



Schulich  
MEDICINE & DENTISTRY

# Presentation

- 63 year-old female, presents with 2 week hx epigastric pain
- Pain radiates to her lower back, aggravated by food
- Associated with fevers, chills, night sweats

# Medical/Surgical History

- Medical history: hypothyroidism, cervical cancer, pancreatic cancer
- Surgical history: Whipple under Dr. Wall, Aug 2014
  - Stage: T3 N0 M0 Ductal adenoca (IIa)



# Social History

- Non-smoker, moderate alcohol intake
- Lives alone, but good social support

# Physical Examination?

# Physical exam

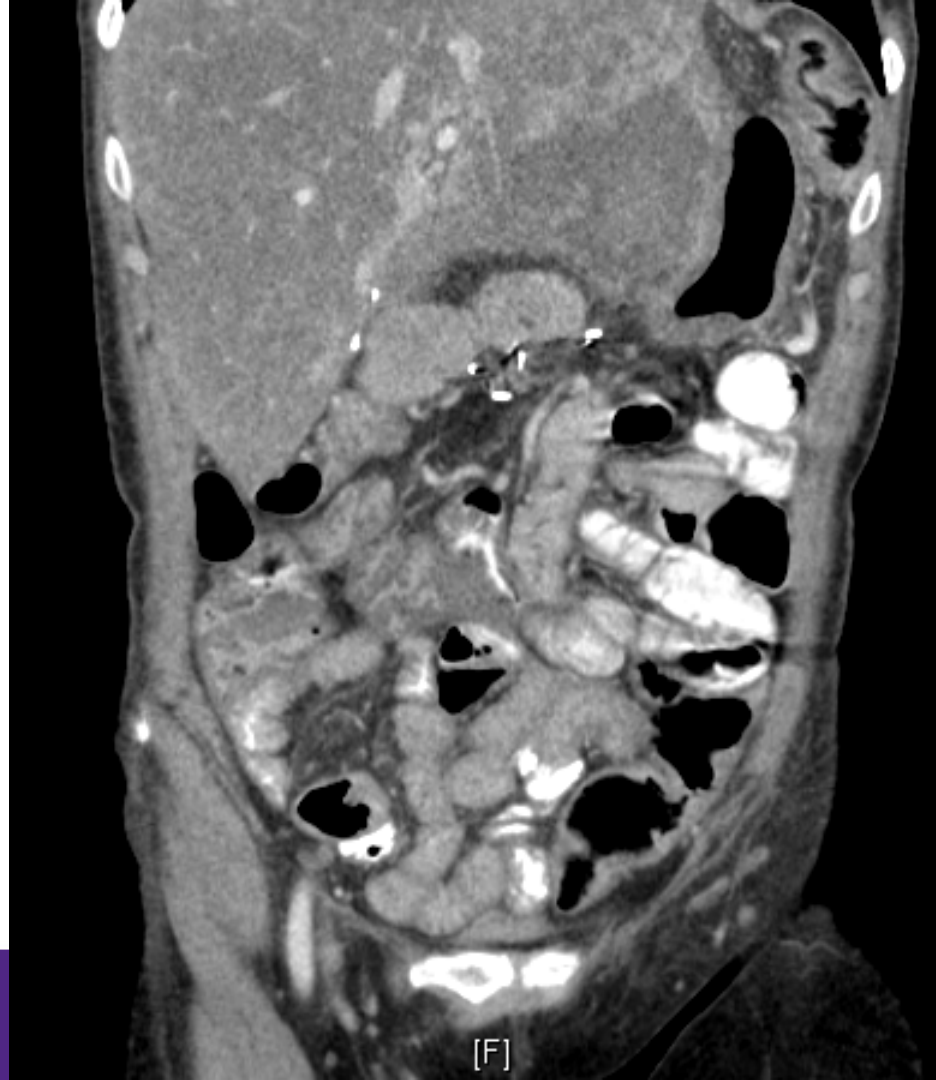
- Abdomen soft, non-distended, tender epigastrium
- Hepatomegaly, no other masses
- No lymphadenopathy
- No evidence of jaundice, signs of liver disease
- No evidence of wasting, sudden weight loss



# Imaging







# Wednesday Teaching

Feb 3, 2016

# Today's Agenda

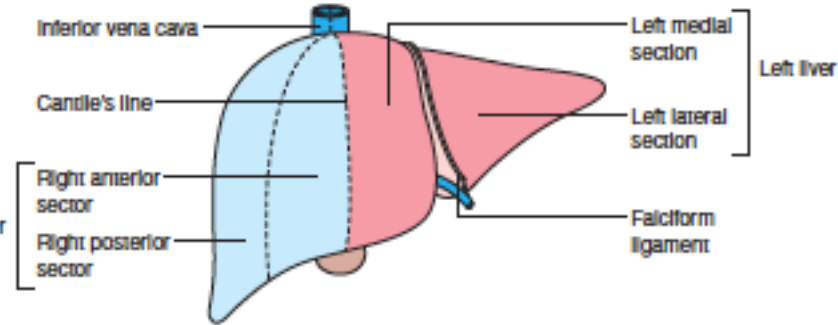
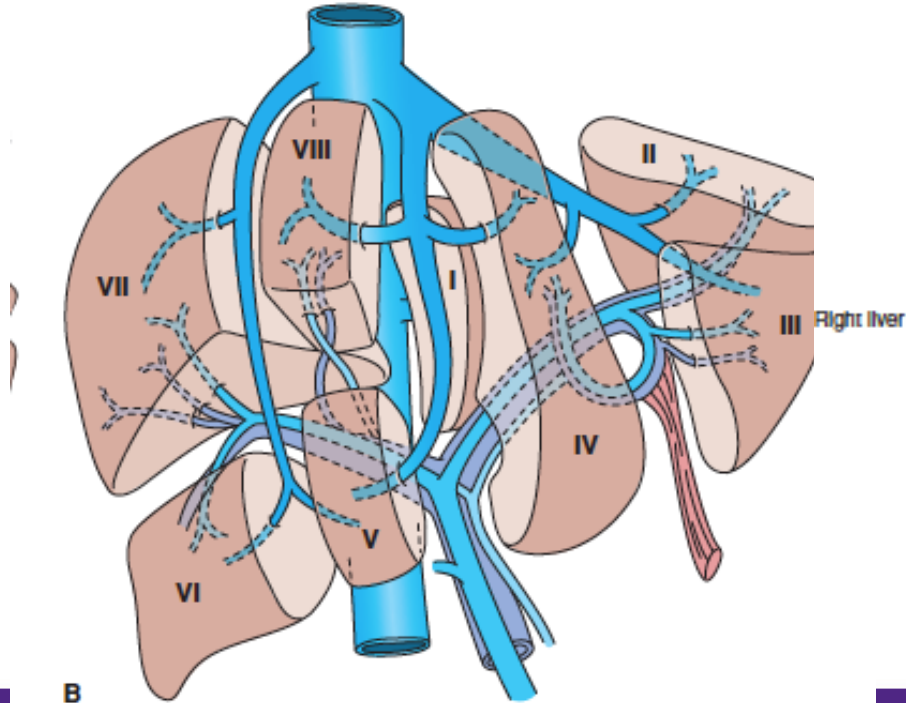
Dept of General Surgery

- Liver anatomy
- Hepatic lesions
  - Abscess, hydatid cysts, HA, FNH, hemangioma
- Workup of a liver mass
- Management of varices (prophylactic)
- Transplant workup
- Discuss some seminal papers

# Liver Anatomy

# Main Points:

- Couinaud's Segments, clockwise, Caudate!



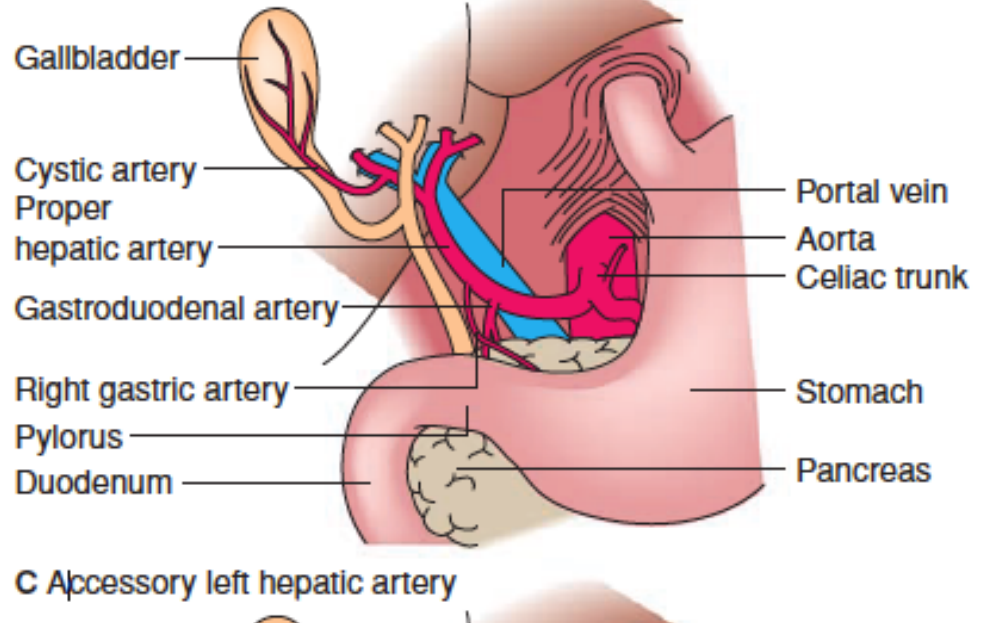
- Arterial blood supply: only normal in 50%

**Table 1. CLASSIFICATIONS OF HEPATIC ARTERIAL TYPES**

	Type	Description	Percent
Michels <sup>3</sup> (n = 200)	1	Normal	55
	2	Replaced LHA from LGA	10
	5	Accessory LHA	8
			18
	3	Replaced RHA from SMA	11
	6	Accessory RHA	7
			18
	4	Replaced RHA + LHA	1
	7	Accessory RHA + LHA	1
	8	Replaced RHA + Accessory LHA or Replaced LHA + Accessory RHA	2
Current Series (n = 1000)			4
	9	CHA from SMA	2.5
	10	CHA from LGA	0.5
	1	Normal	75.7
	2	Replaced or Accessory LHA	9.7
	3	Replaced or Accessory RHA	10.6
	4	Replaced or Accessory RHA + Replaced or Accessory LHA	2.3
	5	CHA from SMA	1.5
	6	CHA from aorta	0.2

LHA—left hepatic artery; LGA—left gastric artery; RHA—right hepatic artery; SMA—superior mesenteric artery; CHA—common hepatic artery.

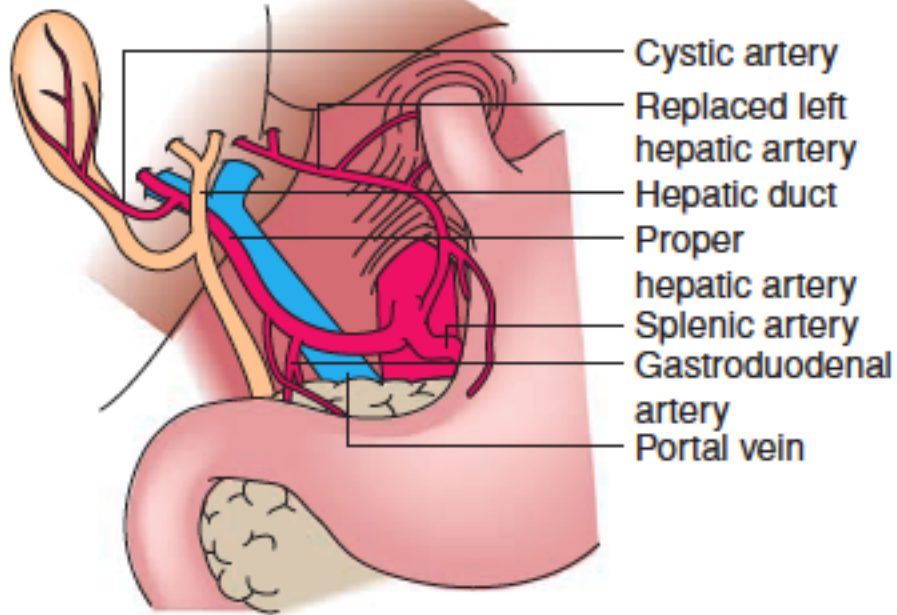
### A Normal anatomy



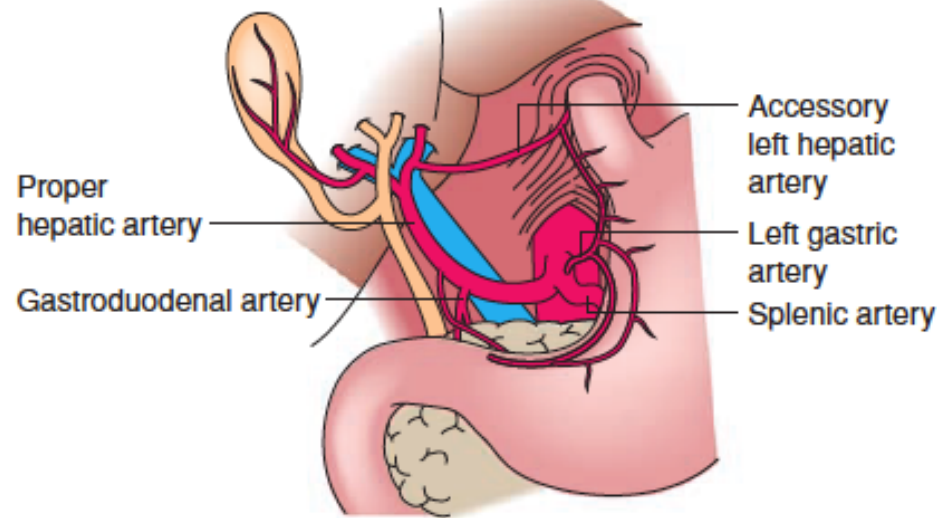
### C Accessory left hepatic artery

- Arterial blood supply: only normal in 50%

**B** Replaced left hepatic artery



**C** Accessory left hepatic artery



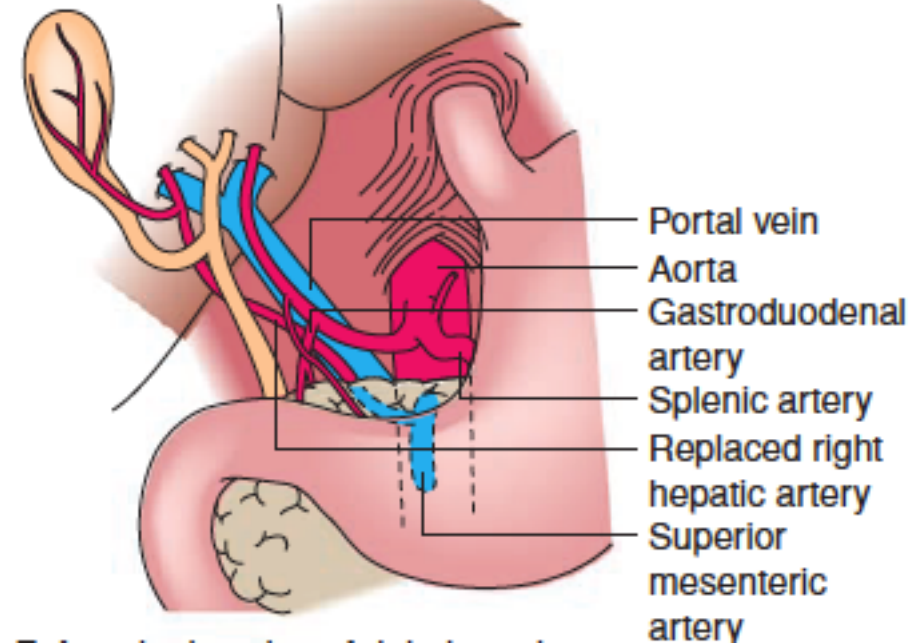
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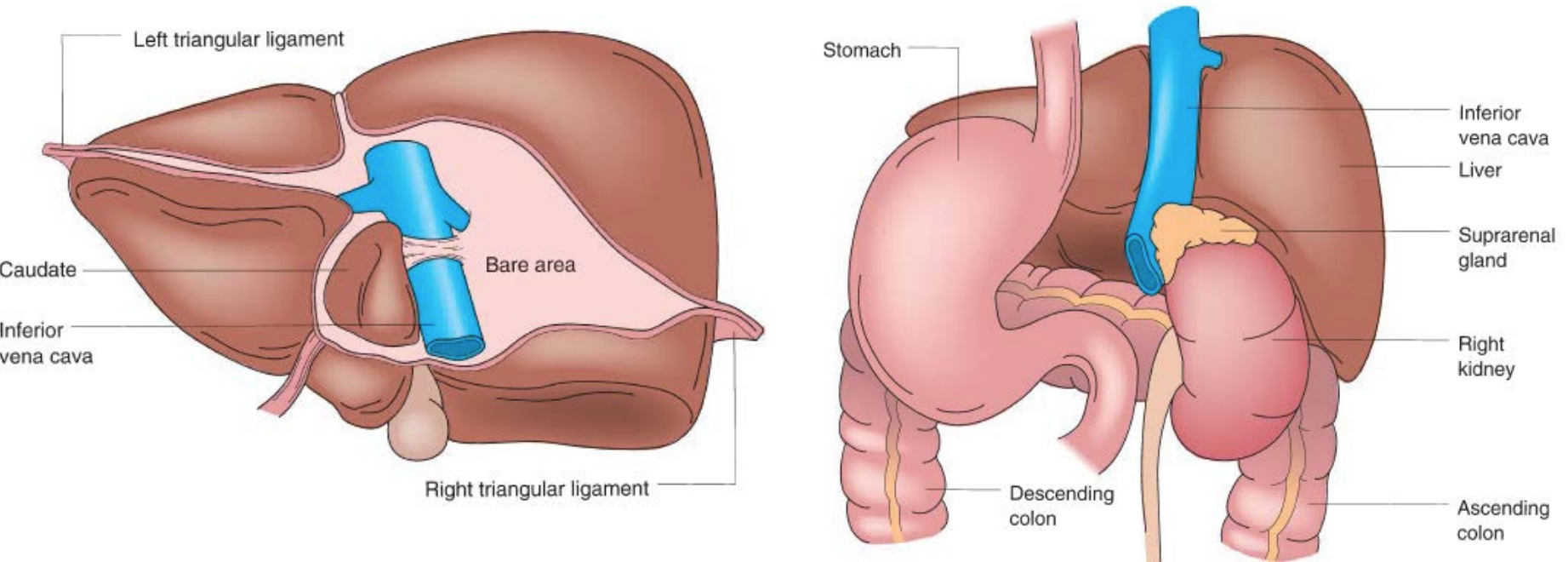
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**D Replaced right hepatic artery**

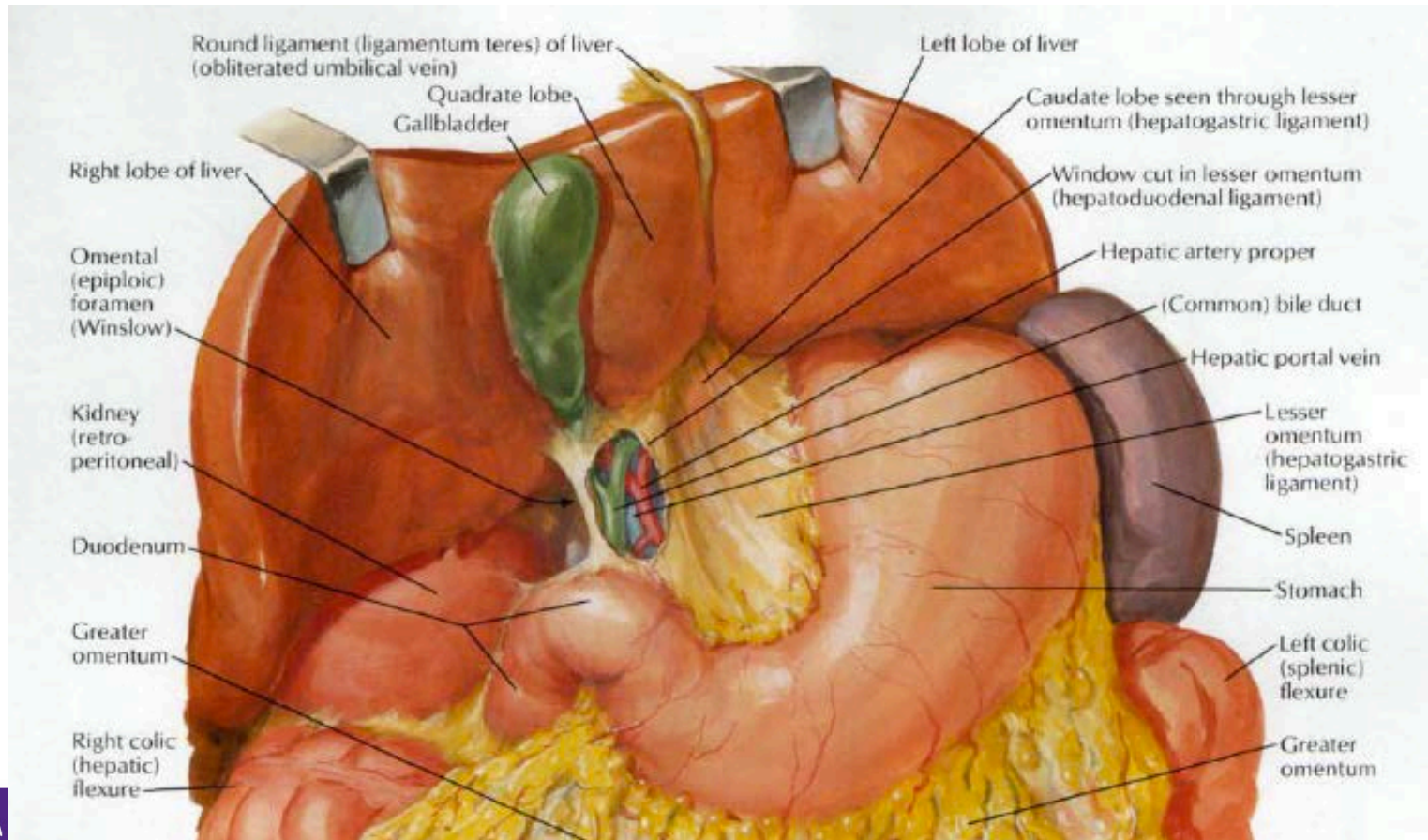




- Morphological anatomy

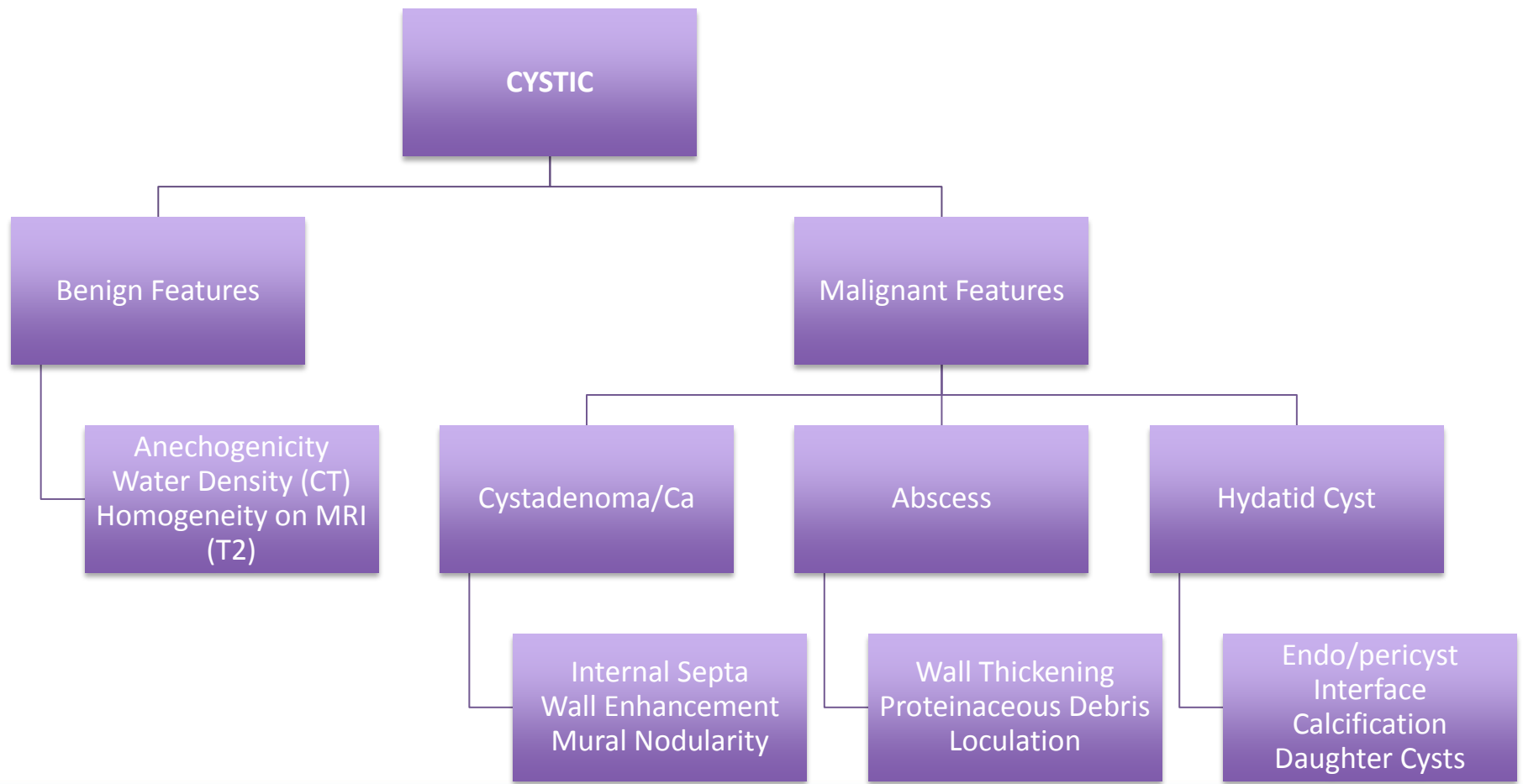


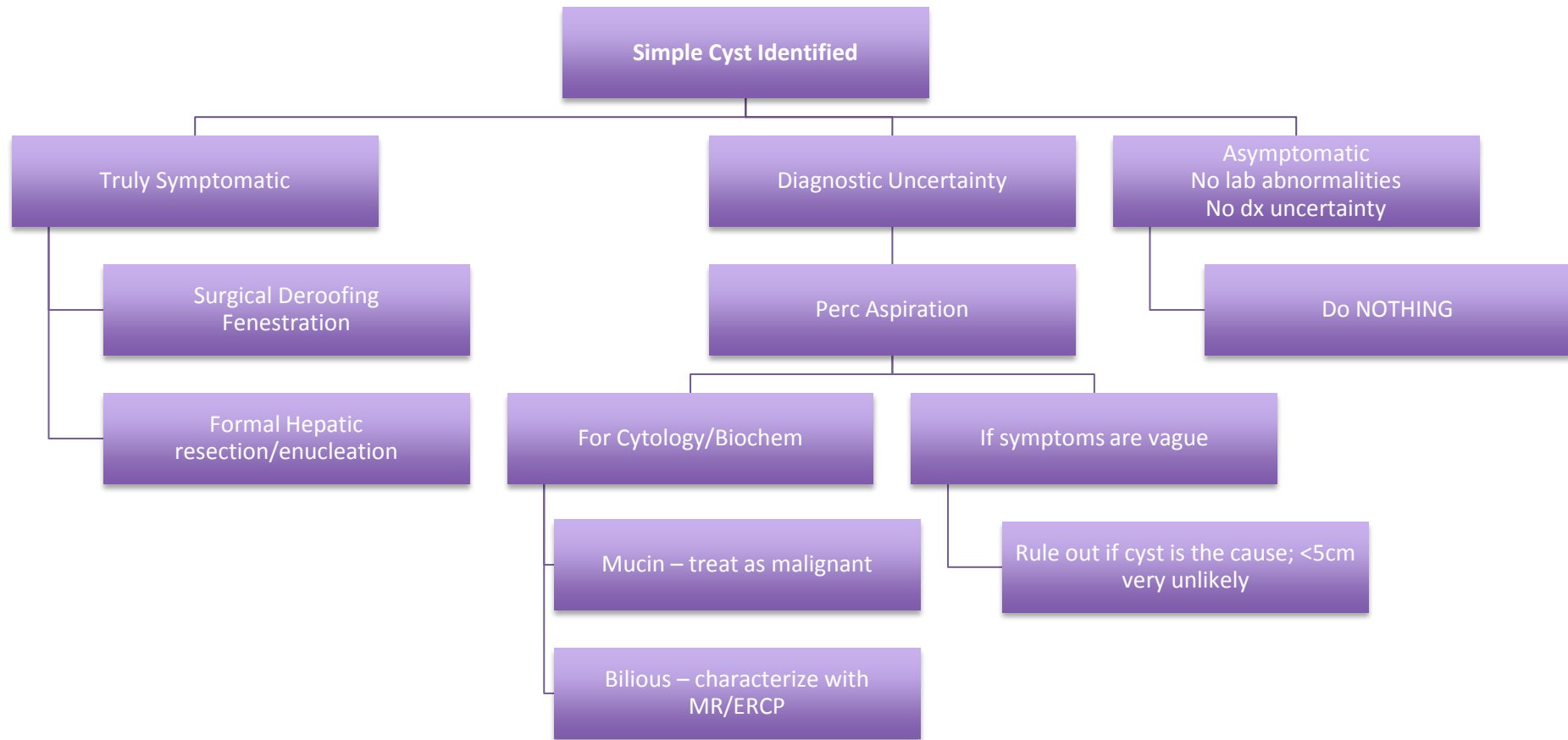
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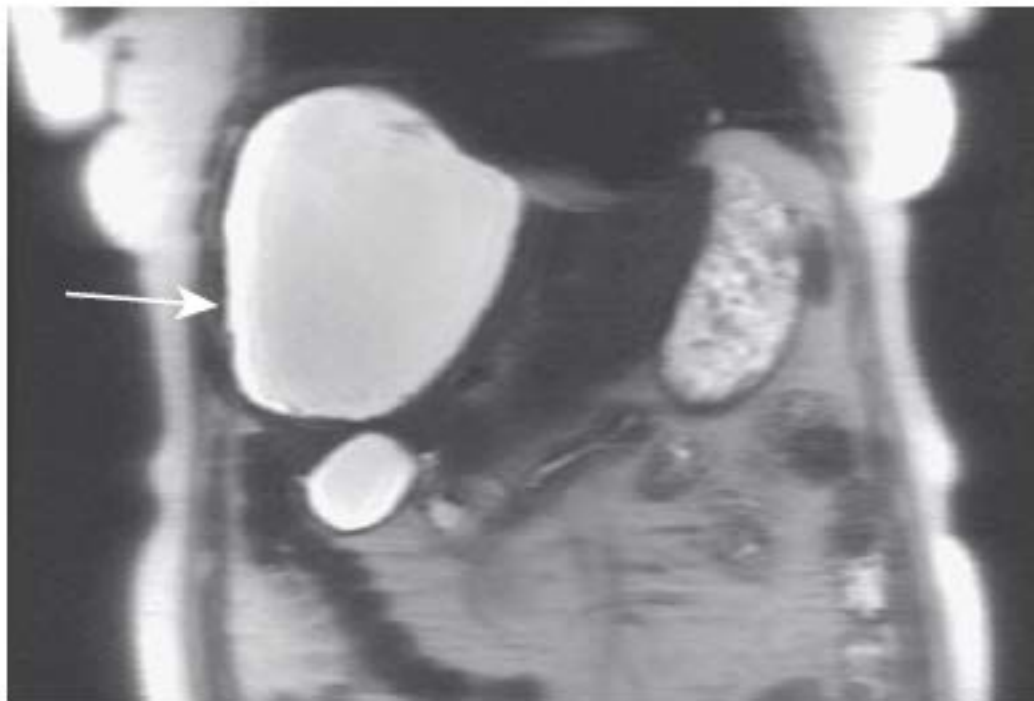


# Approach to Benign Liver Lesions

# Cystic or Solid?







# Neoplastic Cysts



# Epidemiology

- Cystadenomas – 5% of all hepatic cysts
  - more frequently in 40-50s, female
- Cystadenocarcinoma
  - With ovarian stroma – females only
  - Without – equal dist., 50-60s

# Characteristics

- Multilocular, lined by columnar/cuboidal epithelium
- May have dense, cellular ovarian-like stroma (if so, ER/PR + receptors)
- Cystadenomas occur commonly in seg IV
- May have elevated CEA (reliable) / CA-19-9 (unreliable)

# Characteristics

- 3 distinct layers
  - Epithelial (mucin secreting), mesenchymal stroma (atypical spindle cells, intestinal metaplasia gives malignant progression), outer layer of collagen tissue – allows enucleation

# Characteristics

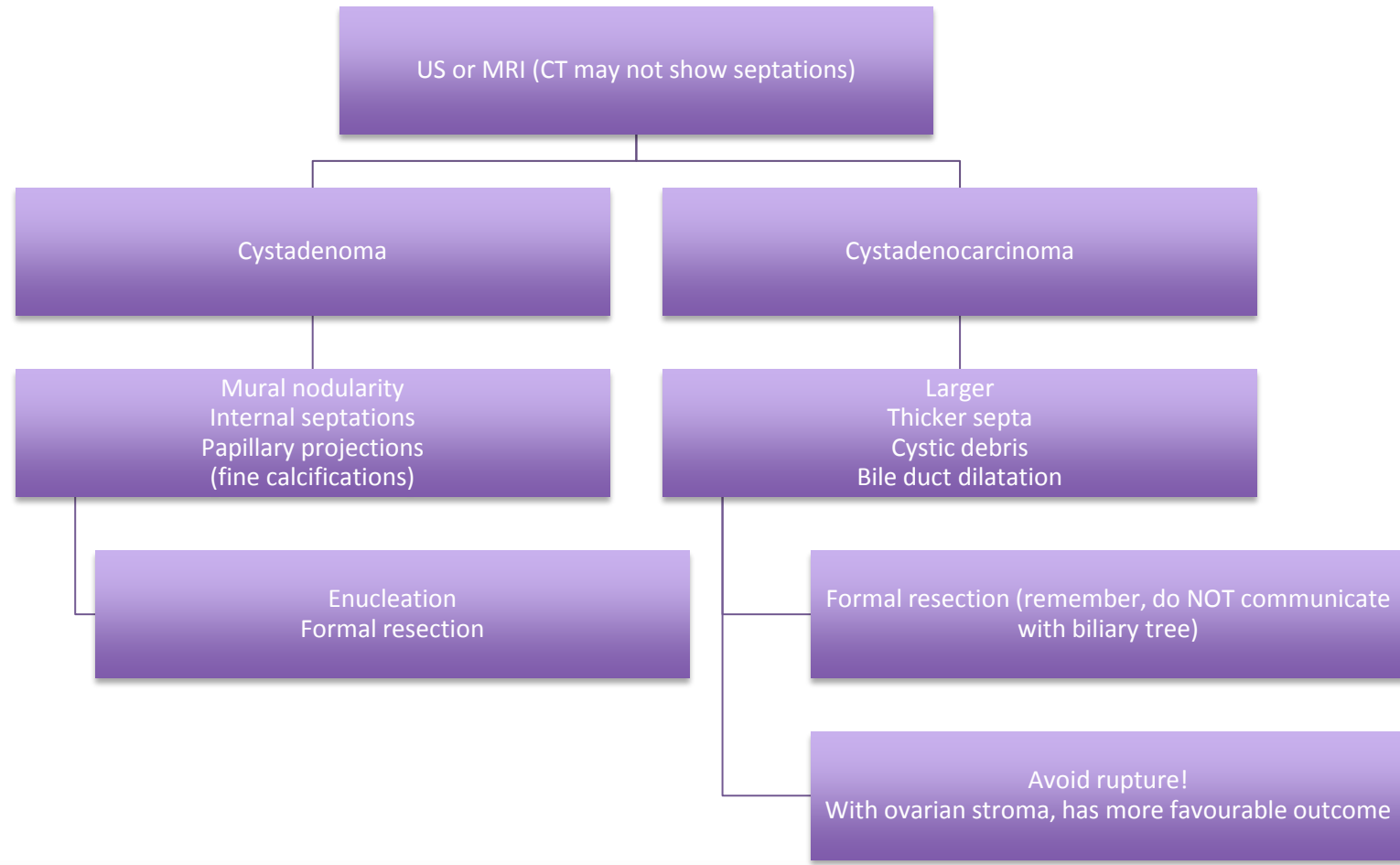
- Non-ovarian stroma variant can occur in both sexes, but only ovarian can occur in women
- These more commonly communicate with biliary tree

# Pathogenesis

- Arise from the biliary system
- Prevailing theory – during embryogenesis ovarian cells shed into liver, cause proliferation, cysts

# Presentation

- Non-specific, vague abdominal pain
- Nausea
- Biliary obstruction
- Incidental finding







# Hydatid Cysts

# Epidemiology

- Endemic in Mediterranean, Africa, Middle East, SA, Asia, European sheepfarmers
- In North Americans, caught either from contact with immigrant, or abroad in endemic area
- Fecal-oral transmission of the larva
- Ingested – portal circ – liver/lung/brain/bone

# Pathogenesis

- Canine tapeworm *Echinococcus granulosus*
- In 1/3 rupture occurs
- Mature cysts may become inactive, calcified, negative serology

# Characteristics

- 2 layer cyst wall in liver
  - Inner single layer where daughter cysts grow
  - Outer, thick, reactive fibrous layer, 'pericyst', calcified in 50%
- Classification – complicated 5-stage WHO, or
- Active, inactive, transitional

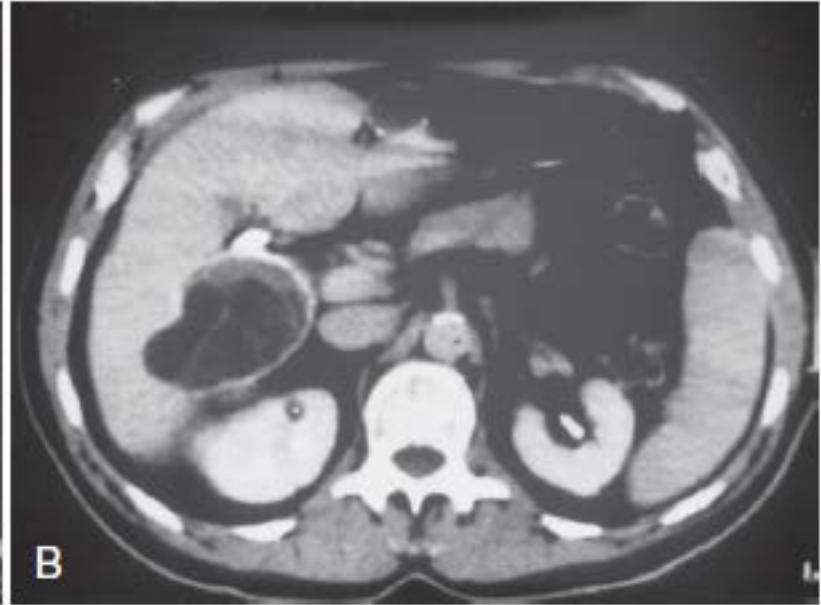
# Presentation

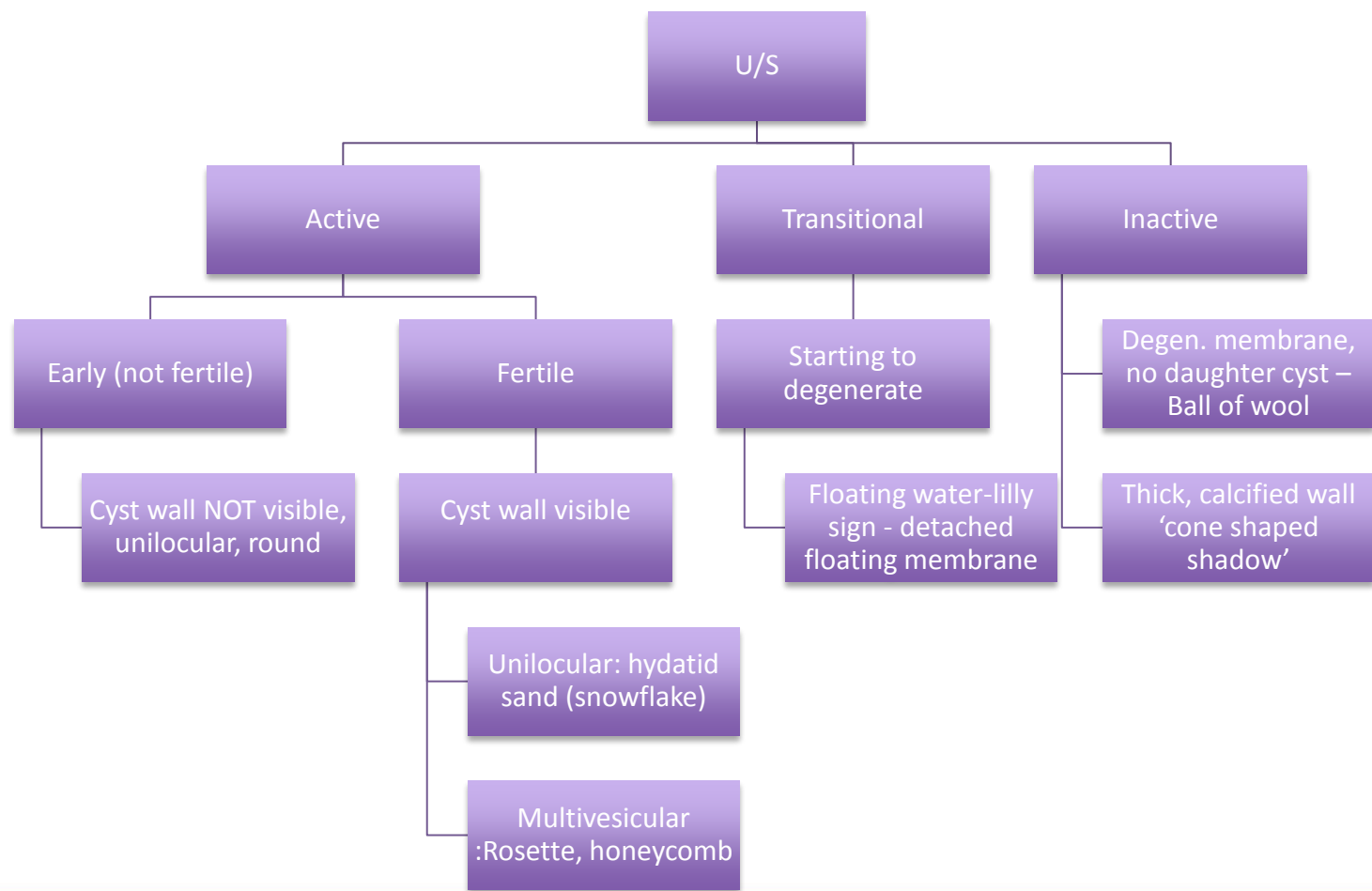
- Asymptomatic (incidental finding)
- RUQ pain, fullness
- Pain from rupture, fever, obstructive jaundice (communication with biliary tree in 25%)
- Labs:
  - CBC (eosinophilia in 40%), ELISA for serology, microscopy of fluid

# Imaging

- Ultrasound is the main modality
- CT – for perioperative planning, additional detail
- MRI to delineate relationship to other structures
- MRCP/ERCP for identification of biliary involv.
- Intra-op ultrasound – commonly used

# Imaging







# Treatment

- 3 main aims
  - Early intervention to prevent secondary comps
  - Eradication of parasite
  - Prevention of recurrence

# Treatment

- Mainstay – systemic antihelminthic drugs
  - DOC – albendazole – concentrated by liver into cysts, most effective
- Non-operative mgt
  - Perc tx: PAIR:  
puncture/aspiration/injection, reaspiration  
(use 20%NS / 95% ethanol) + 1w pre  
albendazole, 1m post

# Treatment

- PAIR CI – biliary fistulae, complicated cysts, inactive, calcified
- Catheter drainage
  - >10cm, unilocular, left in place until <10cc/24h
  - Indicated for high risk, refuse surg, failed medical mgt, infected, recurrence post sx

# Treatment

- Operative therapy indications
  - Multiple daughter cysts
  - Singular, superficial, high risk of rupture
  - Infected / biliary communication
  - Mass effect on adjacent structures

# Treatment

- Cyst evacuation, pericystectomy or surgical resection?
- Resection indications
  - Multiple cysts, complicated (ie risk of rupture, often >7.5cm), biliary fistulae, proximity to vital structures, failed conservative mgt,

# Treatment

- Important points:
  - Line surgical field with saline-soaked gauze (to avoid spread of infection)
  - Never use scolecoidal agent if you suspect biliary fistula
  - Try not to rupture the cyst...why?

# Cyst Rupture

- Disseminated intra-abdominal disease
- Anaphylaxis
- Peritonitis
- Bronchopleural fistula (chest/diaphragm) with spont rupture
- ICU, if spont, require surgery

# Outcomes

- 90-95% cure rate with some kind of surgical intervention
- Recurrence 70-80% with medical therapy alone



# Pyogenic Liver Abscess

# Epidemiology

- Most common visceral abscess
- 48% of visceral abscesses, 13% of intra-abdominal
- Risk factors: DM, hepatobiliary/pancreatic disease, liver transplant
- In Asia: K pneumoniae – colorectal Ca

# Pathogenesis

- Usually follow episode of portal vein pyema (bowel perf or peritonitis)
- Direct route of spread from biliary sepsis
- Gallstones/malignant obstruction 40-60% of cases
- Occasionally from surgical wounds

# Pathogenesis

- Systemic circulation: monomicrobial abscess with strep/staph should prompt hunt for source
- More common in right lobe of liver
- Most commonly Strep anginosus/milleri

# Presentation

- Most commonly fever, abdominal pain (90%)
- 50% have hepatomegaly or jaundice
- Symptoms of right-sided pneumonia in 30%

# Treatment

- Drainage, antibiotics
- <5cm – catheter/aspiration drainage, followed by antibiotics
- >5cm – recommended drainage via catheter, remaining in situ until output minimal

# Treatment

- Indications for surgical drainage
  - Multiple abscesses
  - Loculated abscess/failure of catheter
    - Failure after 7 days
    - Viscous content
  - Treatment of underlying surgical disease

# Treatment

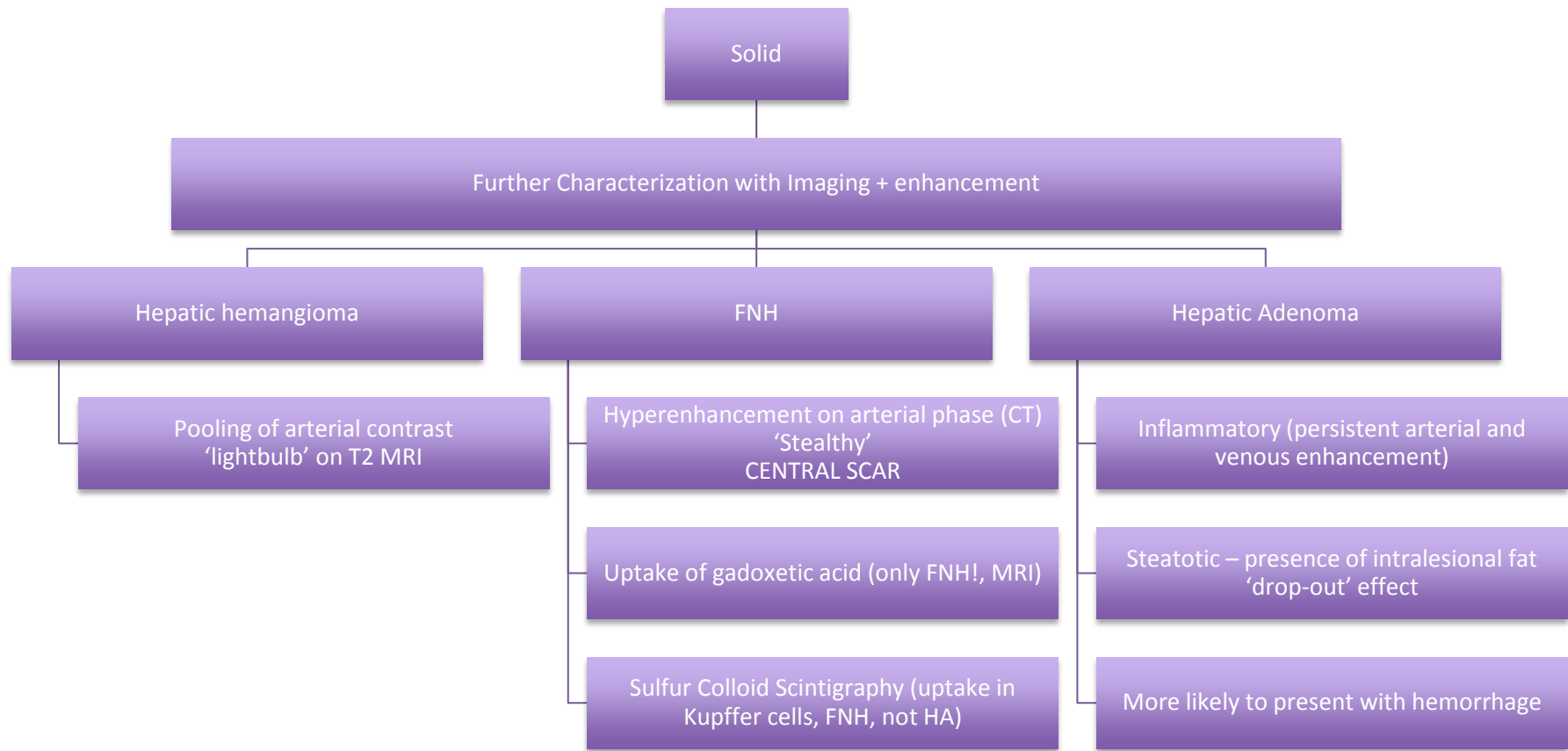
- ERCP indicated for those in communication with biliary tree
- Endpoints:
  - Clinical resolution (imaging takes longer to resolve)
  - WCC, CRP, temperature,



# Treatment

- Mean time to resolution (on US)
  - <10cm – 16 weeks
  - >10cm – 22 weeks
- Abx – 4-6 weeks
- No randomized control trials regarding duration, type of antibiotic, drain time

# Cystic or Solid?



# Hepatic Hemangioma 'cavernous'

# Epidemiology

- Most common benign liver tumour, F:M / 3:1
- NO malignant potential
- >5cm = 'giant' hemangiomas (some texts >10cm)
- ~1% rupture/bleed, mortality 30%

# Pathophysiology

- Unknown, but hormonally driven
- Classically – pregnant woman
- 10-15% regress spontaneously
- Histologically: cavernous/capacious vascular spaces, larger lesions have calcifications

# Presentation

- RUQ pain – maybe due to microthrombi, stretch of Glisson's capsule
- Rare rupture (1%)
- Even more rare arteriovenous shunting with cardiac hypertrophy and CHF

# Diagnosis

- No lab tests
- CT – art/venous/delayed phase = contrast puddling, pathognomonic of the disease
- MRI – classic bright non-con T2
- Biopsies will make stuff bleed bro



# Treatment

- In event of acute bleed:
  - IR embolization
  - Surgical resection if fails, or, Kasabach-Merritt Syndrome (fibrinolysis, thrombocytopenia, more common in children, life-threatening)

# Infantile hepatic hemangioma

- Appears similar to adult, but of course different
  - Histologically composed primarily of endothelial cells 'aka hemangioendotheliomas', rapid growth/involution
  - Don't confuse with 'epithelioid hemangioendotheliomas' which are malignant

# Hepatic Adenoma

# Epidemiology

- Typically young female (20-40y), on OCP, or roiding male
- Other risk factors: glycogen storage disease, pregnancy
- Malignancy risk: starts when >5cm, 4-15% HCC, in men, as high as 47%

# Pathophysiology

- Neoplastic, monoclonal proliferation of hepatocytes, may lead to bleeding/malignancy
- Subtypes: inflammatory, steatotic

# Presentation

- Usually mass effect or bleeding
- Lesions  $<5\text{cm}$  rarely cause symptoms (look for other cause)

# Diagnosis

- Difficult to distinguish between FNH and HA
- See previous algorithm
- When equivocal, may consider perc bx BUT
  - Risk of malignant dissemination through tract, risk of bleeding

# Treatment

- If <5cm, may follow – routine imaging and AFP
- Indications for surgical resection
  - >5cm and/or male
  - Symptoms, including bleeding
  - Inability to R/O malignancy



# Focal Nodular Hyperplasia

# Epidemiology

- Often found in asymptomatic women, not hormonally driven
- No malignant potential
- 23% association with other liver lesions
- Atypical – lack central scar, harder to differentiate

# Pathophysiology

- Aetiology unknown
- Probably due to disturbance of vascular function
- Benign polyclonal proliferation of all hepatocellular elements, thus hard to distinguish from normal parenchyma

# Treatment

- Do not treat if not symptomatic
- No need for surveillance

# Transplant

# Criteria

- Evaluation for liver transplant should occur when:
  - A patient with cirrhosis has experienced an index complication
  - Worsening renal dysfxn or rapid hepatic decompensation (if they match the overall criteria)

# Considerations

- Major comorbidities
- Alcohol or substance abuse ongoing, psychosocial issues, support systems
- Are there pre-existing issues that can be treated pre-transplant to maximize chances of success?

## Liver Transplant Evaluation (O'Leary et al, Gastro 2008)

Financial screening	Hepatology eval
Surgical eval (technical challenges, etc)	Lab testing
Cardiac eval	Imaging
General Health Assessment	Dental assessment
Anesthesia eval	Psych/mental health eval
Financial counselling	Nutritional eval
Infectious disease eval	Social work eval



Contraindications	
MELD <15	Severe cardiac disease
AIDS	HCC with mets
Anatomic abnormality	Hepatic failure with ICP >50mmHG
Non-compliance	Lack of support system

# MELD Score

- Uses Cr, bili, INR
  - $3.78 \times \ln[\text{serum bilirubin (mg/dL)}] + 11.2 \times \ln[\text{INR}] + 9.57 \times \ln[\text{serum creatinine (mg/dL)}] + 6.43$
- 3m mortality – 40 = 70%; 10-19 = 6%

# MELD Exceptions

- Conditions associated with liver disease/death, not accounted for directly in MELD scoring
- HCC, cystic fibrosis, hilar cholangio, hepatopulmonary syndrome to name a few
- Standard set of exception points for each pathology, to bump them up in the transplant list

# Varices

- Recommendations
- In patients with compensated cirrhosis and no varices, do not start B-blockers. Repeat EGD in 3 years (Class A, no risk of bleeding)

# Varices

- For patients with cirrhosis, small varices and risk of bleeding (Child's C/B, red whales)
- Non-selective B-blocker is indicated
- For patients with cirrhosis, small varices and no risk of bleeding, B-blockers can be used but no advantage has been proven

# Varices

- For medium/large varices that have high risk for bleeding
- Non-selective B-blockers or EVL can be used to equal benefit, no difference in mortality
- EVL requires repeat scopes q1-2w to obliteration, FU EGD not necessary for B-blockers

# Group 1

- 45yo female referred to your clinic for mass found incidentally on US for assessment of GB
- ‘well marginated hypoechoic lesion seen with displacement of hepatic vasculature, 3cm maximal diameter. Differential diagnosis includes FNH vs HA. Further imaging is recommended’
- Please describe the further tests you would order to differentiate between FNH and HA

# Group 2

- Please draw/describe the functional divisions of the liver
- Please draw/describe the anatomical variations regarding arterial supply of the liver
- Draw/describe the main portal triad



# Group 3

- Cystic lesion is noted in the liver during an US for intermittent fevers, appy workup. The appy is not visualized. Report states 'dedicated liver US is recommended to characterise this lesion'. The patient is a young long haul flight attendant.
- What further history would you like to elicit? What findings on ultrasound would you like to obtain to distinguish between a simple cyst, and hydatid disease?

# Group 4

- You are asked to help work up 55 year old patient with well-compensated cirrhosis for transplant. What criteria will you use, and what is part of your workup?
- On endoscopy, the patient is found to have varices. Describe the different management strategies for variceal bleeding prophylaxis, depending on the situation.

# Thank you

- I hope you found this session enjoyable.
- Have a nice day.



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