



AMERICAN COLLEGE OF SURGEONS • DIVISION OF EDUCATION
ACCREDITED EDUCATION INSTITUTES
ENHANCING PATIENT SAFETY THROUGH SIMULATION

Accredited Education Institute

SSAT ABSITE Review: HPB

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@DJCSurgEd



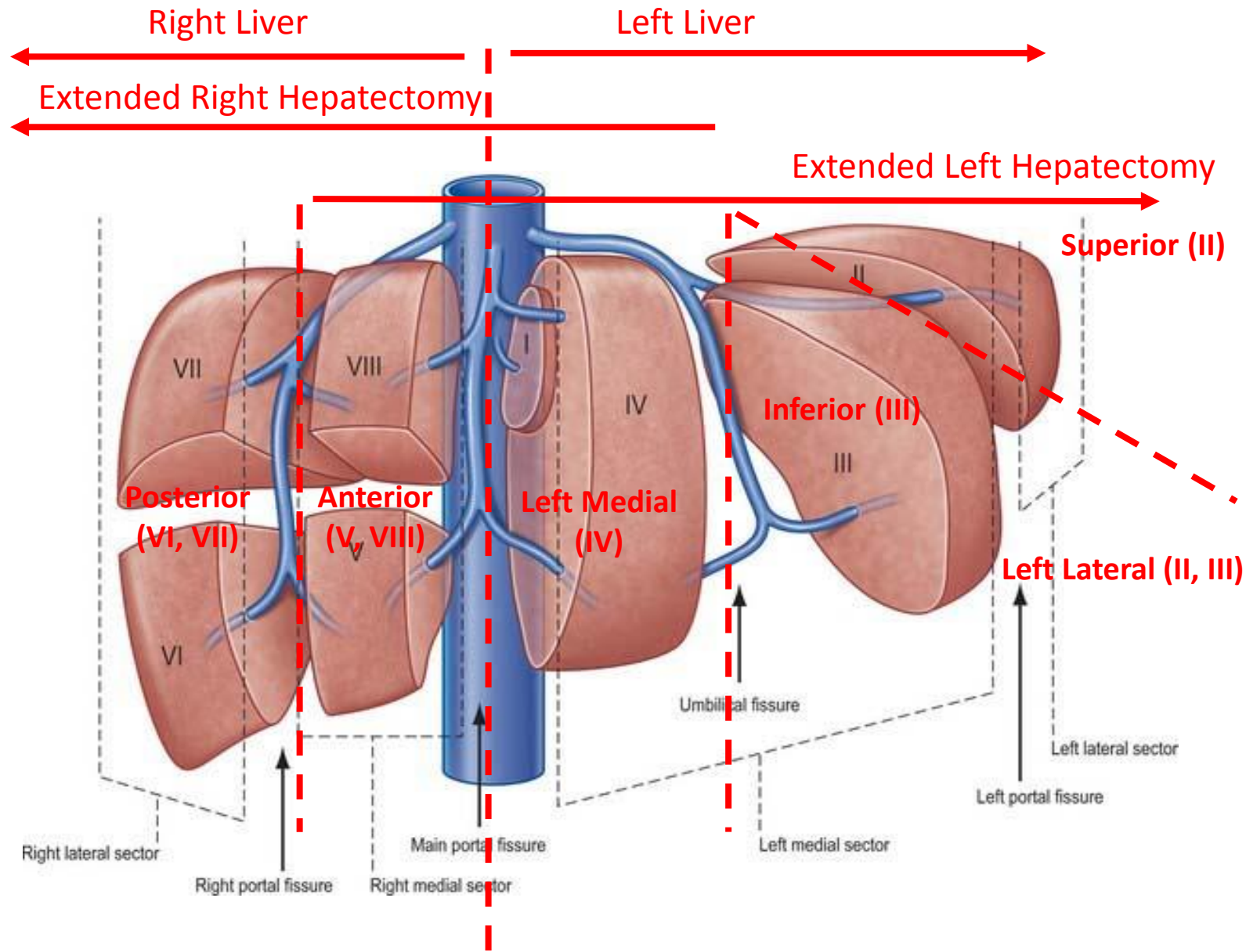
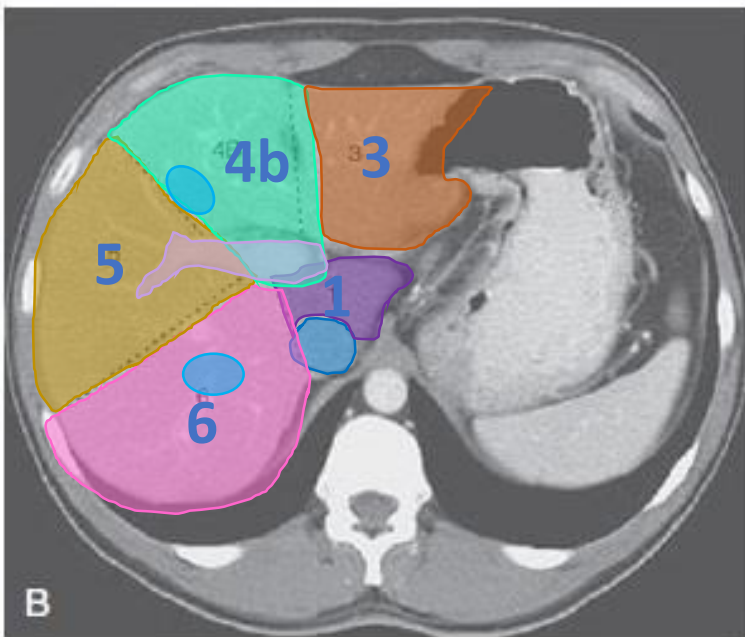
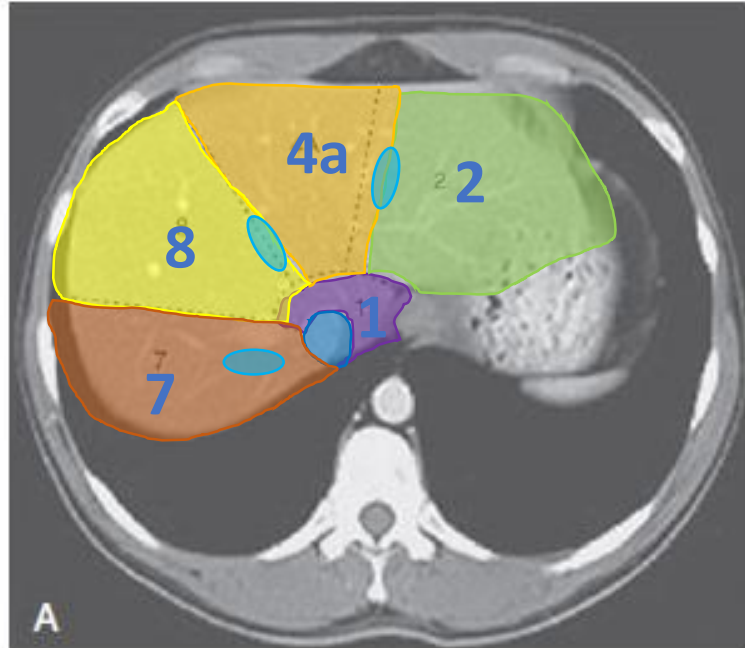
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ABSITE® CONTENT OUTLINE

ABSITE CONTENT TOPICS*		CATEGORY WEIGHTS (%)
SCORE Patient Care Category #	Patient Care Category	72
	ABDOMEN - TOTAL	17
1	Abdomen - General	3
2	Abdomen - Hernia	2.5
3	Abdomen - Biliary	3.5
4	Abdomen - Liver	3.5
5	Abdomen - Pancreas	3.5
6	Abdomen - Spleen	1
	ALIMENTARY TRACT - TOTAL	14.5
7	Alimentary Tract - Esophagus	2.5
8	Alimentary Tract - Stomach	3.5
9	Alimentary Tract - Small Intestine	3
10	Alimentary Tract - Large Intestine	3.5
11	Alimentary Tract - Anorectal	2
12	Endoscopy	2
13	Breast	4
14	Endocrine	4
15	Skin and Soft Tissue	3.5
16	Surgical Critical Care	5
17	Trauma	6

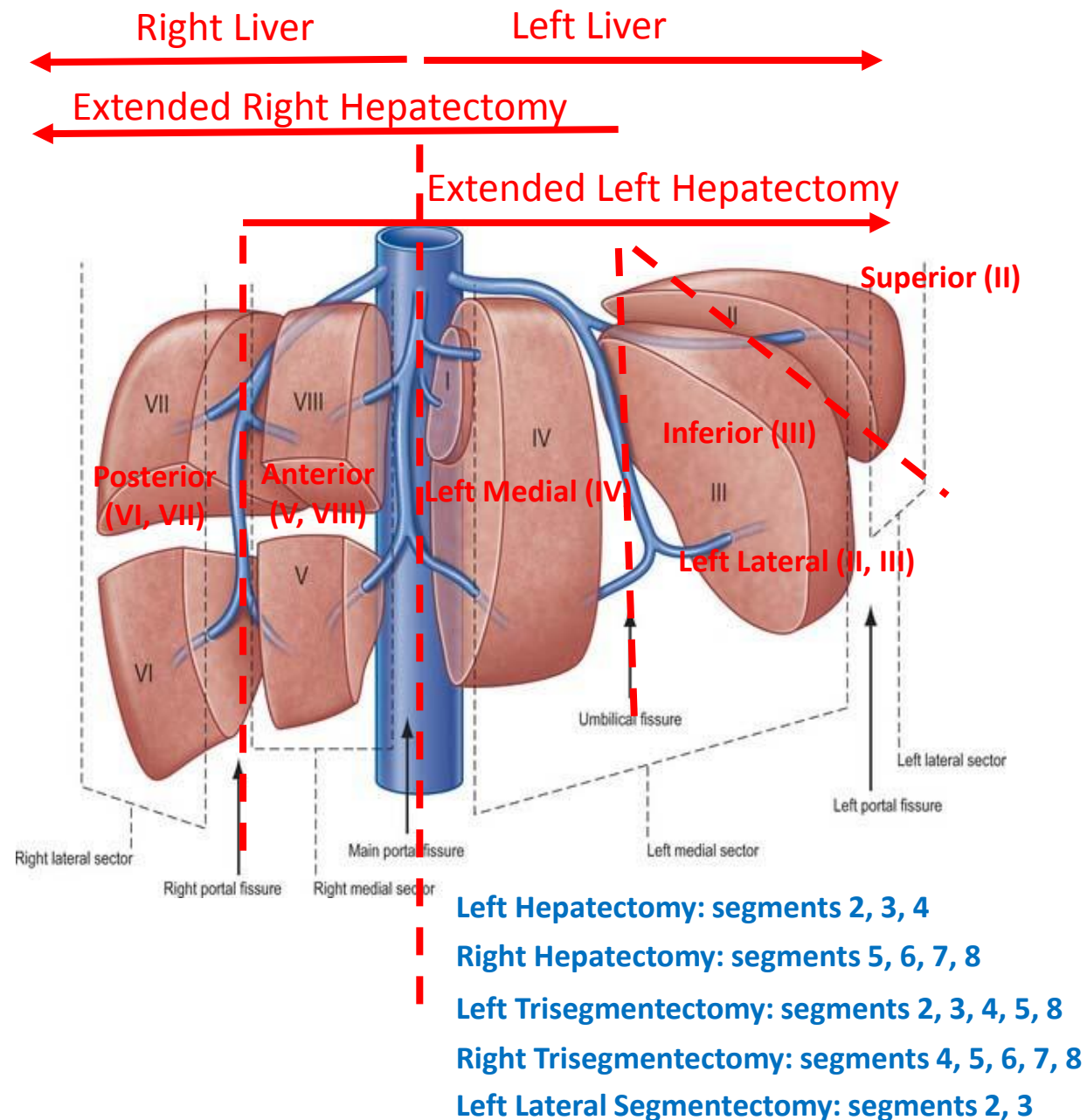
Content Outline

- Liver
 - Anatomy
 - Liver Abscesses
 - Benign Liver Lesions
 - Malignant Liver Lesions
 - Liver Failure + Portal Hypertension
- Biliary
 - Anatomy and Physiology
 - Bile Duct Injury
 - Benign Biliary Pathology
 - Bile Duct and Gallbladder Cancer
- Pancreas
 - Anatomy, Embryology, and Physiology
 - Acute Pancreatitis
 - Chronic Pancreatitis
 - Cystic Lesions of the Pancreas
 - Pancreatic Adenocarcinoma
 - Neuroendocrine Tumors of the Pancreas



Liver Anatomy

- Divided into functional right and left lobes by Cantlie's line
 - Cantlie's Line: vertical plane extending from the middle of the gallbladder fossa to the IVC
- Right Lobe (segments V-VIII)
 - Divided into anteromedial (V, VIII) and posterolateral (VI, VII) segments by the right hepatic vein (right scissura)
- Left Lobe (segments II-IV)
 - Divided into an left medial (IV) and left lateral (II+III) segment by the umbilical fissure
 - Left lateral segment: divided by the left hepatic vein (left scissura)
- Caudate Lobe (I): receives separate right and left portal and arterial inflow; drains directly into the retrohepatic IVC



Liver Abscesses

- Pyogenic

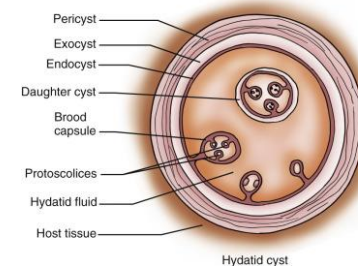
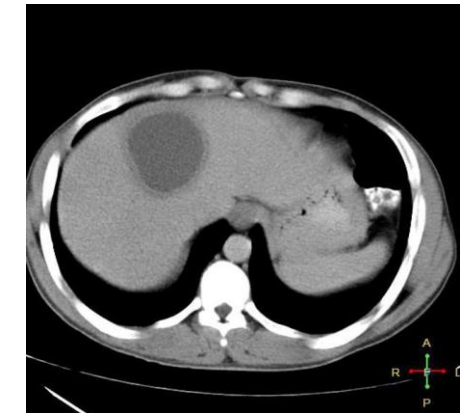
- Most common; GNRs (#1 *E. coli*, #2, *K. pneumoniae*)
 - Direct spread from biliary tree (#1)
- CT: peripheral enhancing, central hypoattenuating, multi-loculated (vs. amebic)
 - FNA with WBCs +/- organisms
- Tx: percutaneous drainage + Abx

- Amebic

- Epi: male predominant; hx of travel (Central/South America)
- Pathogen: *E. histolytica* (fecal-oral); +Abs
- CT: well-defined hypoattenuating lesion with peripheral wall of enhancing edema
 - Central localized hepatic necrosis (no organisms on FNA)
- Tx: metronidazole; drainage if poor response/superinfection

- Echinococcal

- Epi: endemic to Mediterranean, East Africa, Middle East, South America, Asia
- Pathogen: *E. granulosus*; fecal-oral; eosinophilia, +serology, +Casoni
- CT: hypoattenuating lesion, septae/daughter cysts (75%), +/- peripheral calcifications (50%)
 - Pericyst, ectocyst, endocyst (germinal layer -- gives rise to daughter cysts)
- Tx: albendazole, PAIR/surgical resection



Benign Liver Lesions

- Hemangioma

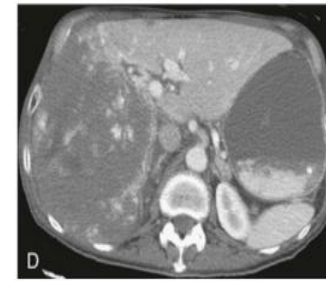
- Epi: most common benign tumor; F > M (3:1)
- CT: late contrast puddling is pathognomonic
 - PERIPHERAL enhancement on ARTERIAL phase
 - CENTRIPETAL filling on PORTAL VENOUS phase
 - Retention of contrast on washout or delayed phase
- Tx: angioembolization for bleeding; enucleation if symptomatic

- Hepatic Adenoma

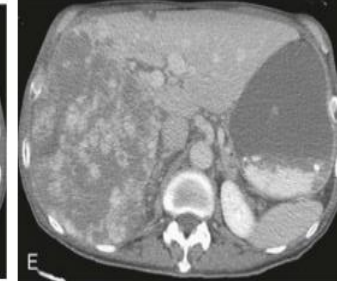
- Epi: female predominant; hormonally sensitive (OCPs, steroids)
- Risk of bleeding and malignant transformation
- CT: well-marginated, isointense on NC CT; HYPERINTENSE on ARTERIAL phase; rapid washout; +/- heterogenous due to hemorrhage
 - No Eovist (Gd) [MRI] or sulfur colloid [NM scan] retention (no Kupffer cells)
- Tx: discontinue offending agent; resection if >5cm, symptomatic

- Focal Nodular Hyperplasia (FNH)

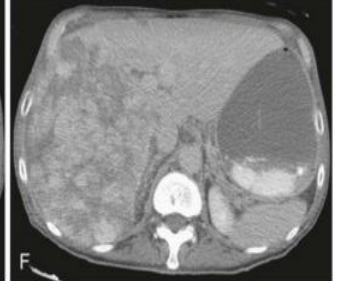
- No risk of bleeding or malignant transformation
- CT: homogenous mass with CENTRAL scar; HYPERINTENSE on ARTERIAL phase
 - Central Scar: hypoattenuating on arterial phase; hyperattenuating on delay
 - Eovist (Gd) and sulfur colloid uptake (+ Kupffer cells)
- Tx: observation



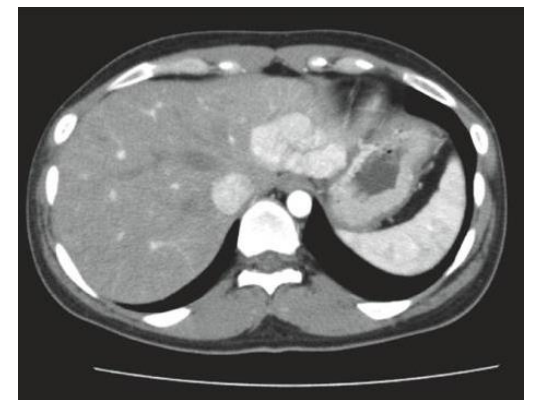
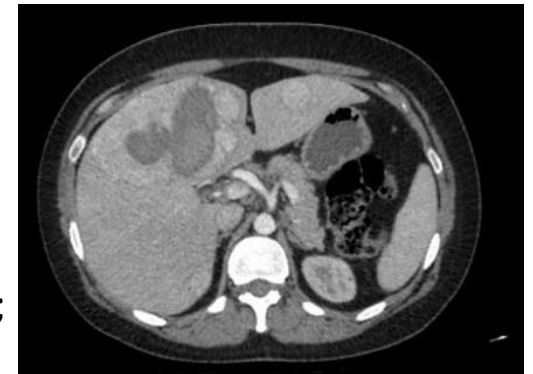
Arterial phase CT scan showing classic peripheral enhancement.



Portal venous phase CT scan showing centripetal enhancement.



Delayed phase CT scan showing washout and equilibration.



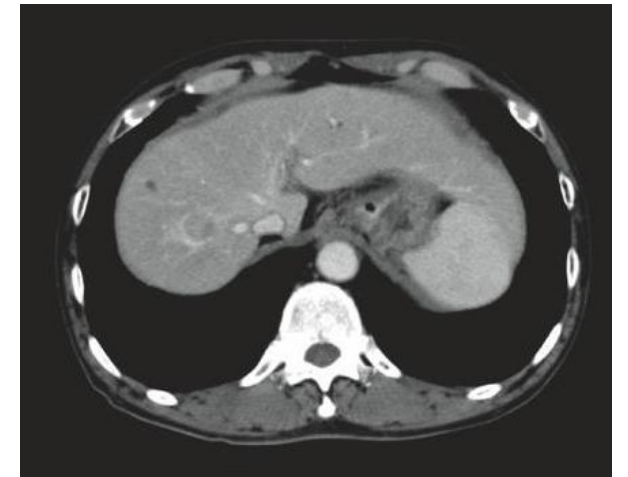
Malignant Liver Lesions

- Hepatocellular Carcinoma

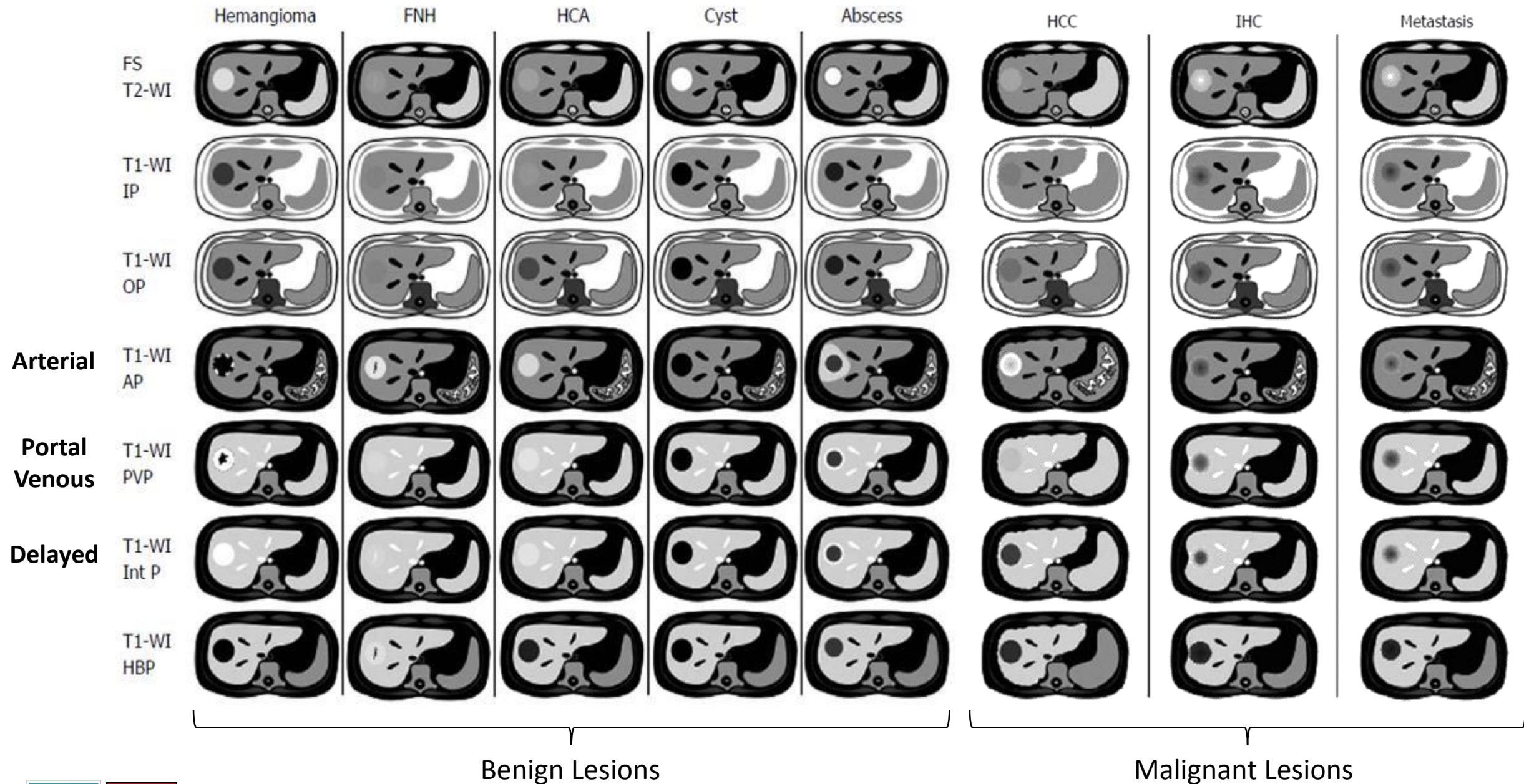
- Most common primary hepatic malignancy
- HBV infection #1 cause worldwide; 80% in setting of cirrhosis
- Screening: AFP + ultrasound every 6 months for AT RISK PATIENTS
- CT: primarily arterial inflow
 - Heterogenous; Early arterial enhancement and washout of contrast on delayed phase
- Tx: resection/ablation (if localized and adequate remnant), OLT (Milan criteria), sorafenib (metastatic/unresectable)

- Metastatic Liver Tumors

- More common than primary liver tumor (20:1)
- CT: Hypoattenuating on NC CT compared to liver; hypovascular lesions with minimal arterial enhancement



Liver Lesion	Non-contrast	Arterial	Portal	Venous
Hepatic Hemangioma	Hypointense to liver	Peripheral enhancement	Centripetal filling	Retention of contrast
Focal Nodular Hyperplasia	Isointense to the liver Homogenous lesion	Rapid homogenous enhancement w/ hypoattenuating central scar	Rapid washout	Isointense w/ delayed enhancement of central scar
Hepatic Adenoma	Isointense to the liver Heterogenous (bleed, necrosis, fat)	Rapid enhancement	Rapid washout	Isointense to liver
HCC	Isointense/hypointense to liver	Homogenous rapid enhancement	Rapid washout	Isointense
Metastases	Hypointense	Hyperintense (NET) Hypointense (CRC)	Central filling (CRC)	Washout



Liver Failure and Portal Hypertension

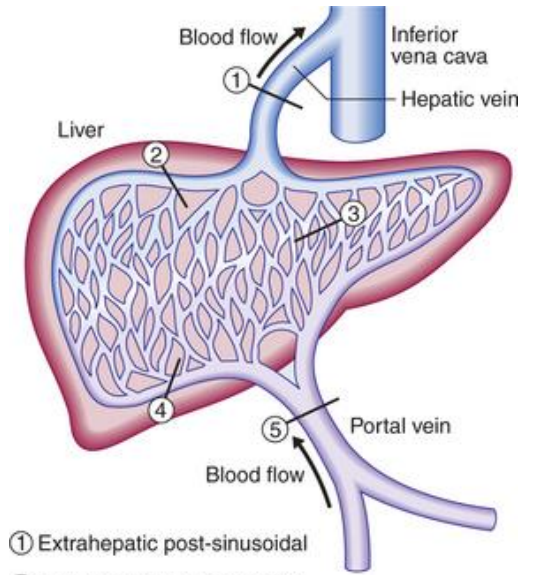
- Portal HTN

- Dx: hepatic venous pressure gradient (HVPG): pressure gradient between the wedge hepatic venous pressure (WHVP) and the free hepatic venous pressures

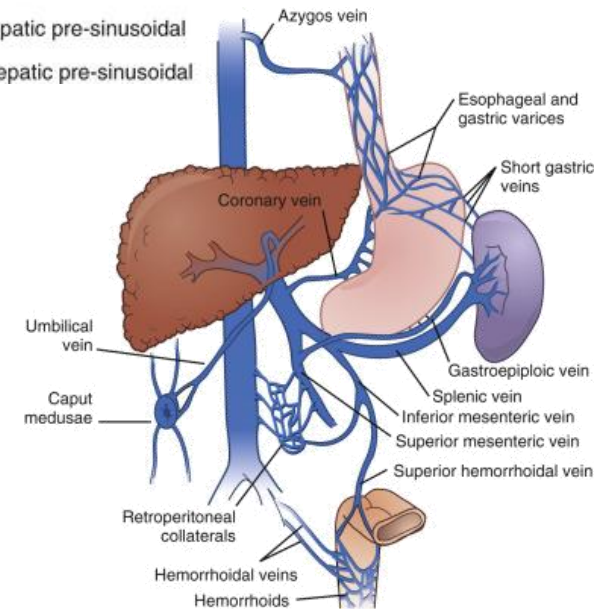
- Estimate of pressure gradient between PV and IVC
- **Portal HTN: HVPG greater than or equal to 5mmHg**; clinically significant when >10mmHg

- Liver Failure

- Causes: Pre-hepatic, Intrahepatic, Post-hepatic
 - Intrahepatic: pre-sinusoidal, sinusoidal, post-sinusoidal
- Child's Pugh Score:
 - Components: total bilirubin, PT/INR, albumin, ascites, encephalopathy
- MELD Score:
 - Components: total bilirubin, PT/INR, creatinine



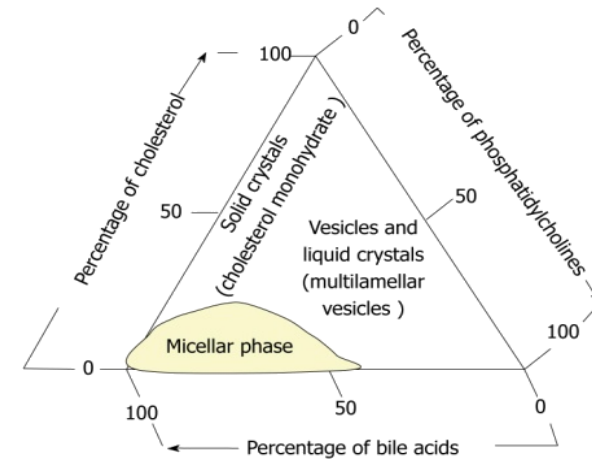
- ① Extrahepatic post-sinusoidal
- ② Intrahepatic post-sinusoidal
- ③ Sinusoidal
- ④ Intrahepatic pre-sinusoidal
- ⑤ Extrahepatic pre-sinusoidal



Biliary Anatomy and Physiology

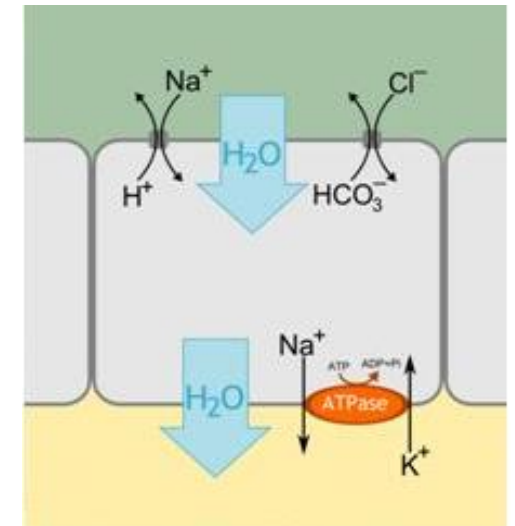
• Physiology

- Bile salts: primary bile salts (cholic and chenodeoxycholic acid) produced from cholesterol; conjugated in liver (taurine, glycine)
 - 95% reabsorbed in terminal ileum
- Bilirubin: produced by breakdown of senescent RBCs and metabolism of heme
 - Conjugated in liver by UDP-glucuronyl transferase
 - 90% excreted in stool (stercobilin), 10% reabsorbed (urobilinogen)



• Anatomy

- Cystic artery: arises from right hepatic artery, travels posterior to CHD and through triangle of Calot
 - 10% double cystic artery
- CBD: segmental blood supply; runs at 3 and 9 o'clock



Biliary Obstruction

- Causes:
 - ****Choledocholithiasis****, benign biliary strictures, malignant biliary structures, choledochal cysts, extrinsic compression, etc.
- Cholangitis: Charcot triad of fever, RUQ pain, and jaundice
 - Reynolds Pentad: + hypotension and altered mental status
- Managing CBD Stones:
 - Endoscopic Retrograde Cholangiopancreatography (ERCP)
 - Intraoperative Cholangiogram (IOC)
 - Saline Flush + Glucagon (1-2 mg)
 - Transcystic CBD exploration (choledochoscope, baskets, Fogarty catheters)
 - CONTRAINDICATIONS: small duct, numerous stones, large stones
 - Choledochotomy and CBD exploration (lap vs. open)

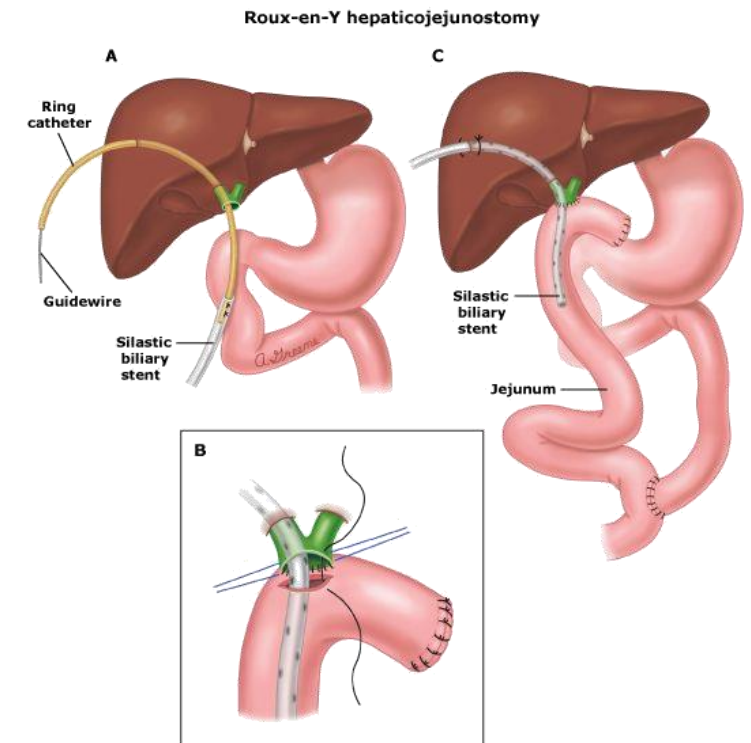
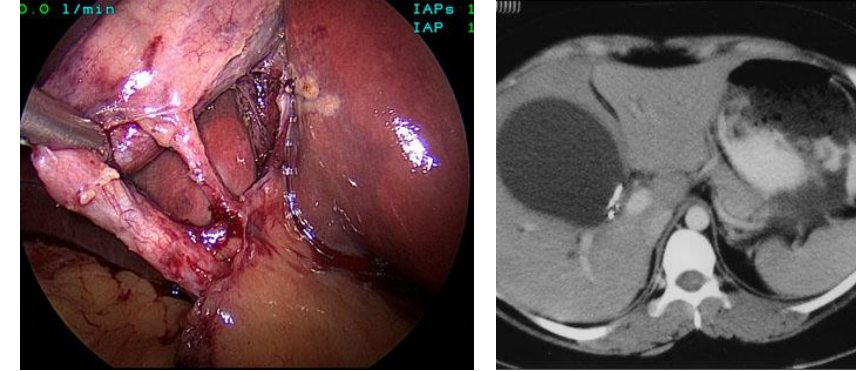
Likelihood	Predictors
Very strong	Common bile duct stone on transabdominal ultrasound Clinical ascending cholangitis Bilirubin >4 mg/dL
Strong	Dilated common bile duct (>6mm) on ultrasound Bilirubin level 1.8–4 mg/dL
Moderate	Abnormal liver biochemical test other than bilirubin Age older than 55 years Clinical gallstone pancreatitis

Presence of any strong or both strong predictors suggests a high likelihood of choledocholithiasis. No predictors suggest a low likelihood, and all other patients have an intermediate likelihood.¹⁸ By definition, all patients enrolled in this trial will have at least a moderate likelihood because of the clinical diagnosis of gallstone pancreatitis.



Bile Duct Injury

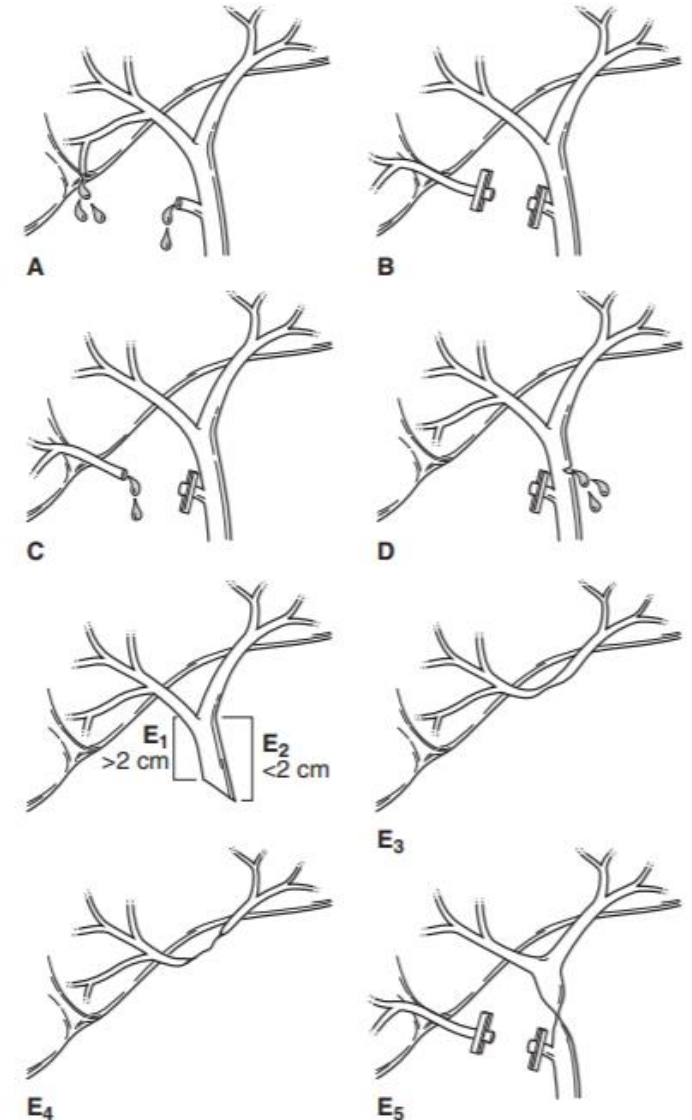
- Critical View of Safety (CVS):
 - Hepatocystic triangle is cleared at fat/fibrous tissue
 - Lower thirds of GB is separated from the liver
 - Two and only two structures are seen entering the gallbladder
- Cause of injury: excessive cephalad retraction of fundus aligns the cystic duct and CBD which are subsequently mistaken
- Work-up: abdominal U/S; consider CT vs. MRCP
- Management:
 - Intra-op:
 - Short segment: primary repair over T-tube
 - Long segment: hepaticojejunostomy
 - Drainage and transfer to HPB center
 - Post-op:
 - Goal is to control sepsis and control drainage prior to delay reconstruction
 - Percutaneous transhepatic cholangiography (PTC): defines the anatomy of the PROXIMAL biliary tree for surgical reconstruction AND decompress the biliary system



Bile Duct Injury

- Bismuth-Strasberg Classification:

- A: leak from cystic duct stump or accessory duct of Luschka --> ERC + endobiliary stent placement
- B: occlusion of ABERRANT right hepatic duct
- C: transection without ligation of aberrant right hepatic duct
- D: lateral injury to extrahepatic bile duct --> risk for stricture over time
- E: complete disruption of biliary-enteric continuity due to transection, excision, and/or ligation
 - E1: common hepatic duct; >2 cm from bifurcation
 - E2: common hepatic duct; <2 cm from bifurcation
 - E3: common hepatic duct at the bifurcation; no residual CHD but confluence preserved
 - E4: involvement of the confluence and loss of communication between the right and left hepatic ducts
 - E5: involvement of aberrant right hepatic duct +/- CHD stricture



Benign Biliary Pathologies

- Biliary Dyskinesia: motility disorder of GB that involves abnormal biliary tract peristalsis muscle in response to dietary stimulation
 - Dx: CCK-stimulated HIDA scan w/ diminished GB ejection fraction (<33%)
 - Tx: cholecystectomy
- Gallstone Ileus: mechanical intestinal obstruction from gallstone after fistulization of GB to duodenum
 - Rigler's Triad: intestinal obstruction, pneumobilia, aberrant gallstone location
 - Tx: longitudinal enterotomy proximal to stone, removal stone and primary transverse closure
- Gallbladder Adenomas:
 - Polyps > 10 mm, polyps (any size) + stones, polyps + PSC ----> cholecystectomy
 - Polyps 6-9 mm ----> serial ultrasound monitoring

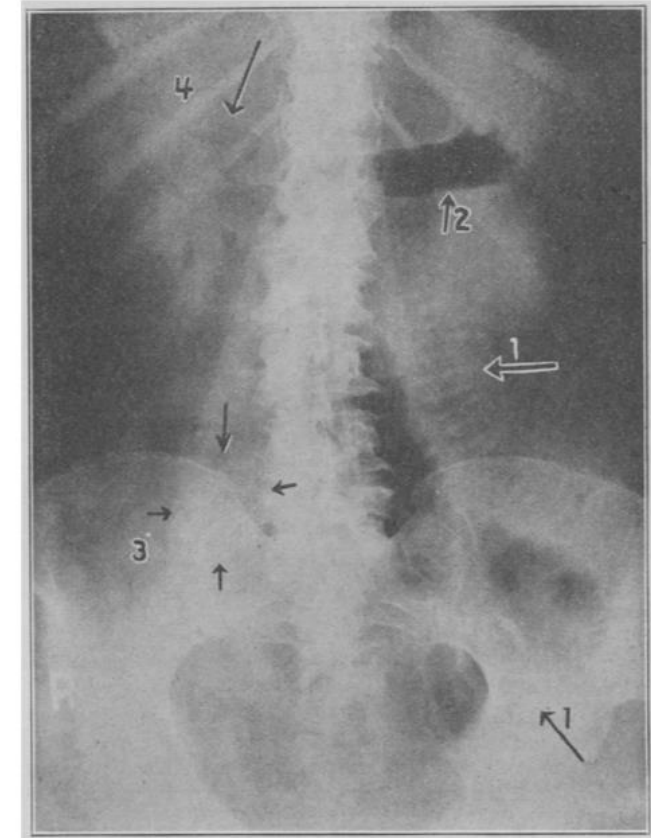


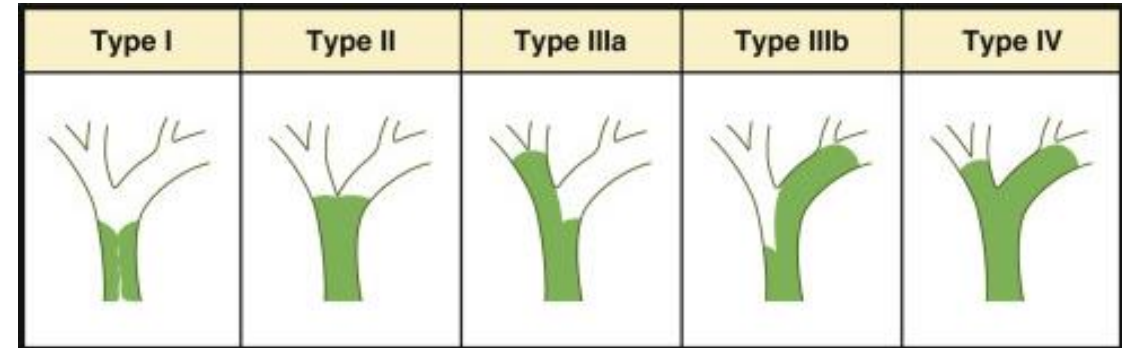
Fig. 6 (case 2).—Roentgenogram of abdomen in supine position, 1, dilated loops of intestine filled with gas; 2, gas in the stomach; 3, shadow of impacted stone with facet on upper surface (this was not observed before operation); 4, faceted stone in gallbladder with gas above it, extending into biliary ducts. Presence of gas in the biliary tract, the remaining faceted stone in the gallbladder and evidences of intestinal obstruction permitted the exact diagnosis of gallstone obstruction.

Bile Duct and Gallbladder Cancer

• Cholangiocarcinoma:

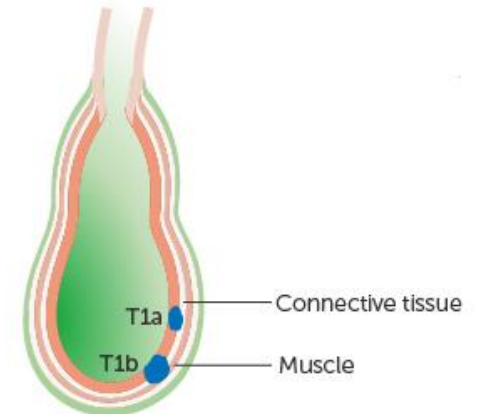
- Arise from biliary epithelium; 3 distinct subclasses [intrahepatic, perihilar, distal]
- 3 distinct morphologic subtypes: sclerosing, nodular (MOST COMMON), papillary (BEST PROGNOSIS)
- Surgical Treatment:
 - Intrahepatic: partial hepatectomy +/- bile duct resection + Roux-en-Y HJ
 - Perihilar: distal duct excised for clear margins, CCY +/- hepatic resection with RnY HJ reconstruction
 - Frozen section of proximal R/L hepatic ducts, RnY w/ HJ
 - OLT: for locally advanced perihilar cholangiocarcinoma
 - Distal: pancreaticoduodenectomy

Bismuth-Corlette Classification of Perihilar Cholangiocarcinoma



• Gallbladder Cancer:

- Risk Factors: cholelithiasis and chronic gallbladder inflammation
- Surgical Treatment
 - Laparoscopic cholecystectomy adequate for Tis or T1a margin-negative GB cancers
 - Tumors >T1a require more extensive resection → extended or radical cholecystectomy
 - Partial hepatectomy (segments IVb/V) + cholecystectomy + regional lymphadenectomy
 - Bile duct margin sent → if (+) → extrahepatic bile duct resection to achieve negative cystic duct margin (+ HJ reconstruction)



Pancreas Anatomy, Embryology, and Physiology

- Pancreas Divisum: failure of fusion of the ventral and dorsal pancreatic ducts

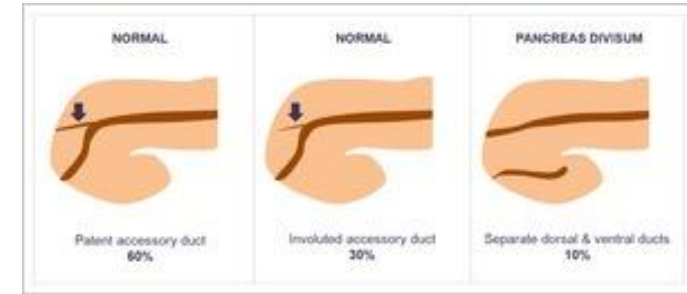
- Accessory duct of Santorini (dorsal bud) drains via minor papillae [body, tail, part of head]
- Major duct of Wirsung (ventral bud) drains via major papillae [uncinate, head]
- Majority asymptomatic, can present with pancreatitis
- Tx: ERCP + papillotomy of minor papillae

- Annular Pancreas: aberrant migration of the ventral pancreatic bud --> extrinsic duodenal obstruction

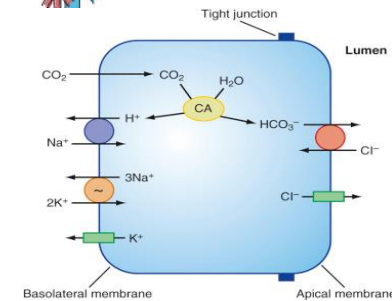
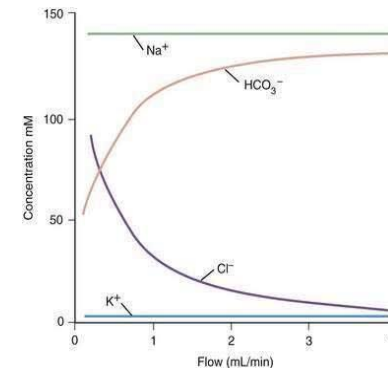
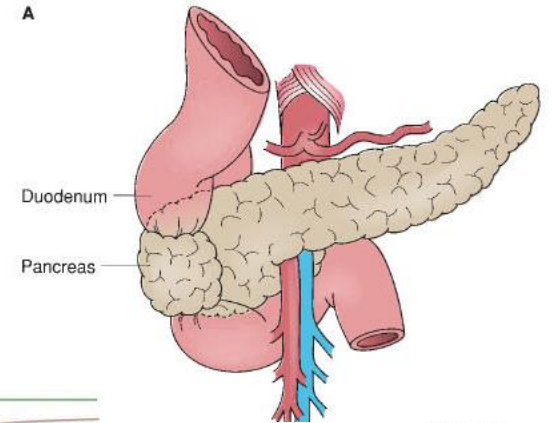
- Tx: duodenal bypass; do not resect obstructing pancreatic segment

- Physiology:

- Secretin --> pancreatic fluid secretion
 - Chloride secretion VARIES INVERSELY with bicarbonate secretion
 - Sum of anions = Sum of cations
- Vagal /CCK --> pancreatic enzyme secretion



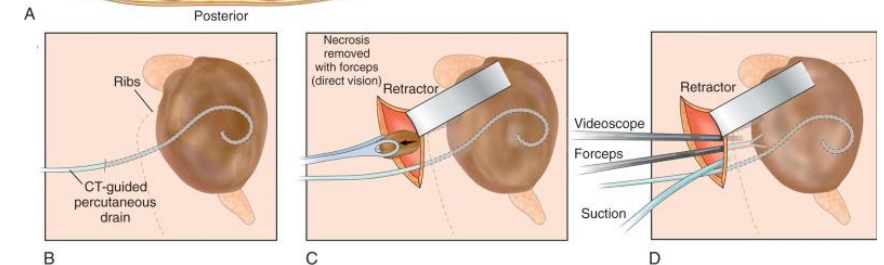
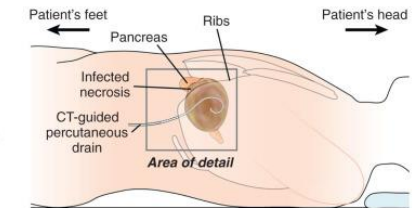
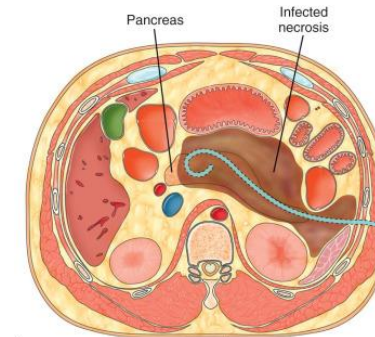
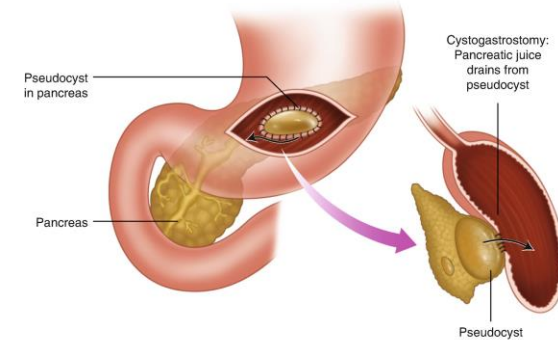
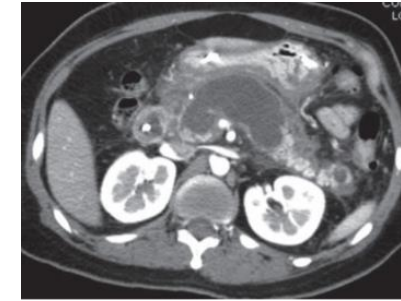
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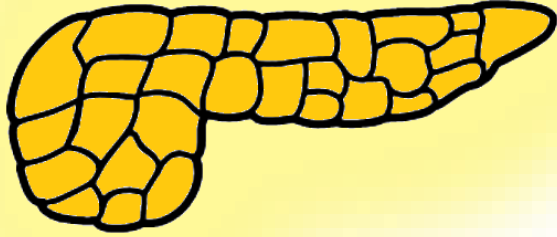
Acute Pancreatitis

- Acute Pancreatitis:

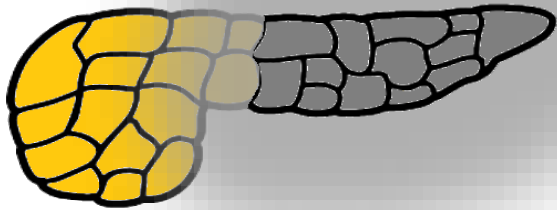
- Causes: gallstones (#1 in US) and EtOH
- Dx: need 2 of 3
 - Clinical presentation (epigastric pain, N/V, radiate to back)
 - Lipase/amylase >3x upper level of normal
 - CT findings consistent with pancreatitis
- Complications: Revised Atlanta Classification
 - Pseudocyst: time vs. drainage (internal preferred)
 - <6 weeks: 40% resolve; >6cm: 2/3 will need drainage
 - Consider ERCP if not resolving (communication w/ main duct vs. downstream stricture)
 - Tx: internal drainage
 - Endoscopic cystogastrostomy, laparoscopic vs. open cyst-gastrostomy/enterostomy, distal pancreatectomy
 - WOPN: step-up therapy [perc drainage, RP/transgastric debridement]



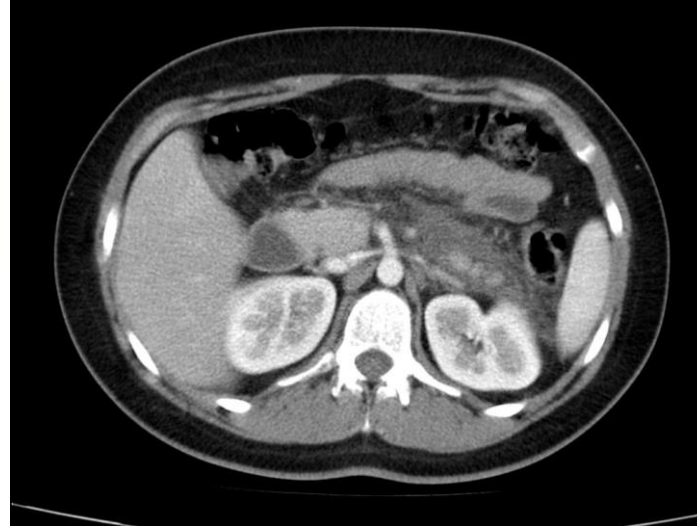
**Acute
Interstitial
Edematous
Pancreatitis**



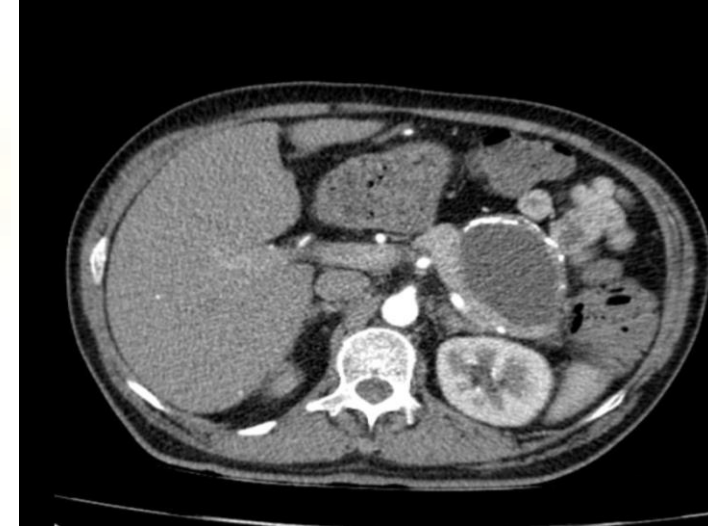
**Acute
Necrotizing
Pancreatitis**



< 4 weeks



> 4 weeks

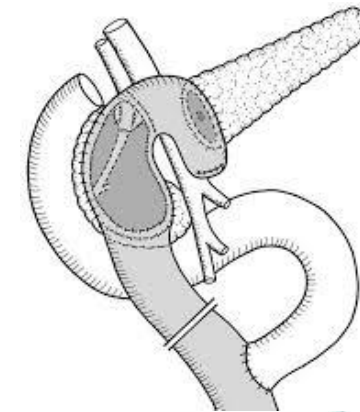
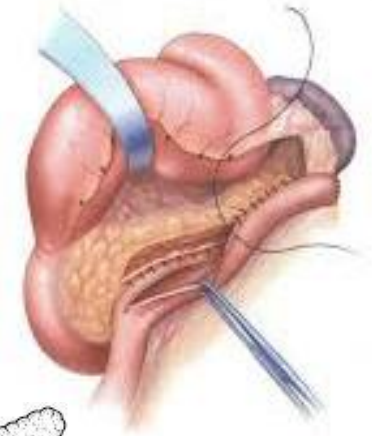


Chronic Pancreatitis

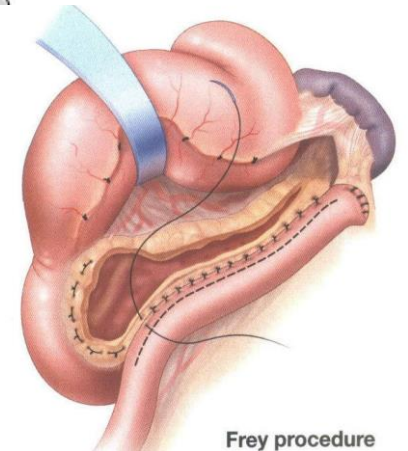
- Chronic Pancreatitis:

- Causes: EtOH (70-80%); hereditary (PRSS1 gene)
- Imaging: CT pancreas protocol, MRCP
- Indications: intractable pain, complications of pancreatitis
 - Pseudocysts, pancreatic ascites, obstruction, neoplasia
- Surgery: determine need for resection vs. decompression (or both)
 - Decompression:
 - Puestow (lateral pancreaticojejunostomy)
 - Resection:
 - Whipple, Beger (DPPHR) – for isolated pancreatic head disease
 - Distal pancreatectomy – for isolated pancreatic body / tail disease
 - Total pancreatectomy and autologous islet cell txp (AIT)
 - Combination:
 - Frey – for isolated pancreatic head disease AND ductal dilatation

Puestow procedure

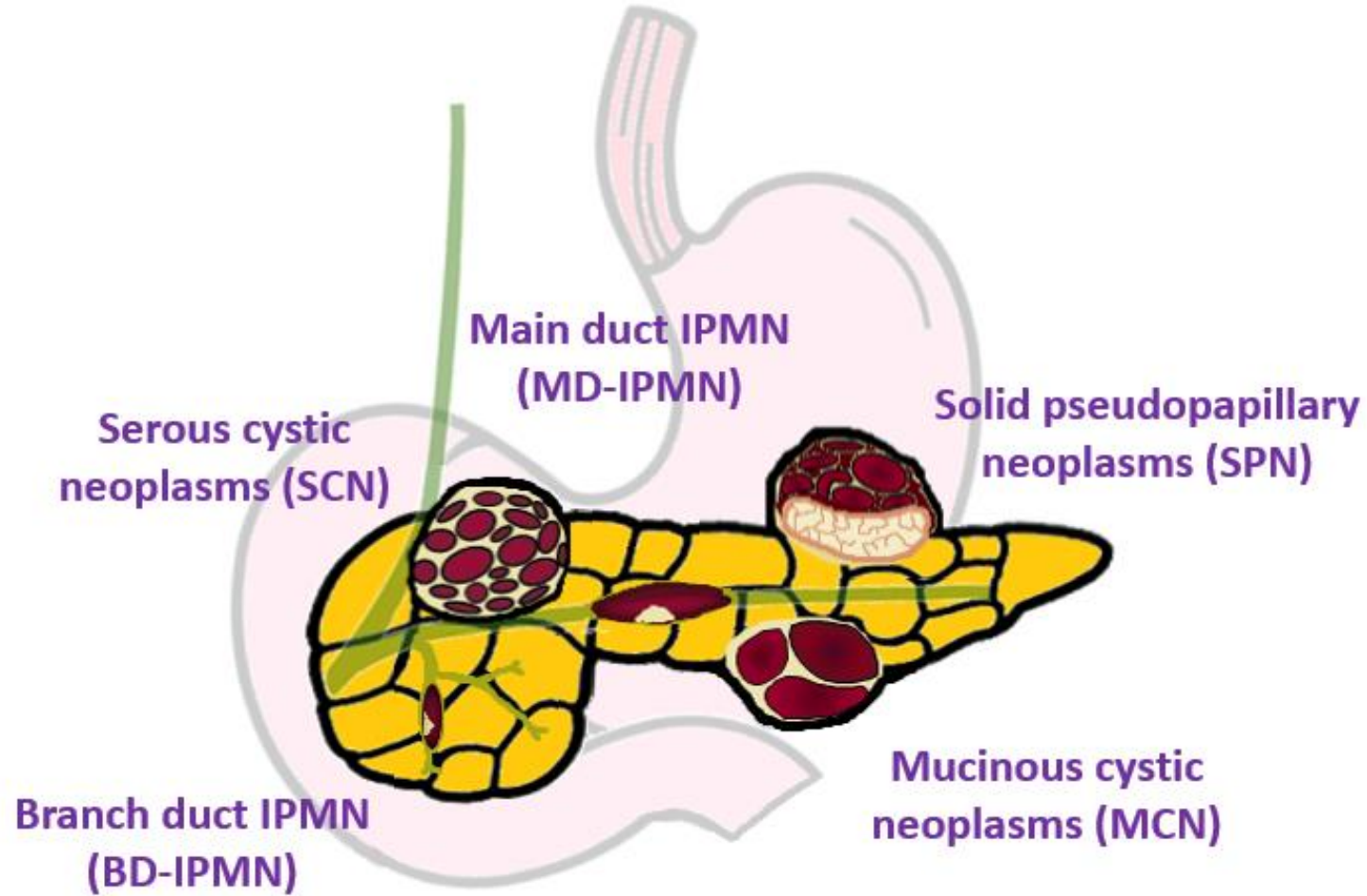


Beger procedure
(duodenal
preserving
pancreatic head
resection)








Frey procedure

Cystic Lesions of the Pancreas

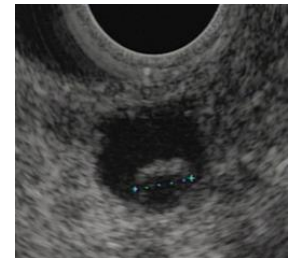
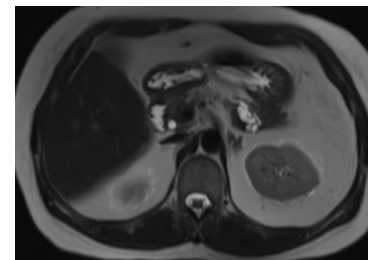
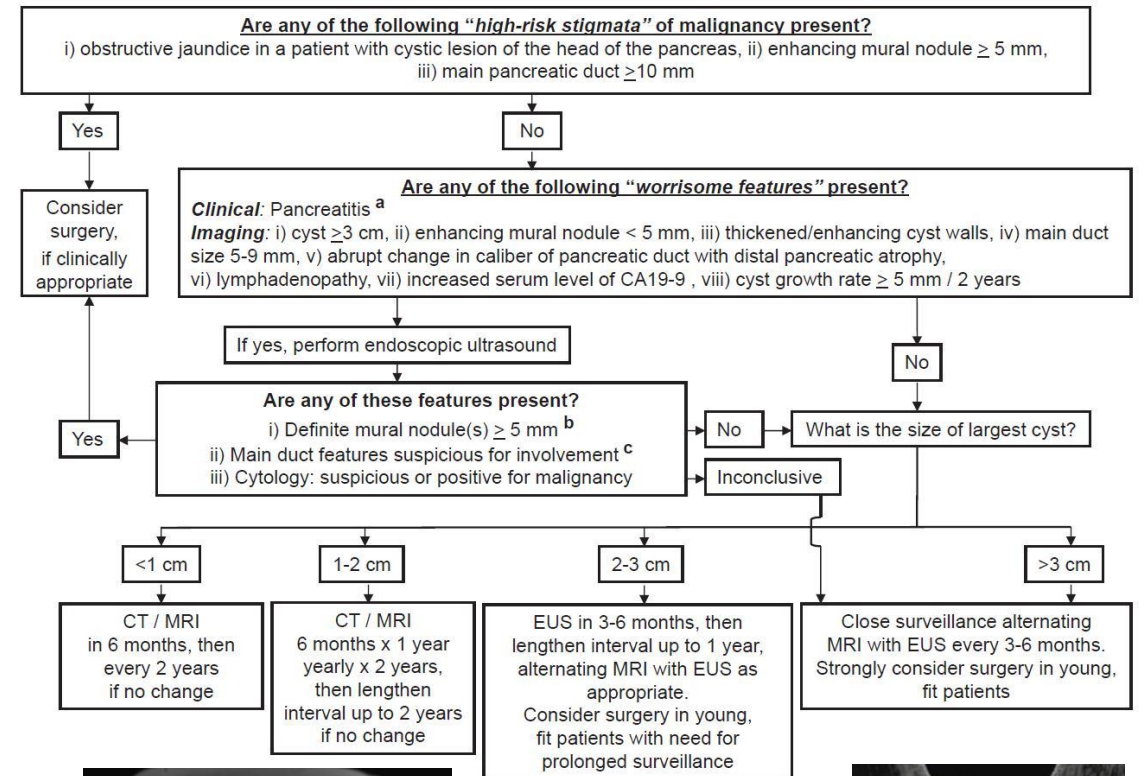


Cystic Lesions of the Pancreas

	Features	Location	Risk of Malignancy	Amylase	CEA	Viscosity	Mucin	Cytology	Radiology
SCN	F>M (4:1); 60-70s "grandmother"	Throughout	No	↓	↓	↓	(-)	Glycogen rich	
MCN	F>M (9:1); 40-50s "mother"	Body/Tail	15%	↓	↑↑↑	↑	(+)	Ovarian-type stroma	
SPT	F; <40 "daughter"	Body/Tail	Low	↓	↓	Solid / cystic	(-)	Necrotic cells	
Pseudocyst	N/A	Throughout	No	↑↑↑	↑/↓	↓	(-)	Inflammatory	
IPMN	F = M, 60-80s	Head; Throughout	MD: 60% BD: 2-10%	↑↑↑	↑	↑	(+)	Tall columnar, mucin producing	

Cystic Lesions of the Pancreas -- IPMNs

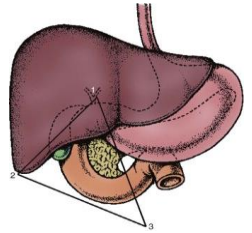
- IPMNs: Resect all MAIN DUCT IPMNs and BD-IPMNs with HIGH-RISK STIGMATA
 - Enhancing solid component
 - Main Duct (MD) ≥ 10 mm
 - Proximal lesion with obstructive jaundice
- Worrisome features: NEED EUS +/- FNA to stratify lesion
 - Cyst ≥ 3 cm
 - Thickened/Enhancing cyst wall
 - Main duct $> 5-9$ mm
 - Enhancing mural nodule
 - Abrupt change in pancreatic duct caliber
 - Lymphadenopathy
 - History of pancreatitis
 - Growth ≥ 5 mm in 2 years
 - \uparrow CA 19-9



Pancreatic Adenocarcinoma

- Risk Factors: smoking; chronic pancreatitis, DM, obesity, genetics [CF, HNPCC, BRCA-2, Peutz-Jegher, Familial Pancreatitis]
- Pathogenesis: PanIN --> PDAC [95% KRAS mutation]
- Tumor Markers: CEA, CA19-9 (10-15% w/o ↑CA19-9)
- Treatment: Whipple (head/neck); distal pancreatectomy + splenectomy (body/tail)
- Complications: DGE, Pancreatic fistula/leak, GDA bleed/pseudoaneurysm
- Palliative Therapy:
 - Biliary obstruction: ERCP + metal stent; HJ if during laparotomy
 - GOO: endoscopic stenting; GJ if during laparotomy
 - Pain: celiac plexus block

Neuroendocrine Tumors of the Pancreas



- Non-Functional PNETs: majority of PNETs, hypervascular on CT, 50% with mets to liver
 - Tx: enucleation (small, <2cm) vs. complete resection (en-bloc resection + metastectomy)

Functional PNETs:

- Axial Imaging [Triple phase CT, MRI]
- Somatostatin Receptor Scintigraphy [no receptors for insulinoma]
- Ga-68 DOTATATE PET-CT
- EUS
- Selective visceral angiography

	Symptoms/Features	Location	Risk of Malignancy	Associations	Diagnosis	Treatment
Insulinoma	Whipple's Triad (Most common fNET)	Evenly distributed	Least Likely <10%	#2 fNET in MEN1 (5%)	↑insulin/C-peptide w/ fast Insulin : glucose > 0.4	Enucleation (benign)
Gastrinoma (Zollinger-Ellison)	Peptic ulcers, diarrhea	Passaro's Triangle	50% malignant	#1 fNET in MEN1 (25%)	Gastrin > 1000 Secretin stimulation ↑gastrin > 200	Resection vs. enucleation; PPI
Glucagonoma	4 D's: dermatitis (necrolytic migratory erythema), diabetes, depression, DVT	Tail	50-80% malignant	95% sporadic MEN1 (5%)	fasting glucagon > 1000 pg/mL	Complete excision/debulking Octreotide for sxs
VIPoma (WDHA; Verner-Morrison)	watery diarrhea, hypokalemia, achlorhydria	Body/Tail	50-70% malignant	95% sporadic MEN1 (5%)	VIP > 200 pg/mL	Complete excision/debulking Octreotide for sxs
Somatostatinoma	DM, Cholelithiasis, Steatorrhea (Least common fNET)	Head, periampullary	Majority malignant	NF-1	somatostatin > 10 ng/mL	Complete excision/debulking + CCY



Thank you and good luck!



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