



BE A LIVER, NOT A HATER

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MAYO CLINIC ARIZONA

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DISCLOSURES

I have no relevant relationships to disclose.





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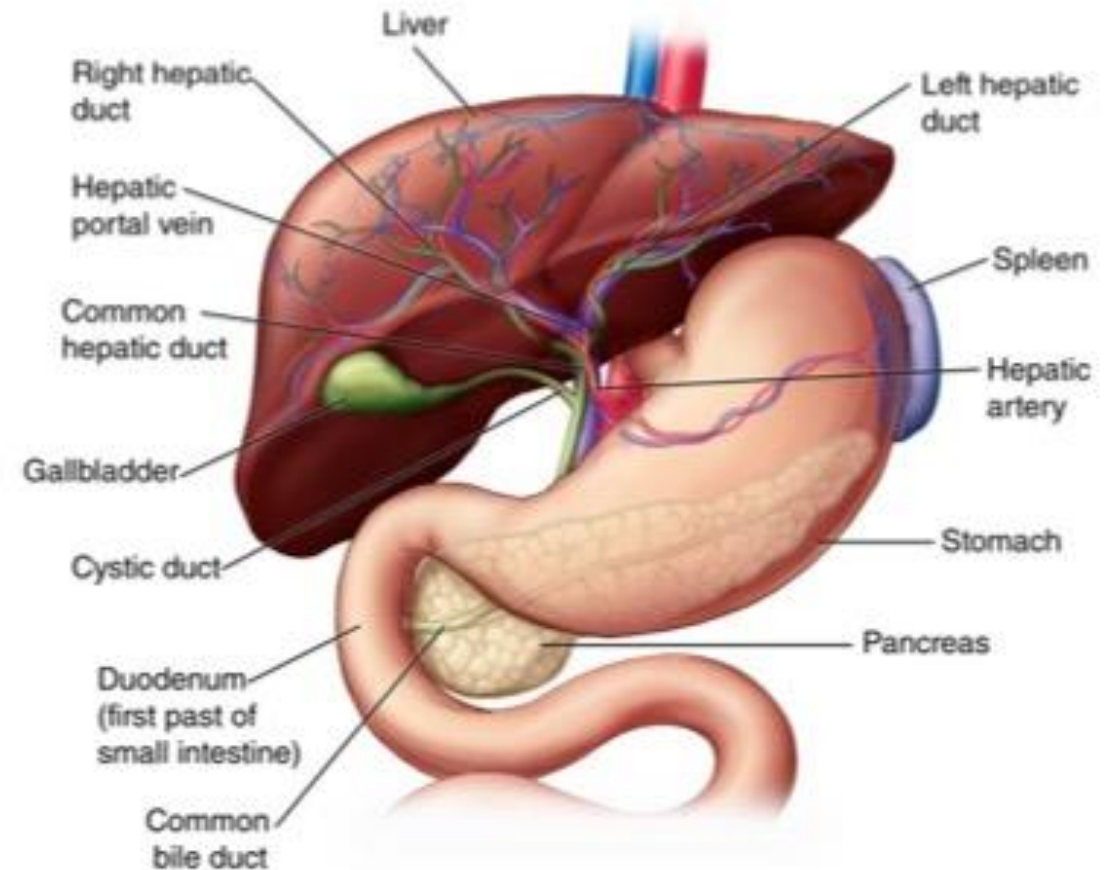
THE GOAL OF THIS SESSION IS TO PROVIDE A GENERALIZED KNOWLEDGE BASE REGARDING HEPATIC AND HEPATOBILIARY DISORDERS TO BE ABLE TO EFFECTIVELY EVALUATE AND TREAT PATIENTS.

LEARNING OBJECTIVES

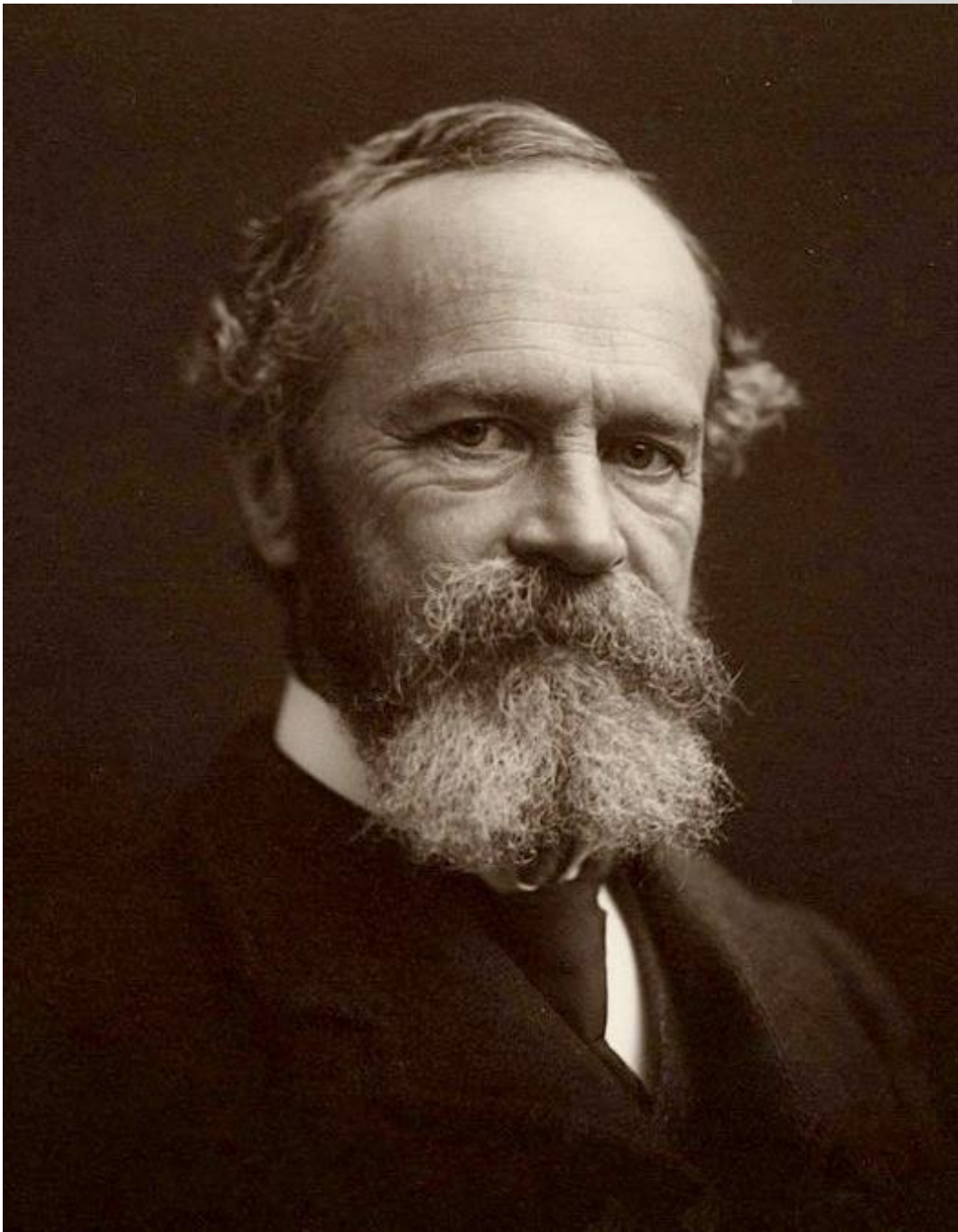
- Describe common hepatobiliary disease processes that are seen inpatient
- Initiate evaluation of common hepatic disorders
- Know when to refer

FUNCTIONS OF THE LIVER

- + Production of bile to break down fats in the small intestine
- + Production of proteins for blood plasma
- + Production of cholesterol
- + Conversion of excess glucose into glycogen for storage
- + Regulation of blood levels of amino acids
- + Storage of iron
- + Conversion of poisonous ammonia to urea
- + Clears the blood of drugs and other poisonous substances
- + Regulates blood clotting
- + Clearance of bilirubin, also from RBCs



https://www.hopkinsmedicine.org/-/media/images/health/1_-conditions/liver-gallbladder-and-pancreas/liver-anatomy.ashx



“Is life worth living? It all depends on the liver.”

- *William James*

SYNTHETIC DYSFUNCTION VS INJURY

UNDERSTANDING LIVER CHEMISTRIES

- Liver chemistries can be separated into those that reflect:
 - Hepatocyte injury
 - Biliary epithelium injury
 - Hepatic synthetic dysfunction
- It is rare for there to be significant hepatocyte injury without some degree of biliary injury (and vice versa)
 - Establish the predominant pattern

DESCRIBING THE LIVER INJURY PATTERN...



- Hepatocellular pattern:
 - Disproportionate elevation in the serum aminotransferases compared with the alkaline phosphatase
 - Serum bilirubin may be elevated
 - Tests of synthetic function may be abnormal
- Cholestatic pattern:
 - Disproportionate elevation in the alkaline phosphatase compared with the serum aminotransferases
 - Serum bilirubin may be elevated
 - Tests of synthetic function may be abnormal
- Mixed pattern

EXAMPLE 1

HEPATOCELLULAR OR CHOLESTATIC?

Lab Result	Value
Bilirubin, Total, S	3.8
Bilirubin, Direct, S	2.9
Alanine Aminotransferase (ALT), S	40
Aspartate Aminotransferase (AST), S	35
Alkaline Phosphatase, S	464

EXAMPLE 2

HEPATOCELLULAR OR CHOLESTATIC?

Lab Result	Value
Bilirubin, Total, S	1.1
Bilirubin, Direct, S	0.2
Alanine Aminotransferase (ALT), S	210
Aspartate Aminotransferase (AST), S	86
Alkaline Phosphatase, S	120

CAUSES OF LIVER INJURY (HEPATITIS)...



*What to
do with
elevated
LFTs?!*

COMMON CAUSES OF ACUTE TRANSAMINITIS (ACUTE HEPATITIS)

Disease	Clinical Clues	Diagnosis
Hepatitis A	Exposure Hx	Anti-HAV IgM
Hepatitis B	Risk factors	HBsAg, HBcAb IgM, HBV DNA
Drugs	Hepatotoxic med and timing	Improves after d/c med
Alcoholic Hepatitis	AST:ALT > 2, EtOH excess	Clinical, improves with abstinence
Ischemic Hepatitis (i.e. "shock liver")	Hx ↓BP, transient ↑↑ ALT	Clinical
CBD stone	Pain, ↑ amylase, transient ↑ ALT/AST (usually mixed pattern)	US, ERCP, MRCP



COMMON CAUSES OF CHRONIC HEPATITIS

Disease	Clinical Clues	Diagnosis
Hepatitis C	Risk factors	Anti-HCV, HCV RNA
Hepatitis B	Risk factors	HBsAg, HBcAb, HBV DNA
Nonalcoholic fatty liver disease (NAFLD)	Metabolic risk factors (obesity, DM, HLD)	U/S, MRE, Fibroscan, Liver Bx
Hemochromatosis/Wilson's Dz	Family Hx, Iron Studies/Kayser–Fleischer rings, High bili low alk phos	Iron studies, HFE gene test, liver Bx/ceruloplasmin, 24 hr copper, liver Bx
Alcoholic Liver Dz	Hx EtOH excess	Bx, improvement with abstinence
Autoimmune hepatitis	ALT 200-500, female, co-morbid autoimmune dz	ANA, AMA, ASMA, liver Bx

CLINICAL PEARLS: HEPATOCELLULAR ENZYMES

- Low levels of AST/ALT elevation (less than 2x ULN) are very non-specific.
- AST/ALT 2:1 -> EtOH?
- Enzymes usually only > 1000 in:
 - DILI, most commonly acetaminophen toxicity
 - Ischemia/shock liver (ischemic hepatitis)
 - Acute viral hepatitis (don't forget HSV, A-E)
 - Autoimmune hepatitis
 - Acute Budd-Chiari syndrome
- Even severe alcoholic injury should not cause AST/ALT greater than 300



COMMON CAUSES OF CHOLESTASIS

Disease	Clinical Clues	Keys to Diagnosis
PBC	Middle-aged female (F>M 9:1)	AMA, Bx
PSC	UC, Male>Female	P-ANCA, ANA, anti-SMA, ERCP, Bx
Large duct obstruction	Jaundice and/or pain with/without fever (Charcot's Triad)	U/S, CT, MRCP, ERCP, PTC
Drug-induced	Compatible agent and timing	Improvement after d/c agent
Infiltrative disease	Hx suggestive of sarcoid, amyloid, Ca	U/S, CT, biopsy
Inflammation-associated/ Cholangitis lenta	Sx of underlying inflammatory disorder	Blood Cx, antibody tests

CHOLEDOCHOLITHIASIS

- Consider when abdominal pain and transient abnormality of ALT/AST
- In acute obstruction, bile ducts may not be dilated on imaging
 - Also, U/S only 50% sensitive for detecting choledocholithiasis
- Diagnosis: MRCP, ERCP
- For sick patients, best to proceed with the most definitive test, also offers chance to provide therapeutic intervention
- Cholecystectomy should be deferred until patient stable

63 y.o. female pt with abd pain, fever, and nausea x 4 hrs.

Exam: febrile to 39.0 C, jaundice and mild epigastric tenderness.

Notable labs: WBC 18,000 with left shift, total bili 3.6 (primarily direct), ALP 150, AST 745, and ALT 650.

US: Multiple small stones in GB, *no biliary ductal dilatation*, normal pancreas.

Started on Abx for medical management but following day still febrile (despite IV Zosyn). Repeat lab: bili 5.8, AST 684 and WBC 25,000. 2 of 2 initial blood cultures pos for E. coli; rpt bl Cx NGTD (on Zosyn).

Which of the following would you advise next?

- 1. Doppler of hepatic vessels
- 2. Laparoscopic cholecystectomy
- 3. Magnetic resonance cholangiopancreatography
- 4. Endoscopic ultrasound
- 5. ERCP

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- 2. Laparoscopic cholecystectomy
- 3. Magnetic resonance cholangiopancreatography
- 4. Endoscopic ultrasound
- 5. *ERCP*



53 y.o. asymptomatic woman sent because of abnormal ALT. Never had liver tests assessed in the past. Hx notable for migraines for which she takes sumatriptan and acetaminophen prn. No risk factors for or Hx of liver disease.

Exam is notable for obesity. AST and ALT are 75. ALP, bilirubin, INR, iron studies, and albumin are normal.

What would be the best next step?

1. Observe and repeat ALT in 3 months
2. Liver biopsy
3. Stop sumatriptan
4. Hepatitis B surface antigen
5. Abdominal CT

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57 y.o. asymptomatic woman sent because of a three yr history of abnormal liver tests. No risks factors or Fam Hx of, liver disease. Med: lisinopril. Exam: BMI 22 kg/m².

Lab: Hgb 13.1, plts 187, AST 84, ALT 86, ALP 56, bili 0.8, chol 245.

Neg or WNL: US, iron, CK, TSH, AMA, ANA, TTG, HBsAg, anti-HCV, A1AT phenotype.

Which of the following would you advise at this point?

1. Liver biopsy
2. Ceruloplasmin
3. Annual liver tests
4. Lipid-lowering agent

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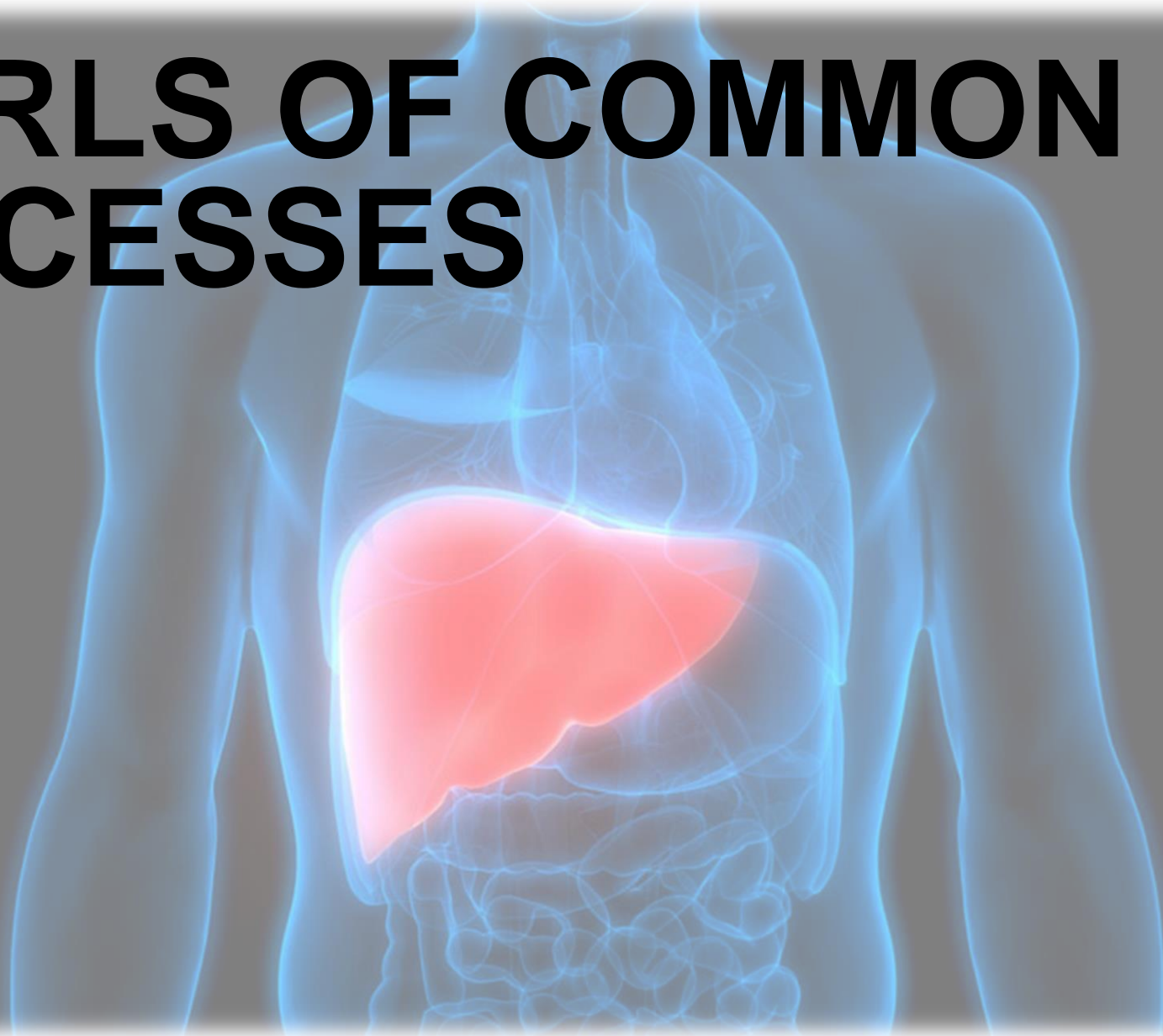
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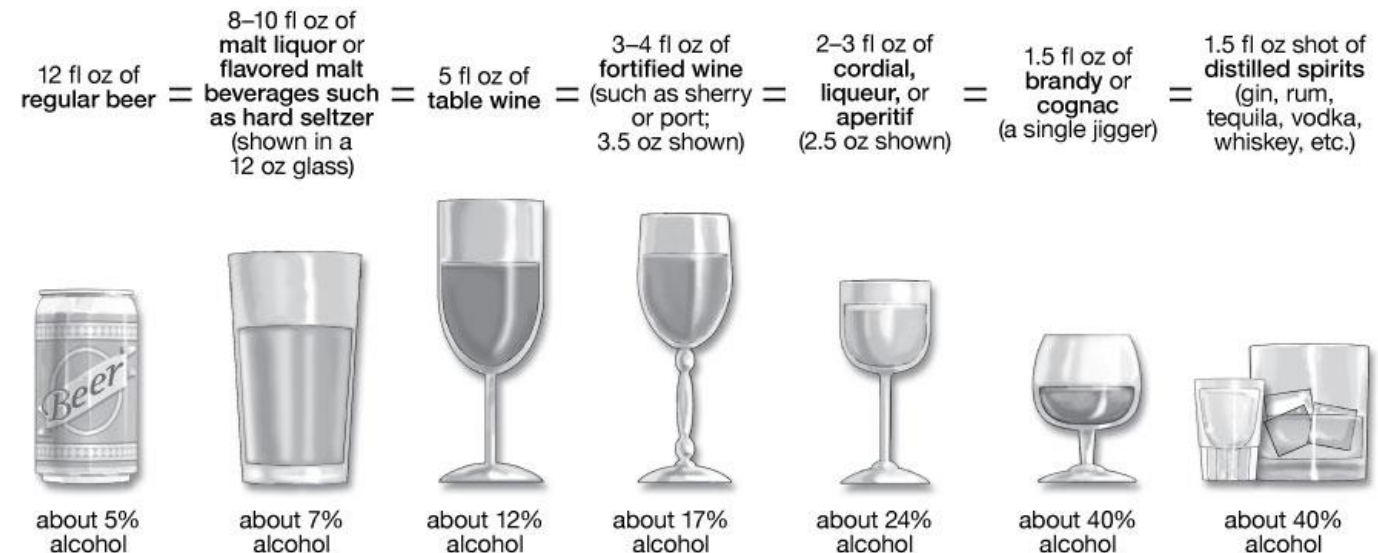
1. *Liver biopsy*
2. Ceruloplasmin
3. Annual liver tests
4. Lipid-lowering agent

PEARLS OF COMMON DZ PROCESSES



ALCOHOL-RELATED LIVER DISEASE

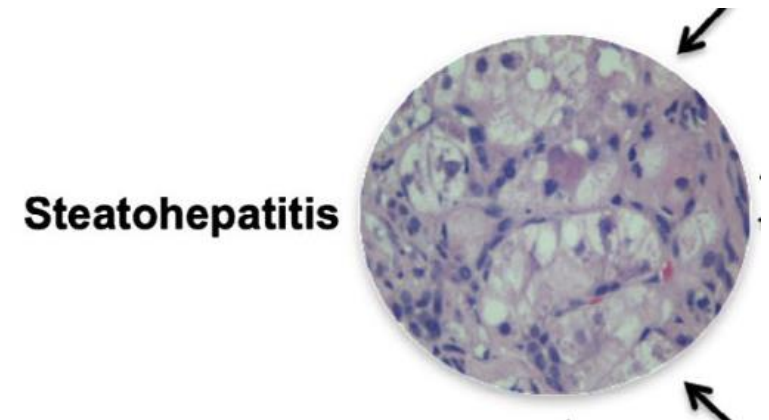
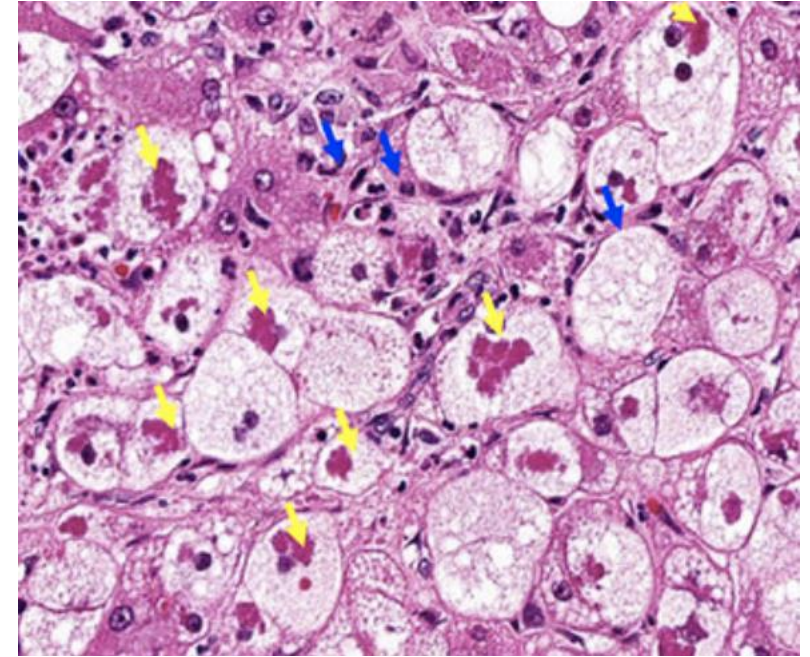
- Ranges from hepatic steatosis to more advanced forms including alcoholic hepatitis (steatohepatitis), alcohol-associated cirrhosis (AC), and acute AH presenting as acute-on-chronic liver failure
- Liver damage at lower doses with other underlying liver disease (i.e. HCV, NAFLD)
- Need to be clear as to “dose” of alcohol used
- A drink is a drink is a drink
 - Beware of “just beer”
- Liver disease associated with:
 - 1-2 drinks/day for women
 - 3-4 drinks/day for men



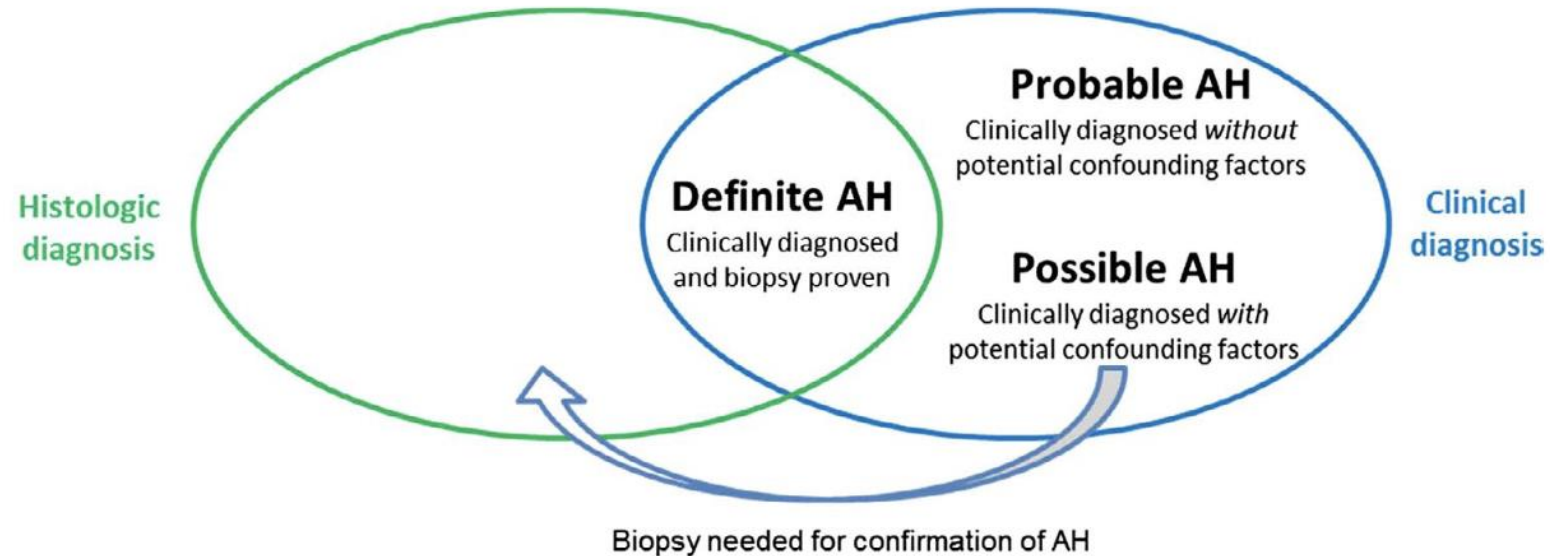
Each drink shown above represents one U.S. standard drink and has an equivalent amount (0.6 fluid ounces) of “pure” ethanol.

ALCOHOL-RELATED HEPATITIS

- Rise in incidence during the COVID 19 pandemic
 - Likely related to rise in AUD
- Dx: pathology
 - Macrovesicular steatosis, lobular infiltration of neutrophils, ballooning hepatocytes that often contain eosinophilic inclusions called Mallory-Denk bodies, and ductular reaction
- Mostly a clinical diagnosis
- Biomarkers, history, r/o other confounding factors
- Treatment



Crabb, D.W., Im, G.Y., Szabo, G., Mellinger, J.L. and Lucey, M.R. (2020), Diagnosis and Treatment of Alcohol-Associated Liver Diseases: 2019 Practice Guidance From the American Association for the Study of Liver Diseases. *Hepatology*, 71: 306-333. <https://doi.org/10.1002/hep.30866>



Clinical diagnosis of AH

- Onset of jaundice within prior 8 weeks
- Ongoing consumption of >40 (female) or 60 (male) g alcohol/day for ≥6 months, with <60 days of abstinence before the onset of jaundice
- AST >50, AST/ALT >1.5, and both values <400 IU/L
- Serum total bilirubin >3.0 mg/dL

Potential confounding factors

- Possible ischemic hepatitis (e.g., severe upper gastrointestinal bleed, hypotension, or cocaine use within 7 days) or metabolic liver disease (Wilson disease, alpha 1 antitrypsin deficiency)
- Possible drug-induced liver disease (suspect drug within 30 days of onset of jaundice)
- Uncertain alcohol use assessment (e.g., patient denies excessive alcohol use)
- Presence of atypical laboratory tests (e.g., AST <50 or >400 IU/L, AST/ALT <1.5), ANA >1:160 or SMA >1:80.

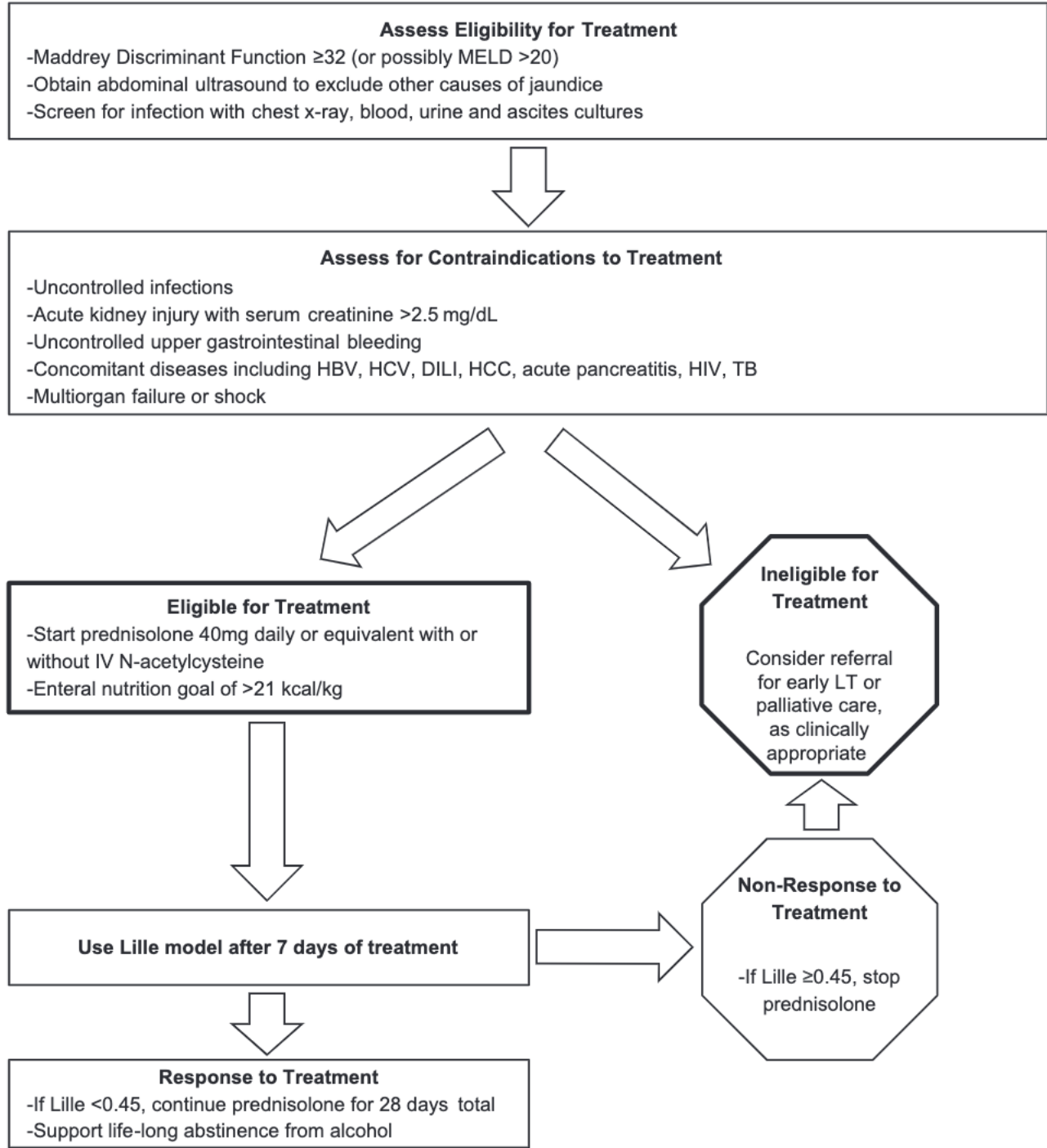
LAB BASED PROGNOSTIC SCORES

- **Maddrey's Discriminant Function (MDF)**
 - $DF = 4.6 * (Pt's \text{PT} - \text{control PT}) + \text{Tbili}$
- MDF > 32 typically suggests poor prognosis and may benefit from corticosteroids
- MDF < 32 can identify mild-mod AH, conferring low (not zero) risk of mortality with supportive care
- Ability to discriminate pts who *may* have a survival benefit from corticosteroids



LAB BASED PROGNOSTIC SCORES

- Predicts 6 mo mortality in pts with AH not responding to steroid therapy (non-responders)
- **Lille Score**
 - Age, albumin, initial bili, day 7 bili, Cr, PT
- >0.45 predicts a 6 mo survival of 25% (d/c steroids)
- <0.45 predicts a 6 mo survival of 85%
- Risk stratifies pts already receiving steroids for AH to predict who will not improve and should be considered for other management strategies
- Supportive measures
 - Prevent infection
 - Nutrition/protein
 - **ABSTINENCE!**



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NAFLD

- Common cause of liver disease in the U.S.
- Most common cause of abnormal liver tests, usually mild elevation of ALT and AST
- Commonly associated with obesity, DM, and/or dyslipidemia (metabolic syndrome)
- Other associated co-morbidities: PCOS, hypothyroidism, OSA, hypopituitarism, hypogonadism
- Prevalence 2x higher in men male symbol
- Diagnosis: Evidence of steatosis on either imaging or histology
 - Ultrasound (change in echotexture), Fibroscan, MRE
- Prognosis: variable, < 20% progress to cirrhosis
- Treatment: control risk factors/lifestyle modifications; SGLT2i; bariatric surgery; FIB-4/Fibroscan monitoring; *statins are okay!*
 - *Weight loss of at least 3%-5% of body weight appears necessary to improve steatosis, but a greater weight loss (7%-10%) is needed to improve the majority of the histopathological features of NASH, including fibrosis.*

45 y.o. woman presents to ED with jaundice for two weeks. She has also lost 10 lb but was previously well. Drinks one glass of wine each day; two or three glasses on weekends. Has taken two 500 mg Tylenol tabs daily over the last 2 weeks.

Exam: 37.8 C, HR 100, BP 100/65, icteric sclera, and tender hepatomegaly. Alert and oriented without asterixis.

Lab: Hgb 12.5, MCV 108, plt 120,000, WBC 14,900 with left shift, AST 125, ALT 59, ALP 290, total bili 22.8, and INR 1.5. ANA, viral serologies, ceruloplasmin are all normal.

US: GB sludge, coarse liver echotexture, no ascites, no bile duct dilatation.

What is the most likely diagnosis?

- A. Celiac disease
- B. Alcoholic hepatitis
- C. Nonalcoholic fatty liver disease
- D. Autoimmune hepatitis
- E. Acetaminophen hepatotoxicity

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HEPATITIS A VIRUS

- Causes acute but not chronic hepatitis
- Transmitted by fecal-oral route
- High-risk groups: travelers, day care attendees, MSM, IVDU
- Incubation period 2-6 weeks
- Diagnosis: IgM anti-HAV (IgG anti-HAV indicates immunity)
- Treatment: supportive, monitoring

HEPATITIS B SEROLOGIES

- Hepatitis B surface antigen (HBsAg): current infection
 - Different from active viremia (HBV DNA)
- Antibody to hepatitis B surface antigen (anti-HBs, HBsAb): immunity
- HBcAb: think prior infection
 - IgM antibody to hepatitis B core protein (IgM anti-HBc): recent infection or “flare” of chronic HBV
 - IgG anti-HBc: remote infection
- Hepatitis B e antigen (HBeAg): high infectivity (active viral replication)
- (HBV DNA = viral load/active viremia)

Interpretation of Hepatitis B Serologic Test Results

HBsAg	Anti-HBc	IgM Anti-HBc	Anti-HBs	Interpretation
-	-	-	-	Susceptible to HBV infection
-	+	-	+	Immune due to natural hepatitis B infection
-	-	-	+	Immune due to hepatitis B vaccination
+	+	+	-	Acute HBV
+	+	-	-	Chronic hepatitis B infection
-	+	-	-	Interpretation unclear; four possibilities: 1. Resolved infection (most common) 2. False-positive anti-HBc, thus susceptible 3. "Low level" chronic infection 4. Resolving acute infection

HEPATITIS B – ACUTE

- Risk factors: high risk sexual behavior, injection drug use, birth in high endemic areas of Asia and Africa
 - Incubation period: 30-180 days
 - Prognosis after acute infection: 95% of adults develop immunity; 5% remain chronically infected
- *Important to screen for co-infection: HCV, HIV, delta virus
- *Important to monitor closely/assess for fulminant liver failure (?H.E.)

HEPATITIS B – CHRONIC

- Definition: HBsAg for > 6 months
- Risk factors for chronic infection: neonatal or early childhood acquisition, immunocompromise
- Prognosis: variable (dependent on HBeAg and HBV DNA); can have spontaneous loss of HBsAg
 - HBV DNA $>10^4$ IU/mL at risk for progression to cirrhosis

*Important to screen for HCC (not just in pts with cirrhosis)

HEPATITIS C

- Almost always chronic, acute presentations are very rare and more likely to be a “flare” (acute on chronic)
- Similar risk factors as HBV
- No vaccine however curable; screen >18yo (USPSTF) or risk factors
- HCV Ab with reflex to HCV RNA
- Prognosis: curable with DAA therapy!
 - transplantation of organs from HCV-viremic donors into HCV negative recipients
 - Mayo Clinic Arizona cohort: 100% SVR



32 YEAR OLD MALE

A CASE REPORT

CC/HPI

- Several days of GI symptoms, including abdominal pain, N/V, fevers, and chills
- Initial labs at prison: elevated LFTs
- Sent to hospital in Winslow for further eval

PMHx

- Known hepatitis C
 - Treatment-naive, was told he had spontaneous clearance
- Known hepatitis B, on entecavir
 - No missed doses or lapses in treatment recently
- No additional MHx
- No other meds or supplements

Social Hx

- Currently incarcerated
- Hx IVDU as well as current drug use (heroin and meth)
- Denies any recent EtOH or pruno intake
- Remote Hx of alcohol use in the past as well as marijuana, no reported alcohol abuse

EVALUATION

BOTH BIOCHEMICAL AND RADIOGRAPHIC

At OSH:

- Upon presentation:
 - Transaminitis in the 3000s, total bili of 8.9 initially
 - Abd u/s: patent portal vein, no thrombus
 - APAP: low
 - HIV undetected
- UA suspicious for UTI -> Levaquin, ceftriaxone, doxycycline (concern for STI)
 - Tx initiated s/p initial LFTs
- Total bili incr to 10.9 following day, INR 2.12 -> EMTALA txf

Upon arrival to MCA:

- Initial labs: total bili of 14.9 (primarily direct), ALT 6475, AST 6422, alk phos 130, and INR 2.5
- U/S repeated and WNL
- NAC initiated ovrn prior to Hepatology consultation
- Viral studies sent: HCV Ab and RNA, HBV DNA, HBsAg, HBcIgM, Hep A IgM, HEV IgG/IgM, EBV DNA, CMV DNA, HSV DNA
- **No asterixis and no evidence of sub-clinical HE** (No evidence of ALF)
- Q6h LFTs, PT/INR, lactate, serum phos. Q2h serum glu, mental status.

Favorable biochemical trend...

1m ago All Rows

Time Mark

	2022 9/5/22 09:28	9/5/22 04:05	9/4/22 04:31	9/3/22 17:17	9/3/22 15:03	9/3/22 05:48
Calcium, Total, S		8.3 ▼	7.7 ▼			
Glucose, S		90	84			
Magnesium			1.9			
Bilirubin, Total, S		10.2 ▲	11.3 ▲		14.0 ▲	
Bilirubin, Direct, S					12.0 ▲	
Alanine Aminotransferase (ALT), S		2950 ▲	3873 ▲		4937 ▲	
Aspartate Aminotransferase (AST), S		878 ▲	2059 ▲		3364 ▲	
Alkaline Phosphatase, S		116	104		112	
Protein, Total, S		4.9 ▼	4.2 ▼		4.6 ▼	
Albumin, S		3.2 ▼	2.9 ▼		3.3 ▼	
Lactate			1.8	3.2 ▲		4.6 ▲












Time

ASSESSMENT:

- # Acutely elevated LFTs in a mixed hepatocellular cholestatic injury pattern with known Hx of chronic HBV on entecavir and HCV (treatment-naive), unclear etiology of acute hepatitis however perhaps an acute HCV flare



 <1m ago <input checked="" type="checkbox"/> All Rows »»	
    Time Mark	
HEPATITIS C  	
HCV RNA Detect/Quant, S	799000 ! 
HCV Ab, S	React... ! 

- **Treatment plan:**

- Mavyret or no Mavyret?
- Close lab monitoring (LFTs in particular – assessing liver injury)
- Fibroscan to assess if any chronic fibrosis
- Prison/Health Dept notification, contact screening

HCV/HIV CO-INFECTION

- Most (if not all) HCV patients should be screened for HIV (and HBV)
- Infections share routes of transmission
- Approximately 30% of HIV infected patients are also co-infected with HCV
- Approximately 10% of HCV infected patients are also co-infected with HIV
- HIV/HCV co-infection accelerates the rate of hepatic fibrosis
- Patients with advanced HIV should be treated with HAART before initiating HCV therapy

40 yo male with jaundice, fatigue, and nausea x 2 weeks. Had sexual intercourse with a prostitute in Mexico approx. 3 months ago.

Notable findings:

Exam: jaundice; no encephalopathy.

Labs: ALT 2210, bilirubin 5.8, and INR 1.4.

HAV IgG +, HAV IgM-, HCV Ab -, HBsAg +, IgM anti-HBc +, IgG anti-HBc -

What is the most likely diagnosis?

1. Acute hepatitis A
2. Acute hepatitis B
3. Autoimmune hepatitis
4. Acute hepatitis E
5. Chronic hepatitis B

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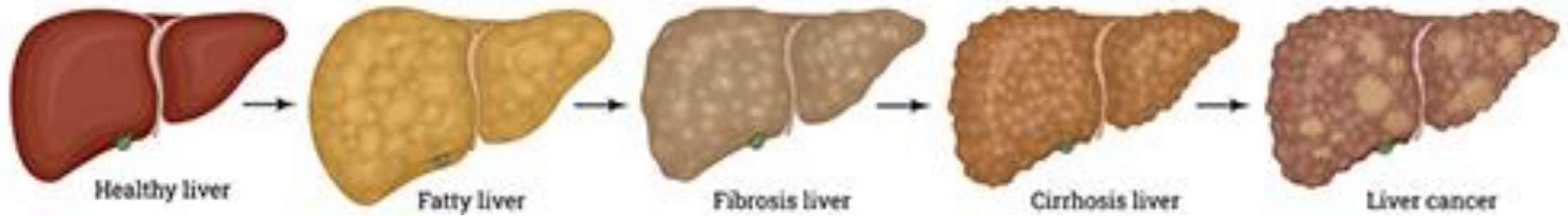
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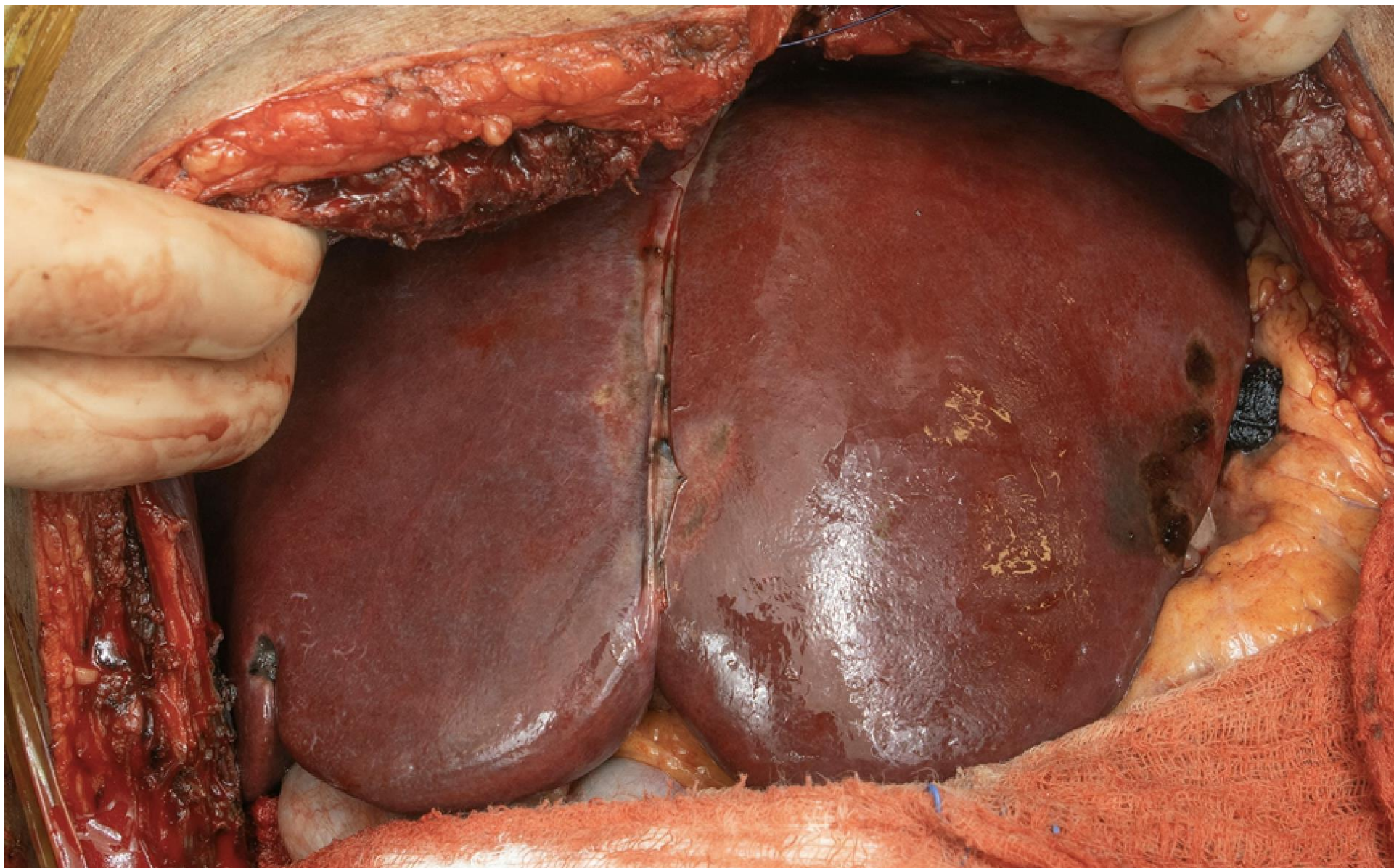
EVOLUTION OF LIVER DISEASE



<https://gi.md/resources/articles/are-fatty-liver-and-cirrhosis-serious-health-concerns>

WARNING





DIAGNOSIS OF CIRRHOSIS

- Histology from liver biopsy
- Portal pressure measurements*
- Clinical grounds
 - Presence of a risk factor for chronic liver disease
 - Radiographic evidence of parenchymal changes
 - Clinical evidence of portal hypertension
 - Esophageal varices
 - Splenomegaly with thrombocytopenia
 - Clinical evidence of hepatic synthetic dysfunction
 - Hypoalbuminemia
 - Elevated PT/INR
 - Physical exam findings specific to chronic liver Dz
 - Sarcopenia
 - Spider angiomas, telangiectasis

SCREENING/SURVEILLANCE FOR CIRRHOSIS

- All patients should be enrolled in HCC surveillance
 - Abdominal imaging (US) every 6 months indefinitely
 - AFP alone not sufficient (poor sensitivity)
- All patients should be screened for esophageal varices at diagnosis with routine screening endoscopy thereafter
- Update immunizations to include pneumococcus, HAV and HBV



43 y.o. asymptomatic woman with abnl liver tests. Drinks 1 bottle wine daily. Remote Hx IVDA.

Exam: jaundice, spider angiomas, splenomegaly.

Bili 5.4, AST 121, ALT 58, INR 1.6, platelets 34k, HCV RNA pos.

US: GB stones, nodular-appearing hepatic parenchyma, recanalized periumbilical vein, splenomegaly.

In addition to cessation of alcohol, which of the following would you advise?

1. Pegylated interferon and ribavirin
2. Liver biopsy
3. Splenectomy
4. Laparoscopic cholecystectomy
5. US every 6 months

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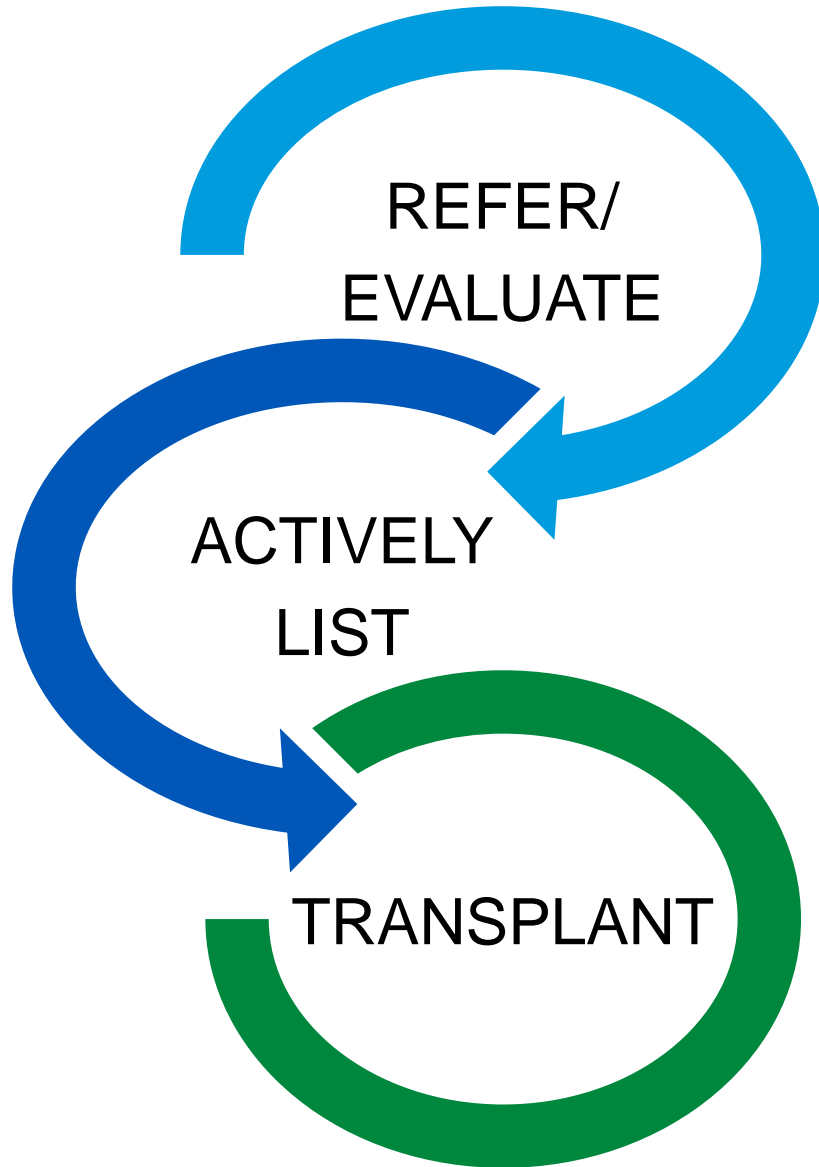
1. Pegylated interferon and ribavirin
2. Liver biopsy
3. Splenectomy
4. Laparoscopic cholecystectomy
5. *US every 6 months*

MANAGEMENT OF CIRRHOSIS

- Can live with cirrhosis for years if compensated
- HCC screening/esophageal variceal surveillance
- Manage complications (i.e. SBP, GI bleeding, pruritis)
- Prevent infection
- Prevent/treat HRS physiology
- Adequate nutrition

WHEN TO REFER TO A TRANSPLANT CENTER

- Cirrhosis/ESLD
 - Especially MELD-Na > 15 but really any time
- Acute fulminant hepatic failure/acute liver failure (ALF)
 - Fulminant Wilson's Dz can be diagnosed most effectively not by waiting for copper levels (too slow to obtain) or by obtaining ceruloplasmin levels (low in half of all ALF patients, regardless of etiology) but by simply looking for the more readily available bilirubin level (very high) and alkaline phosphatase (very low). Bili/ALP ratio exceeds 2.0.
 - Any patient with very high aminotransferases and low bilirubin on admission (with no evidence of ischemic injury): must r/o acetaminophen toxicity.
 - King's College criteria
 - Low threshold for biopsy in patients with indeterminate ALF, given that autoimmune hepatitis may be the largest category of indeterminate, after unrecognized acetaminophen poisoning.



NUMBER OF OLT AT MAYO CLINIC ARIZONA [THROUGH 9/13/22]



- 176 YTD
- 2048 total

QUESTIONS?

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