standard for acute appendicitis (NEJM 2023).

Antibiotics alone risk perforation, while discharge or non-surgical referrals are inappropriate

Regarding appendicitis, all of the following statements are true EXCEPT:

- A. The risk of developing the illness is greatest in childhood
- B. Mortality increases with age and is greatest in the elderly
- C. 20% of appendices are extraperitoneal in a retrocecal position
- D. Fecaliths are present in the majority of resected specimens
- E. CT has a diagnostic accuracy > 90%.

the answer in the file was A because it peaks in teens and young adults -20-30 YO
but deepseek says that D is also false. Also in slides it is 25%

Appendicitis is most difficult to be diagnosed in which one of the following:

- A. Infant 1 year old
- B. Pregnant woman
- C. Woman between 18-35
- D. Patient on anti-inflammatory drugs

the file says B due to the pain being retro hepatic or in the RUQ

but DeepSeek says it is A.

Appendicitis is hardest to diagnose in infants due to nonspecific symptoms (e.g., irritability, vomiting) and inability to communicate pain

Diagnostic challenges:

Cannot localize pain → presents with vomiting, fever, lethargy (sepsis-like).

High perforation rate (delayed diagnosis due to nonspecific symptoms).

Evidence: Perforation rates are >80% in infants

stupid question :

You were assisting the senior surgeon in appendectomy for twenty years old patient, at the end of the procedure the nurse in charge gave you the specimen, you should: ***

- A. Refuse the action by the nurse and ask her to keep it
- B. Carefully label it and send it for histological examination
- C. Send it to the surgical pathology museum
- D. Give it back to the nurse to put it in the medical disposable
- E. Give it to the patient to keep it

answer: B

The standard of care mandates labeling and sending all surgical specimens to pathology. This ensures diagnostic accuracy, rules out malignancies, and fulfills legal requirements. Disposal or patient handover is never appropriate.

About meckel's diverticulum, which is wrong:

- A. Fresh bleeding
- B. Causes Painful hematemesis
- C. Contains gastric mucosa
- D. Contains pancreatic mucosa

Answer:B

lower GI:

inflammatory bowel disease:

UC vs. Crohn's:

1. Distribution

UC: Continuous inflammation from rectum → proximally (always involves rectum).

Crohn's: Patchy (skip lesions), anywhere mouth-to-anus (rectal sparing common).

2. Endoscopy/Imaging

UC:

Mucosal erythema, erosions, pseudopolyps.

Backwash ileitis

(Backwash ileitis occurs in UC with pancolitis due to reflux of colonic inflammation into the terminal ileum.

Never seen in Crohn's, where ileitis is primary)

Crohn's:

Deep ulcers, cobblestoning, strictures, fistulas.

Terminal ileitis (primary, not "backwash").

3. Histology

UC:

Crypt abscesses, plasma cell infiltrates.

No granulomas (if present, think Crohn's).

Crohn's:

Non-caseating granulomas (30% of biopsies).

Transmural inflammation (fissures, fistulas).

4. Complications

UC:

Toxic megacolon perforation

Crohn's:

Fistulas (enteroenteric, perianal), strictures, malabsorption.

5. Extraintestinal Manifestations

Both:

Arthritis, uveitis, PSC, erythema nodosum.

6. Surgery

UC: Curative with total proctocolectomy.

Crohn's: Not curative (recurrence common post-resection).

UC

Ulcerative colitis is primarily managed medically, but 25-30% of patients may require surgery:

indications:

1- Fulminant Colitis with Toxic Megacolon

Definition: Colonic dilation (diameter > 5.5 cm or cecum > 9 cm) + systemic toxicity (fever, tachycardia, hypotension, leukocytosis).

2- Colonic Perforation:

Presentation: Sudden severe abdominal pain, rigidity, rebound tenderness, free air on imaging.

3- severe hemorrhage

4- failed medical treatment.

now for the types of fistulas in Crohn's

1- Colovesical (Enterovesical) - between the colon and the bladder-

Hallmarks: Pneumaturia, fecaluria, recurrent polymicrobial UTIs.

Diagnosis: CT cystogram (>95% sensitive).

note:

Diverticulitis causes 65-80% of colovesical fistulas (due to perforated diverticulum adhering to bladder). so it is the most common cause not Crohn's - past idea-

Mechanism:

Peridiverticular abscess → erosion into bladder → fistula.

2. Colointestinal

Often silent (no symptoms).

Risk: Can cause malabsorption (e.g., bypassing ileum).

3. Colovaginal

Pathognomonic: Feces/flatus per vagina.

Common in: Hysterectomy patients or severe pelvic Crohn's.

4. Colocutaneous

Draining abdominal wound/sinus.

HY Point 1: Colovesical → polymicrobial UTIs (not single-organism).

HY Point 2: Pneumaturia = colovesical until proven otherwise.

HY Point 3: Asymptomatic fistulas = likely colointestinal.

Feces/flatus per vagina → Colovaginal.

Draining skin abscess → Colocutaneous.

With regard to Crohn's disease, all of the following statements are true, EXCEPT:

- A. Bloody diarrhea is a frequent symptom
- B. The absence of granulomas does not exclude the diagnosis
- C. Intestinal obstruction is the commonest indication for surgery
- D. Malignancy occurs less frequently in comparison with ulcerative colitis
- E. Crypt abscess is not characteristic for crohn's disease

answer: A

Most common extraintestinal in crohn's:

- A. Ankylosing spondylitis

B. Arthritis *

C. Erythema nodosum

D. Iritis

answer:B

Peripheral arthritis occurs in 20-30% of Crohn's patients (most frequent extraintestinal manifestation).

also in UC --according to DEEPSEEK-

Regarding the pathology of ulcerative colitis, one is TRUE:

A. Is characterized by mesenteric creeping

B. The rectum is rarely involved

C. 10% patients have terminal ileal disease

D. Enterocutaneous or intestinal fistulae are common

E. Pseudopolyps are premalignant

answer:C

explaining E

when there is chronic inflammation of the colon it will develop some defects that look like polyps but they are not polyps -they are called pseudopolyps- these are due to the recurrent bouts of healing and inflammation seen in IBD they are benign and non malignant.

All seen with Crohn's disease except:

- A. Lead pipe appearance on barium enema
- B. Serosal involvement
- C. Skipped lesions
- D. Cobblestone
- E. Cryptitis

answer A.

lead pipe is seen in UC

crohn's is associated with string sign.

cryptitis is seen in UC.

but it can be seen in crohns in less than 30% of patients

Acute Colonic Pseudo-Obstruction (Ogilvie's Syndrome):

Pathophysiology

Autonomic imbalance:

↓ Parasympathetic tone (lack of peristalsis) or ↑ Sympathetic output → colonic paralysis.

Results in gas/fluid accumulation → massive dilation (cecum/right colon most affected)-past idea about the most affected sites-.

Who gets it? Hospitalized patients (post-op, trauma, sepsis, electrolyte imbalances).

Diagnosis:

AXR/CT: Diffuse colonic dilation without mechanical obstruction.

No transition point (vs. true obstruction).

we fear the risk of perforation which ranges from 3-15%.

Mortality jumps to 50% if perforated → act fast.

Never give laxatives (↑ rupture risk).

Not an indication for surgery in UC:

- A. Toxic mega colon
- B. Massive gl hemorrhage
- C. Refractory to medical
- D. Responsive to medical but persisted more than 7 years.

answer: D

diverticular disease:

Diverticular Disease Basics:

Prevalence: ~50% of adults >60 years in Western countries.--Young age (<50) predicts aggressive disease (higher recurrence/complications).--

Asymptomatic in 70–80% of cases; only 15–25% develop complications.

Diverticular disease predominantly affects Western populations due to low-fiber diets -not Asian or African(right sided diverticula is more common in asia -this is from slides-, with sigmoid colon involvement in >90% of cases.

While diverticulitis occurs in only 15–25% of patients -from slides and deepseek-, it remains a key diagnostic consideration for left lower quadrant pain. Diverticular bleeding is the most common cause of lower GI bleeding.

With angiodysplasia are the most common cause of massive lower GI bleeding - this is from slides and deepseek-

Complications:

Diverticulitis, bleeding, perforation, abscess.

Bleeding is acute but not the most common complication.

Key Distinctions:

Diverticulosis \neq cancer risk (no precancerous potential).

Colonoscopy contraindicated during acute diverticulitis (risk of perforation).

for imaging :

Barium enema is contraindicated in acute diverticulitis (risk of perforation → barium peritonitis).

Gold standard: CT abdomen/pelvis with contrast (shows wall thickening, fat stranding, abscess).

Barium is used only for elective evaluation of diverticulosis (e.g., chronic symptoms).

Diagnosis of acute diverticulitis (to exclude it):

A. U/s

B. Ct

C. Colonoscopy

D. Barium

the answer is B

Gold standard: CT with IV contrast (sensitivity >95%, specificity ~99%).

Key findings:

Colonic wall thickening (>4 mm)

Pericolic fat stranding

Diverticula with abscess (if complicated)

CT also stages severity (uncomplicated vs. abscess/perforation) → guides treatment (antibiotics vs. drainage/surgery).

Diverticular disease complications include bleeding, abscesses, strictures, and fistulae—all stemming from inflammation or mechanical effects. Carcinoma is not a direct complication but must be ruled out when symptoms persist.

HY Point 1: Painless hematochezia = classic diverticular bleeding (vasa recta rupture).

HY Point 2: Colovesical fistula presents with pneumaturia/fecaluria (UTI symptoms + fecal matter in urine).

HY Point 3: Strictures require biopsy to exclude malignancy.

HY Point 4: Delay colonoscopy until 6 weeks post-attack to exclude malignancy safely.

indications for surgery are:

---1 complicated diverticulitis attack

----2 uncomplicated attacks.

All the followings are complications of diverticular disease of the colon EXCEPT:

A. Carcinoma

B. Stricture

C. Lower gastrointestinal bleeding

D. Paracolic abscess

E. Fistulae

Answer: A

One of the followings is TRUE about diverticular disease:

A. Most of patients will have complications during their life

B. Young age is a good prognostic sign for the disease course

C. Bleeding is the most common complication

D. Colonoscopy should be done during the acute attack to exclude concomitant colon cancer

E. It is not precancerous

answer is E.

COLORECTAL CA:

Clinically Essential Facts About Colorectal Cancer (CRC):

CRC peaks in adults 65-74 years old young patients are <10% of diagnoses.

there are some proven life style risk factors: obesity, smoking, high red meat intake

The TNM staging system doesn't include bone metastases (DeepSeek says otherwise)

Remember that colonoscopy does double duty as both our best diagnostic tool (with biopsy capability) and primary prevention method through polypectomy. While tenesmus can signal rectal cancer, it's more often caused by benign conditions like hemorrhoids

staging predicts the survival -- 90% at Stage I which drops to just 15% at Stage IV, making early detection critical. there was a past choice about stage 1 being 90% 5year survival.

Surgical goal is to achieve a free margin locally, remove local LNs and establish safe anastomosis

the free margin concept is called R0 resection (clean margins)/ R1 means leaving behind micrometastasis at the margins/ R2 is leaving macromets.

CEA is a tumor marker for CRC but lacks sensitivity/specificity for diagnosis. It is used post-diagnosis to establish a baseline for monitoring recurrence.

For suspected rectal cancer, colonoscopy confirms the diagnosis and screens for synchronous tumors (3–5%)

. Pelvic MRI is critical for local staging - to assess mesorectal fascia involvement (critical for surgical planning).-

, while CEA provides a baseline for surveillance.-CEA is not diagnostic but used for post-treatment monitoring.- a rise will indicate recurrence

Distant metastasis evaluation requires CT (not bone scan unless symptomatic).

HY Point 1: Colonoscopy + biopsy is diagnostic for CRC and detects synchronous tumors (~5%).

HY Point 2: Pelvic MRI is gold standard for rectal cancer staging (evaluates mesorectal nodes/fascia).

HY Point 3: CEA is for monitoring, not diagnosis.

HY Point 4: Bone scans are not routine (only for symptoms/labs suggesting bone mets).

the most common site for mets is the liver

Unexplained microcytic anemia in patients >50 warrants urgent malignancy workup (e.g., colonoscopy) due to high risk of GI bleeding from cancer. Younger patients with anal symptoms or short-duration constipation rarely require malignancy evaluation unless accompanied by red flags (weight loss, obstruction).

NICE guidelines recommend urgent investigation for unexplained iron-deficiency anemia in patients >50.

Risk factors for colorectal cancer (CRC):

High-Risk CRC Factors:

IBD (UC/Crohn's): Risk ↑ with duration/extent (e.g., pancolitis >15–20 years).

Adenomatous polyps: Villous/tubulovillous histology, size >1 cm, or ≥3 polyps.

Serrated polyps: Traditional serrated adenomas (TSAs) are premalignant.

Low-Risk Lesions:

Hyperplastic polyps: No malignant potential (unless in serrated polyposis syndrome).

Critical Distinctions:

Villous adenomas have ↑ CRC risk (20–40%) vs. tubular (5–10%).

Crohn's colitis risk ≈ UC if similar extent/duration.

HY Point 1: Hyperplastic polyps = benign (no surveillance unless serrated polyposis).

HY Point 2: Villous adenomas > tubular for CRC risk (20–40% vs. 5–10%).

HY Point 3: IBD CRC risk: Starts at 8–10 years of pancolitis

All of the following parameters influence the risk of metastatic spread after resection of colorectal cancer, except

A. Degree of differentiation

B. Lymphovascular invasion

C. Positive circumferential margin

D. Lymph node positivity

E. T stage

After removing colon cancer, doctors look at certain tumor features to predict if it might spread. Most features matter:

1-Lymph Node Positivity: Cancer in lymph nodes = already starting to spread.

2- Lymphovascular Invasion:

Tumor cells in blood/lymph vessels = highway to liver/lungs.

3-Cancer at the cut edge = leftover cells = recurrence risk.

positive margins mean that when we remove the cancer we look at the margins of the cut

if there is left over cancer then the recurrence risk is high

that is why doctors do what is called a safety margin (they cut much more around the cancer in case of microcells left over)

4-Poor Differentiation (A):

Messy, aggressive-looking cells = more likely to metastasize.

T Stage:

Only shows how deep the tumor grew locally (e.g., through bowel wall). doesn't effect metastatic spread

so the answer is E.

Familial adenomatous polyposis (FAP), an autosomal dominant condition causing colorectal cancer (CRC):

Genetics:

APC gene mutation on chromosome 5q21.

Autosomal dominant inheritance; 100% penetrance for polyposis.

Clinical Features:

Hundreds to thousands of colorectal adenomas by teens/early adulthood.

Near 100% CRC risk by age 40 if untreated.

Management:

Screening starts at age 10–12 (colonoscopy/sigmoidoscopy annually).

Prophylactic total proctocolectomy is standard. we remove the rectum and all the colon

Extraintestinal Manifestations:

Osteomas (mandible, skull), desmoid tumors, CNS tumors.

HY Point 1: FAP = APC gene (5q21) → 100% CRC risk without colectomy.

HY Point 2: Start screening at 10–12 years with annual colonoscopy.

HY Point 3: Total proctocolectomy is curative; left hemicolectomy fails.

high yield note

it will have clinical manifestations in late years not in teens

meaning that if a patient doesn't perform colectomy then he will have clinical symptoms not in his teens but as an adult --early adulthood not late--

Regarding FAP, which is wrong:

- A. Polyps are adenomatous
- B. All patient will have cancer at some point
- C. Autosomal dominant, APC gene on chromosome five mutation
- D. Clinically present in teens
- E. Mostly the surgery is, colectomy with ileorectal anastomosis

answer:D

HNPCC -hereditary non polyposis colorectal cancer-(Lynch Syndrome):

Genetics & Pathogenesis:

Autosomal dominant mutation in MMR -mismatch repair- genes (MLH1, MSH2, MSH6, PMS2).

Microsatellite instability

Cancer Risks (Lifetime)

Colorectal (70-80%): Early onset (~45y), proximal/right-sided.

it is associated with other tumors:

Endometrial (40-60%): Most common extracolonic cancer.

Others: Ovarian (10%), gastric (5-10%), urothelial, sebaceous skin tumors.

from the name U might think that there is no polyps at all but this is wrong there are polyps but they are less than a 100.

important note:

Both familial and sporadic CRC involve mutations in tumor suppressors (APC, p53) and DNA repair genes (e.g., sporadic MSI-H tumors lack MLH1 due to methylation).

Key Difference:

Familial: Germline mutations (inherited).

poradic: Somatic mutations (acquired).

A 40-year-old male with cecal mass diagnosed by colonoscopy, his father, paternal grandmother, and paternal uncle all developed colon cancer by their fifth decade. Mutation of which of the following genes is associated with this disease?

- A. APC
- B. BRCA1
- C. BRCA2
- D. hMSH2
- E. K-Ras

now in this question U need to know the genes of disease and U need to know that in APC which causes FAP the cancer will appear much more earlier than 50 and the colonoscopy will show thousands of polyps that is why the answer is D

Familial adenomatous polyposis, one is TRUE:

- A. Is inherited as an autosomal recessive condition
- B. Is characterized by polyp formation in late adulthood
- C. Is best treated by total proctocolectomy and ileal pouch construction
- D. Is due to a mutation on the short arm of chromosome 15
- E. Malignant transformation is occurs in 75% of untreated patients

answer: C

One of the following is correct about familial adenomatous polyposis syndrome:

- A. Screening start at the teenage
- B. Presence of hamartomatous polyps in colon and rectum
- C. Is due to mutation at APC gene at chromosome number 8
- D. Hemicolectomy is the gold standard operation.
- E. The risk of malignancy in small bowel is 100%

answer us A

B: from the name familial adenomatous polyposis --adenomatous so the polyps are adenomatous polyps--

Which is true about Familial adenomatous polyposis:

- A. Problem on ch15
- B. 75% will develop into malignancy
- C. Polyps in late adulthood
- D. Panproctocolectomy with pouch is curative

answer:D

Anorectal:

Anatomy of the rectum:

Rectal length: ~12–15 cm in adults.

Arterial supply:

Superior rectal artery (branch of inferior mesenteric artery).

Middle rectal artery (branch of internal iliac artery).

Inferior rectal artery (branch of internal pudendal artery).

Venous drainage:

Superior rectal vein → portal system (via inferior mesenteric vein).

Middle/inferior rectal veins → systemic system (via internal iliac veins).

Innervation:

Parasympathetic: S2–S4 (pelvic splanchnic nerves).

Sympathetic: L1–L2 (hypogastric plexus).

Lymphatic drainage:

Upper rectum → inferior mesenteric nodes.

Lower rectum → internal iliac nodes.

anal anatomy:

Two Definitions of Anal Canal

Anatomical anal canal (3 cm):

Anal verge → dentate line

Surgical anal canal (5 cm):

Anal verge → anorectal ring (where puborectalis forms the anorectal angle).

Why it matters: Surgeons preserve the anorectal ring during resections to maintain continence.

Anal verge:

The opening of the anus on the surface of the body

Transitional zone between the moist, hairless, modified skin of the anal canal and the perianal skin

Dentate line (Pectinate line):

A mucocutaneous line separating proximal pleated mucosa from distal smooth anoderm

Located 1–1.5 cm above the anal verge

Formed by a series of cusps; the spaces between cusps are called crypts

Ducts of mucus-secreting anal glands open into these crypts

it is a Watershed area: divides two embryologically distinct regions with differences in:

Epithelium

Sensation

Blood supply

Lymphatic drainage

Epithelial boundaries:

Proximal to dentate line: Columnar epithelium

Distal to dentate line: Specialized squamous epithelium (anoderm), devoid of skin appendages

there is a Transitional area (Cloacogenic zone):

this is the True mucocutaneous junction (not the dentate line)

Located 1 cm above the dentate line

Lined by variable epithelium (columnar, squamous, or mixed)

Anal Glands and Sphincters

Anal glands:

8 – 12 in number.

Lay in the intersphincteric plate.

Their ducts open in the crypts.

Most of them are located in the anterior part of the anus.

Anal sphincters: Internal and External.

The internal sphincter:

Specialized rectal smooth muscle (from inner circular layer)

Involuntary

Contracted at rest

Responsible for 80% of resting pressure

The external sphincter:

Striated muscle

A continuation of puborectalis muscle

Responsible for 20% of resting pressure and 100% of voluntary pressure

Regarding the anatomy of the anorectum all the following statements are correct EXCEPT:

- A. The anorectal angle is usually obtuse in females
- B. The anal glands are mostly located in the intersphincteric space
- C. The anoderm is devoid of sweat glands
- D. The dentate line is the true embryologic squamocolumnar junction
- E. The internal sphincter is involuntary

answer is D

While traditionally taught as the embryologic junction, the true histologic transition is actually 1-2 cm ABOVE the dentate line (at the anal transitional zone).

Dentate Line \neq True Embryologic Junction (transition zone is higher).

hemorrhoids :

Hemorrhoids are normal vascular cushions in the anal canal (present in everyone) that aid in fine continence.

hemorroidal disease:

Epidemiology

Peak age: 45–65 years (not young adults).

Risk factors: Chronic constipation, pregnancy, prolonged sitting.

Hemorrhoid Anatomy:

Primary hemorrhoids: Classically at 3, 7, and 11 o'clock (left lateral, right anterior, right posterior).

Internal hemorrhoids: Above dentate line (visceral innervation = painless).

External hemorrhoids: Below dentate line (somatic innervation = painful).

External hemorrhoids are covered by skin whereas internal hemorrhoids are covered by mucosa

Most common symptom: Bleeding (bright red, painless, on wiping or in bowl). 80% of cases.

Pain occurs only with thrombosis, prolapse, or strangulation.

Grades of Internal Hemorrhoids:

1st: Bleed but do not prolapse.

2nd: Prolapse with straining but reduce spontaneously.

3rd: Prolapse requires manual reduction.

4th: Irreducible prolapse (risk of strangulation).

Treatment:

Medical (1st–2nd grade): Fiber, fluids, topical steroids.

Procedural (failed medical Rx 1st and 2nd degree, some 3rd degree): Rubber band ligation, sclerotherapy.

Surgical (3rd–4th grade): Hemorrhoidectomy (excisional or stapled).

anal fissures:

Anal fissures are linear tears in the anoderm.

they are caused by hypertonic internal anal sphincter this is usually the primary pathology but it is aided by other factors like straining and constipation.

the disease consists of a cycle of pain/ spasm/ constipation each one causes the other.

in order to treat the disease U need to cut the cycle.

Location: Most anal fissures are posterior midline (90%) due to anatomical weakness in this area. Anterior fissures are less common but more frequent in women.--lateral fissures are usually seen in IBD.

Multiple fissures → suspect Crohn's

Chronic vs. Acute: Chronic fissures develop sentinel piles (skin tags), hypertrophied anal papillae, and exposed internal sphincter fibers.

-Sentinel Pile-

What is it?

A skin tag (fibroepithelial polyp) at the distal end of a chronic anal fissure.

Pathognomonic for chronicity (not seen in acute fissures).

Why does it form?

Chronic inflammation → repeated healing/tearing → hypertrophic skin at fissure edge.

Acts as a "sentinel" (guard) marking the fissure site.

Clinical Features:

Painless (vs. fissure itself = painful).

treatment of anal fissure is either conservative or pharmacologic or surgery

conservative: high fiber diet, laxatives, warm sitz bath, anal hygiene also U can give something for the pain- like a topical anesthetic e.g lidocaine-

pharmacologic sphincterotomy: nitrates/ calcium channel blockers/ botulinum toxin -botox-

surgical: sphincterotomy it has a success rate of 90% --Risks: 5–15% minor incontinence (for flatus/liquid stool). and it is a last resort for chronic fissures (not acute).

the first line is conservative management. only go for surgery if it fails.

Acute fissures: 50% heal with conservative measures (fiber, sitz baths, topical nitrates/CCBs).

Chronic fissures: Often require sphincterotomy.

remember please:

Acute anal fissures are managed non-surgically with stool softeners, topical anesthetics (lidocaine), and vasodilators (GTN/CCBs) to break the cycle of pain-

spasm-ischemia. Surgery (LIS) is reserved for chronic fissures with fibrosis/exposed sphincter fibers.

Wrong about anal fissures:

- A. In males, it's most commonly anterior median
- B. Multiple fissures are associated with Crohn's disease
- C. Primary fissures underlying pathophysiology is increased internal sphincter tone
- D. It's equally prevalent in males and females

answer: A

anal fistula:

Definition & Pathogenesis

Abnormal tract between anal canal and perianal skin.

Cause: 90% from cryptoglandular abscess (other 10%: Crohn's, TB, trauma).

Men predominate in most series with a male to-female ratio varying from 2:1 to 7:1

Park's Classification:

Intersphincteric (70%) - most common/ low risk of incontinence.

Transsphincteric (23%) - crosses external sphincter/higher incontinence risk.

Suprasphincteric 5% Above puborectalis

Extrasphincteric (rare) Outside sphincters

Needs MRI, often from Crohn's

Trauma/post-op, hardest to treat

- An external opening adjacent to the anal margin may suggest an intersphincteric tract
- A more laterally located opening would suggest a transsphincteric one
- The further the distance of the external opening from the anal margin, the greater is the probability of a complicated upward extension
- increasing complexity and increasing laterality and multiplicity of external openings also has been observed

Clinical Features

Constant seropurulent drainage

Palpable tract on exam

External opening: Granulation tissue ("red bump") near anus

Diagnosis

Clinical exam (90% accurate for simple fistulas)

MRI fistulogram: Gold standard for complex cases

treatment:

Goal: Eliminate fistula while preserving continence. -most important thing is to preserve continence-.

Simple Fistulas:

1. Fistulotomy

Procedure: Lay open entire tract (cure rate >90%).

2. Fistula Plug/Advancement Flap

For: High-risk patients (avoid sphincter cutting).

Complex Fistulas:

1. Seton Placement:

in a new slide the doctor explained the different types of setones and asked about them during the rotation

Three Types of Setons

1-Draining Seton (Non-cutting)

Purpose: Keeps fistula open for drainage (controls infection)

Use: First step for complex fistulas

2-Cutting Seton

Purpose: Gradually cuts through sphincter muscle to eliminate tract

Technique: Tightened every 2-4 weeks in clinic

3-Medicated Seton (e.g., Silk/Vicryl)

Purpose: Induces fibrosis/scarring to close tract

rarely used.

one of the side

there is a rule to predict the path of the fistula tract it was explained by the doctor during rotation and present in slides:

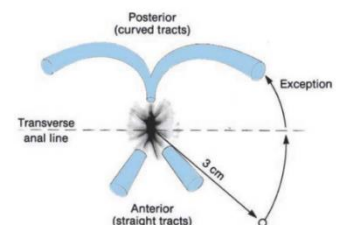
it is called Goodsall's rule:

A predictive rule for determining the path of an anal fistula tract based on the location of the external opening relative to the anal verge.

Posterior External Openings (behind a transverse anal line):if the tracts opens posteriorly

Fistula tract curves toward the posterior midline (6 o'clock position).

Exception: If >3 cm from verge → may have direct radial tract.
if the tract is more than 3 cm away from the anal canal it might be straight tract



Anterior External Openings (in front of transverse line):

Fistula tract follows a straight radial path to the nearest anal crypt.

now anal fistulas are most commonly caused by abscesses -remember?-

U can think of it this way:

Acute vs. Chronic Anal Conditions:

Abscess = acute infection (pain, fever, swelling).

Fistula = chronic complication (intermittent drainage).

Pain Characteristics:

Abscess: Continuous, throbbing pain (pus under pressure).

Hemorrhoids/Fissure: Pain triggered by defecation.

Systemic Signs: Fever/chills indicate infection (abscess),

A 34-year-old man presents with fresh painless rectal bleeding, he is constipated, his weight is steady, and his appetite is normal, he has no family history of large bowel cancer. The most likely diagnosis is?

- A. Anal fissure.
- B. Haemorrhoids.
- C. Diverticulosis.
- D. Thrombosed piles.
- E. Colon cancer

the answer is B

A will be painful

B will be in an older patient typically not in this age group

thrombosed pile -thrombosed hemorrhoid will be very painful!!!-

A young man presented with a lump in the anal region for 2 weeks, continuous throbbing pain, not related to defecation, fever and chills, most likely dx is:

A. Perianal abscess

B. Fistula in ano

C. Haemorrhoids

D. Tumor

E. Diverticulum

answer is A.

weird Q:

Concerning lower gastrointestinal bleeding (LGIB), all of the following statements are true EXCEPT:

A. If the bleeding is brisk and massive, upper GI bleeding and right sided colonic bleeding may present with bright red blood per rectum.

B. Resuscitation and initial assessment should be followed by localization of the bleeding site.

C. Radionuclide scanning is associated with a very low false localization rate for the bleeding site.

D. In addition to its success in identifying the site of severe LGIB (in $\geq 70\%$ of patients), colonoscopy offers the opportunity for therapeutic intervention.

E. Selective mesenteric angiography can detect bleeding at a rate of more than 0.5 mL/min.

choice A is correct

massive upper GI bleeding can present as fresh blood per rectum

also massive right sided colon bleeding will present as fresh blood

B: the first step is always to stabilize the patient

C this is the wrong one; it has very high false up to 30% that is why we don't really use it that much

D: colonoscopy has high success rate in identifying the location of bleeding and offers intervention

E: Angio detects bleeding at a rate of more than 0.5 mL/min

You need to memorize these. You have no choice.

Types of anal cancers:

Anal skin cancers (Anal margin tumors):

Located at the anal verge and extending up to 5 cm onto perianal skin.

Anal canal cancers:

Epidermoid carcinoma (most common).

Malignant melanoma.

Located proximal to the anal verge up to the border of the internal sphincter.

Collectively, the types are:

Squamous cell carcinoma (SCC) – Most common (80%).

Cloacogenic (transitional cell) carcinoma.

Adenocarcinoma.

Malignant melanoma.

Mucoepidermoid carcinoma.

Key Notes:

Anal cancer is rare (1% of colorectal cancers).

SCC in situ is called Bowen's disease.

Adenocarcinoma in situ is called perianal Paget's disease.

ETIOLOGY

Risk Factors:

HPV infection (Types 16/18).

HIV/AIDS (30x increased risk).

Smoking.

Immunosuppression (e.g., transplant patients).

Chronic inflammation (anal fistulas, Crohn's disease).

Multiple sexual partners / Anal intercourse.

Herpes simplex virus (HSV).

Peak incidence: 50-60 years

More common in WOMEN

Regarding squamous cell carcinoma of the anal canal, which is true??

- A. Related to HPV
- B. Most common in teenage
- C. More common in males

Answer:A

now about colonic polyps

there were 2 questions about it in the past:

it is a very long subject and to me not worth it so here are the 2 questions:

Regarding colonic polyps, one is TRUE:

- A. Hyperplastic polyps are usually large sessile polyps (>2 cm)
- B. Adenomatous polyps are most commonly sessile
- C. Villous adenomas are more common than tubular adenomas
- D. Genetic mutations can result in epithelial metaplasia
- E. Almost all carcinomas arise in pre-existing adenomatous polyps

answer:E.

All of the following statements are true regarding colonic polyp EXCEPT:

- A. Hyperplastic polyps are the most common non-neoplastic polyps in the colon.
- B. Villous histology, increasing polyp size, and high-grade dysplasia are risk factors for focal cancer within an adenoma.
- C. Distal small hyperplastic polyps rarely, if ever, develop into colorectal cancers.
- D. Hamartomatous polyps are polyps that are made up of tissue elements normally found at that site, but which are growing in a disorganized mass.
- E. Aspirin increases the incidence of metachronous adenomas and probably cancer.

answer:E

Adenoma-Carcinoma Sequence

Most GI cancers arise from adenomatous polyps.

Two-stage process:

Normal mucosa → Adenoma: Mutations in APC gene (initiates polyp formation).

Adenoma → Carcinoma: Sequential mutations in KRAS → DCC → TP53 (drives malignancy).

2. Key Mutations (Mnemonic: "A King Dies Poorly")

APC → KRAS → DCC → TP53 → Final step in malignant transformation.

3. Aspirin & Colon Cancer Prevention

Mechanism: COX enzyme inhibition reduces inflammation-driven carcinogenesis.

Dose Effects:

Low-dose aspirin (81 mg): Mild reduction in adenoma recurrence (no significant cancer prevention).

Full-dose aspirin (325 mg): Reduces colon cancer risk by 20–30%.

Protective Factors:

Higher dose (>300 mg/day).

Longer duration (>5 years).

Frequent use (daily).

GI lymphoma:

the most common site is the stomach: gastric lymphoma --most common in the distal stomach --associated with H pylori--.

second most common site is the small intestine -more common in the ileum since it has more lymphoid structure (peyers patches)

in the large intestine the most common site is the cecum.

Rectal prolapse, all are true except:

- A. More common in elderly
- B. More common in females
- C. Can be associated with constipation or incontinence
- D. Abdominal operation is associated with higher recurrence rate
- E. Endoscopy should be done for patients

now U need to know that organ prolapse is more common in elderly compared to young patients

it is more common in females -due to pelvic floor weakness from child birth-

so the typical scenario is an elderly woman

constipation and straining are known triggers

but when the prolapse happens U might get incontinence

so both are associated with the disease

now the final 2 choices are where the debate happens

the file says the answer is E

but DeepSeek says: Endoscopy (colonoscopy) is mandatory to rule out malignancy/solitary rectal ulcer syndrome.

and that Abdominal approaches (e.g., ventral mesh rectopexy, resection-rectopexy) are gold standard for fit patients due to:

Lower recurrence rates 5-10%

Perineal procedures have higher recurrence.

so DeepSeek says the answer is D

Sigmoid volvulus:

Epidemiology & Anatomy:

Most common site of GI volvulus (60–75% of cases).

Risk factors: Chronic constipation, neuropsychiatric disorders, elongated sigmoid colon.

Pathogenesis:

Redundant sigmoid + narrow mesenteric attachment → torsion.

Colonic dysmotility (e.g., Parkinson's, Hirschsprung's) predisposes.

Diagnosis:

Abdominal XR: "Coffee bean" sign or omega sign).

CT: Confirms diagnosis + rules out perforation/ischemia.

if U cant diagnose with imaging U can use enema : which will show birds peak sign which is pathognomonic in enema for sigmoid volvulus.

Treatment of Sigmoid Volvulus

1. Initial Non-Operative Management

If no signs of strangulation → Sigmoidoscopic reduction (success rate ~85%).

Contrast enema is less effective (reduction in only ~5% of cases).

Recurrence rate after reduction: ~40%.

2. Surgical Indications

Emergency surgery if:

Strangulation suspected (peritonitis, ischemia, perforation).

Unsuccessful endoscopic reduction.

Elective surgery after successful reduction:

Sigmoid resection (due to high recurrence risk).

so even after reduction is successful we plan for elective surgery:

Regarding sigmoid volvulus, all of the following statements are true EXCEPT:

- A. Sigmoid colon is the most common site of volvulus in the gastrointestinal tract.
- B. Suggested pathogenesis includes a redundant sigmoid colon that has a narrow mesenteric attachment and the presence of colonic dysmotility.
- C. Abdominal CT can be done to establish the diagnosis of sigmoid volvulus and to rule out other causes of abdominal pain and intestinal obstruction.
- D. Immediate laparotomy is done in patients with signs and symptoms suggestive of bowel necrosis.
- E. Surgery is not recommended after successful endoscopic detorsion.

answer:E

other topics:

The most common arrhythmia seen during laparoscopy is: (General)

- A. Sinus bradycardia. (peritoneal traction)
- B. Sinus tachycardia.
- C. Premature ventricular contraction.
- D. Atrial fibrillation.
- E. Ventricular tachycardia.

A

memorize ittt

All are correct about C difficile colitis except:

- A. Most likely affect elderly patients with comorbidities
- B. The use of cephalosporin based abx is a risk factor
- C. Surgery is the first line of management
- D. Oral but not intravascular vancomycin is of help in this case
- E. Can be diagnosed by performing flexible sigmoidoscopy

repeated in general surgery

answer:C

All of the following are risk factors for C. difficile, except :

- A. PPI
- B. Cigar smoking
- C. steroids
- D. Comorbidities
- E. Broad-spectrum antibiotics

repeated from general:

answer:B

All of the following are risk factors for developing clostridium difficile colitis, except.

- A. Prolonged intravenous antibiotics
- B. Contraceptive pills
- C. Mal-nutrition

D. Steroids

E. Proton pump inhibitor

answer:B

All of the following are associated with increased likelihood of surgical site of infection after major elective surgery, EXCEPT:

A. Age over 70 years.

B. Chronic malnutrition.

C. Controlled diabetes mellitus.

D. Long-term steroid use.

E. Infection at a remote body site.

answer:C

One of the following is correct about groin hernia:

A. Femoral hernia is more common in males.

B. The inguinal hernia appears medial and below to the pubic tubercle.

C. Direct inguinal hernia is lateral to the inferior epigastric artery.

D. Hernioplasty is the surgical treatment for inguinal hernia in adult men.

E. The risk of strangulation is more common in inguinal compared to femoral hernia

answer: D

55 male patient with inguinal pain, he has had a swelling that was reducible .. Now there's absent cough impulse what to do:

- A. Exploration (because pain is a sign of strangulation)
- B. U/S
- C. CT scan
- D. Iv antibiotics in the surgical ward

answer:A

A 50-year-old man presents with a complaint of a 1-cm moderately painful, tender mass situated one-third of the way between the xiphisternum and the umbilicus. What is the most likely diagnosis?

- A. Fat necrosis
- B. Omphalocele
- C. Epigastric hernia
- D. Spigelian hernia
- E. Fibrosarcoma of the abdominal wall

answer:C

Regarding abdominal surface anatomy, all of the following are true EXCEPT:

**

- A. The abdomen can be divided into 4 quadrants.
- B. The trans pyloric plane is at the level of L1
- C. The deep inguinal ring is 1.25cm below the mid inguinal point
- D. McBurney's point is located one third distance between anterior superior iliac spine

and the umbilicus

E. The umbilicus is normally situated mid way between the xyphoid process and the symphysis pubis

same question was in general

answer:C

One of the following is a bulk forming laxative:

Stimulant SodiumTicoSenna

- A. Fiber
- B. Castor oil (stimulant)
- C. Polyethylene glycol (osmotic)
- D. Lactulose (osmotic)
- E. Bisacodyl (stimulant)

answer: A

bulk forming laxatives: fibers

stimulant laxatives:,sodium ticosulfate/ senna/ bisacodyl/ caster oil

osmotic: magnesium citrate or hydroxide// polyethelyne glycol// lactulose

Which one of the followings is a good predictor for spontaneous closure of enterocutaneous fistula?

- A. Previous radiation therapy
- B. Presence of underlying abscess

- C. Long fistula tract
- D. Short fistula tract
- E. Presence of foreign body in the fistula tract

Answer:C

general question:

A major problem in nutritional support is identifying patients at risk. All of the following can identify the patient at risk, EXCEPT: (general)

- A. Weight loss of greater than 15% over 2 to 4 months.
- B. Serum albumin.
- C. Malnutrition as identified by Physical examination.
- D. Serum transferrin.
- E. Hemoglobin Level.

answer:E

All of the following is correct about Femoral hernia EXCEPT:

- A. More common in women
- B. The risk of strangulation is more as compared with inguinal hernia
- C. It present as a swelling below and medial to Pubic tubercle
- D. The sac may contain omentum
- E. Can be a cause of small bowel obstruction

answer: C -it is lateral-

Regarding abdominal wall hernias:

- A. Are 2nd to adhesions as a cause of strangulated intestinal obstruction
- B. 20% of inguinal hernias are indirect
- C. In women inguinal hernias are less common than femoral hernias
- D. The mortality associated with bowel strangulation is over 10%
- E. Trial reduction of pediatric inguinal hernias is not recommended

answer:D

if untreated mortality is 100%

if treated with in 24-48 hours mortality reaches 10%

year old male patient presented to the accident and emergency department with painful groin swelling that was reducible before. Exam showed stable vital signs. His abdominal exam was unremarkable apart from a tender swelling at the right groin and absent cough impulse. The most appropriate next step in his management is:

- A. CT scan to look for the cause of this swelling
- B. Start IV antibiotics and Observe in surgical ward for 8 hours
- C. Apply a truss gently and observe
- D. Arrange for Ultrasound scan
- E. Arrange for exploration

answer:E

Irreducible + tender hernia mandates immediate surgical exploration to rule out strangulation (even if vitals are stable).

Diaphragmatic Openings & Contents:

Aortic hiatus (T12):

Aorta

Thoracic duct

Azygos vein (sometimes)

Esophageal hiatus (T10):

Esophagus

Vagal trunks (anterior/posterior)

Caval opening (T8):

Inferior vena cava (IVC)

Right phrenic nerve branches

Which of the following structures does not pass through one of the three main diaphragmatic openings?

A. Aorta

B. Left vagus nerve

C. Left phrenic nerve

D. Inferior vena cava

E. Esophagus

answer:C

Which of the following structures is NOT retroperitoneal?

- A. Ascending colon.
- B. Seminal vesicles.
- C. Descending colon.
- D. Duodenum (D2).
- E. Ovaries.

answer:E

All of the following are formed by external oblique and its aponeurosis except:

- A. Inguinal (poupart's) ligament
- B. External spermatic fascia
- C. Conjoint tendon
- D. Superficial ring
- E. Deep ring

answer:C and E

Wrong about peritonitis:

- A. Is inflammation of peritoneum
- B. Most common surgical cause is secondary bacterial contamination
- C. Can be septic or aseptic
- D. Primary peritonitis is more common in adults than in children
- E. TB peritonitis can be with or without ascites

answer:D

Primary Peritonitis

Usually uncontrolled

Bacteria transported from bloodstream to peritoneal cavity

usually monomicrobial

More common in children

Common Microorganisms:

E. coli

Streptococci

Enterococci

Klebsiella

Secondary Peritonitis

Secondary to entry of bacteria or enzymes into the peritoneum from the gastrointestinal or biliary tract.--usually polymicrobial--

Caused by:

Perforated DU (duodenal ulcer)

Perforated appendix

Perforated diverticulitis

TB Peritonitis:

Occurs with or without ascites (dry vs. wet forms)

Classic "doughy abdomen" on exam

Wrong about peritonitis:

- A. Is inflammation of peritoneum
- B. Most common surgical cause is secondary bacterial contamination
- C. Can be septic or aseptic
- D. Primary peritonitis is more common in adults than in children
- E. TB peritonitis can be with or without ascites

answer:D.

Which of the following organisms is not a gastrointestinal source of peritonitis?

- A. Bacteroids
- B. Chlamydia
- C. Escherichia coli
- D. Clostridium
- E. Streptococci

answer:B

it is a sexually transmitted organism

Visceral Pain Stimuli:

Ischemia (e.g., infarct) → lactic acid buildup.

Stretch/Distention (e.g., bowel obstruction).

Inflammation (e.g., appendicitis).

Chemical irritation (e.g., bile/pancreatic enzymes).

Non-Stimuli:

Heat/cold → Visceral organs lack thermoreceptors.

Sharp cutting (e.g., liver biopsy is painless unless capsule stretched).

Not a stimuli for visceral pain:

A. Infarction

B. Inflammation

C. Heat

D. Stretch

E. Distention

answer:C

another general question:

A 60-year-old TPN-dependent male with short gut syndrome and diarrhea

presents with non- healing leg wound. Which trace element he may need supplementation with?

- A. Manganese.
- B. Fluorine.
- C. Selenium.
- D. Copper.
- E. Zink.

answer:E

All of the following are indications for postoperative chemotherapy after anterior resection for upper rectal adenocarcinoma EXCEPT:

- A. Node positive
- B. Lympho-vascularinvasion
- C. T4 stage
- D. Tumour size above 3 cm
- E. Bi-lobar liver metastasis

there was an idea like this one explained in colon Ca

we dont use tumor size as a factor in determining mets

answer:D.

