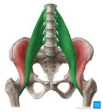
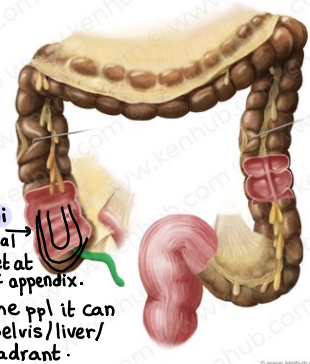
Appendicitis

*Appendix → can be 2cm-20cm normally, connected to cecum.
It is considered a lymphatic organ, it's full of lymph tissue & called "the tonsil of the abdomen."
It's body moves freely but the m/c loci is retro-cecal.

*remember: Psoas major muscle is behind cecum, it flexes the hip joint.



*Note: due to extensive variations in it's loci bet. ppl, it's pain doesn't always have to be in the right lower quadrant.



Taeniea coli → 3 longitudinal muscles meet at the base of appendix.

*in some ppl it can reach pelvis/liver/left quadrant.

*sharp constant pain.

*7% of people have it, can happen at any age, but peak=teens-30s.

*pathophysiology:

viral disease causing lymph node hyperplasia. m/c in children.	fecalith (m/c=adults) (stool obstructs it).
idiopathic/tumors	① Appendith: foriegn body causing obstruction (↓ common). ② Helminth: worms. (rare).

* Blockage of it's fluid secretion's drainage → intraluminal HTN → venous congestion & infarctions → micro perforations → frank perforation within 3 days.

*venous congestion (stasis) → opportunity for E.coli, pseudomonas & anarobes. (PEa).

*perforation: happens in 10% of p's. within 24-72hrs of pain onset.

*perforation m/c: <10 yrs old - >50 yrs. (age extrimities)

*presentation:

vague periumbilical pain (reffered) (can be epigastric) then → RLQ pain then → Anorexia (loss of appetite) then → tenderness then → fever ⑥

*when inflammation sp. reads to surrounding peritonium it becomes somatic & we feel it in the RLQ

then → nausea & vomitting + food intolerance.

*N/V → cuz peritonitis → ↑ sympathetic stim. → ↓ gi motility → N/V.

* Note: if perforation happened: ↑↑ pain, diffuse peritonitis, ↑↑ fever & ↑↑ WBC (>18K). & in abdominal x-ray → air under diaphram.

*if appendix touches rectum p can present with tenesmus or diarrhea.

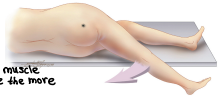
*physical: (p has some depending on site).

① Mc burney's sign:

→ when u ask p to point at pain he will put his finger at 1 & if you draw a line 1 will link umbilicus to ant sup iliac spine (ASIS).

② Rouvsing's sign: Pressing on LLQ induces pain in LRQ.
(+ve cuz of peritonitis).

③ psoas sign:



*the more you stretch a muscle by doing it's opposite move the more it hurts.

Psoas Sign: pain upon passive extension of hip, cuz we're causing tension on psoas major.
Flexion causes relaxation of psoas major muscle.
(cuz inflammation ↑ it's pain when it's stretched → extended, to ↓ pain it destretches (flexes)).

④ obturator sign: RLQ pain internal rotation of R. hip.
(this stretches obturator internus muscle (it also lies near to appendix).

(happens if appendic is pelvic)/inflammation in pelvis.

⑤ Pain in pelvis/scrotum: peri-rectal exam.
(if it was in pelvis).

* in pregnant p's pain is in the upper abdomen cuz uterus pushes app upwards. (periumbilical/upper right abdomin).

⑥ Signs of peritonitis:
Gaurding = local peritonitis (voluntary). (all abdomen). + Rigidity = involuntary spasm of muscles (localized).
Rebound tenderness: when u remove ur hand from site pain ↑↑.

-Labs: mild leukocytosis (12K-18K), or normal WBC in 10%. Urinalysis can have RBC and some WBC. (in pelvic appendicitis, cuz it can irritate bladder)

normal WBC: 4.5K-11K.

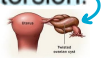
-Imaging: AXR: ① normal (abdominal x-ray) & usually looks (sometimes shows ↑). loss of Psoas shadow, fecalith, ② decreased RLQ gas. US shows inflamed appendix or pelvic fluid in children or thin adults (signs: thick wall >3mm, non-compressible >6mm outer diameter, periappendicial fluid, appendicolith, hyperemia, reactive LN, target sign, etc). CT Scan: dilated appendix or thick wall, pelvic fluid, fecalith, perforation, fat stranding. (a sign of omental thickening).

*CT is the best way. But sometimes for childrens safety/lean adults we use US.
*most imp US sign: non-compressible dilated appendix.
*RLQ loss of gas: ① patient doesn't eat from pain. ② inflammation can cause temporary cessation of ileus function.

Alvarado score: MANT2REL2S (① migrating pain, ② anorexia, ③ nausea, ④ RLQ tenderness 2 points, ⑤ rebound tenderness, ⑥ elevated temperature, ⑦ leukocytosis 2 points, and ⑧ shift to left). If <5 appendicitis is unlikely, if >8 it is highly likely. (now we depend on imaging more).
(⑧ WBC (↑ pmns).

Differential diagnosis:

- GI disease in RLQ: Meckel's diverticulitis (① in SI), mesenteric adenitis (②), epiploic appendicitis (③), cecal (④) +IBd. diverticulitis, GE. Atypical: cholecystitis (like in pregnancy), PUD (peptic ulcer disease), SBP. Typhlitis (↳ cecal inflammation in ppl who have Neutropenic fever from chemotherapy). (shifting pain).
- UroRenal: ① pyelonephritis, ② stones, ③ testicular torsion, epididymitis.
- Gynecologic: ① PID (pelvic inflammatory disease), ② EP (ectopic pregnancy), ③ ovarian cyst, ④ ovulatory pain, ⑤ torsion.



* mesenteric adenitis: Lymph node inflammation due to a recent infection (bacterial, yersinia) mainly in children. mc differential diagnosis in children.
* epiploic appendicitis: inflammation in epiploic appendages. (aka: appendigitis).

Treatment

Complicated (perforation/abscess)

- no peritonitis → antibiotics + immediate appendectomy
- ✓ peritonitis (surgical abdomen) (gaurding, rigidity...). surgical exploration + wash out of peritonium. + delayed appendectomy.

non-complicated

Antibiotics (25% have recurrence after a year).

* standard txR: ① periop. Antibiotics. ② appendectomy.

* Note: we never do any surgery when there abscess cuz 1- ↑ risk to open. 2- Suturing inflammed tissue will reopen & fail.

* Note: when we have appendix perforation (base) we can't apply a patch, we have to remove the whole ascending colon!

* Note: viscera doesn't feel pain, (they have ↓ pain receptors) so their pain is very dull & vague; if we stabbed our visceral organs we won't feel a thing, but when the inflammation spreads to surrounding peritonium we start feeling pain AKA: Somatic pain.

* Note: Omentum is the gaurdian of the abdomen, if there's an inflammation it moves/↑ in that area. (so in x-ray it obstructs rays, looks dark).

Small bowel obstruction: (mechanical).

Pathophysiology:

- Initially: peristalsis increases proximally (causing pain), later bowel can fatigue resulting in proximal dilation. The distended bowel will sequester fluids and electrolytes inside causing third spacing. This can cause hypovolemic shock.

(1) periumbilical + colicy. (2) (3) (4) (5)

↳ distal, prox. sequester: take fluids from blood

3rd space = fluid is inside lumen, not vessel/tissue (edema)

- Ischemia and bacterial translocation can lead to perforation (ischemia due to ↑pressure on wall).

(1) (2)

- More proximal obstruction is associated with excess vomiting (and hypochloremic hypokalemic metabolic alkalosis). Distal obstruction: more water sequestration and less electrolyte abnormalities. (decreased vomitting). (Both have hypovolemia).

→ due to vomitting.

Presentation:

- Nausea/vomiting, distention, pain, constipation/obstipation. If blood seen may suggest ischemia from strangulation. Proximal obstruction has more vomiting and less distention. Obstipation and more distention in distal obstruction. (colicky pain).

(1) (2) (3) (4)

with time pain decreases, cuz it's colicy → related to peristalsis → peristalsis fatigue with time.

- Physical: (1) fever (↑ sympathetic stimulation) (2) tachycardia, (3) abdominal distention. (4) Do PR. (↳ perineal exam (nothing/bleeding)). + in early → ↑peristalsis & bowel sounds. + look for signs of systemic perforation (rigidity, rebound t.).

- Labs: high WBCs (↳ bacterial translocation), electrolytes abnormalities (↓K, ↓Cl, ↑HCO_3^-) and loss of fluid (high BUN, creatinine and Hct) (↳ prerenal acute injury). (↳ Bleeding = ↓Hct. ↳ dehydration = ↑Hct)

Causes:

- Adhesions are MCC in adults. Incarcerated hernia is MCC in children and 2nd in adults. Other causes: neoplasia, intussusception, volvulus, strictures (Crohn's, postoperative, ischemic, radiation), gallstone ileus, bezoar, external (annular pancreas (↳ pancreas surrounds duodenum), SMA, pancreatic pseudocyst, abscess, abdominal compartment syndrome, pregnancy).

* colicy pain: comes & goes every ≈ 20 mins (related to peristalsis).
* Adhesions: mainly in ps who underwent many surgeries.
* Bezoar → foriegn body accumulation (p who takes many drugs/eats hair/حشيش/حشيش بزر).
* Constipation → no motility, present flatus.
  obstipation → no motility, no flatus → worse.
* Adhesions can compress organs/move them out of place/form kinks. (inner scars).
* when there's vomitting = 100% presence of nausea, but not the opposite.

Imaging:

- Upright AXR: AF levels in small intestine, decreased gas in colon. Look for pneumoperitoneum if perforation.
  (air fluid)

- Small bowel follow through: if in doubt, and suspected partial obstruction.
  (like UGi series).

- CT with IV and oral contrast: test of choice to diagnose obstruction: proximal dilation, transition point, distal collapse, (thick wall and fat stranding suggest ischemia/inflammation), pneumatosis intestinalis suggests necrosis. (gold standard → ct). (can tell us cause & area & type).

Treatment:

① NPO and ② bowel rest. ③ IVF replacement. ④ NGT decompression (lavage).

Partial obstruction resolves in 70% without surgery. If suspected ischemia (fever, rising WBC, increased abdominal pain) needs exploration. → Basic managment & follow up.

↳ cuz necrotic tissue is a breeding ground for many micro-organisms.

Complete obstruction requires exploration after stabilization → surgery.

*Note: UGi series only to doudenum, small bowel flow to ilium.

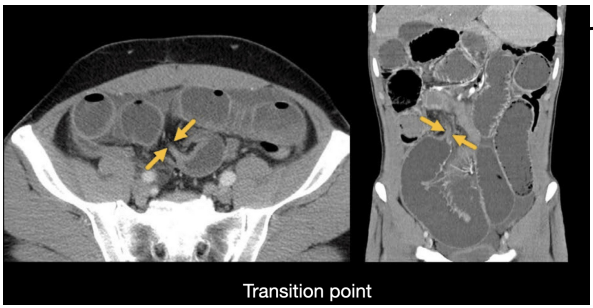
Paralytic ileus: (non-mechanical bowel obst.).

- Picture like mechanical obstruction but due to reduced motility (less or no pain, no peristalsis). ↑↓ bowel sounds.

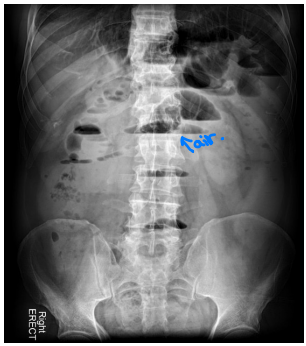
- MCC: ① laparotomy. Other: ② drugs (opioids, anticholinergics), ③ electrolyte (low K, low Na, low Mg, uremia), ④ autonomic neuropathy, ⑤ infections/sepsis, ⑥ ischemia. (↳ inhibit vagus.)

- Treatment: ① bowel rest. ② Correct fluids and electrolyte. ③ Stop causes. ④ Laxatives, ⑤ ambulation, ⑥ gum chewing.
  (↳ ↑ motility. ↳ walk. ↳ vagal stimulation.)

*Note: we watch p's motility after laparoscopy.



→ ct scan.
(you can locate small intestines from plicae circularis (distal doud → proxi ilieum).



x-ray.

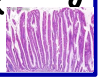
Small intestine neoplasia

Benign

'HHAL'.

Adenoma 35%

- m/c → ilium.
- villious ↑ malignant risk
- glandular tissue



Hamartoma

- Seen in Peutz Jeghers syndrome (AD).
- Peri-oral melanocytic maculae.
- m/c in jeujenum & ileum.
- X risk of malignancy but ↑ of bleeding & intussusception.
- 50% have colorectal lesions.
- 25% have gastric polyps. (it's in mucosa).

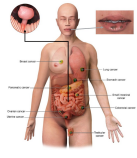
Lieomyoma / lipoma

(submucosa).

Hemangioma

- Seen in Turners
- Seen in heridatory hemorrhagic Telengectasia.
- sub/mucosa.

* Telengectasia = angioma.
* Turners: one of females X is fully/partially absent.



* peutz jegher's (a genetic syndrome)
* intususception → one part of intestine enters the other.

malignant lesions

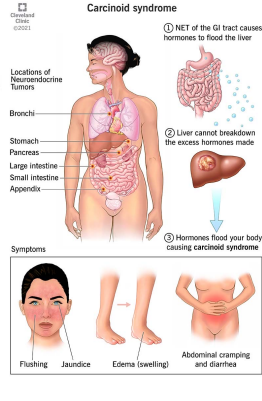
Carcinoma (50%)

- glands.
- Risk f: ① fap (familial adenomatous).
  ② HNPCC (heridatory non-polyposis colorectal cancer).
  ③ Celiac & crohn's.
- doudenal.
- can cause: obstructive jaundice/bleeding/obstruction.

Carcinoid (40%):

- Carcinoid (40%): from enterochromaffin cells of Kulchitsky in crypts of Lieberkuhn. MC in appendix. Up to 30% have multiple lesions. Diagnose with 25-h urine (5-HIAA) or pentagastrin stimulation test. Octreotide scan locates tumor. Do echo for carcinoid heart disease. Treatment small bowel carcinoid (if >1cm or LN, do wide resection. Otherwise do local resection), appendix (tip and 2cm or less: simple appendectomy/ if >2cm, involves base or has LN: right hemicolectomy).

- Carcinoid syndrome: if liver metastases: episodic flushing, sweating, bronchospasm, watery diarrhea, and rarely right heart disease. Treat with somatostatin. (anti diarrhea).
(it's a complication of neuroendocrine tumor (NET)).

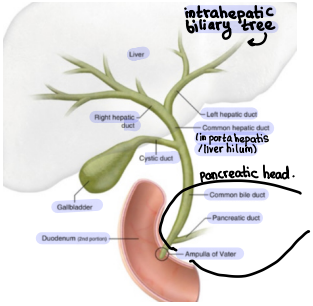


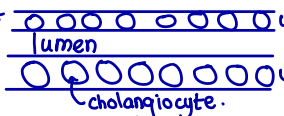
Biliary system anatomy

*liver participates in the biliary system in 2 ways:

① containing biliary tree inside it (tree → a network of biliary ducts).

② synthesizes bile in hepatocytes → tree → gallbladder.



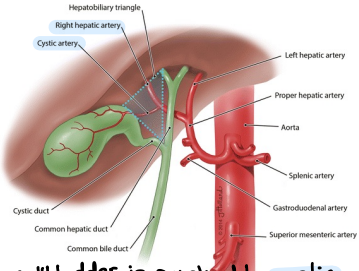


Biliary duct.

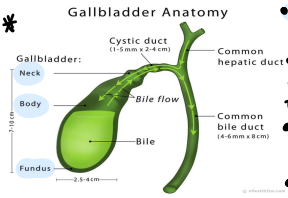
hepatocyte makes bile cholangiocyte tranports it.

*Gallbladder is considered extra hepatic.

*The superficial part of the gallbladder that appears ant. can be felt on The 9th right rib midclavicular spot upon palpation.

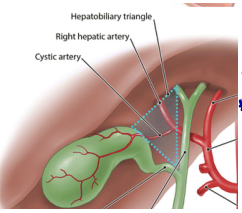


gallbladder is supplied by cystic artery (branch of right hepatic artery) (it's vessels are in serosa & muscularis).



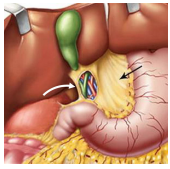
- Hartmann pouch is the widest part of the neck.
- inside the wall of the gallbladder is a smooth irrigular shaped muscle that contracts to squeeze gallbladder.
- mucosa of gallbladder contains folds to ↑ surface area.
- it doesn't have a submucosa, so if it was scarred it's so hard for it to heal (no lymph).

*it's main function is concentration of bile by absorbing water in it (this makes it more efficient). (capacity = 20-30 ml).



It's imp to be able to recognize hepato biliary triangle, in order to ligate cystic artery & cystic duct.

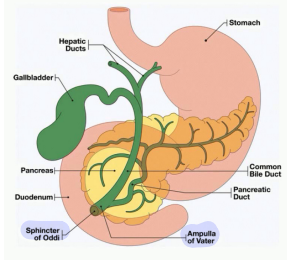
*AKA: calote triangle.



Hepatodoudenal ligament

*not an actual ligament, it's a peritonial fold that connects liver & doudenum, it contains: common bile duct, hepatic artery & portal vein (these 3 are called portal triad).

*common bile duct goes distally through hepatodoudenal ligament to reach doudenum, → post to first part of doudenum → inside head of pancrease. (it never runs inside doud.) → meets pancreatic duct → ampulla of vater → opens in 2nd doudenum. (in doud. it's cont rolled by sphincter of Odi).



*Bile → ≥90% water
- ⑥ Bile acids → conjugated with acid = bile salt → fat digestion.
- cholesterol (extra unwanted, to be excreted in bile.
- phospholipids
- conjugated bilirubin (to be excreted in bile).
- calcium salts (Ca^{+2}).

*Bile salts are reabsorbed back to the liver from the terminal ilium (this full cycle is called Entero-hepatic circulation).

- Bacteria present in SI can do deconjugation of bile salts, liver does reconjugation again.

*cholestrol isn't H_2O soluble, so phospholipids & bile salts emulsify it in gallbladder.

+ conjugated bilirubin → urobilinogen → stercobilin (brown colour of stool) {this hoppens in doudenum by bacteria).

- Some urobilinogin is left out, it's reabsorbed by liver & secreted again. & if anything still remains it's taken by kidney → in urine as urobiline. (So if there's high unconjugated bilirubin release → ↑urobilin in urine & if ↓release → pale stool + ↓urobilin in urine. (dark urine).

(bilirubin is produced unconjugated, but it's conjugated in gallbladder).

Biliary Diseases

Biliary stones = cholithiasis (stones in gallbladder).

- 10-20% of people develop it.

- **Risk factors:** more with age, more in women. Estrogen reduces bile acid (prevents stone formation) and increases cholesterol content in bile. Female, fat, fertile, forty: 3x risk.

- **Other risk factors:** transplant recipients (liver), chronic hemolysis (hereditary spherocytosis, sickle cell, thalassemia), prolonged TPN (total parenteral nutrition), and rapid weight loss (m/c) → (Bariatrics) → ↓ food. + meds like fibrates & cholestyramines. + Crohns disease.

- **Most commonly asymptomatic** <20% develop colic. 4% develop cholecystitis, obstructive jaundice, or pancreatitis. (formation of stones ≠ obstruction, that's why it's mostly asymptomatic).

*cholestrole is like oil if it's not emulsified it forms cluster → Stones.
*Stones = 50-100% cholestrole. (rest = unconjugated bilirubin & calcium salts → these make it rigid).
*Estrogen ↑ amount of cholestrole in relation to bile acids → ↑risk of sto

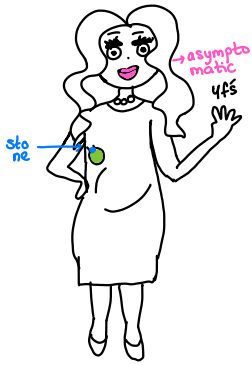
Pathophysiology:

- Bile is composed of water, bile salts, cholesterol, phospholipids (mainly lecithin), bilirubin, and salts (calcium and others). Bile salts and phospholipids act as anti-stone factors. Dehydration of bile (low water), loss of bile salts or PLs, and/or excess of cholesterol/bilirubin causes stones. Gallbladder stasis plays an important role.

- Types: non-pigmented (cholesterol) stones: MC in West (75%), and pigmented (UB+Ca-bilirubinate) MC in East; they include black stones (from excess UB, as in hemolysis) and brown stones (from bacterial deconjugation/ or foreign bodies).

* gallstones
  - ↓ in anti-stone factors
    1. Bile salts.
    2. phospholipids
    3. ↓ motility (stasis).
    4. dehydration.
  - ↑ in stone forming components.
    1. Cholestrol ↑.
    2. unconjugated bilirubin ↑.

*TPN: Total parenteral nutrition: pat. relies completely on routes other than GI for feeding. (↑ risk of stones cuz no food = ↓ stimulation of gallbladder).
* gallbladder is activated by vagus & cholecystokinen (CCK)



* Stone types
  - Cholestrol/mixed (non-pigmented). (m/c in west). (mixed = cholestrol + Ca^{++} salts/ unconjugated bilirubin).
  - Pigment stones
    - Black ● from unconjugated Bilirubin only (more common). cause: Hemolysis.
    - Brown ● unconjugated bilirubin + calcium salts. cause: infection/foriegn body in biliary system/bacterial deconjugation. (m/c in east)

*bacterial infection can cause swelling of ducts → biliary stasis → stones.

*Note: colithiasis doesn't usually cause jaundice, cuz the obstruction is only in gallbladder, so bile can't be stored anymore, but it can still be released from ducts to doudenum.

*obstruction either causes inflammation → acute cholysistitis or colicy pain.
* pain because gallbladder muscles try to push against the stone.
* Cholesystitis = inflammation (caused because trapped bile causes irritation to the walls).
*main way to know if obstruction caused inflammation?
① ↑ WBC ② fever ③ ↑ enzymes (ALT, AST, ALp).

Biliary colic:

- Transient obstruction of GB outlet (cystic duct-CBD) with stone ① or sludge ② causing distention.
  ↳highly concentrated bile.

Presentation:

- Biliary colic pain RUQ-epigastrium (may radiate to back ① or right scapular angle ② (Boas sign)), abrupt (sudden) in onset and constant (not a true colic). Promoted by meals. Also, nausea. (can radiate to right shoulder).

- RUQ-epigastrium tenderness ①. NO fever ②, no signs of peritonitis ③, no jaundice ④.
  ↳if obstruction caused pain only without inflammation.

- Normal labs with possible rise in ALP.

Imaging:

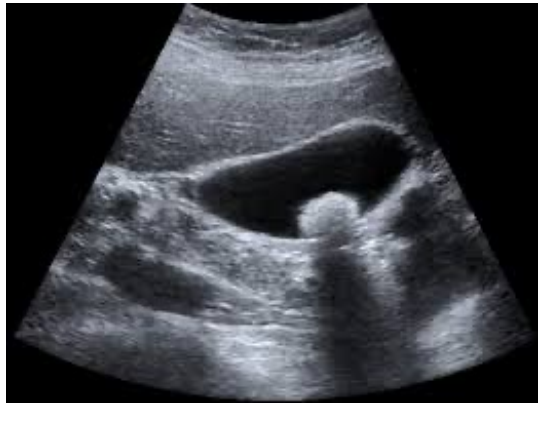
- X-ray: 10% of stones are opaque.

- RUQ US: 1st choice: 95% sensitivity. Echogenic ①, mobile ②, shadow ③, inside GB ④.
  ↳appears white in US.

- treat with cholysistectomy.

* Biliary colic pain isn't constant it comes in waves (starts & stops suddenly). (↑ mainly after fatty meals).
* Not a true colicy pain= constant.

* not relieved with analgesics.



Choledocholithiasis: (stone in ducts).

- 5-10% of patients with biliary colic have CBD stones as well.

Presentation:

- Same as biliary colic but with obstructive jaundice (① icterus → yellow skin, ② acholic stool → pale, ③ dark urine → it accumulates in blood → Kidneys → excreted in urine.)

- **Labs:** elevated ① bilirubin, ② ALP (plays a role in bile flow) and ③ GGT (antioxidant that protects cells from damage).

Imaging:

① - **RUQ US:** poor sensitivity and operatory dependent for CBD stones (unlike gallstones). But high sensitivity for CBD dilation (normal is 6mm, up to 8mm in elderly). (with age they naturally widen).

② - **MRCP and EUS:** good for diagnosis, but no therapy. (magnetic resonance cholangiopancreatography / Endoscopic us).

③ - **ERCP:** diagnostic and therapeutic. Endoscopic retrograde cholangiopancreatography

④ **Intraoperative cholangiography IOC and intraoperative US:** good sensitivity for CBD stones.

Treatment:

- Must remove stones to prevent cholangitis. This is either done before cholecystectomy (preoperative, with minimally invasive procedures (i.e. ERCP)), or intraoperatively (CBD Exploration (transcystic or transductal), and if intraoperative clearance fails, with post-operative ERCP.

*we do cholesystectomy to avoid complications (cholesistitis) & to prevent recurrence). & we remove the stones before surgery because the surgery may be done after a few days but until then the stones may cause cholangitis.

- ERCP: perform sphincterotomy and stone extraction. Failure ranges 4-10%, mortality is <1%. The MC complication of ERCP is ① pancreatitis (10-15%), others include ② bleeding (hemobilia), ③ duodenal perforation, ④ cholangitis.

*we remove the stone through sphincter of Oddi. (sphincterectomy)

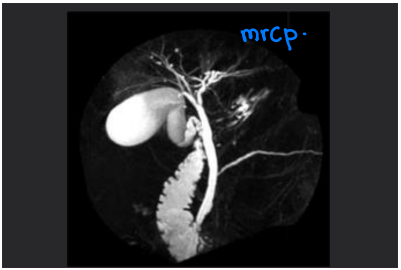
*stone in common Biliary duct → cholidocholithiasis, causes → obstructive jaundice. / cholangitis (inflammation).

*If stone reached pancrease → Biliary pancreatitis.

Symptom	cholithiasis colic	cholesystitis	cholidocholithiasis colic	cholangitis.
pain	✓	✓	✓	✓
jaundice	X	X	✓	✓
systemic symptoms (↑WBC, fever, ↑ALT, Ast).	X	✓	X	✓

when 3 are present = charcot triad.

*Remember → any stone that causes pain / infection → cholysestectomy.



Acute cholecystitis:

* Acute cholecystitis
  - Calculous: stasis + ischemia.
  - Acalculous: stasis → TPN · ischemia → shock.

* stones → ↑ pressure inside gb → biliary colic → ↑ venous pressure + ischemia → inflammation + stasis + bacterial overgrowth.

Etiology:

1. Calculous (due to stones).

- From GB obstruction with stone or sludge that leads to distention and stasis. Increased pressure leads to venous congestion and ischemia/inflammation. Also, stasis allows bacterial overgrowth (bile culture is positive in 50-75%, MCC being ① GNB (G-ve), + ② anaerobes and ③ Ecoli + ④ enterococcus).

2. Acalculous (rare)

- In the critically ill (stasis), ② shock (ischemia), or in ③ prolonged TPN (stasis).
  ① Gmeds + ↓food.

Presentation:

- RUQ-epigastric pain (not true) constant with right shoulder/scapula radiation, persisting and increasing over hours (unlike colic)). With additional fever, N/V, and anorexia.

- Physical: ① fever ↓grade, ② tachycardia, ③ RUQ tenderness. ④ Murphy sign. + ⑤ mild leukocytosis.

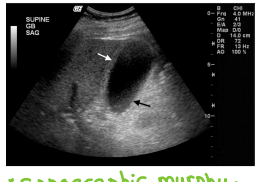
- Labs: mild leukocytosis and high ALP/GGT. ① High WBC suggests gangrene, ② cholangitis, or ③ pancreatitis. + crp.

+ pain is associated to signs of local peritonitis → Murphy's sign + rebound tenderness + local tenderness.
(murpheys; if you put ur hands on the site of pain in the abdomen & ask the patient to inhale, he won't be able to).

Imaging:

- RUQ US: 1st choice. Operator dependent with 60% sensitivity. Look for wall thickening (>4mm), pericholecystic fluid, sonographic Murphy (best sensitivity)! can see stones/sludge.

- HIDA scan: gold standard. Use if US negative but still suspected. Failure of GB filling in ④ hours is diagnostic.



* sonographic murphy: when us handle is put on the area, p stops breathing
* pericholysistic fluid → inflammation of GB wall can cause some bile to leak.

Treatment:

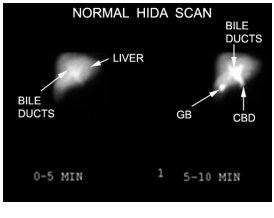
① Mainly antibiotics and ② cholecystectomy.

- Start broad spectrum antibiotics once diagnosed, stop after surgery.

- Do cholecystectomy within ④ 72 hours of onset (Golden Window).

- Do percutaneous cholecystostomy if cannot do surgery. Can Later do cholecystectomy if became possible.
  ↳ stoma (external drainage of GB).

- Cut-down cholecystostomy can be done in the critically ill at the bedside in the ICU.
  ↳ drainage by stoma.



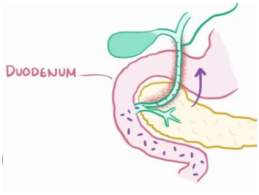
* HIDA is a substance that's given orally to p, it goes to blood → liver → secreted in bile. (it's radioactive so normally we can see it going through all these parts, in cholysistitis → inflammation causes bile to be blocked so HIDA can't reach GB).

* Note: can be relieved with analgesics.

* Elective cholecystectomy cases:
(No symptoms yet).
① peds with stones. ② Dm patients with stones.
③ porcelain GB (Ca^{+2} in GB walls).
④ old patients.

Cholangitis: = Ascending cholangitis

Pathophysiology:



- Bacteria normally ascends from duodenum into biliary tree, this, however, gets cleared with bile flow. Obstructed bile flow allows bacterial growth (ascending cholangitis, MC). Or from hematogenous seeding of bacteria in bacteremia patients.

- Causes of obstruction: ① choledocholithiasis (MCC). ② Pancreatitis/pseudocyst, ③ Mirizzi syndrome, ④ choledochal cysts, ⑤ neoplasia. Iatrogenic causes include: ① strictures, ② foreign bodies, ③ instrumentation.

*it's scary because it has a high risk of causing sepsis
*some patients dont show all Charcott!

Presentation:

- Charcot triad: ① fever (MC), ② RUQ pain, ③ jaundice. Seen in 50-75%. Reynold pentad (sepsis): ~~add~~ Charcott + altered mental state and hypotension. Can also see local peritonitis (Murphy) or diffuse peritonitis (severe).

*sepsis: spread of infection to liver → bloodstream.

- Labs: ① high WBC, possible ② coagulopathy. ③ High ALP/GGT/④ bilirubin. ⑤ High AST/ALT can be seen in severe infection or liver microabscess formation.

suggest involvement of liver.

Treatment:

① Stabilize, ② antibiotics and ③ biliary drainage. Later do ④ cholecystectomy.

- ① Aggressive fluids, correct ② coagulopathy. (plasma, vit K, platelets).

- IV empiric antibiotics: similar spectrum to cholecystitis (① GNB+ ② anaerobe), but more anaerobes.
  - ① Bacilli.
  - → Ambicillin-sulbactam. / Quinolone + metronidazole.

- Biliary drainage:

- ERCP is standard. Percutaneous transhepatic cholangiography if no ERCP, more complications. Operative decompression: high mortality, last resort, only drain the ducts.

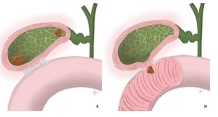
- Definite treatment is cholecystectomy later. (has to be delayed).

CHARCOT'S TRIAD

REYNOLD'S PENTAD

Gallstone ileus: (rare complication of gallstone). (ileus = obstruction in GI).

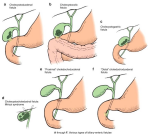
- A large stone (>2.5cm) causes pressure on GB wall > necrosis > fistula (with colon, duodenum or antrum) > passage of stone to bowel > GI obstruction.



- MC site is ileocecal valve causing SBO. Rarely, GOO happens (Bouveret syndrome). (if fistula with antrum).

AXR:

- Rigler triad on imaging: pneumobilia, AF level in small bowel, and gallstone in IC valve. CT shows SBO + pneumobilia. ↳gas inside ↳biliary system.



- Surgically remove stone and gallbladder.

* in gallstone ileus, when the fistula is with the duodenum; stones keep sliding down until they reach the narrowest part of SI → ileocecal valve, during the 'sliding' p complains of intermittent pain but when it reaches → small bowel obstruction symptoms.

↳ileo cecal.

Mirizzi Syndrome:

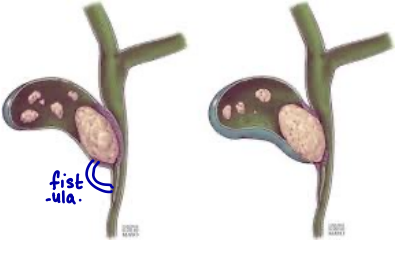
- A large stone or multiple small ones cause pressure on GB infundibulum or cystic duct > CBD obstruction > obstructive jaundice and also fistula formation. The GB and CBD are both obstructed: can cause biliary colic, jaundice, cholecystitis and/or cholangitis.

- Classification: Type I (hepatic duct obstruction). Type II (<1/3 of bile duct involved by fistula), type III 1-2/3, and Type IV: >2/3.

- Treatment: subtotal cholecystomy (leaving fibrosed GB) with fistula closure (in types II-IV)

↳it's already closed.

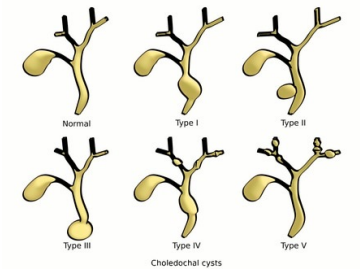
* mirizzi: when cholithiasis causes local fibrosis that reaches CBD. it's the only condition where a GB stone causes jaundice.



Choledochal Cysts:

- Congenital dilation of biliary ducts (bile can go upwards (turbulence) & some of it get stuck in the dilated area).

- Complications: jaundice, strictures, recurrent cholangitis and pancreatitis, choledocholithiasis, cholangiocarcinoma, cyst rupture and peritonitis.



Choledochal cysts

Types:

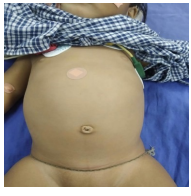
- Type I (85%): saccular, cystic, or fusiform dilation. Extrahepatic.

- Type II: True, single, extrahepatic diverticulum. (true diverticulum).

- Type III: choledochocele, cystic dilation of distal CBD into duodenum

- Type IV (10%): multiple: intra and extra (IV A), only extra (IV B).
hepatic.

- Type V: Single or multiple intrahepatic only. If with liver fibrosis: Caroli disease.



Presentation:

- Usually presents with painful jaundice. In children, a palpable mass can also be seen. In adults, usually diagnosed after recurrent pancreatitis or RUQ pain after cholecystectomy. Can also cause secondary biliary cirrhosis if longstanding.

Imaging:

- In a child with obstructive jaundice, do RUQ US to role out atresia. Can also do HIDA scan in a neonate if the anomaly was detected prenatally.

- In adults, CT scan is the first study. Do MRCP for pre-op planning or ERCP for potential of intervention.

Treatment:

If possible, resect in everyone, due to complications and malignant risk. Resect and re-anastomose with choledochojejunostomy or hepatojejunostomy.
*Resect even if assymptomatic.

GB Cancer: (not imp).

- Risk factors: MC is gallstones (>2.5cm). Porcelain GB, PSC, chronic infection (salmonella, helicobacter), anomalies, medications (methyldopa, isoniazid), obesity.

- 80-95% is adenocarcinoma. Low 5-year survival.

- Presents incidentally (1-2% of cholecystectomy), risk of spillage after lap chole. Can cause colic or jaundice. May see high ALP, AST, ALT, CEA, CA19-9.

- EUS is better than RUQ US. **Mid-bile duct obstruction in ERCP is GB cancer unless proven otherwise.** CT/MRCP for staging.

- There is need for staging laparoscopy.

- If lesion extends beyond lamina propria in routine cholecystectomy, and disease is resectable, re-exploration is needed. If limited to lamina propria, cholecystectomy with negative margins is enough. If beyond lamina propria, radical cholecystectomy (GB, 2 cm of liver, and surrounding LN).

Cholangiocarcinoma (not imp).

- RFs: PSC, choledochal cysts, intrahepatic biliary stones, Clonorchis sinensis infection, hepatitis B or C, cirrhosis, Lynch syndrome, obesity.

- Classification: proximal (intrahepatic and hilar), central extrahepatic, distal extrahepatic (intrapancreatic), hilar carcinoma (at common hepatic duct, connecting right and left ducts, Klatskin tumor).

- Presents vaguely, pain in intrahepatic, obstructive jaundice in more distal disease. Can see high CEA, CA19-9, AFP, ALP, GGT.

- Imaging and need for staging laparoscopy (similar to GB cancer).

Pancreas:

*Retroperitoneal =
①it's pain comes in the back.
②no mesentery only covered by peritonium ant.
③if it has hemorrhage, it comes as retroperitoneal hemorrhage (unclear→only symptom ↓Hct in a 'normal individual).

Anatomy: (retroperitoneal).
① ② ③ ④ ⑤
- Made of head (and uncinate), neck, body, and tail.

- The neck as anterior to the origin of the superior mesenteric vessels, the head is to the right surrounded by 1st and 2nd part of duodenum, uncinate is inferior to head, surrounded by 3rd and 4th parts of duodenum, and is posterior to the superior mesenteric vessels and anterior to IVC. Body is to the left of neck, and tail reaches splenic hilum.

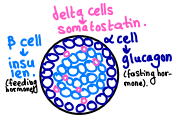
- Supply is via celiac and SMA. Celiac: GDA forms superior pancreaticoduodenal artery to head, uncinate and neck, splenic artery forms dorsal pancreatic artery to neck (right branch) and body (transverse pancreatic artery), splenic artery also forms pancreatica magna artery to body and tail (caudal artery). SMA: forms inferior pancreaticoduodenal artery to uncinate and head.

①- Main pancreatic duct (of Wirsung), originates embryologically from ventral bud, begins in tail and opens in ampulla of Vater. Accessory duct (of Santorini), originally from dorsal bud, starts from main duct and ends in duodenum via minor papilla (above sphincter of Oddi).

*main pancreatic duct is the one joined by Biliary duct.

- Pancreas divisum is failure of ventral and dorsal buds to fuse, most of the pancreas drains through accessory duct to minor papilla, can lead to recurrent pancreatitis. MC congenital anomaly of pancreas (10%). (most ppl are assymptomatic).

Function: (it's both an Endo & an Exocrine organ).

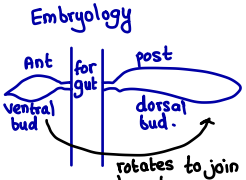


①- Endocrine islets of Langerhans form 2% of mass. Alpha cells make glucagon, beta cells make insulin, D cells make somatostatin, PP cells make pancreatic polypeptide. Alpha and beta cells are prominent in the body and tail.

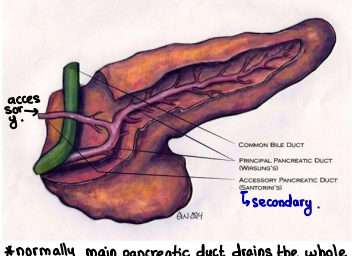
②- Exocrine: production of digestive enzymes (via acinar cells, 80% of mass). Enzymes are released inactive) and need enteropeptidase (in brush border) to be active.
↑for fats, carbs & protiens.

* Enzymes secretion is stimulated by cck wich is released by doudenum when it's full of food. & duct secretion by secretin (also stimulated by doudenum when it's full of food).

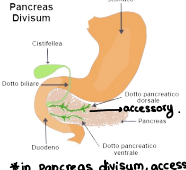
Embryology



(it originates from forgut, that's why it's pain is epigastric).

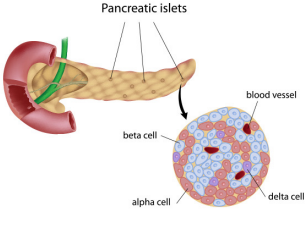


*normally main pancreatic duct drains the whole pancreas, & the accessory drains the upper part of the head

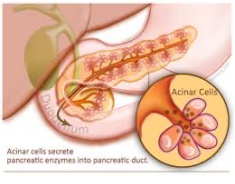


*in pancreas divisum, accessory duct becomes the drain for the whole pancreas.
* accessory duct is small, so >risk of obstruction.

Endocrine (2%)



(Exocrine 80%). asinar cells.

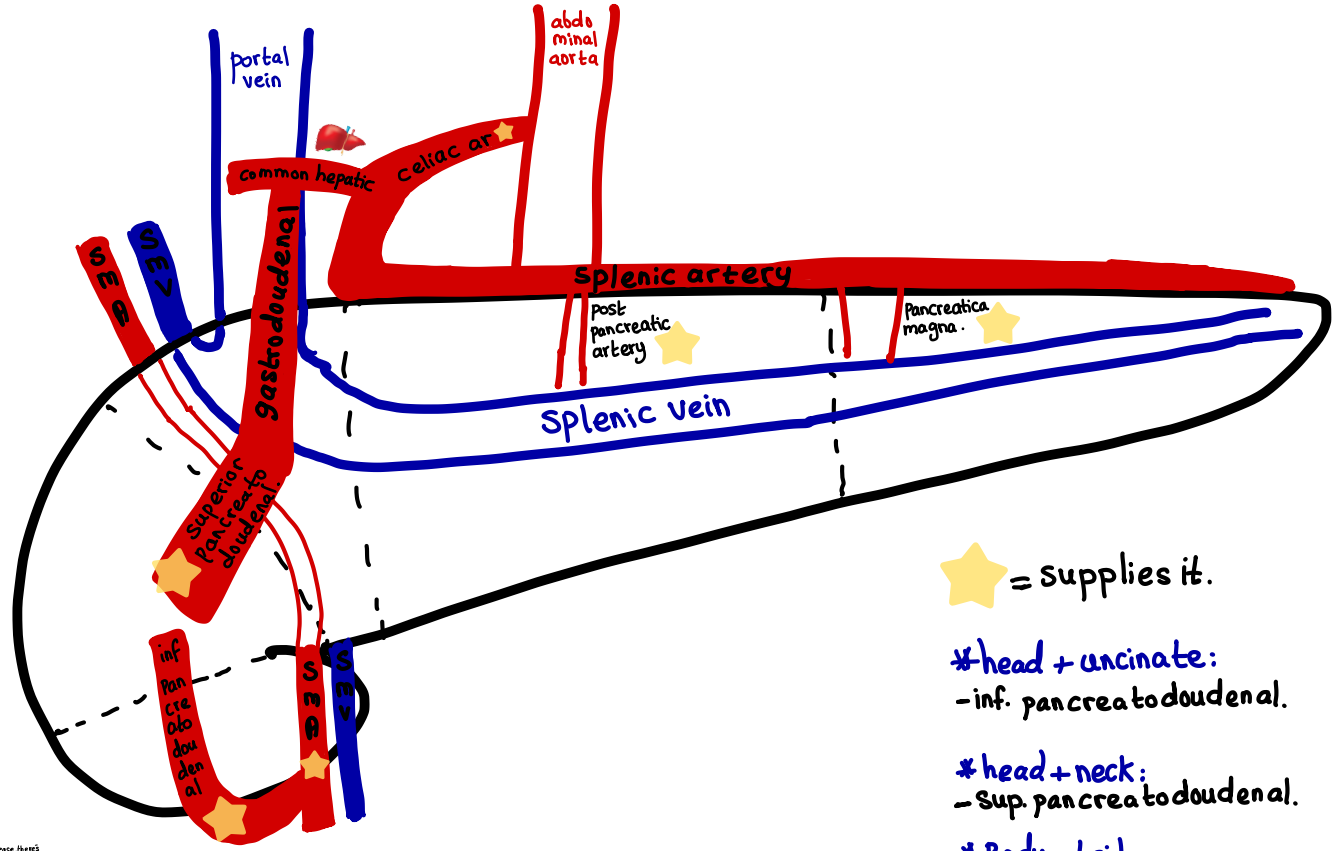


*Note: Enzymes secreted by acinar cells Proteases & lipases, can digest body tissues, so pancreas produces them in an inactive form also it produces inhibitors, to inhibit the Enzymes while not in action site, & it produces bicarbonate (HCO_3) in ducts, so that it reduces their activity as they pass through acinar cell ducts.
in doudenum, doud's lower alkaline environment + EnteroKinase (=enteropeptidase) secreted by brush border cells transforms Trypsinogen → Trypsin & trypsin activates proteases & lipases.

Enzymes: 'PAL'

*Amylase → carbs.

* food in doudenum → CCK → enteroKinase → Trypsinogen to trypsin → activates 'PAL'.



= supplies it.

*head + uncinate:
- inf. pancreatoduodenal.

*head + neck:
- sup. pancreatoduodenal.

*Body + tail:
- post. Pancreatic.
- pancreatica magna.
- SMA.

(main suppliers → celiac, SMA).

*Behind the neck of pancreas, there's ① SMA. ② SMV ③ formation of portal vein.
* SMA & SMV descend anterior to uncinate process.
* Behind the pancreas: inferior vena cava & abdominal aorta.
* celiac artery that supplies pancreas gives splenic artery (this branch lies in the upper border of pancreas).
* celiac artery also gives us common hepatic artery & this gives gastroduodenal artery → gives us superior pancreaticoduodenal artery with supplies head & neck of pancreas.
* SMA gives us inf. pancreatoduodenal. (head, neck & uncinate).
* also, splenic vein runs vertically behind pancreas, to join SMV & form portal vein.
* splenic → gives pancreas supply → dorsal pancreatic & pancreatica magna a. } post to pan. supply body + tail.
GDA SA SMA

Acute pancreatitis:

- Pathophysiology: the MC etiologies (alcohol and gallstones) result in ductal obstruction and accumulation of enzymes in acini, with inappropriate activation of enzymes leading to digestion of pancreas, and inflammation. Release of tissue factor and enzymes in circulation leads to systemic manifestations. (ROS).

* with time accumulation of Enzymes inside acini due to gallstones obst. will perforate it & it will leak. Since the pancreas's interstitium is full of fat & has a PH, the enzymes will become activated, so enzymes will literally digest the pancreas = autodigestion.

- Etiology: MC are alcohol and gallstones. Others: iatrogenic (especially ERCP), pancreas divisum, medications (steroids, valproate, sulfa (sulfonamides, furosemide, thiazides, azathioprine, etc.)), trauma, autoimmune, familial, infectious, hypertriglyceridemia, hypercalcemia, malignancy, duct obstruction, idiopathic. antibiotics. AD. (accumulate in acini). (↑Ca activates enzymes).

* in ERCP the contrast accumulates in ducts & causes obstruction.

* infections
↙ direct mumps
↘ obstruction helminths. round worm liver fluke?

Presentation:

- Symptoms: epigastric pain with radiation to the back, improves on leaning forward. With nausea, vomiting, anorexia, bloating (from secondary ileus and gastric irritation), fever.

Vomitting + epigastric Pain radiating to back so imp.

- Signs: tachycardia, epigastric tenderness, abdominal distention, ecchymosis (from retroperitoneal hemorrhage, including: Gray-Turner (flank) and Cullen (periumbilical)), shock.

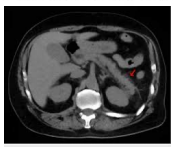
- Labs: elevated amylase and lipase (more specific) 3x normal range. Elevated ALT suggests biliary cause. ↳can be high in sialadinitis.

Imaging:

- AXR: loss of psoas margins, dilated proximal jejunum and gas in duodenum (sentinel loop) from ileus.

- US: look for gallstones and CBD dilation, pseudocyst formation.

- CT: most accurate: fat stranding, fluid collection, necrosis.



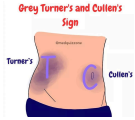
Fat stranding ct.

Diagnosis is based on combination of presentation, labs, and imaging findings. (atleast 2/3).

* Alcohol ↑ protien content & ↓ water in pancreatic secretion, this causes secretions to become concentrated → main pancreatic duct obstruction → leakage of acinar cells → autodigestion.
* Alcohol can also directly cause perforation in acinar cells.
* Alcohol can also close sphincter of oddi, by causing it to spasm → triggers pancreatitis.

* Note: ↑Ca^{+} can cause pancreatitis, cuz Ca^{+2} activates pancreatic enzymes. & pancreatitis causes ↓Ca^{+2} cuz it produces ↑ amounts of enzymes wich needs Ca^{+2} to be activated.

* Acute pancreatitis is associated with high bleeding (due to it's ↑ vascularity). It comes as retroperitoneal hemorrhage. Blood will flow retroperitoneally then settle in the ant. abdominal wall → Appears as Ecchymosis (bruising around umbilicus = cullin sign or on sides = gray Turner).



* "I Get Smashed" → causes of pancreatitis.

idiopathic — gallstone. — trauma (ethanole (alcohol)). — scorpion bite. — mumps — autoimmune. — steroids. — ↑Ca^{+2}. — ↑TG. — ERCP. — Drug induced.

* abdominal aortic aneurysm rupture pain is also epigastric & radiates to the back, to make sure it's not AAA, we look for paralytic ileus & ↓ Gastric emptying (↑nausea & vomitting → dehydration).

↓/↑ωBC ↓/↑temp
SIRS
tachypnea. tachycardia

* it's considered a systemic disease, cuz enzymes & ROS & free fatty acids leaking due to it trigger a systemic response = SIRS (systemic inflammatory response syndrom). (SIRS also happens in sepsis). it can progress to organ failure.

* that's why m/c cause of death in pancreatitis patients is Acute respiratory distress syndrome (ARDS).

Treatment:

(supportive).

- Rest the bowel: NPO and NGT. Analgesia. Correct fluids. ICU monitoring if severe. May use acid suppression to reduce gastric discomfort. (like ppi).

*it's supportive cuz at the end of the day the enzymes will become depleted on their own.

- Biliary pancreatitis: if severe, with cholangitis, or with persistent obstruction: ERCP. Cholecystectomy **after** resolution, in the same admission (high recurrence).

↳contraindicated in mild pancreatitis.

Prognosis:

- 10-15% develop severe pancreatitis.

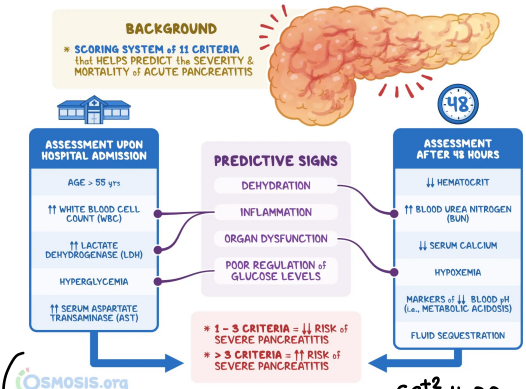
- Ranson criteria can be used to predict prognosis: ① On admission (GA LAW: Glucose >200, AST >250, LDH >350, Age >55, WBC >16k), after 48 ② hours (Ca HOBBS: Calcium <8, Hct fall by >10%, Oxygen <60mmHg, BUN increase by >5, base deficit > -4, Sequestration of >6 L of fluid.). If 3 or more, severe pancreatitis. Mortality if more than 50% if more than 6. Criteria values are different for biliary pancreatitis.

- Complications include: pseudocyst, endocrine/exocrine dysfunction, necrosis, duct strictures, hemorrhage, chronic pancreatitis (associated with risk of splenic vein thrombosis, GI obstruction, biliary obstruction, pseudoaneurysm). +Dm + Chronic pancreatitis.

*You have to monitor fluid output by monitoring urine by folly's catheter (1ml/kg/hr). This tells us if pancreatitis is severe or not.

*in mild biliary pancreatitis many patients have a spontaneous resolution & the gallstone is excreted in bile.

* pancreatitis causes hemorrhage → ↑ROS → DIC (disseminated intravascular coagulation). pancreas bleeding directly.



GALAW

G: glucose ↑
A: Ast ↑
L: LDH. ↑
A: age ↑ >55
W: WBC ↑.

*ALT very specific to liver (AST can rise due to other diseases).

Ca^{+2} HoBBs

Ca^{+2}: ↓
Hct: ↓
O2: ↓ (ARDs).
BUN: ↑ (Kidney injury).
Blood pH: ↓
Sequestration: ↑ (hypovolemia)
(↓ urine output due to fluid sequestration)

Chronic pancreatitis:

- An inflammation leading to tissue scarring and/or duct strictures. (pancreatic output affected).

- Presents with chronic intermittent pain, nausea, and vomiting. Associated with pancreatic insufficiency, weight loss, and diabetes. (recurrent attacks of pain similar to acute pancreatitis)

- Imaging shows atrophy, calcification, and duct dilation (chain of lakes)

Treatment:

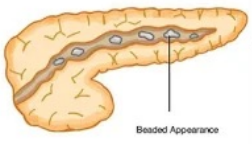
- In acute attacks, treat like acute pancreatitis (bowel rest, analgesia, fluid correction, monitoring).

- Seek and remove cause (e.g. alcoholism), treat insufficiency (with enzyme replacement) and diabetes (with insulin).

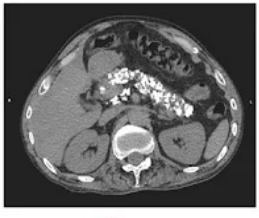
- Surgical resections for chronic pain. Can do celiac block as last resort.(to cut pain sensation).

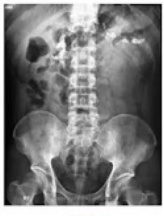
* m/c cause is recurrent acute pancreatitis mainly due to alcoholism in adults & cystic fibrosis in children.

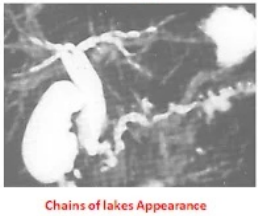
* pancreatic insufficiency → ✕ Exocrine function → fat malabsorption → steatorhea + weight loss.
✕ Endocrine → DM 1.

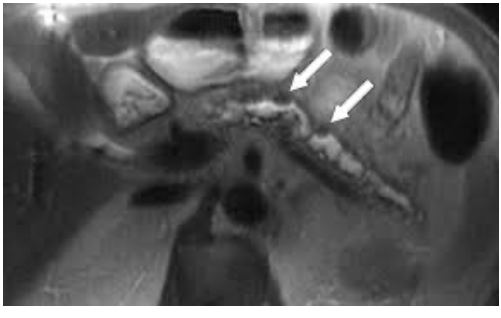


Pancreatic Calcifications


CT


X-ray


Chains of lakes Appearance



Pseudocyst:

- Walled-off collection of pancreatic juice, the wall is made of granulation tissue/fibrosis, not epithelium, hence the name pseudo. Usually caused by acute pancreatitis or other causes of duct rupture.

Presentation (can be assymptomatic).

- Symptoms: abdominal pain, secondary obstruction of gastric outlet, can have fever, and weight loss. ↳vomitting.

- Signs: CBD obstrucation leading to jaundice, epigastric mass, possibility of bleeding.

Differential diagnosis:

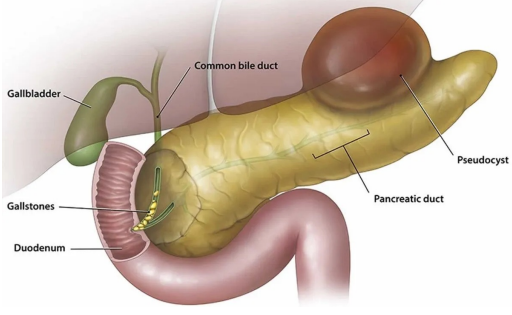
- Inflammatory conditions: abscess or phlegmon. (puss without wall surrounding it).
↳puss+wall.

- Neoplasia: cystic tumors of the pancreas or adenocarcinoma.

Treatment:

- 50% of asymptomatic pseudocysts resolve in 4-6 weeks, can observe. Drain if >5cm or persists beyond 8-12 weeks.

- Symptomatic: endoscopic or surgical drainage into bowel, to use the juices for digestion. Biopsy wall looking for epithelium (role out neoplastic cysts). Resection is rarely done, usually if neoplasia cannot be excluded. (we open the cyst into bowel so we can use it for digestion).



* simply when pancreatic juices leak a scarring tissue appears around the leaking area as a response to the inflammation the juice caused.

* theres no epithilium in its wall thats why its called pseudo.

(when theres epithilium → cyst → neoplastic).

Pancreatic necrosis:

- Usually due to pancreatic ischemia. MCC in acute pancreatitis with arterial thrombosis (days to weeks after acute attack). Other causes include chronic pancreatitis or global ischemia (shock, mesenteric ischemia).

- Presents with pain, fever, N/V, with possibility of sepsis and shock. CT scan shows lack of contrast enhancement. Can have superimposed infection (showing fluid collection or gas).

- Treatment: if not infected, aggressively improve fluids, and monitor for infection. If infected, start antibiotics and perform surgical debridement.

* Necrosis → ↑ risk systemic complications → ARDS/DIC/sepsis/superimposed infection.
* we establish diagnosis with ct with contrast (contrast doesn't reach dead areas).
* on imaging infected necrosis shows as extra fluid inside area + gas bubbles. we can make fine needle aspiration to confirm.

Pancreatic neoplasia:

Ductal carcinoma subtypes
↓ ↓ ↓ ↓
Adeno carcinoma. | Scc | Adenosquamous. | medullary.

Ductal carcinoma: (m/c malignancy).

- 95% of primary pancreatic cancer. Can be adenocarcinoma (MC), medullary, adenosquamous.

- Risk factors: Smoking is most important. Alcohol (causing chronic pancreatitis), genetic (more in older, men, and African), familial (Lynch), high fat diet, DM, obesity. Exposure to radiation.

↳ were overusing pancreas.

Presentation:

- Symptoms: vague abdominal pain, insufficiency (diarrhea, steatosis), diabetes, weight loss, obstructive jaundice. (symptoms appear late). (very ↑ mortality).

↳ new onset.

* ppl with new onset of diabetes at an unusual age; ex: 65 have pancreatic cancer unless proven otherwise

- Signs: Courvoisier sign: painless palpable GB and obstructive jaundice. Sister Mary Joseph nodule. Virchow node. Migratory superficial vein thrombosis (Trousseau syndrome). Chronic DIC.

↳ specialy if in tail. (keeps appearing in places).

- Can have high CA19-9 (good sensitivity, poor specificity). (tumor marker).

Imaging:

- CT angiography (triple phase) or MRCP. Endoscopy with ERCP or US for lesion, biopsy, and potentially biliary stenting. Staging laparoscopy.

Treatment:

- Unresectable if: distant mets. Contact with SMA or celiac over 180 degrees. SMV or Portal vein involvement that is unreconstructable.

- Resect if possible with adjuvant. Neoadjuvant if borderline resectable.

Pancreaticoduodenectomy (Whipple) operation: for head tumors.

- Remove GB, CBD, portal LN, stomach pylorus, part of jejunum, head/neck/uncinate of pancreas, and duodenum.

- Anastomoses: pancreaticojejunostomy, hepaticojejunostomy, gastrojejunostomy

- Complications: Leak/fistula (increased drain output, fever, high WBC, delayed gastric emptying. Treat initially with octreotide, NPO. Then ERCP or surgery.) Others: wound infection, incisional hernia.

* If the cancer was in the head of the pancreas & it caused CBD obstruction, it will cause CBD & GB painless dilation. so "painless obstructive jaundice is pancreatic cancer unless proven otherwise". = courvosier sign.

* FAP, lynch syndrome & BRCA 1 & Dpc are related to it (KRAS too).