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 adenocarcinoma (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
Today’s Agenda

- 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
Main Points:

- Couinaud’s Segments, clockwise, Caudate!
• Arterial blood supply: only normal in 50%
- Arterial blood supply: only normal in 50%
• Arterial blood supply: only normal in 50%

Table 1. CLASSIFICATIONS OF HEPATIC ARTERIAL TYPES

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>55.0%</td>
</tr>
<tr>
<td>2</td>
<td>Replaced LHA from LGA</td>
<td>10.0%</td>
</tr>
<tr>
<td>5</td>
<td>Accessory LHA</td>
<td>8.0%</td>
</tr>
<tr>
<td>3</td>
<td>Replaced RHA from SMA</td>
<td>11.0%</td>
</tr>
<tr>
<td>6</td>
<td>Accessory RHA</td>
<td>7.0%</td>
</tr>
<tr>
<td>4</td>
<td>Replaced RHA + LHA</td>
<td>1.0%</td>
</tr>
<tr>
<td>7</td>
<td>Accessory RHA + LHA</td>
<td>1.0%</td>
</tr>
<tr>
<td>8</td>
<td>Replaced RHA + Accessory LHA</td>
<td>2.0%</td>
</tr>
<tr>
<td>9</td>
<td>CHA from SMA</td>
<td>4.0%</td>
</tr>
<tr>
<td>10</td>
<td>CHA from LGA</td>
<td>0.5%</td>
</tr>
<tr>
<td>1</td>
<td>Normal</td>
<td>75.7%</td>
</tr>
<tr>
<td>2</td>
<td>Replaced or Accessory LHA</td>
<td>9.7%</td>
</tr>
<tr>
<td>3</td>
<td>Replaced or Accessory RHA</td>
<td>10.6%</td>
</tr>
<tr>
<td>4</td>
<td>Replaced or Accessory RHA + Accessory LHA</td>
<td>2.3%</td>
</tr>
<tr>
<td>5</td>
<td>CHA from SMA</td>
<td>1.5%</td>
</tr>
<tr>
<td>6</td>
<td>CHA from aorta</td>
<td>0.2%</td>
</tr>
</tbody>
</table>

LHA—left hepatic artery; LGA—left gastric artery; RHA—right hepatic artery; SMA—superior mesenteric artery; CHA—common hepatic artery.
• Morphological anatomy
• Morphological anatomy
Approach to Benign Liver Lesions
Cystic or Solid?
CYSTIC

Benign Features
- Anechogenicity
- Water Density (CT)
- Homogeneity on MRI (T2)

Malignant Features
- Cystadenoma/Ca
- Abscess
- Hydatid Cyst
  - Internal Septa
  - Wall Enhancement
  - Mural Nodularity
- Wall Thickening
- Proteinaceous Debris
- Loculation
- Endo/pericyst Interface
- Calcification
- Daughter Cysts
Simple Cyst Identified

- Truly Symptomatic
  - Surgical Deroofing Fenestration
  - Formal Hepatic resection/enucleation

- Diagnostic Uncertainty
  - Perc Aspiration
  - For Cytology/Biochem
    - Mucin – treat as malignant
    - Bilious – characterize with MR/ERCP

- Asymptomatic
  - No lab abnormalities
  - No dx uncertainty
  - Do NOTHING

- If symptoms are vague
  - Rule out if cyst is the cause; <5cm very unlikely
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
US or MRI (CT may not show septations)

Cystadenoma
- Mural nodularity
- Internal septations
- Papillary projections (fine calcifications)
- Enucleation
- Formal resection

Cystadenocarcinoma
- Larger
- Thicker septa
- Cystic debris
- Bile duct dilatation
- Formal resection (remember, do NOT communicate with biliary tree)
- Avoid rupture!
  With ovarian stroma, has more favourable outcome
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
- **U/S**
  - **Active**
    - **Early (not fertile)**
      - Cyst wall NOT visible, unilocular, round
    - **Fertile**
      - Cyst wall visible
      - Unilocular: hydatid sand (snowflake)
      - Multivesicular: Rosette, honeycomb
  - **Transitional**
    - Starting to degenerate
      - Floating water-lilly sign - detached floating membrane
  - **Inactive**
    - Degen. membrane, no daughter cyst – Ball of wool
    - Thick, calcified wall ‘cone shaped shadow’
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
- Bronchoplerual 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 angionosus/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?
Solid

Further Characterization with Imaging + enhancement

Hepatic hemangioma
- Pooling of arterial contrast ‘lightbulb’ on T2 MRI

FNH
- Hyperenhancement on arterial phase (CT) ‘Stealthy’ CENTRAL SCAR
- Uptake of gadoxetic acid (only FNH!, MRI)
- Sulfur Colloid Scintigraphy (uptake in Kupffer cells, FNH, not HA)

Hepatic Adenoma
- Inflammatory (persistent arterial and venous enhancement)
- Steatotic – presence of intralesional fat ‘drop-out’ effect
- More likely to present with hemorrhage
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, pathognomomonic 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 ‘eptheliod 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 <5cm 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 treated pre-transplant to maximize chances of success?
<table>
<thead>
<tr>
<th>Liver Transplant Evaluation (O’Leary et al, Gastro 2008)</th>
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<tbody>
<tr>
<td>Financial screening</td>
</tr>
<tr>
<td>Surgical eval (technical challenges, etc)</td>
</tr>
<tr>
<td>Cardiac eval</td>
</tr>
<tr>
<td>General Health Assessment</td>
</tr>
<tr>
<td>Anesthesia eval</td>
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<tr>
<td>Financial counselling</td>
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<tr>
<td>Infectious disease eval</td>
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<tr>
<td>Contraindications</td>
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<td>-----------------------------------------</td>
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<tr>
<td>MELD &lt;15</td>
</tr>
<tr>
<td>AIDS</td>
</tr>
<tr>
<td>Anatomic abnormality</td>
</tr>
<tr>
<td>Non-compliance</td>
</tr>
</tbody>
</table>
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

Sclerotherapy and TIPS are contraindicated
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
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.