



Chapter 90 – Liver and Biliary Tract

Episode Overview:

- 1) List 8 differential diagnoses for hepatitis
- 2) Complete the following table for Hepatitis A, B & C: Transmission, Risk Factors, Carrier State, Acute Infection, Previous Infection, Chronic Infection, Previous Vaccines, Transmission Risk, Vaccination. What is hepatitis E? Where is it commonly found (geographically)? What is the significance of hepatitis D?
- 3) Describe the post-exposure prophylaxis for exposure to HepA, HepB, HepC
- 4) Compare the expected lab work in acute viral hepatitis vs alcoholic hepatitis
- 5) What liver diseases are associated with alcohol abuse? What non-hepatic conditions are associated with alcohol abuse? Describe the management of alcoholic hepatitis
- 6) List 6 stigmata of chronic liver disease and list 3 complications
- 7) How are chronic cirrhosis and ascites managed in the ER?
- 8) Describe a grading scale for hepatic encephalopathy and list 5 management considerations
- 9) Describe the ER diagnosis and management of SBP.
- 10) List 3 types of drug-induced liver disease.
- 11) What are two types of hepatic abscesses? How are they diagnosed and treated?
- 12) What is Budd-Chiari syndrome? How is it managed?
- 13) What is primary sclerosing cholangitis (PSC)? What is primary biliary cirrhosis? What is PSC associated with?
- 14) List 6 risk factors for cholelithiasis
- 15) Describe the clinical presentation of cholecystitis. List lab, X-ray (3) and US (4) findings
- 16) List 4 patients that get acalculous cholecystitis
- 17) List 4 considerations in the management of acute cholecystitis. When is surgery performed early?
- 18) What is the classic presentation of ascending cholangitis? What two clinical eponyms are described? How is ascending cholangitis managed?

Wisecracks:

- 1) Which conditions are associated with transaminases in the 1000's?
- 2) How do you approach a patient with a needlestick injury? What is the risk of transmission following a needlestick?
- 3) What are underlying causes of hepatic encephalopathy in patients with known liver disease?
- 4) What are the typical investigations performed on ascites fluid? What is the SAAG and how is it interpreted?
- 5) What is the significance of a calcified gallbladder?



1) List 8 differential diagnoses for hepatitis

The term hepatitis is general. Usually it refers to one of the TWO most common causes of hepatitis, which are:

1. **Viral hepatitis**
2. **Alcoholic hepatitis**

But there are other causes, including:

3. **Bacterial**
 4. **Fungal**
 5. **Parasitic**
 6. **Medication induced (e.g. tylenol)**
 7. **Nutritional / herbal supplements (e.g. mushrooms)**
 8. **Autoimmune**
- Most viral causes are from one of five viruses:
 - Hepatitis A = infectious
 - Hepatitis B = serum
 - Hepatitis C = post-transfusion / serum
 - Delta viruses
 - Epstein-Barr virus

Viral hepatitis can present in many ways, and many people can be asymptomatic.

- Look for:
 - Malaise, fever, anorexia, nausea, vomiting, diarrhea. Most people come to medical care once JAUNDICED (scleral icterus occurs first).

Fulminant hepatitis:

- Acute onset of hepatic failure with encephalopathy over days to weeks. Usually occurs with HBV-HDV co-infection.

Usually we can't find the precise cause of hepatitis in the ED, but tests should include:

- Transaminases, ALP, Bilirubin, Hepatic synthetic function (INR, Albumin), **Viral hepatitis serology**

2) Complete the following table for Hepatitis A, B & C: Transmission, Risk Factors, Carrier State, Acute Infection, Previous Infection, Chronic Infection, Prev Vaccine, Transmission Risk, Vaccine. What is hepatitis E? Where is it commonly found (geographically)? What is the significance of hepatitis D?

Ugh, this is painful, but it's a table you may need to know for your next quiz!



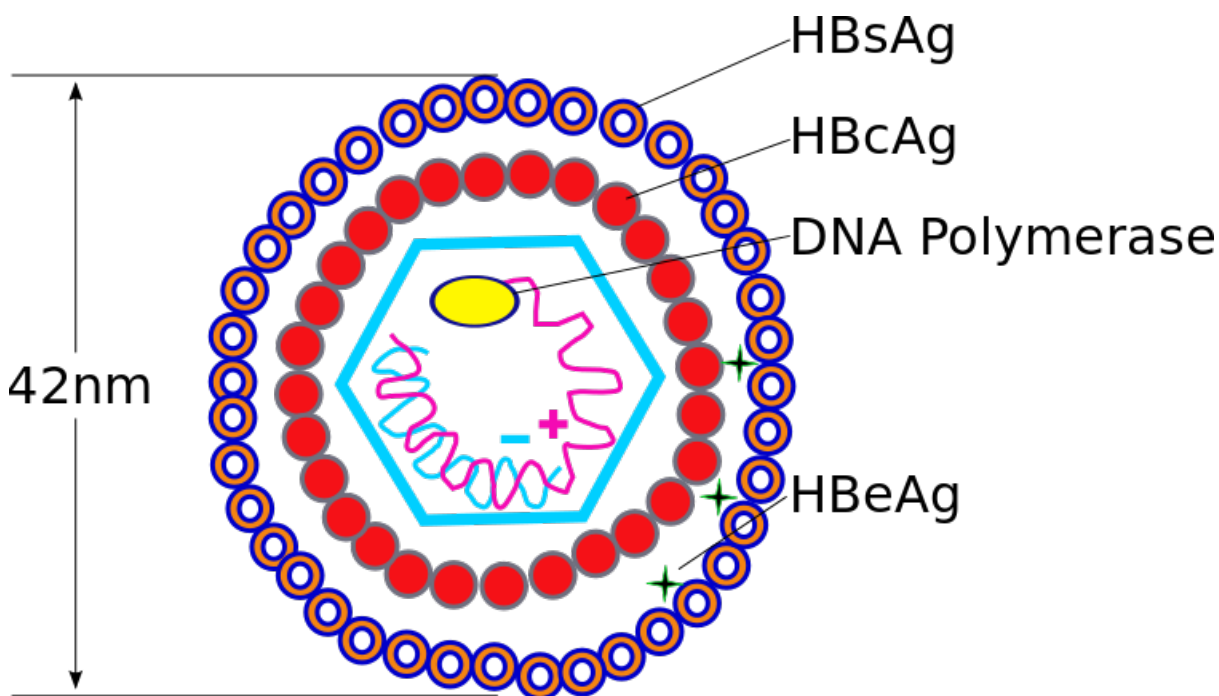
Ag = antigen, Ab = antibody

| | Hep. A | Hep. B* | Hep. C |
|---|--|---|---|
| Transmission | Fecal-Oral (water, food) | Parenteral; intimate contact | Parenteral; intimate contact |
| Risk factors | <u>Travel outside the USA</u> Children, MSM, IVDUs, Food handling / daycares | IVDU, homosexual men | Blood transfusions / organ transplant before '92 IVDU, > 20 lifetime sex partners, |
| Carrier state | NONE | 90% of neonates, and 10% of adults will become asymptomatic chronic carriers = HBsAg | Yes. |
| Acute infection (N/V/D symptoms, jaundice, ALT) | High ALT Anti-HAV IgM | High ALT HBsAg HBcAb IgM** HBeAg = HIGH infectivity | (symptomatic in 70% of cases) |
| Prev. infection | Anti-HAV IgG (may also have anti-HAV IgM) | HBsAb → HBsAb IgG Anti-HBcAg antibody (IgM/G)*** | HCV RNA (lower levels) |
| Chronic infection | NOT applicable! | HBsAg in serum; HBV DNA in serum, persistent ALT and AST elevation, liver biopsy + HBeAg (highly infective) | HCV RNA (higher levels) 90% of cases progress to chronic hepatitis. |
| Previous vaccine | Anti-HAV | anti-HBsAg antibody (prev. Vaccine or immunity to HBV) | NONE |
| Transmission risk | Fecal shedding and max. Infectivity occur BEFORE the onset of disease and/or jaundice Meticulous personal hygiene - no sharing anything in contact with blood/body fluids. [^] | 30% transmission risk from percutaneous exposure. 20-40% risk from sexual exposure. | Risk of seroconversion from HCV +ve source = 1.8% (percutaneous exposure) |
| Vaccine | Available | HB Vaccine. 1 ml. Three series of IM injections into the deltoid | None available |
| Misc. | Very common worldwide, 50% of americans are seropositive for HAV. 70% of people infected may be asymptomatic 15-45 day incubation period | Found in bodily secretions: saliva, semen, stool, tears, urine, vaginal secretions. Incubation is 60-90 days; serum is positive in 1-3 weeks. <i>Less than 5% of Hep B. infections in healthy adults will progress to chronic</i> | Leading cause of cirrhosis 30-90 day incubation period. Increasing in prevalence. |



| | | | |
|--|--|---|--|
| | | <p>hepatitis.</p> <p>HBeAb = resolving HBV infection</p> | |
|--|--|---|--|

^May not return to work while symptomatic and until jaundice is gone. *see figure below. **need both HBsAg AND HBVcAb-IgM (because HBsAg can be absent in late acute disease or chronically present). ***best marker of previous HBV infection



By Original uploader was TimVickers at en.wikipedia - Transferred from en.wikipedia, Public Domain,
<https://commons.wikimedia.org/w/index.php?curid=5670720>

Transmission:

B - loo - D

- Virus B, C and D are blood borne. And B always comes before HDV

What is hepatitis E? Where is it commonly found (geographically)?

- Fecal-oral transmission.
- Asia, Africa, Russia.

Hepatitis G:

- The most recently discovered virus. Blood-borne transmission. Manifests after co-infection with another virus.

What is the significance of hepatitis D?



- An infection that only occurs in people with Hepatitis B that actively produce HBsAg (chronic HBV infection).
- Superinfection of HBV-HDV can occur leading to fulminant hepatitis.

3) Describe the post-exposure prophylaxis for exposure to HepA, HepB, HepC

| | Post-exp. Prophylaxis | notes |
|--------------------------------|---|--|
| Hep A -- Rarely give it! | ISG, 0.02 ml/Kg IM HAV Immunoglobulin reserved for: <ul style="list-style-type: none"> • immune-naive pts. With increased risk from hepatitis • allergic to Hep. A vaccine. • Close personal contacts • Day care centre workers • Within 2 weeks of foodborne exposure | Healthcare, school contacts, workplace contacts, food-borne source workers not routinely immunized against HAV |
| Hep B ---- Give it prn! | Ideally give within 24 hrs. If not immediately available, give serum Ig within 7-14 days of exposure (HBIG) Treat (ideally) based on known source (HBsAg status) and exposee (anti-HBs titre) If unknown source and unvaccinated/unknown exposee = give HBIG | HBIG Indicated for people not immunized or with an unknown immune response to the Hep B vaccine (0.06 mL/kg IM) Combine with HBV vaccine series if unvaccinated or inadequate titre. If exposee anti-HBs titre is adequate, NO treatment is needed |
| Hep C -- Doesn't exist! | Unknown benefit from pre/post exposure prophylaxis | No vaccine is available ISG 0.06 mL/Kg can be considered |

Key is: universal precautions are the way to go!



4) Compare the expected lab work in acute viral hepatitis vs alcoholic hepatitis

Viral: $ALT > AST$ (both usually 10-100 x ULN), elevated bilirubin, elevated WBC, prolonged INR, fluid and electrolyte imbalances due to vomiting/diarrhea.

ETOH: $AST > ALT$ (values LESS than 10 times normal), elevated bilirubin, elevated WBC, prolonged INR, fluid and electrolyte imbalances due to vomiting/diarrhea.

(Stella > L)

Most people should have an ultrasound to rule out common bile duct obstruction, as well as anti-HAV IgM and HBcAb IgM.

5) What liver diseases are associated with alcohol abuse? What non-hepatic conditions are associated with alcohol abuse? Describe the management of alcoholic hepatitis.

Chronic alcohol use progresses to: steatosis (after 2 weeks), fibrosis, cirrhosis (after 5 yrs) and then can lead to hepatocellular carcinoma.

- Watch for:
 - GI bleeding
 - Hepatorenal syndrome
 - Sepsis
- Non-hepatic conditions:
 - Gastritis
 - Pancreatitis
 - Malnutrition
 - Polysubstance abuse

Management:

“FOR ALL”

- Advise to stop all ETOH use! (arrange addictions help)
- Supportive:
 - Replace fluid and electrolyte imbalances (check Mg, Phos, Ca!)
 - Magnesium should probably be given regardless of serum levels (unless the patient is in renal failure) due to alcohol induced Mg wasting.
 - 1-3 g IV
 - Antiemetics
 - HIGH Calorie, vitamin supplemented diet
 - Watch for hypoglycemia!
 - Replace thiamine - 100 mg IV

“Maybe’s”

- Treat gastritis with



- H2 blocker or PPI
- Aggressively manage any suspected variceal bleed
 - Octreotide / Somatostatin / Vasopressin
 - Endoscopy +/- TIPS / stent placement
- Start corticosteroids on anyone with an mDF score > 32
- Watch for infections (SBP)
 - Empiric ceftriaxone ?

6) List 6 stigmata of chronic liver disease and list 3 complications.

This stuff is all due to loss of hepatic metabolic or synthetic function → decreased portal vein blood flow → portal hypertension.

Stigmata

- Scleral / cutaneous icterus +/- pruritus
- Hepatomegaly
- Spider angiomata
- Caput medusa
- Patchy ecchymosis with thin skin
- Splenomegaly
- Gray / acholic stools
- Gynecomastia
- Muscle wasting
- Palmar erythema
- Dupuytren's contractures
- Testicular atrophy

Complications:

- Hepatocellular carcinoma
- GI bleeding (portal hypertension → variceal hemorrhage)
- Spontaneous bacterial peritonitis
- Encephalopathy
- Ascites

7) How is are chronic cirrhosis and ascites managed in the ER?

Cirrhosis is a generic term for ESLD (end stage chronic liver disease) - normal hepatocytes are replaced by fibrotic tissue. The cellular changes relate to the underlying cause. For example, infectious causes of hepatitis can lead to post-necrotic cirrhosis whereas cryptogenic cirrhosis occurs after NAFLD.

Common lab findings:

- Minimally elevated ALT/AST, increased INR, low albumin, anemia, thrombocytopenia, elevated BUN/creatinine.

ED management big picture: the three C's (See table 80.3 in Rosen's 9th Ed.)

1) Correct fluid/electrolyte abnormalities



2) Complications that need ER treatment:

a) Encephalopathy

b) GI bleeding

- Plts > 50k
- Fibrinogen > 100 mg/dL (use Cryoprecipitate)
- Vitamin K for bleeding (and elevated INR)
- Ceftriaxone 1 g IV daily until patient is taking food orally - this is for SBP prophylaxis!

c) Spontaneous bacterial peritonitis

- Paracentesis*
- Consider antibiotic prophylaxis if high risk for SBP recurrence
- Stoppage of PPI's and BB's

d) Hepatorenal syndrome

- Renal failure in the setting of cirrhosis, with no other renal pathology
- May need albumin infusions, midodrine, octreotide, IV norepinephrine

e) Consider replacing vitamin / nutrient deficits

- Thiamine
- Mg
- K

f) Ascites

- Occurs due to portal HTN, impaired lymphatic flow, hypoalbuminemia, and renal salt retention.
- Removed for symptomatic treatment
- Do not remove > 5 L without giving albumin
- Low salt diet is crucial
- Spironolactone +/- lasix for fluid balance
- Alcohol cessation
- Avoid ACEi/ARBs/NSAIDS
- Refractory ascites:
 - Midodrine
 - TIPS

8) Describe a grading scale for hepatic encephalopathy and list 5 management considerations

Hepatic encephalopathy refers to altered cerebral function because the liver can't do its normal metabolic functions. Ammonia (produced in the liver and normally converted to urea in the liver so it can be excreted) builds up and forms glutamine in the brain. **However, serum ammonia levels correlate inconsistently with the severity of encephalopathy.**



Hepatic encephalopathy exists on a spectrum: mild cognitive dysfunction → irritable → confusion → coma.

Clinical signs:

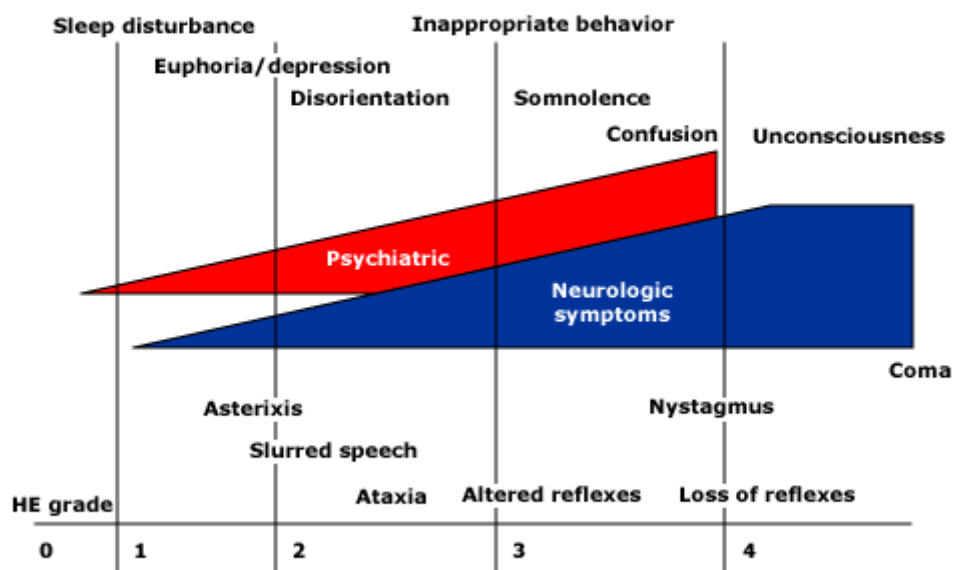
- Asterixis of the extended hands or feet or neck
- Fetor hepaticus
- Stigmata of chronic liver disease

“Hepatic encephalopathy is categorized based on four factors: the underlying disease, the severity of manifestations, the time course, and whether precipitating factors are present” (UpToDate)

Rosen’s doesn’t give us a table on encephalopathy grades in the 9th Ed. But here’s one from Uptodate and MDCalc.

©2017 UpToDate®

Evolution of hepatic encephalopathy



Graphic 58163 Version 1.0



Hepatic Encephalopathy Grades/Stages ☆

West Haven Criteria. Defines criteria for the stages of hepatic encephalopathy.

| | |
|---------------|-----------|
| When to Use ▼ | Why Use ▼ |
|---------------|-----------|

| | |
|----------------------|--|
| West Haven Criteria: | |
| Signs/Symptoms | Changes in behavior with minimal change in level of consciousness +1 |
| | Gross disorientation, drowsiness, possibly asterixis, inappropriate behavior +2 |
| | Marked confusion, incoherent speech, sleeping most of the time but arousable to vocal stimuli +3 |
| | Comatose, unresponsive to pain; decorticate or decerebrate posturing +4 |

See: <https://www.mdcalc.com/hepatic-encephalopathy-grades-stages>

Management considerations:

1. Resuscitate and intubate if unable to protect airway!
2. Blood products for GI bleeding
3. Antibiotics for sepsis/infection
4. Correct fluid / electrolyte imbalances:
 1. hypoK, hypoNa, hypoglycemia, etc.
5. Discontinue all CNS sedatives / drugs / toxins
6. Initiate lactulose 30-60 mL for four BM's a day for ammonia excretion
 1. Consider concurrent neomycin, vancomycin, rifaximin or metronidazole PO
 2. PEG3350 may also help
7. Complementary therapies:
 1. Lactobacillus probiotics, eradication of H. Pylori, Zinc replacement
8. High calorie diet, prevention of sarcopenia

9) Describe the ER diagnosis and management of Spontaneous Bacterial Peritonitis (SBP).

SBP is due to transmural migration of enteric organisms or from iatrogenic sources (E coli, Klebsiella, gram positive bacteria).

Clinical presentation can be subtle (especially with encephalopathy) or overt. ***need to consider the diagnosis of SBP in anyone with ascites + abdominal pain OR a patient with ascites and unexplained clinical deterioration.***

ED treatment should begin **after paracentesis** (don't forget to check the coags/plts first!):



If neutrophils (PMN's) > 250 cells/mm = ceftriaxone 2 g IV x 5 days. If PMN's < 250 cells/mm and infectious signs/symptoms = cefotaxime 2 g IV q8hrs x 5 days

Be aware that a patient with ESLD may also be on peritoneal dialysis, and develop secondary peritonitis from a UTI, GI or Lung infection. These patients may require *intrapertoneal* antibiotic administration.

Other ascitic labs may also help:

- Protein, LDH, glucose, CE antigen, ALP, gram stain and culture.
- Rural tip: ***may also use a urine leukocyte esterase test strip to screen the fluid; if it's positive there is a high likelihood that the ascitic fluid has a significant neutrophil count.***

Other positive findings:

- pH <7.34
- pH gradient between ABG and ascites fluid
- SAAG - serum ascites albumin fluid gradient > 1.1 g/dL (>11 g/L) can be an early indicator of SBP.
 - (subtract the albumin in the ascitic fluid, from the serum albumin level)
 - This can also be used to determine whether the ascites is from portal hypertension or not

10) List 3 types of drug-induced liver disease.

- **Cytotoxic** ("ALT")
 - Acetaminophen
 - Lovastatin
 - Tetracycline
- **Cholestatic** ("ALP")
 - Azathioprine
 - haLdoL
 - Phenobarbital
- **Veno-occlusive** ("AAA")
 - Anabolic steroids
 - Azathioprine
 - Anti-pregnancy (OCP's)

11) What are two types of hepatic abscesses? How are they diagnosed and treated?

Two main types:

1) Pyogenic

- Related to biliary tract obstruction, cholangitis, diverticulitis, appendicitis, bacteremia, pneumonia, or any other infection! (ie. they are secondary!)
- Can be any aerobic/anaerobic organism
- Clinical features: fevers, chills, RUQ pain, N/V
- Requires septic workup, liver panel labs.



- **CT with contrast is the test of choice.** U/S can be done to exclude biliary tract obstruction/cholangitis
- Management:
 1. Resuscitation
 2. Broad spectrum antibiotics for 2-6 weeks
 1. Ampicillin + gentamicin + metronidazole
 2. Cefotaxime + metronidazole
 3. Pip-tazo / imipenem / meropenem (may add Vancomycin)
 3. Pain control
 4. Abscesses > 3 cm may need drainage (usually image guided rather than open)

2) Amebic

- From protozoa via fecal-oral route from contaminated food/water
- Entamoeba histolytica is responsible for this secondary invasive disease
- Clinical findings: fever, chills, N/V, history of diarrheal illness in the past, may have a cough. (children commonly present with diarrhea!)
- Serum lab findings are non-specific related to infection and hepatic dysfunction; look for amoeba in the stool (ELISA protozoa in the stool); CXR for pulmonary involvement
 1. Ultrasound is usually adequate for diagnosis of round mass, with a homogenous hypoechoic centre. CT / MRI may be performed as well.
- Management:
 1. Supportive care
 2. Metronidazole (amebicidal) 750 mg PO / IV for 7-10 days TID
- RARELY need drainage
 - Often can be managed as outpatients

12) What is Budd-Chiari Syndrome? How is it managed?

Budd-Chiari Syndrome refers to hepatic vein outflow obstruction (not portal vein obstruction). Usually associated with disorders that promote excessive clotting, such as Factor V Leiden, Protein C/S deficiency, thrombophilia, antithrombin III deficiency, leukemia, Behcet's disease, OCP use, nocturnal hemoglobinuria. Can present with fulminant hepatic failure or insidious jaundice/ascites.

The key is to think of this diagnosis in the patient with liver dysfunction/failure. Doppler U/S of the hepatic vein is 85-95% sensitive.

Management:

- **Acute decompensation**
 - Consider anticoagulation (LMWH → bridged to Warfarin) if no varices present
 - Transjugular intrahepatic portosystemic shunt placement (TIPS)
 - Percutaneous angioplasty
 - Consider thrombolytic therapy
- **Chronic clot**
 - Diuretics / paracentesis
 - May need liver transplantation



13) What is primary sclerosing cholangitis (PSC)? What is primary biliary cirrhosis (PBC)? What is PSC associated with?

PSC: idiopathic inflammatory disorder of the biliary tree leading to diffuse fibrosis and narrowing of intra/extrahepatic bile ducts. Usually associated with **IBD (especially UC)**.

PBC - associated with other immune disorders. Granulomatous inflammation destroying intrahepatic bile ducts. May have signs or symptoms of scleroderma or CREST syndrome (calcinosis cutis, Raynaud's phenomenon, esophageal motility disorder, sclerodactyly, telangiectasia).

14) List 6 risk factors for cholelithiasis

Gallstones can be cholesterol or pigmented stones. Cholesterol stones form when there is an imbalance between cholesterol in the bile and other solubilizing constituents (bile acids, phospholipids, lecithin, etc.)

Risk factors for **cholesterol stones**:

- Age
- Female
- Massive obesity
- Rapid weight loss
- Cystic fibrosis
- Parity
- Drugs (clofibrate, OCPs)
- Familial tendency

Risk factors for **pigmented stones**:

- Black stones:
 - **Intravascular hemolytic diseases (sickle cell, spherocytosis)**
- Brown stones:
 - **Infections from**
 - **Ascaris lumbricoides**
 - **Clonorchis sinensis**

15) Describe the clinical presentation of cholecystitis. List laboratory, X-Ray (3) and US (4) findings

Clinical presentation:

- Constant, steady upper abdominal pain (cystic duct is obstructed!)
 - May radiate to back or shoulder
- Nausea and Vomiting
- Often similar self-limited recurrences in the past related to eating (biliary colic)
- Tenderness to the upper abdomen
- May have fever, tachycardia



Laboratory findings:

- No pathognomic lab tests are recognized
- Leukocytosis (as good as a coin toss - 50/50)
- ALT/AST may be normal or mildly elevated (rule out hepatitis!)
- Bilirubin / ALP normal (elevated if common duct obstruction)
- Lipase normal

X-Ray:

- Stones in the RUQ (usually the pigmented type if they contain >4% calcium and are visible on plain film)
- Pneumobilia (air in the biliary tree)
- Air in/around the gallbladder wall (emphysematous cholecystitis)
- Upper Quadrant sentinel loop (indicating localized ileus due to inflammation from gallbladder)

U/S findings (5 signs, 5 mm)

- **Sonographic Murphy's sign**
- **Gallbladder wall thickening (>5 mm)***
- **Gallbladder wall edema (double wall sign)**
- **Pericholecystic free fluid***
- **Gallstones***

***these three = 90% PPV for acute cholecystitis.**

16) List 4 patients that get acalculous cholecystitis

- **Elderly**
- **Admitted patients recovering from non-biliary tract surgery**
- **AIDS patients with secondary CMV or cryptosporidium infection**
- **Men, with uncontrolled diabetes (high risk for emphysematous cholecystitis as well)**

Other potential etiologies:

- Tumours / lymphadenopathy
- Fibrosis
- Parasitic disease

These people are sick! And this is a tough diagnosis to make! Imaging tests are less sensitive and specific, but you need to suspect it!

17) List 4 considerations in the management of acute cholecystitis. When is surgery performed early?

Important to exclude other entities in the differential diagnosis:

- Hepatitis
- Hepatic abscess



- Pyelonephritis
- RLL pneumonia
- Pancreatitis
- PUD of the duodenum +/- perforation
- Appendicitis
- Coronary artery disease - unstable angina

Management:

- Optimize volume status
 - Replace electrolytes
 - Treat with antiemetics
- Pain control
 - NSAIDS, narcotics, NG suctioning
- Antibiotics
 - Ancef + Flagyl
 - Pip-tazo if septic
- Consult for surgical treatment
 - Endoscopy if there is a common bile duct stone!

Early surgery for suspected emphysematous cholecystitis (especially with perforation!)

These patients are at high risk for gangrene and perforation. Should get ceftriaxone 2 g IV and metronidazole 500 mg IV OR Pip-tazo OR meropenem

Simple cholecystitis admitted for:

- Unremitting pain (look for fever, leukocytosis, sick)
- Intolerable PO intake
- Electrolyte imbalances
- Diagnosis of gallstone obstruction
- Antibiotics
- Surgery

18) What is the classic presentation of ascending cholangitis? What two clinical eponyms are described? How is ascending cholangitis managed?

Ascending Cholangitis usually occurs after sustained blockage of the common bile duct (gallstone, malignancy, stricture). Bacteria then propagate (from the duodenum) and invade the surrounding tissues. Usually E coli, klebsiella, enterococcus, bacteroides.

Presentation:

- Fevers, chills, nausea, vomiting, abdominal pain

Charcot's Triad: RUQ pain, fever, jaundice (also applies to hepatitis and cholecystitis!)



Reynold's Pentad: Charcot's Triad + hypotension and altered mental status

Ultrasound in cholangitis shows dilated common bile ducts/intrahepatic ducts (unlike cholecystitis which should have normal ducts).

CT may be preferred imaging for emphysematous cholecystitis or hemorrhagic cholecystitis.

Management:

- Resuscitation
 - Fluids, lytes, vasopressors!
- Broad spectrum ABx:
 - Pip-tazo 3.375 g IV QID
 - Ceftriaxone 1 g IV and Metronidazole 500 mg IV
 - Cipro + Flagyl
 - Moxifloxacin + Flagyl
 - Carbapenem
- **Biliary tract decompression (Surgery, GI, or interventional RAD!)**
 - Surgery
 - Percutaneous transhepatic cholangiography (THC)
 - Endoscopic retrograde cholangiopancreatography/scopy (ERCP)
 - Cultures, stone retrieval, sphincterotomy, stent placement

AIDS CHOLANGIOPATHY

- CD4 count < 200/mm
- Bile duct strictures, papillary stenosis, PSC

Wisecracks

1) Which conditions are associated with transaminases in the 1000s?

- Acute Viral Hepatitis (HAV, HBV, HDV co-infection, but NOT HCV)
- Drug/Toxin Induced Liver Injury (Tylenol, herbs, supplements, chemical ingestions)
- Ischemia (Shock Liver)
- Acute Budd-Chiari
- HELLP
- Autoimmune Hepatitis

2) How do you approach a patient with a needlestick injury? What is the risk of transmission following a needlestick?

Ok, this is a super common problem. Your hospital will probably have a protocol all set up already. But here are some key questions:

- **Who is involved?**
 - **Source**
 - **Exposed (i.e. the patient you're seeing in the ER!)**



- **What happened?**
 - Sex trade work, abandoned needle, knife blade, face splash, etc.
- **Where did it happen?**
 - Occupational vs. non-occupational
- **When did it happen?**
 - If > 72 hrs HIV prophylaxis won't help the exposed!

Really there are a huge number of diseases that can be spread via percutaneous exposure.

- Blastomycosis
- Brucellosis
- Cryptococcus
- Diphtheria
- Cutaneous gonorrhea.
- Herpes
- Malaria
- Mycobacterial infections
- Mycoplasma caviae
- Rocky Mountain spotted fever
- Sporotrichosis
- Staphylococcus aureus
- Streptococcus pyogenes
- Syphilis
- Toxoplasmosis
- Tuberculosis

However, we really care about three in the ED: HIV, Hep B, Hep C.

Steps:

- Initiate basic wound care (if not already done). Wash with water and mild soap thoroughly!
- Have baseline “exposed” labs drawn per protocol
 - If possible get source labs tested
 - **The only real actionable lab on the day of exposure is a source with positive rapid HIV test.**
- Figure out if there is any actionable treatment that needs to be given to the source
 - HIV PEP
 - If NOT possible, do your best detective work (“if the source's HIV status is unknown at the time of the exposure, we suggest post-exposure prophylaxis if the source is at high risk for having HIV (eg, man who has sex with men [MSM], injection drug user, sex worker), or if the patient has been sexually assaulted (Grade 2C).”) - Uptodate2017.
 - Get baseline labs as well while starting HIV PEP: CBC, C7, LFTs, Pregnancy test
- Determine the source and exposed patient's Hep B status.
 - If exposed is immune - no need to act
 - “Patients who were previously vaccinated and are known to have responded to vaccine (ie, a post-vaccination anti-HBs ≥ 10 mIU/mL), regardless of when they were vaccinated.) For such patients, testing to confirm the continued presence of antibody is not needed as



- vaccine responders should have lifelong protection even if their antibody level has subsequently declined.” Uptodate2017
- If exposed has unknown immune status/non-immune consider HEP B PEP:
 - “The use of post-exposure prophylaxis with hepatitis B vaccine and/or HBIG can reduce HBV transmission by 70 to 90 percent after an exposure to a patient with chronic HBV when administered within 12 to 24 hours of an exposure. “ Uptodate 2017
 - **“Post-exposure prophylaxis should be administered to patients without previously documented HBV immunity who are exposed to blood or body fluids from a source who is HBsAg-positive or whose HBV status is unknown (ie. HBV-unknown)” Uptodate 2017**
 - Determine the risk for Hep C exposure.
 - “There are no medications or immunizations to reduce the risk of acquiring HCV after a possible exposure. Thus, post-exposure management depends upon close monitoring and referral for treatment if infection does occur. We perform HCV testing in patients who have had a percutaneous exposure or a high-risk sexual exposure (eg, condomless receptive anal intercourse, sexual assault).” - Uptodate 2017
 - Call your local public/occupational health personnel for for help!

Risks of transmission of...if the source is positive!:

- HIV: ~0.3%
- Hep C: ~3%
- Hep B: ~30%



Post-exposure prophylaxis to hepatitis B virus after a nonoccupational exposure

| Vaccination and/or antibody response status of exposed person* | Treatment when source patient is: | |
|---|--|-----------------------------|
| | HBsAg-positive/source unknown or not available for testing | HBsAg-negative† |
| Unvaccinated/nonimmune | HBIG ^Δ × 1; initiate HBV vaccine series | Initiate HBV vaccine series |
| Previously vaccinated [◊] , known responder [§] | No treatment | No treatment |
| Previously vaccinated [◊] , known nonresponder [§] | HBIG ^Δ × 1 and initiate revaccination [×] or HBIG ^Δ × 2 | No treatment |
| Previously vaccinated [◊] , antibody response unknown [‡] | Single vaccine booster dose | No treatment |
| If still undergoing vaccination | HBIG ^Δ × 1; complete series | Complete series |

anti-HBc: antibody to hepatitis B core antigen; anti-HBs: antibody to hepatitis B surface antigen; HBsAg: hepatitis B surface antigen; HBIG: hepatitis B immune globulin; HBV: hepatitis B virus.
 * Persons who have previously been infected with HBV are immune to reinfection and do not require post-exposure prophylaxis.
 † A source is considered HBsAg-negative if at the time of exposure they are known to have either of the following: anti-HBs ≥10 milli-international units/mL after immunization or resolved HBV (anti-HBs-positive and anti-HBc-positive).
 Δ For specific recommendations on dosing and administration of HBIG, refer to the topic that discusses post-exposure prophylaxis after a nonoccupational exposure to HBV and the drug information topic within UpToDate.
 ◊ Vaccinated with full three-dose series.
 § Based on information available at presentation. Responder is defined as person with previously documented adequate levels of serum antibody to HBsAg (serum anti-HBs ≥10 milli-international units/mL); nonresponder is a person with previously documented inadequate response to vaccination (serum anti HBs <10 milli-international units/mL). It is recommended that decision-making not be delayed while testing for anti-HBs at presentation.
 × The option of giving one dose of HBIG and reinitiating the vaccine series is preferred for nonresponders who have not completed a second three-dose vaccine series. For persons who previously completed a second vaccine series but failed to respond, two doses of HBIG are preferred.
 ‡ The need for additional prophylaxis (eg, hepatitis B vaccine and HBIG) depends upon serologic testing performed at the time of the initial evaluation. Refer to the UpToDate topic that discusses post-exposure prophylaxis for HBV.

References:

1. HIV Clinical Resource. UPDATE: HIV prophylaxis following non-occupational exposure. Available at: <http://www.hivguidelines.org/clinical-guidelines/post-exposure-prophylaxis/hiv-prophylaxis-following-non-occupational-exposure/> (Accessed on February 2, 2016). Copyright © 2014 New York State Department of Health AIDS Institute's Clinical Guidelines Development Program.
2. CDC Guidance for Evaluating Health-Care Personnel for Hepatitis B Virus Protection and for Administering Postexposure Management. *MMWR Recomm Rep* 2013; 62:1.
3. Workowski KA, Bolan GA, Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep* 2015; 64:1.

UpToDate®

3) What are underlying causes of hepatic encephalopathy in patients with known liver disease?

This is directly from Box 80.1! Remember just like anemia isn't a diagnosis, encephalopathy is just a syndrome that needs an underlying cause!

Underlying causes of hepatic encephalopathy in patients with known liver disease

Acute:

- GI bleeding (upper / lower leading to hypovolemia or increased ammonia levels)



- Infection
- Venous thrombosis (hepatic / portal vein).

Metabolic:

- Electrolyte imbalance (*hypokalemia, metabolic alkalosis)
- Ileus / constipation
- Sedative medications causing elevated drug toxicity (increased volume of distribution)
 - Phenytoin, antibiotics, tylenol, narcotics, benzo's, ETOH
- Dehydration / hypovolemia (V/D/diuretics)
- Acute / chronic kidney injury
- Primary HCC

All of these diagnoses fall into these broad categories of things that worsen encephalopathy:

- Drugs
- Increased ammonia production, absorption, entry into the brain
- Dehydration
- Portosystemic shunting
- Vascular occlusion
- Primary HCC

4) What are the typical investigations performed on ascites fluid? What is the serum ascites albumin gradient (SAAG) and how is it interpreted?

“The SAAG is easily calculated by subtracting the ascitic fluid albumin value from the serum albumin value, which should be obtained the same day. The SAAG generally does not need to be repeated after the initial measurement.

- The presence of a gradient ≥ 1.1 g/dL (≥ 11 g/L) predicts that the patient has portal hypertension with 97 percent accuracy.
- A gradient < 1.1 g/dL (< 11 g/L) indicates that the patient does not have portal hypertension.

The SAAG will be elevated with any disorder leading to portal hypertension and is not specific to ascites due to cirrhosis. Other testing may be needed to differentiate cirrhotic from noncirrhotic portal hypertension. Additional testing will depend upon the clinical setting and may include an evaluation for heart failure, hepatic metastases, or Budd-Chiari syndrome.” - From Uptodate 2017.

5) What is the significance of a calcified gallbladder?

Porcelain gallbladder, most often in elderly women. May be palpable, but isn't usually tender. Most should be referred for surgical removal due to the high incidence of associated cholangiocarcinoma.

This post was uploaded and copyedited by **Andrew Guy** (@andrew_s_guy)

CrackCast Show Notes – Liver and Biliary Tract – July 2017
www.canadiem.org/crackcast

