# Managing Cirrhosis

## and Understanding When to Refer for Transplantation

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# Nothing to disclose

# Learning Objectives

- Understand the role of hepatocyte injury leading to cirrhosis
- Learn how to identify and diagnose end stage liver disease
- Review the most common causations for end stage liver disease
- Discuss common etiologies and presentation of portal hypertension
- Identify indications for liver transplantation
- Discuss the role of palliative care/hospice care when transplant is not an option

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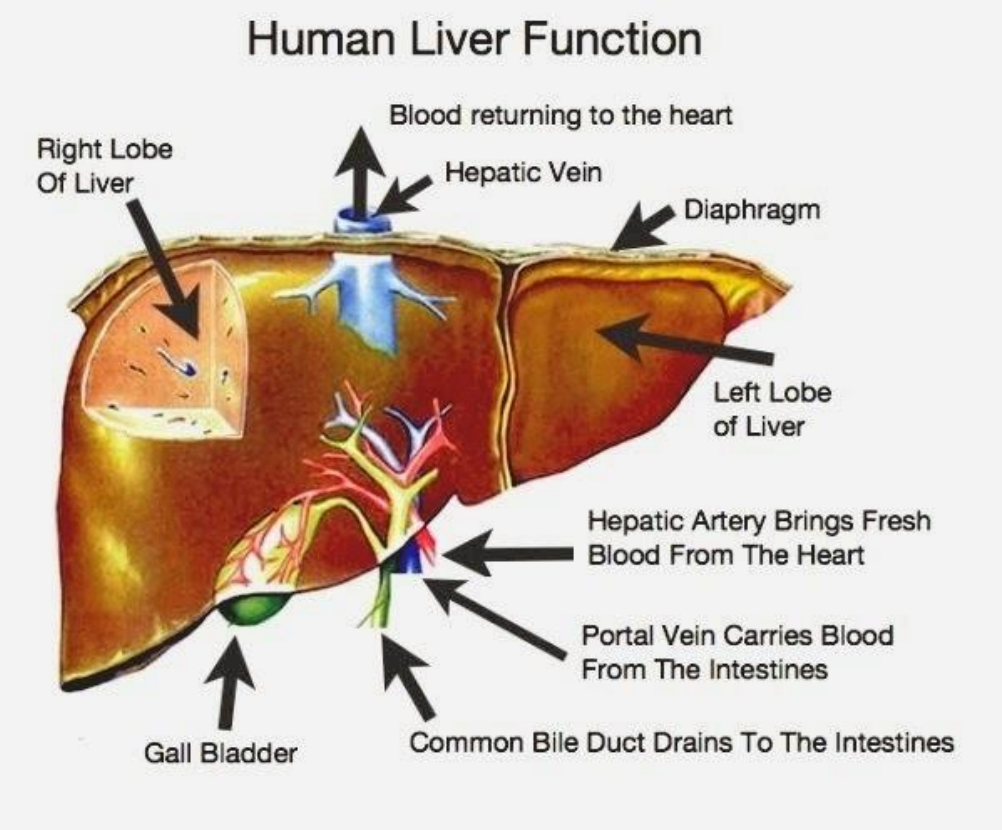
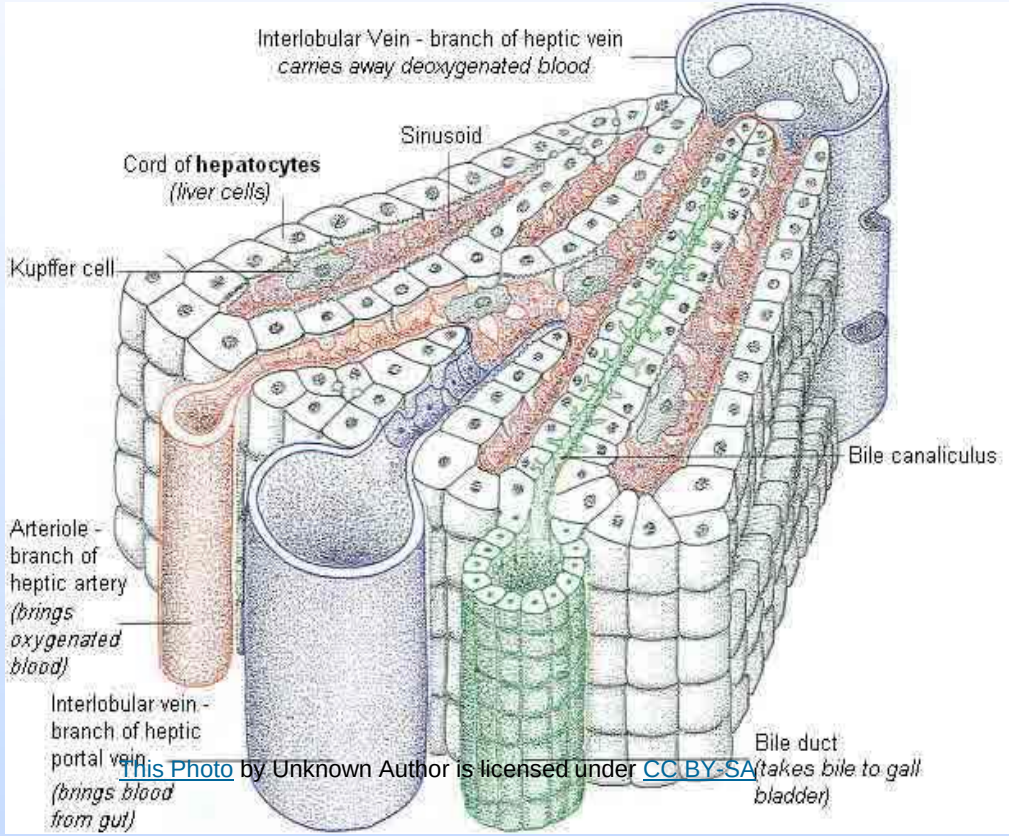
# Liver Anatomy

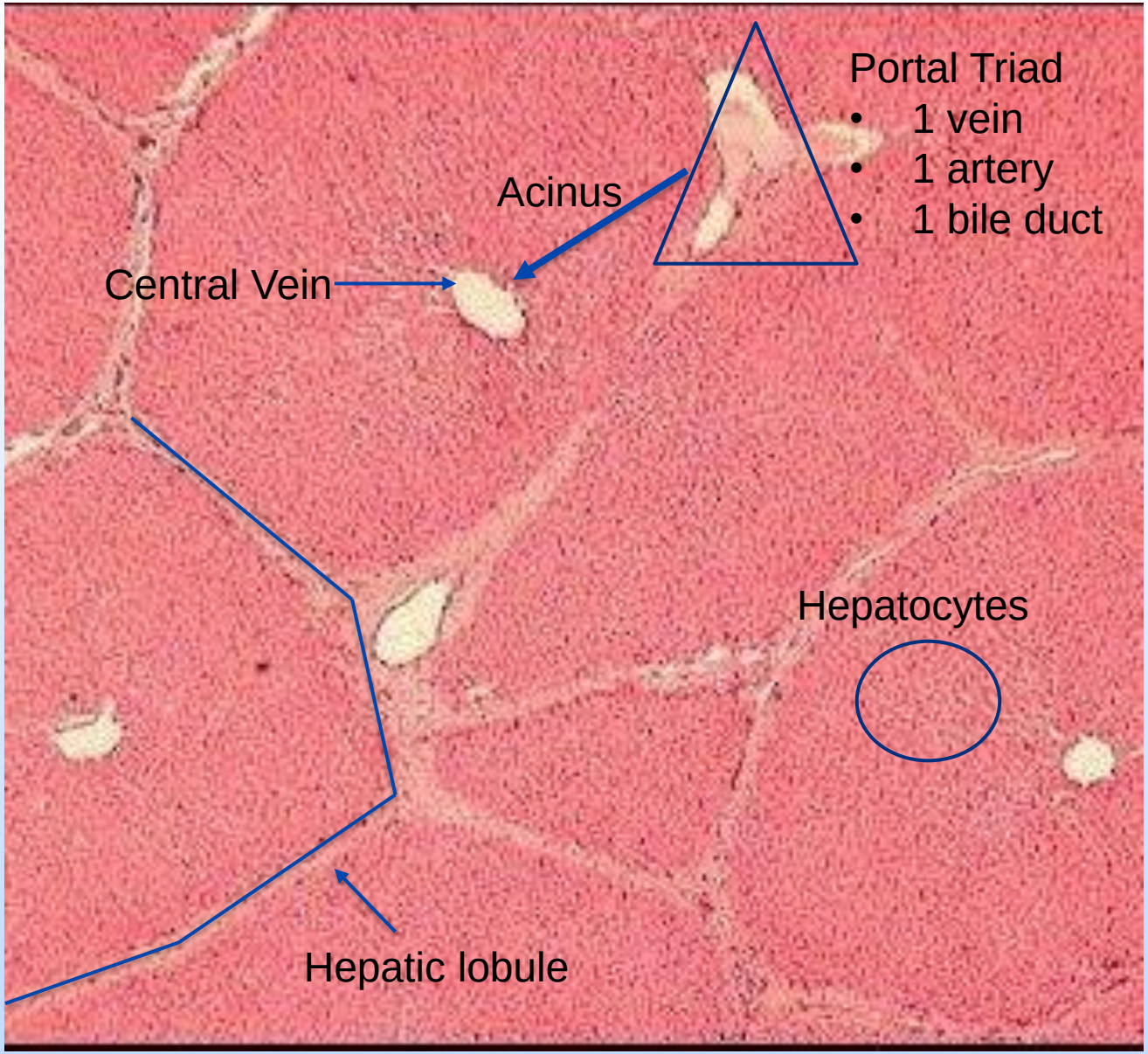




# Liver Anatomy

- Tissue is divided into lobules around a central vein
- Liver tissue is divided into lobules organized around a central vein
- Portal triads exist containing a vein, artery, and bile duct
- The functional unit of the liver is the acinus

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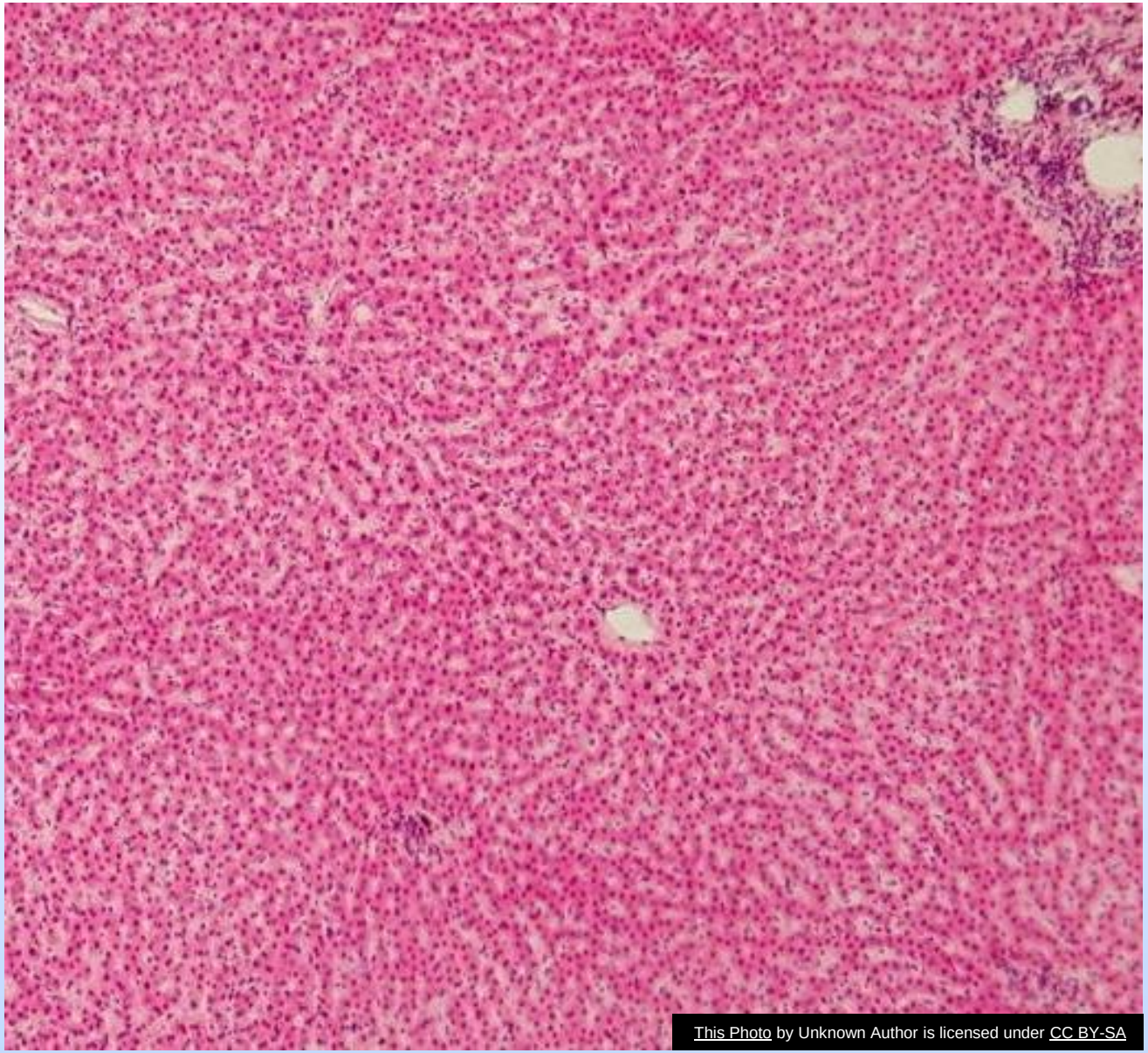
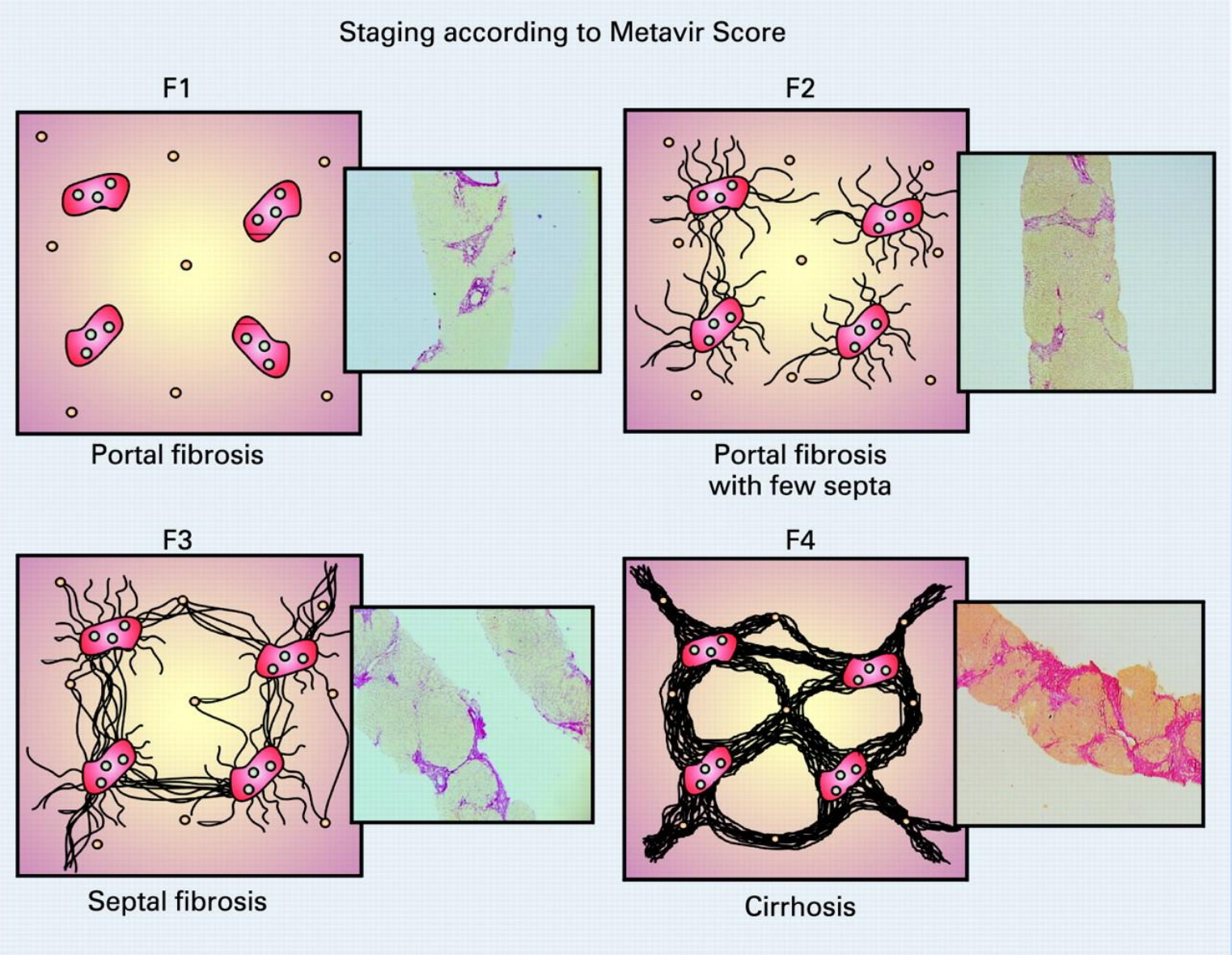
# Histology

Normal Parenchyma



# Progression of Fibrosis

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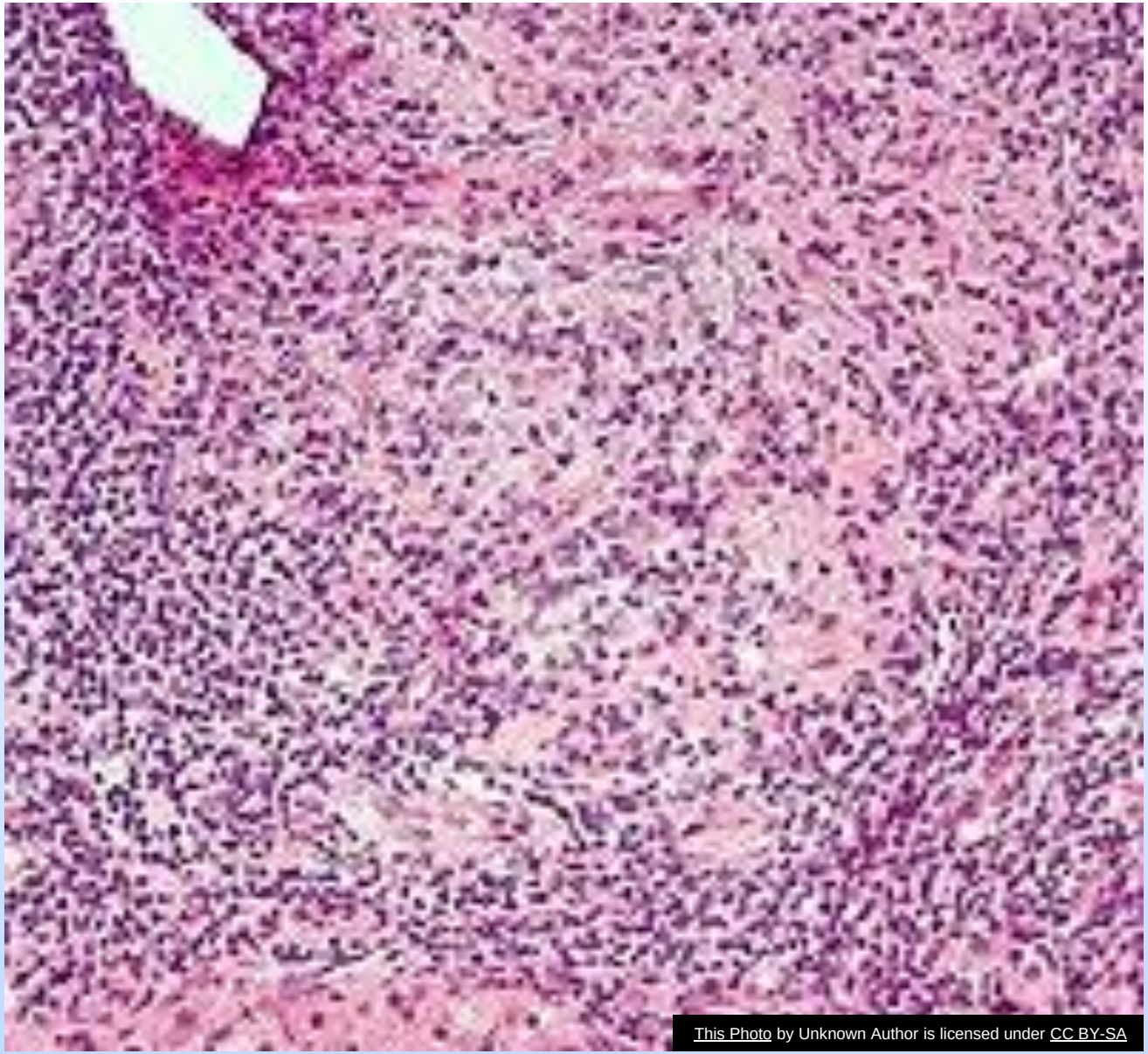
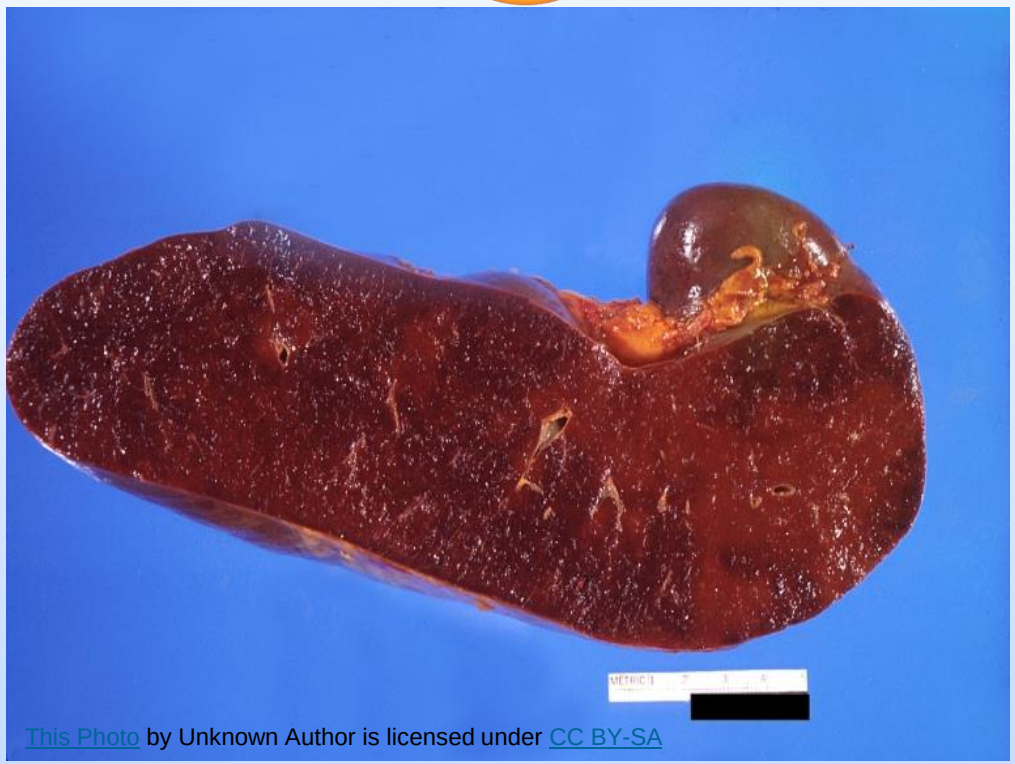
# Histology

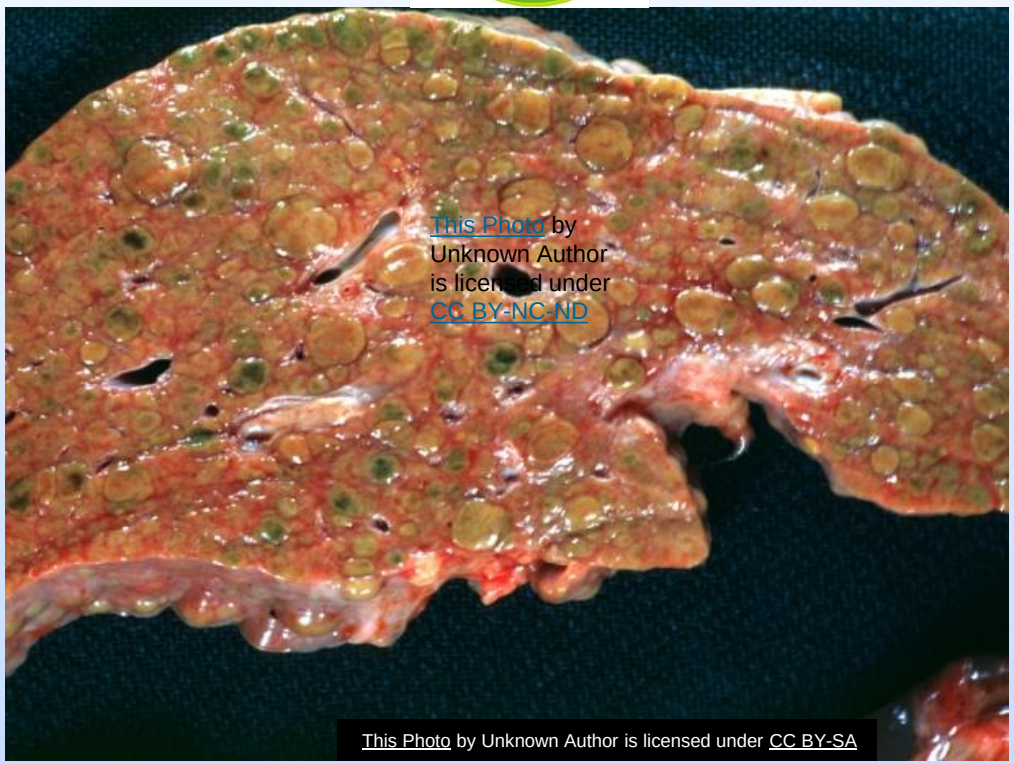
Cirrhosis



# Anatomy





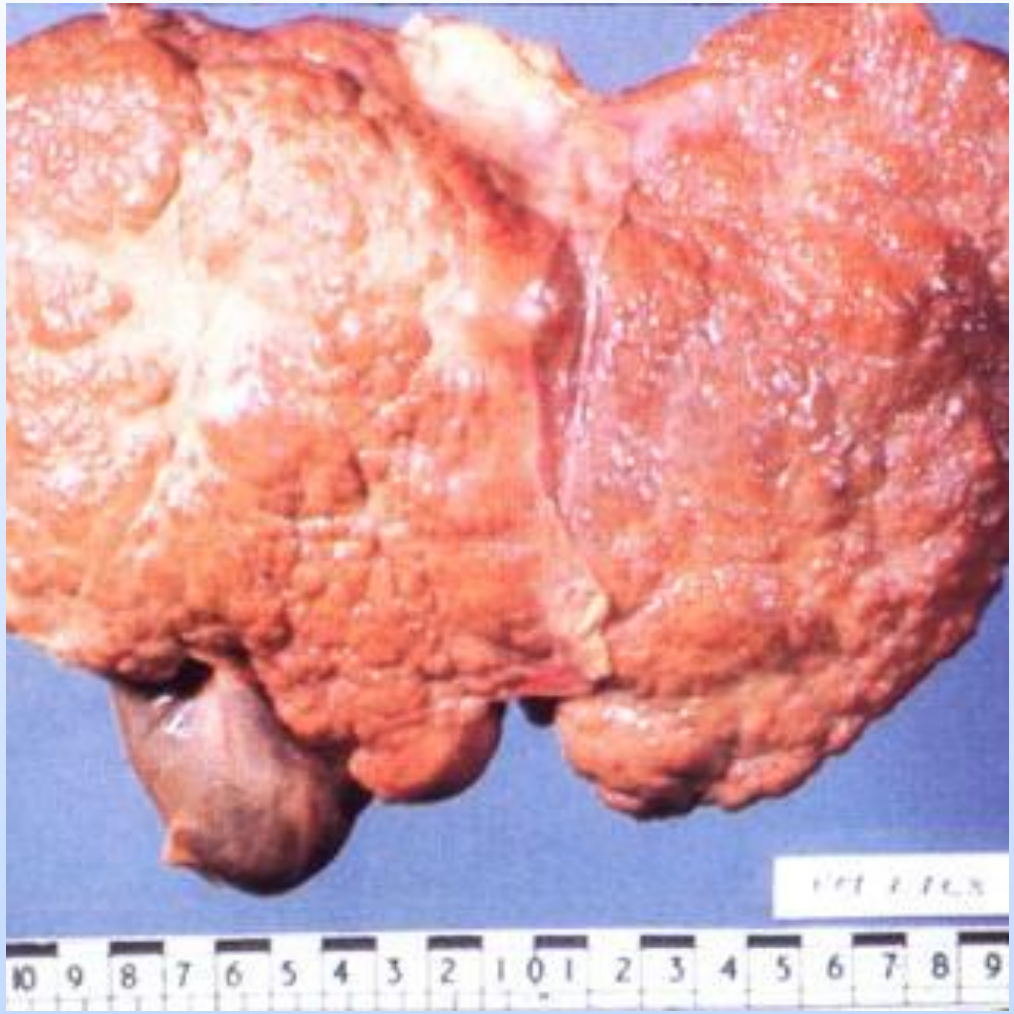






# What is Cirrhosis?

- End stage of progressive fibrosis
  - Leads to distortion of liver parenchyma
  - Characterized by regenerative nodules
- 12 leading cause of death in the U.S. with more than 27,000 deaths annually
- Prevalence= 4.5 to 9.5% of general population



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# How do we diagnose it?

- History
- Physical Exam
- Laboratory Findings
- Imaging
  - US, CT, MRI
- Biopsy
  - Transabdominal vs Transjugular
- Fibroscan



 

# Diagnosis

## History

- Family Hx
- Obesity with elevated LFTs
- Excessive ETOH use
- Unsafe sexual practices

## HCV Specific

- Born between 1945 and 1965
- Autoimmune disease
- Blood transfusion/organ tx prior 1992
- Received clotting factor concentrates prio 1987
- Hx IV or intranasal drug use
- Have HIV
- iHD
- Incarceration
- Born to a HCV positive mother

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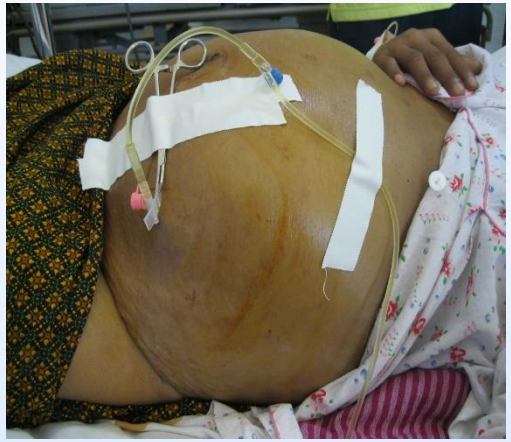
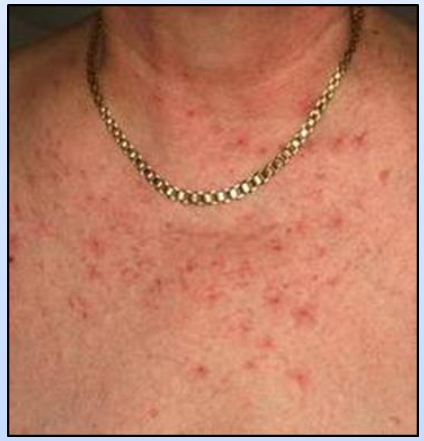
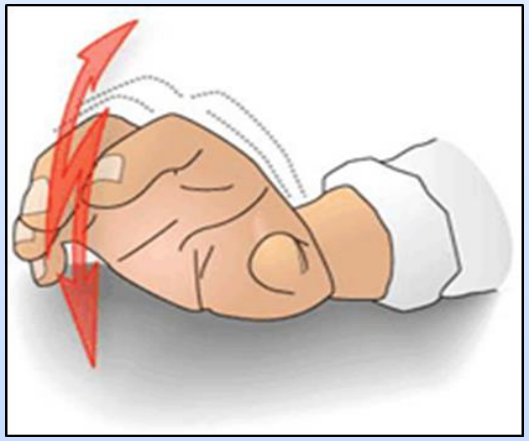
# Physical Exam





- Spider angiomata/telangiectasias
- Palmar erythema- secondary to abnormal estradiol levels
- Temporal/masseter muscle wasting
- Ascites
- Asterixis
- Jaundice- (Tbili>3.0mg/dl)

# Diagnostics

## Laboratory Findings

- Normochromic, normocytic anemia (occasionally macrocytic with ETOH )
- Leukopenia
- Thrombocytopenia
- Hyponatremia
- Moderate elevation is AST/ALT/Alk phos
- Elevated T bilirubin with correlating direct bilirubin
- Hypoalbuminemia
- Elevated PT/INR

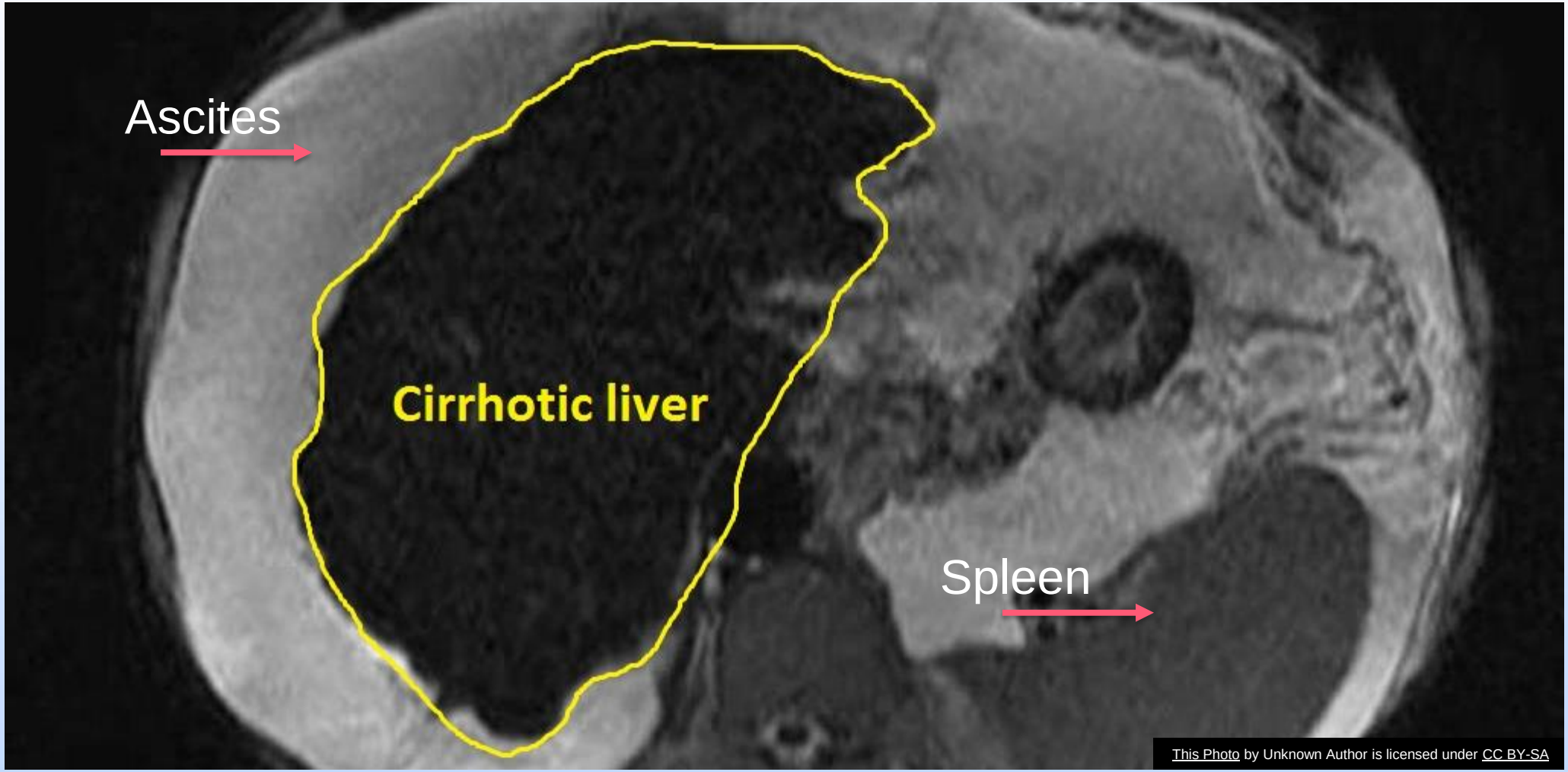
## Imaging

- Hepatomegaly OR a shrunken, collapsed picture
- Splenomegaly
- Enlargement or venous obstruction of the portal splenic veins with portal hypertension
- Cavernous transformation of the portal vein
- Presence of esophageal varices/ascites



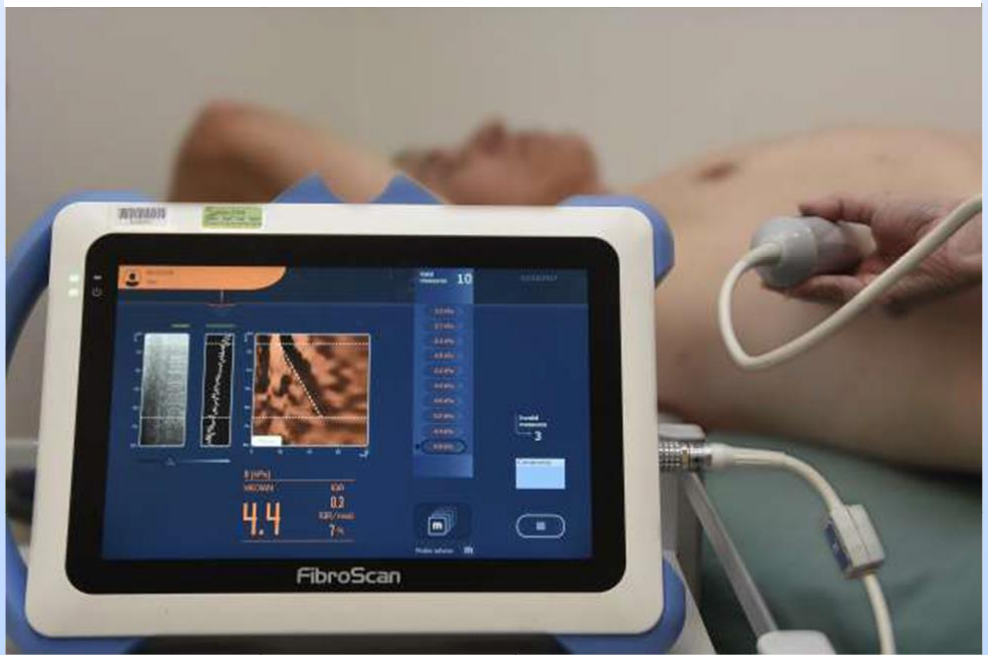
 

# MRI



# Diagnostics
## Biopsy or Fibroscan

- Gold Standard
- Direct transabdominal or indirect transjugular approach
- Fibroscan- Ultrasound based Elastography
- Interpretation:
- Fibrosis interpretation is based on Kilopascal(kPa)
- Data derived in the non liver transplant recipients:
  - <7kPa=no significant fibrosis
  - >11kPa for stage 4 fibrosis (cirrhosis)

# Types of Liver failure

## Chronic

- Accounts for majority of cases
- Progressive fibrosis over many years
- Often asymptomatic: Compensated
- Clinical manifestations: Decompensated
- Decompensated cirrhosis → Liver failure
- Potential for reversal in some cases

## Acute (Fulminant)

- Rapid development of liver failure <26 weeks in a previous healthy organ
- More common in young people

## Acute on chronic

- Underlying liver disease
- Acute decompensating event
  - Bleeding
  - Infection
- Leads to worsening liver failure + other organ failure
- Very high short-term mortality

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# Etiologies of Liver Disease

## Chronic

- Chronic viral hepatitis (B, C, E)
- Alcoholic liver disease +/- viral hepatitis
- Fatty liver disease
- Autoimmune hepatitis
- Primary biliary cholangitis
- Primary sclerosing cholangitis
- Alpha-1 antitrypsin deficiency
- Heart failure
- Sarcoidosis
- Hereditary Hemochromatosis
- Cryptogenic

## Fulminant

- Viral Hepatitis A and B
- Acute alcoholic hepatitis
- Acute autoimmune hepatitis
- Acute Heart Failure
- Drug induced liver injury (DILI)
- Tylenol toxicity

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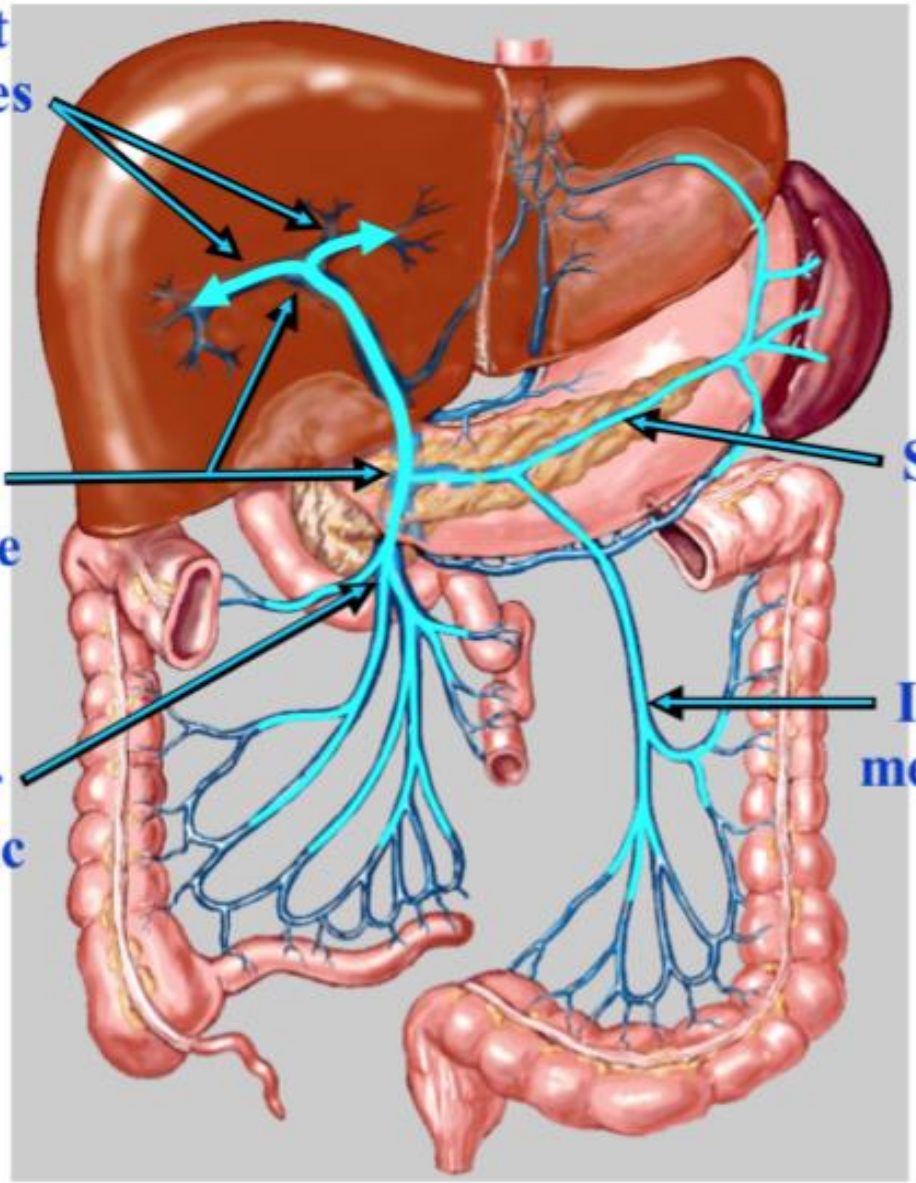
# Hepatic Portal System



# Pathophysiology of Portal Hypertension

1. Increased hepatic resistance to portal inflow
   - Architectural [illegible] (fibrosis)
   - In[illegible] vasodilate in response to inc[illegible] from spleen
2. Increased splanc[illegible]tion
   - Excess Nitri[illegible]
3. Increase in systemic vas[illegible]
   - Excess Nitric oxide in circula[illegible]
4. Release and increased response t[illegible]ictors i.e. norepinephrine and angiotensin II
5. Increased Na+ retention by the kidney and hyperdyna[illegible]ic response by the heart to increase CO/CI

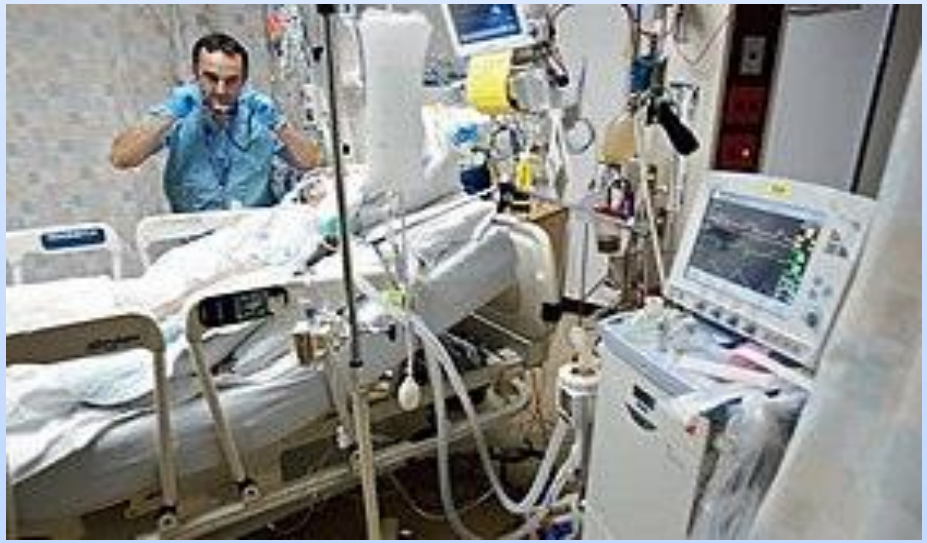
**Portal Hypertension**



# Manifestations

- Synthetic Dysfunction
  - Elevated PT/INR
  - Hyperbilirubinemia
  - Hypoalbuminemia
- Portal Hypertension
  - Hepatic Encephalopathy
  - Ascites
  - Esophageal Varices
  - Hepatorenal Syndrome
  - Hepatopulmonary Syndrome



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# Prognosis of Cirrhosis

- Compensated patients: prior to complications
  - 90% 5-year survival
  - Marked by mild- moderate portal hypertension
  - Presence of varices define early versus advanced stage
- Decompensated patients: post complications
  - 50% 5-year survival
  - Marked by the portal hypertension >10 mmHg
  - Develops at 4-12% per year
    - Encephalopathy
    - Ascites
    - Variceal bleed
    - Jaundice
    - Hyponatremia
    - Hepatocellular carcinoma

D'Amico et al. J Hepatol 2017



 

# Hepatic Encephalopathy


- Grade I: Changes in behavior, mild confusion, slurred speech, disordered sleep
- Grade II: Lethargy, moderate confusion, asterixis
- Grade III: Marked confusion (stupor), incoherent speech, sleeping but arousable
- Grade IV: Coma, unresponsive to pain

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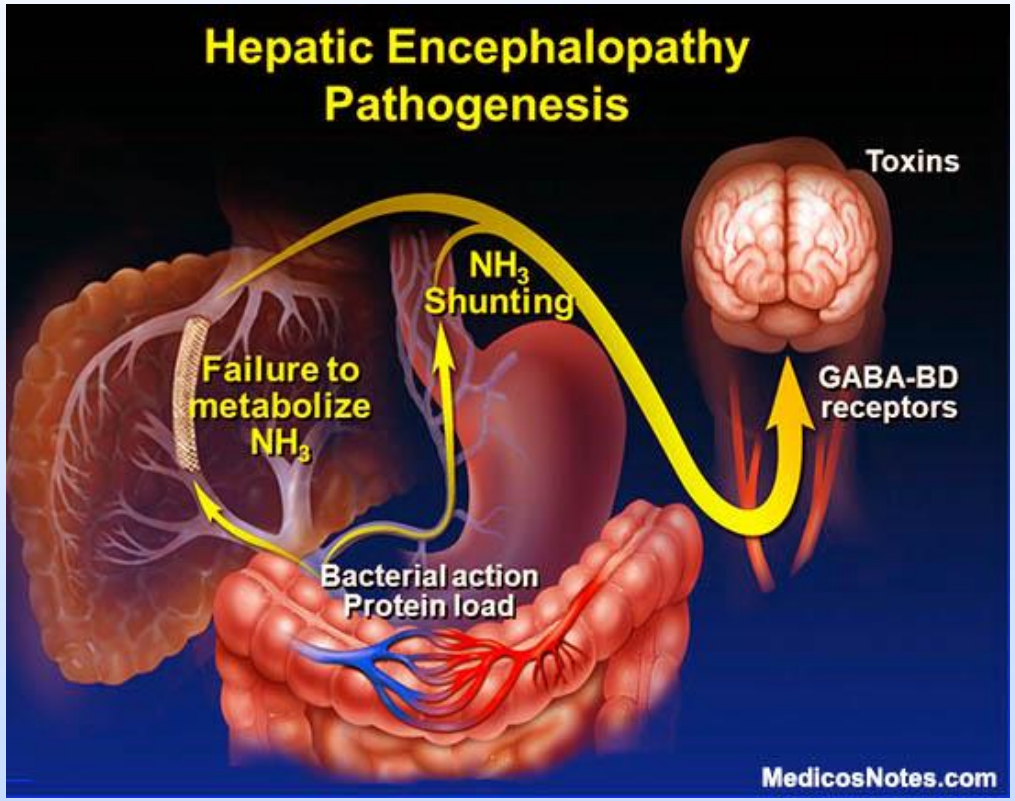
# Hepatic Encephalopathy




- Noted in 27-75% of patients with ESLD
- Develops at rate of 2% - 3% per year
- Median survival post onset is 1-2 years
- Caused by a combination of Porto systemic shunting and hepatic dysfunction
- Plasma ammonia taken up by astrocytes
- up regulation of peripheral type benzodiazepine receptors (PBRs). These in turn stimulate neuro-steroids which are the main modulators of GABA
- GABA causes cortical depression and thus HE

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# Hepatic Encephalopathy

- Do not monitor, follow, or treat the ammonia level!!!
  - Not prognostic
  - Treat your pts. physical and clinical exam



 

# Treatment of Hepatic Encephalopathy

- Correct the Cause!
  - Remove any confounding variables i.e. drugs, toxins
  - Infection
  - Bleeding
- Treat renal insufficiency
  - Uremia can mimic HE
- Correct electrolyte imbalance
  - Na+ <119

- Lactulose
  - Acidifies colon contents to a pH of 5 favoring the formation of the non-absorbable $NH_4$+
  - 20-30g 2-3x daily
  - Goal 2-4 BM per day
- Rifaximin
  - Reduce burden of ammonia producing GI bacteria
  - 550mg po BID
- Zinc sulfate
  - Significant zinc deficiency with ESLD
  - 220mg TID
- Neomycin
  - Side effects of ototoxicity and nephrotoxicity
- Dialysis (CRRT)

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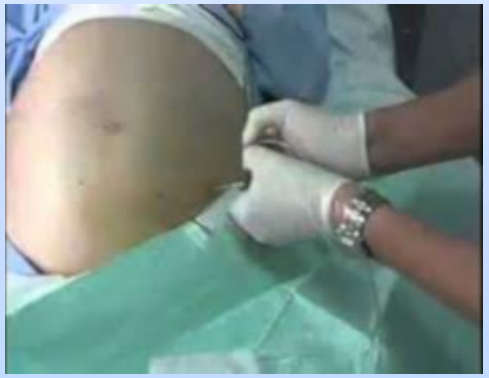
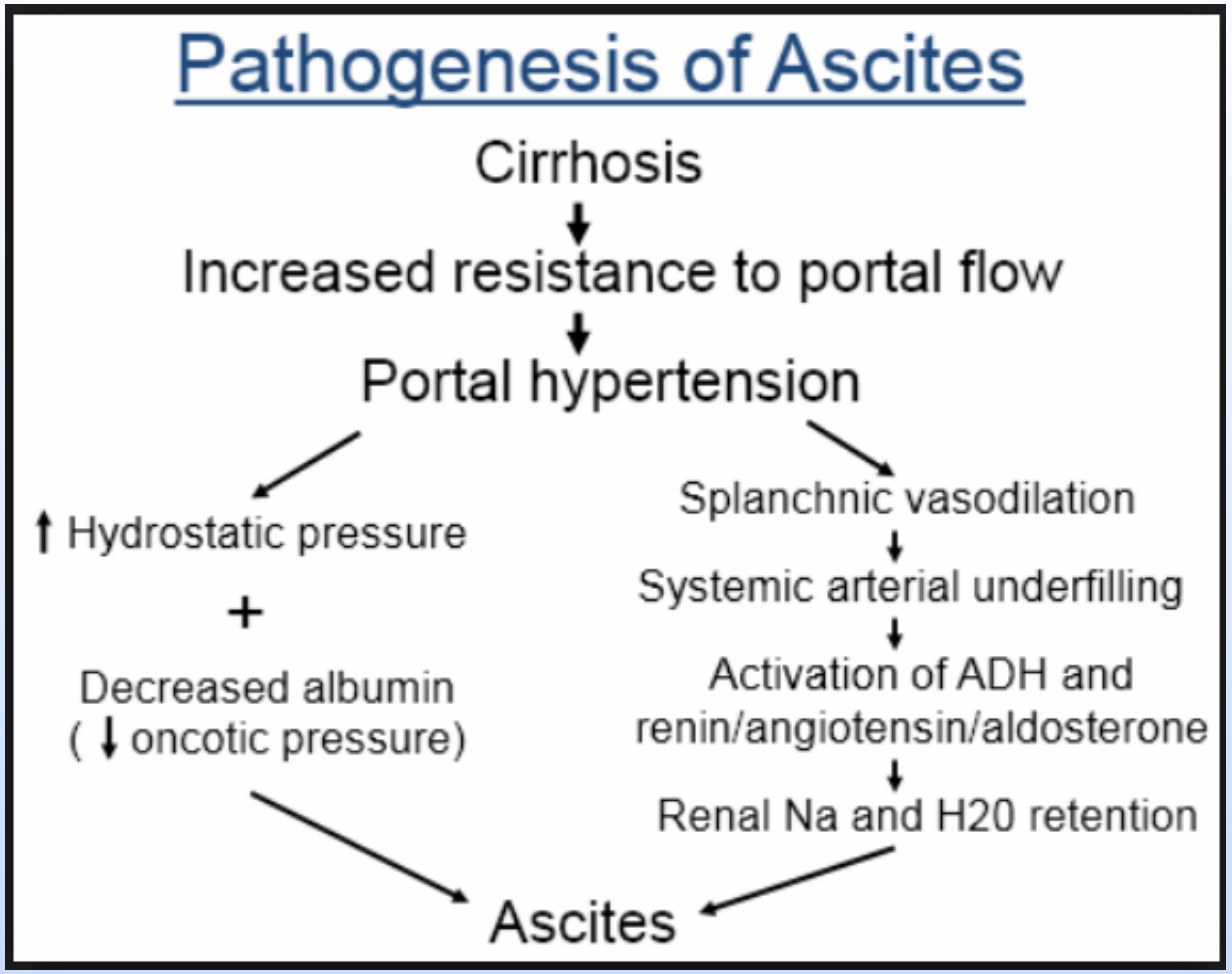
# Ascites

- The most common complication of cirrhosis
- Within 10 years >60% develop ascites
- Physical exam is 83% sensitive 56% specific

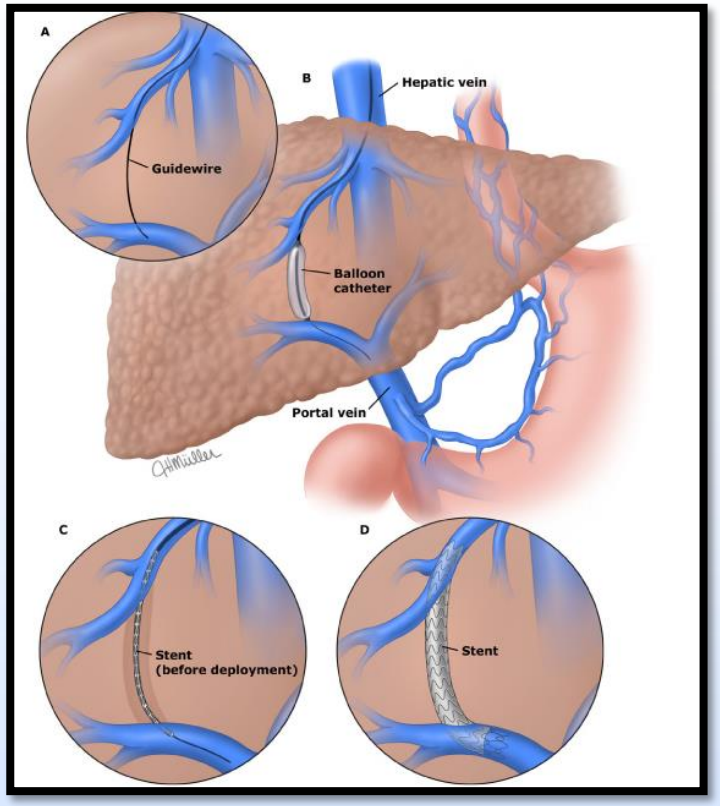


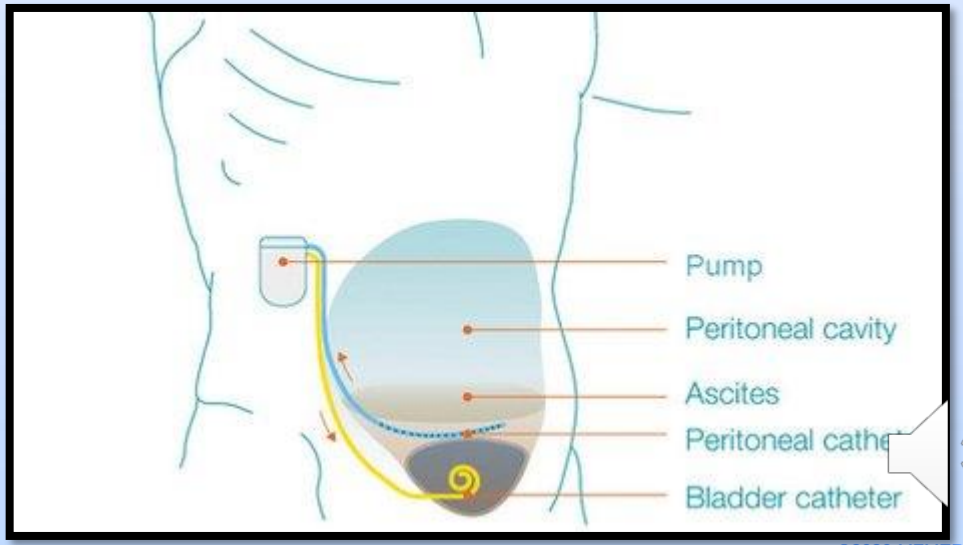



D'Amico et al. J Hepatol 2017
Planas et al. Clin Gastroenterol Hepatol 2006

# Ascites

- Management:
  - Sodium Restriction- <2g per day
  - Diuretic therapy: Stepwise approach to Lasix and Spirolactone based on renal function
  - Paracentesis
    - Caution with frequency
  - TIPS
  - Alpha Pump



# Transjugular Intrahepatic Portosystemic Shunt

- The good…
  - Instant reduction of portal hypertension
    - Eventual reduction or complete resolution of refractory ascites
    - Immediate stop to a variceal bleed
- The not so good…
  - Increased hepatic encephalopathy
  - Decompensation post procedure

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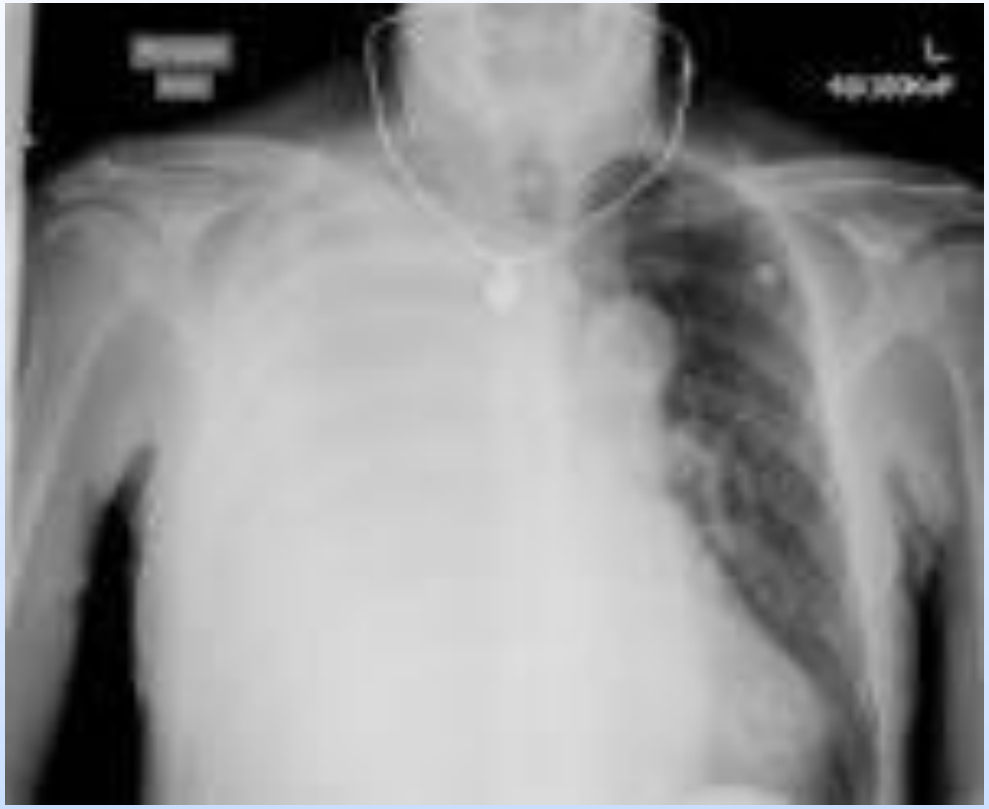
# Ascites…there's more

- Hepatic Hydrothorax
- Transudative pleural effusion
- Absence of cardiac and/or pulmonary disease
- 5%-10% prevalence in cirrhosis
- Defects in diaphragmatic wall
- Passage of ascites into lung space
- Increased with malnutrition
- If infected-spontaneous bacterial empyema



NO CHEST TUBE!

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# Spontaneous Bacterial Peritonitis (SBP)

- Translocation of bacteria from the bowel, lung, or bladder
  - E coli, Klebsiella pneumoniae, and Pneumococci
- 12% of patients admitted with ascites
- Risk increases as liver function worsens
  - 65% one-year incidence in high risk groups
- 1-year mortality after first episode 20%-50%
- Failure to diagnose occurs in 10-13% of patients asymptomatic
- Diagnosed when the absolute polymorphonuclear leukocyte count (PMN) is >250 cells/mm
- OR PMN>500 and neg. culture
  - Neutrocytic ascites

Borzio et al. Dig Liver Dis 2001
Sort et al. N Engle J Med 1999
Fernandez et al. Gastroenterology 2007
Rimola et al. J Hepatol 2000.

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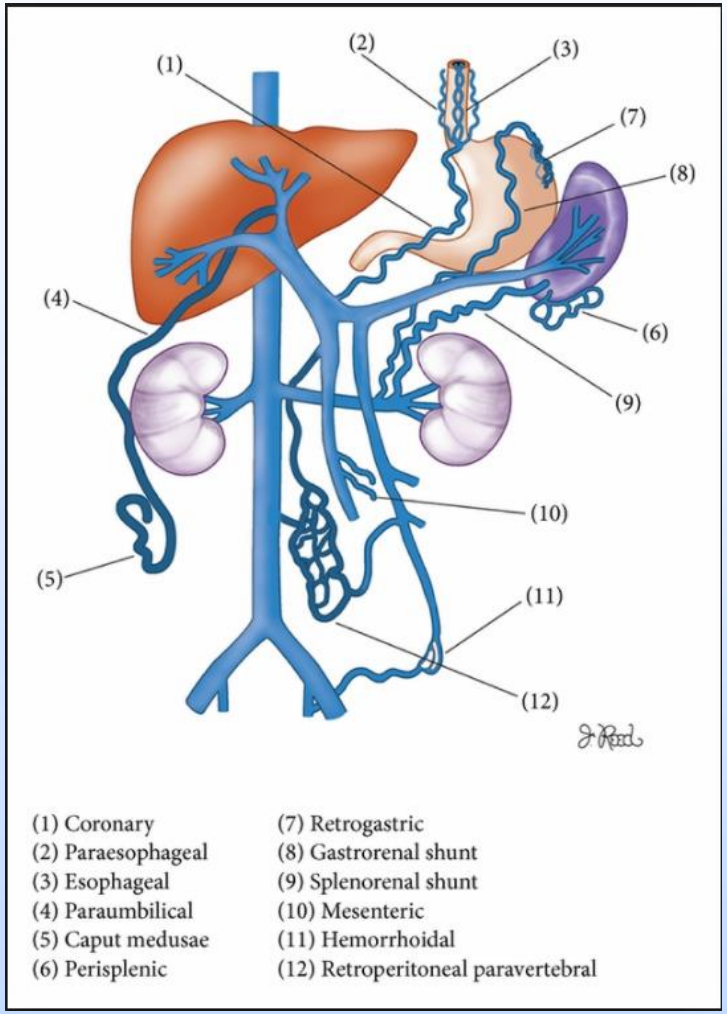
# SBP continued…

- Treated with third-generation cephalosporins (IV ceftriaxone x 7 days)
- Hold diuretics and beta blockers during therapy to prevent AKI
- Follow AASLD albumin replacement protocol to help prevent hepato-renal syndrome
- Day 1 albumin replacement 1.5g/kg of 25%
- Day 3 albumin replacement 1g/kg of 25%
  - Repeat cell ct. and culture day 3
- Ciprofloxacin or trimethoprim/sulfamethoxazole can be given for for prophylaxis

| Body Fluid Analysis | | | |
|---|---|---|---|
| BF Sample Type | Ascites fluid | Ascites fluid | Ascites fluid |
| BF Appearance | Clear | Hazy | Slighty Hazy |
| BF Color | Yellow | Yellow | Yellow |
| BF Nucleated Cell Count | 100 * | 1,206 * (H) | 1,210 * (H) |
| BF Neutrophils % | 11 | 10 | 77 |
| BF Eosinophils % | 0 | 1 | 0 |
| BF Basophils % | 0 | 1 | 0 |
| BF Mono/Macroph % | 61 | 11 | 23 |
| BF Lymphocytes % | 2 | 77 | 0 |
| BF Plasma Cells % | 0 | 0 | 0 |
| BF Blasts % | 0 | 0 | 0 |
| BF Mesothelial Cells % | 26 | 0 | 0 |
| BF Synovial Lining Cells % | 0 | 0 | 0 |
| BF Other Cells % | 0 | 0 | 0 |
| BF Cells Counted | 100 | 100 | 100 |
| BF Comment | See Comment * | See Comment * | |

# Esophageal Varices




- Incidence of 5%-8% per year
- Present in 25-40% of patients with cirrhosis
- Largest complication: Bleeding
- Highest time interval of mortality following a bleed is within the first 2 weeks
- Size of varices proportional to risk of bleeding
- EGD at time of cirrhosis diagnosis
- If compensated cirrhosis and no varices, repeat EGD in 3 years
- If decompensation occurs, repeat EGD and then annual EGD

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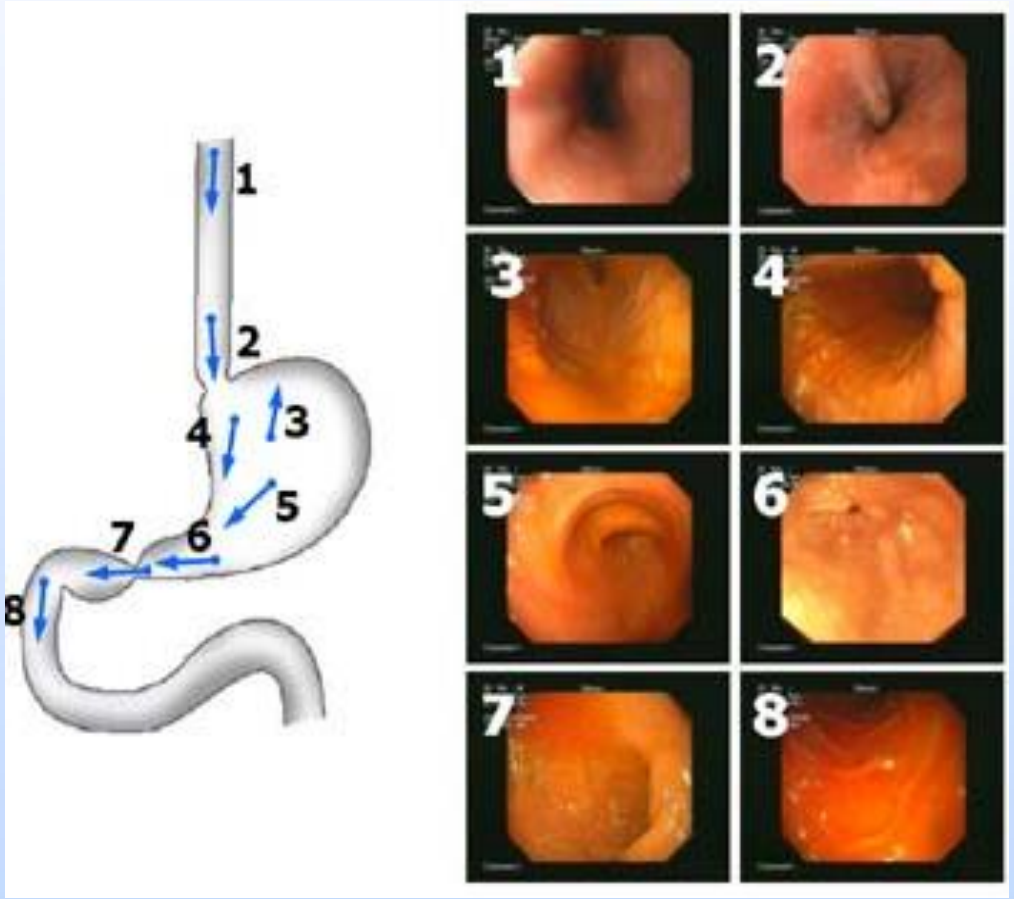
# Anatomy of Upper Endoscopy

- Bleeding stops spontaneously 40%-50%
- 85% controlled with treatment
- Overall mortality
  - 12.9% at 6 weeks
  - Advanced liver disease have worst outcomes (25% versus 55-80%)

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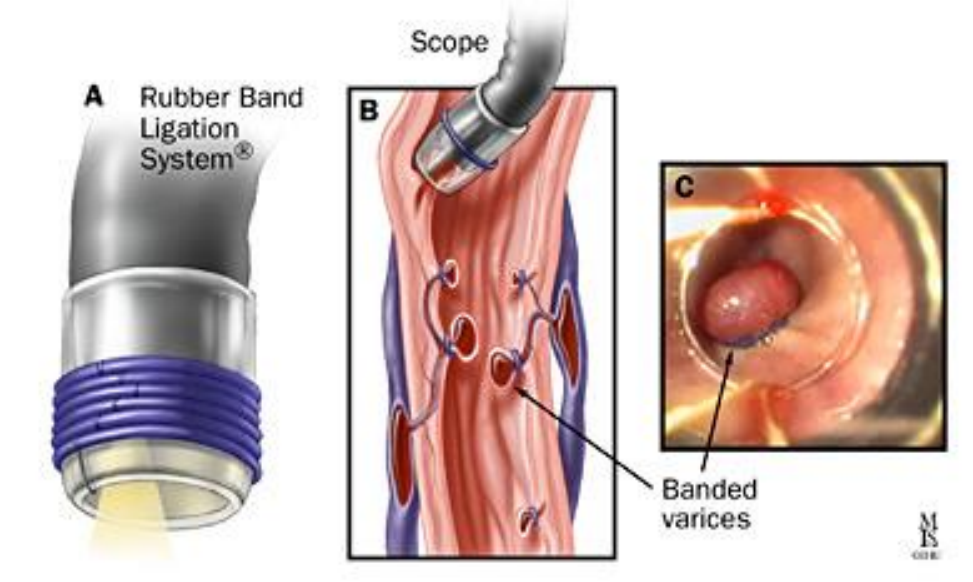
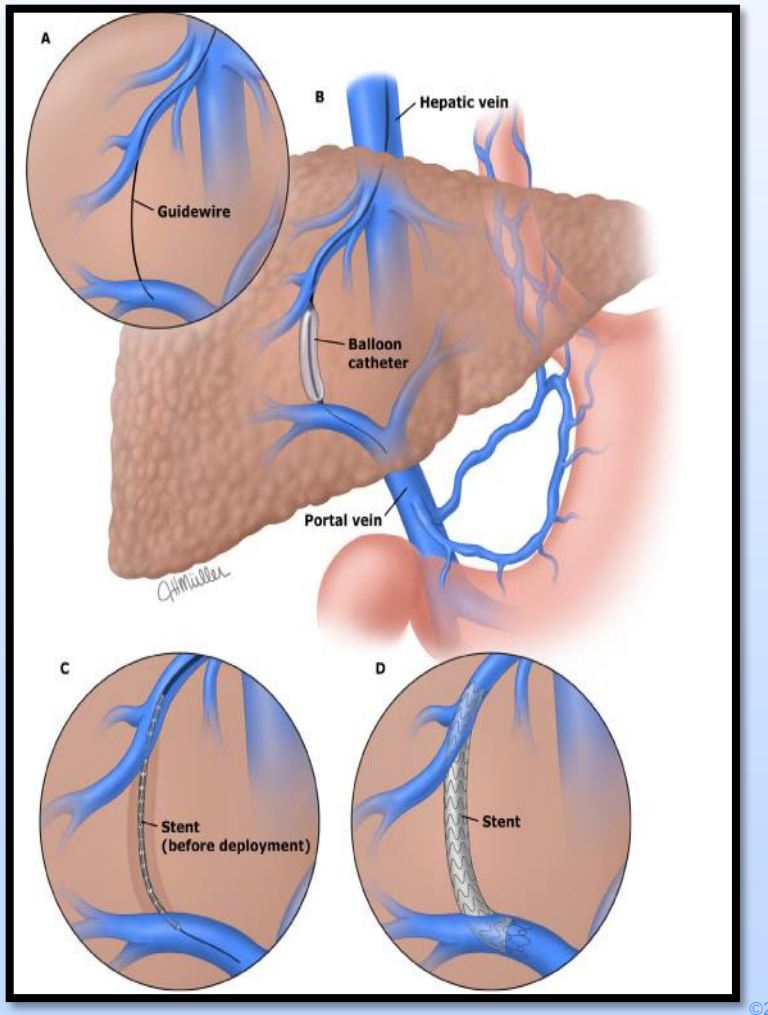
# Management of Variceal Bleeding

- Esophageal variceal ligation
- Non-selective beta blockers
  - (propranolol or nadolol)
- Combination therapy
- Restrictive approach to blood transfusions- Goal 7g/dL Hg




- Transjugular intrahepatic portosystemic shunt (TIPS)



# Transjugular Intrahepatic Portosystemic Shunt

- The good…
  - Stoppage of bleeding varices by instantly reducing portal hypertension (<12mmHg)
  - Eventual reduction or complete resolution of refractory ascites
- The not so good…
  - Increased hepatic encephalopathy
  - Decompensation post procedure

| Contraindications to Placement of TIPS |
|---|
| **Absolute** |
| Primary prevention of variceal bleeding |
| Congestive heart failure |
| Multiple hepatic cysts |
| Uncontrolled systemic infection or sepsis |
| Unrelieved biliary obstruction |
| Severe pulmonary hypertension |
| **Relative** |
| Hepatoma especially if central |
| Obstruction of all hepatic veins |
| Portal vein thrombosis |
| Severe coagulopathy (INR > 5) |
| Thrombocytopenia of < 20,000/cm$^3$ |
| Moderate pulmonary hypertension |

Boyer et al. Hepatology 2010

# Hepatorenal Syndrome

**Definition**

- A potentially ***reversible, functional renal failure*** that occurs in patients with cirrhosis, ascites, and liver failure, consisting of impaired renal function, marked abnormalities in cardiovascular function, and intense overactivity of the endogenous vasoactive systems
  - Extreme renal vasoconstriction with Na+ retention and increased Nitrous Oxide in the portal system
  - Reduced peripheral vascular resistance and vascular shunting
  - Increased systemic hypotension with activation of renin-angiotensin system
  - Renal vasoconstriction with decreased renal perfusion

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# Defining HRS

## The old…

- Type 1:
  - rapidly progressive with median survival of <30 days
  - Doubling of serum creatinine to >2.5mg/dl in < 2 weeks
- Type 2:
  - Serum creatinine levels between 1.5g/dl and 2.5g/dl

## The new... (AKIN/KDIGO criteria)

- An absolute increase in SCr ≥0.3 mg/dL (26.5 µmol/L) within 48 hours
- ***and/or***
- Urinary output ≤0.5 mL/kg BW for ≥6 hours
  (urinary catheterization)
- ***or***
- Percent increase in SCr ≥50% within 3 months using the last available value of SCr

AKIN, Acute Kidney Injury Network; KDIGO, Kidney Disease Improving Global Outcomes.

Arroyo V, et al. *Hepatology.* 1996;23:164-176; 2. Angeli P, et al. *Gut.* 2015;64:531-537; 3. Nadim M, et al. *J Hepatol.* 2016;64:717-735.

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# Hepatorenal Syndrome

- Ascites
- Unresponsive to albumin volume expansion
- Absence of shock
- No recent nephrotoxic drugs
- Absence of proteinuria/hematuria
- Normal renal ultrasound

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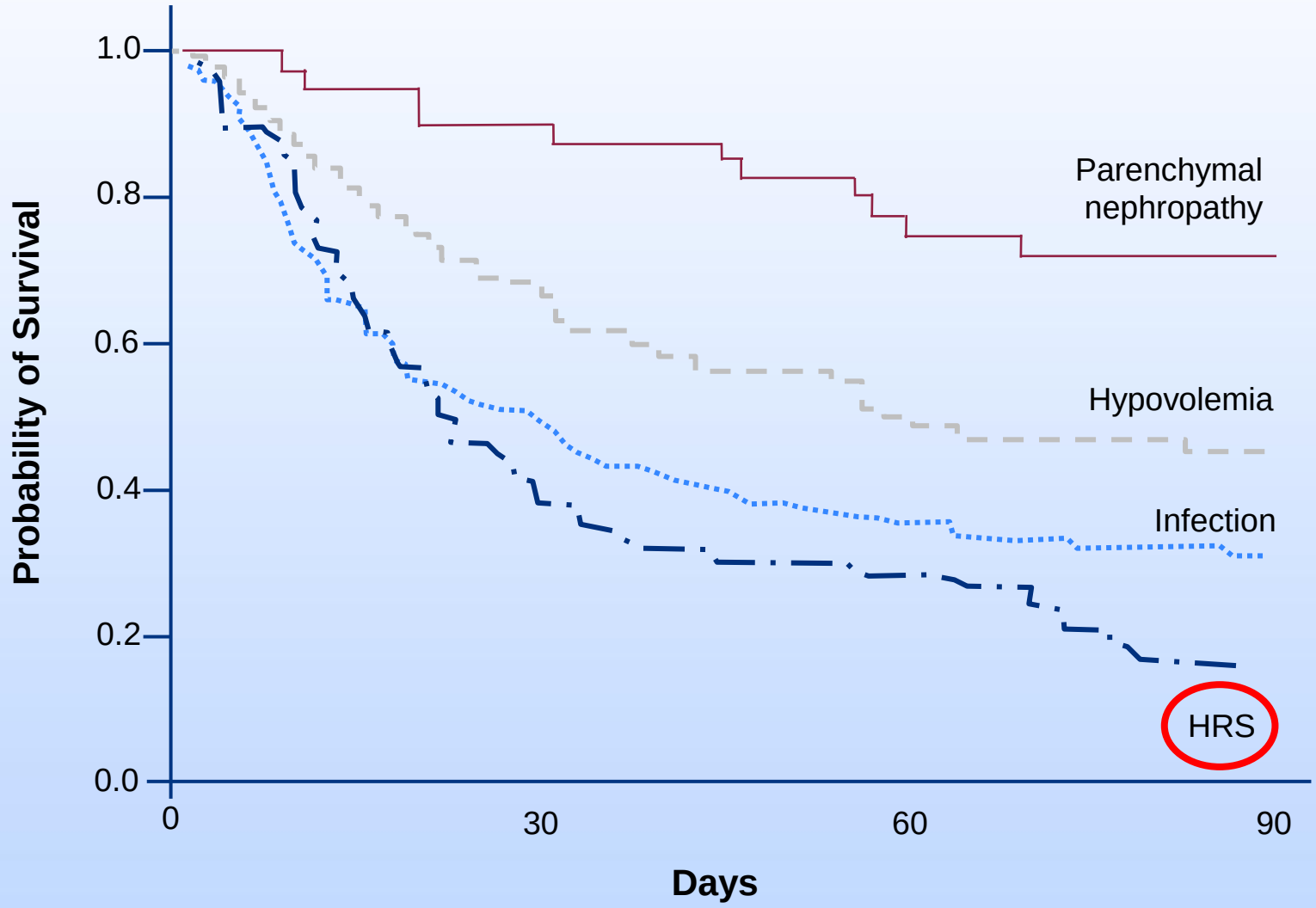
# AKI HRS Is Associated With the Highest Mortality




Martin-Llahi M, et al. *Gastroenterology.* 2011;140:488-496

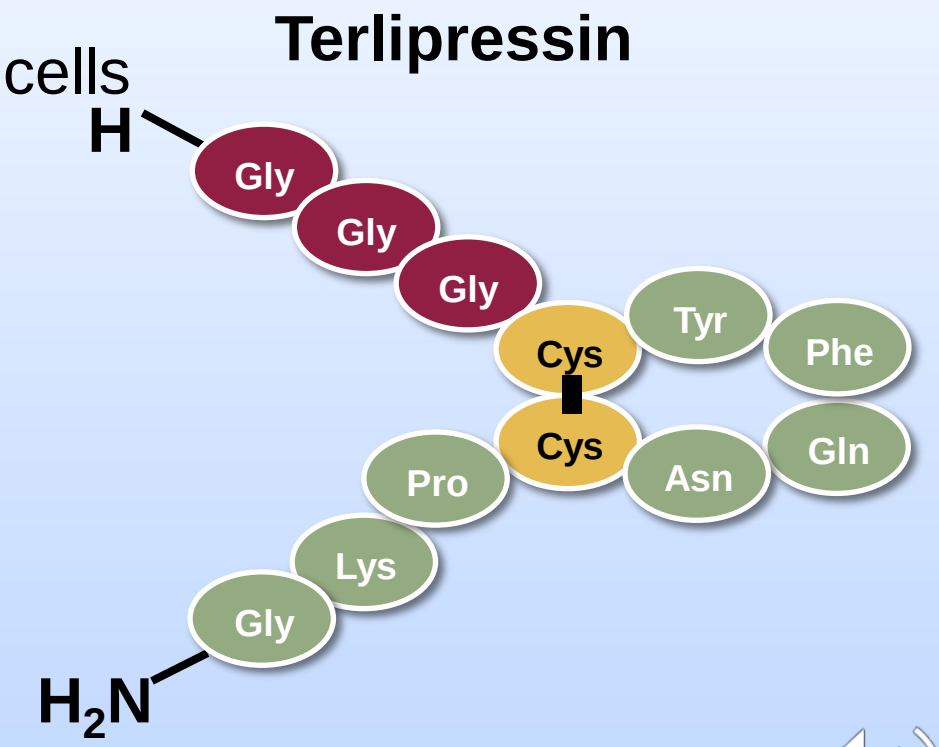
# HRS Therapy


- Treatment Depends on Type of AKI
- **HRS** should be treated with volume expansion, vasoconstrictors, and liver transplant, but not typically with dialysis
- Goal of therapy is to diagnose early (increase in creatinine
  ≥0.3 mg/dL) and treat early to prevent progression
- Avoid nephrotoxic drugs
- Albumin infusion + midodrine is recommended
  - 25g of 25% Q6-8hrs
  - Midodrine 5-15 mg po TID
- Terlipressin has been FDA approved
- Expedited consult for liver transplantation is recommended

# Terlipressin

- Synthetic 12-amino acid peptide
- Prodrug, with pharmacologic activity of its own
- Constrictive activity via V1 receptors
  - Vascular & extravascular smooth muscle cells
- Splanchnic vasoconstriction
  - ↓ portal flow
  - ↓ portal pressure
- Systemic vasoconstriction
  - ↑ effective blood volume
  - ↓ renin and angiotensin
  - → renal vasodilatation
  - → improvement in serum creatinine



# Critical Terlipressin Studies

| Study | Definition | n | Terlipressin Dose | Duration | Albumin | HRS Reversal |
|---|---|---|---|---|---|---|
| Martin-Llahi<br><br>2008 | IAC 1996 | Terli 23<br>Pbo 23 | 1 mg q4h, increased to 2 mg q4h if SCr not decreased by 25% | 15 d | 1 g/kg first 24h, 40 g/day (CVP dependent) | 39% vs 4.3% |
| Sanyal<br><br>2008 | IAC 1996 | Terli 56<br>Pbo 56 | 1 mg q6h increased to 2 mg q6h if SCr not decreased by 25% | 14 d | 100 g on day 1, 25 g/day thereafter (optional, 88% in each group received) | 25% vs 12.5% |
| Boyer<br><br>2016 | ICA 2007<br>Excluded SCr ≥7 mg/dL | Terli 93<br>Pbo 95 | 1 mg q6h, could increase to 2 mg q6h (no more than 8 mg/24h if SCr not <70% of baseline | 14 d | 20–40 g/day (optional)<br><br>May have received 1 gm/kg at pretrial | 19% vs 13% |

CVP, central venous pressure.

Martin-Llahi M, et al. *Gastroenterology* 2008;140:488-496; Sanyal [illegible], et al. *Gastroenterology* 2008;134:[illegible]-1368;
Boyer TD, et al. *Gastroenterology.* 2016;150:151[illegible]-1529

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# Last but not least…

## Hepatopulmonary Syndrome

- Combination of liver disease and intrapulmonary microvascular dilation causing hypoxia
- Intrapulmonary shunt noted on echocardiogram + PaO2 on arterial blood gas (ABG) is ≤ 60 mm Hg
- Pts. with SpO2 <96% need ECHO with shunt study AND repeat ABG on 100% O2
- A PO2 on room air <50 has significant morbidity in the post transplant setting
- May require home oxygen
- May have pleural effusions/Hepatic hydrothorax

## Portopulmonary Syndrome

- a severe, local hypertensive complication that can result from pulmonary vasoconstriction in patients with cirrhosis and portal hypertension; portopulmonary hypertension can progress to right heart failure and death.
- -moderate to severe portopulmonary hypertension is associated with increased post-transplant mortality, but it can be considered in selected situations, if pulmonary artery pressures have been lowered to less than 35 mm Hg with vasodilator therapy.
- -For RVSP ≥45 mm Hg right heart cardiac catheterization is indicated.

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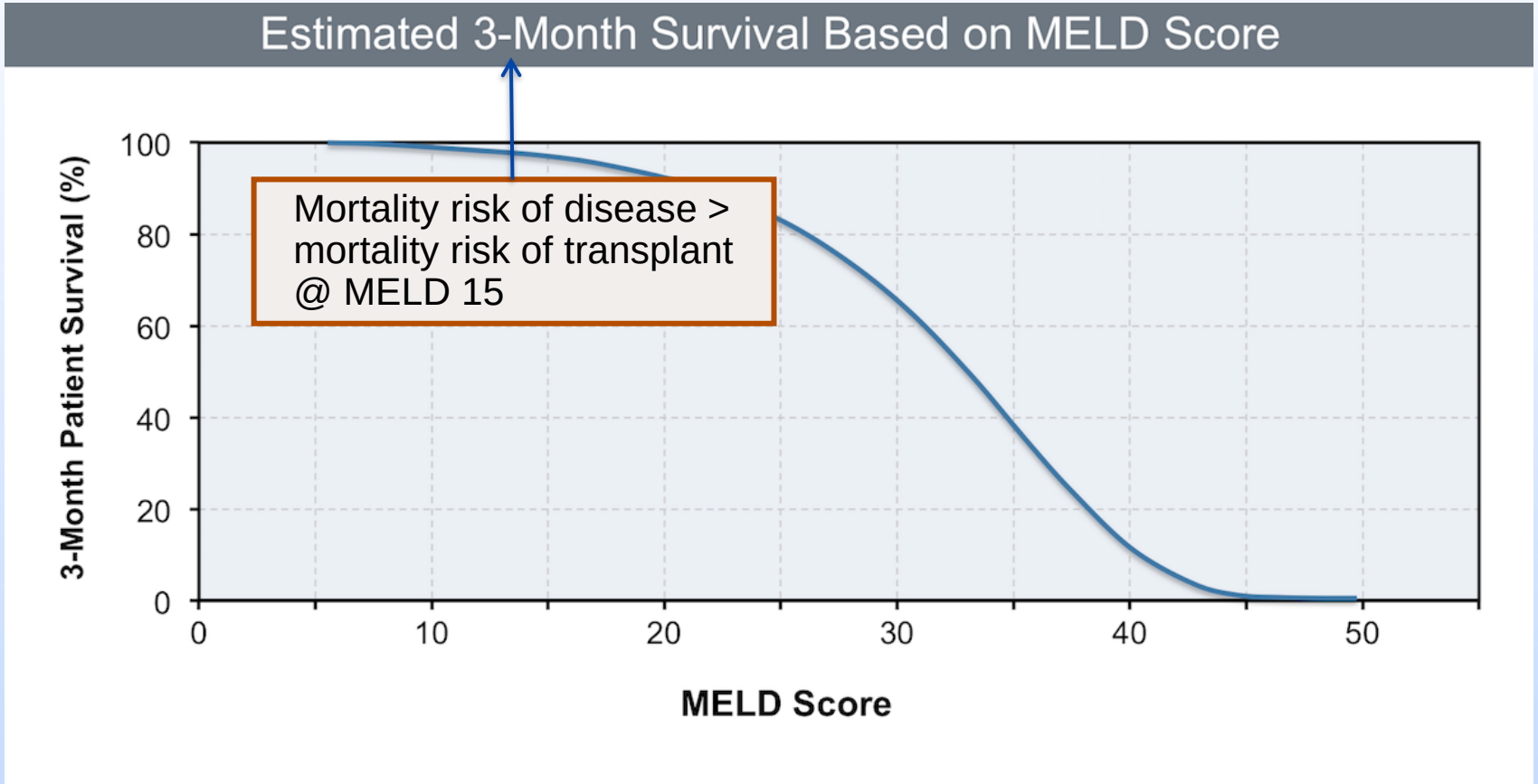
# So let's talk referral for transplant!!




Martin P, DiMartini A, Feng S, Brown R Jr, Fallon M. Evaluation for liver transplantation in adults: 2013 practice guideline by the American Association for the Study of Liver Diseases and the American Society of Transplantation. Hepatology. 2014;59:1144-65.

# Indications for Transplant Referral

- ESLD with complications related to decompensation
  - Ascites, HE, Variceal Hemorrhage
- Hepatopulmonary Syndrome (HPS)
  - Pulmonary microvascular dilatation with shunting
- Portopulmonary Hypertension (POPH)
  - Pulmonary vasoconstriction with increased RVSP
- AKI-including *all causes* of acute deterioration of renal function
  - increase in serum creatinine of >50% from baseline, or a rise in serum creatinine ≧0.3mg/dl in <48 hrs
    - Sepsis, hypovolemia, parenchymal renal disease
    - Hepatorenal Syndrome

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# Indications for Transplant Referral

- Decompensated Primary Biliary Cholangitis
  - Cases with severe pruritus
- Decompensated Primary Sclerosing Cholangitis
  - ICU status ≥2 times in a 3-month period
  - Cirrhotic
  - Highly resistant organism
  - Stricture nonresponsive to PTC/ERCP
- Alcohol related liver disease
  - 6-month sobriety (monitored) or exceptions pathway
- Acute Alcoholic Hepatitis
  - MELD>20, DF score >32
  - non responder to steroid therapy utilizing the Lille score
  - No prior complications of HE, Ascites, GI bleed, jaundice
  - Accepts ETOH as the causation of liver disease
- Decompensated Autoimmune Hepatitis
  - 9% fail therapy connotated by clinical, laboratory, and histological deterioration despite compliance with conventional treatment schedule

# Indications for Transplant Referral

- Hepatocellular Carcinoma
  - Milan Criteria
  - UCSF Criteria
  - Downstaging Protocol
- Cholangiocarcinoma



 

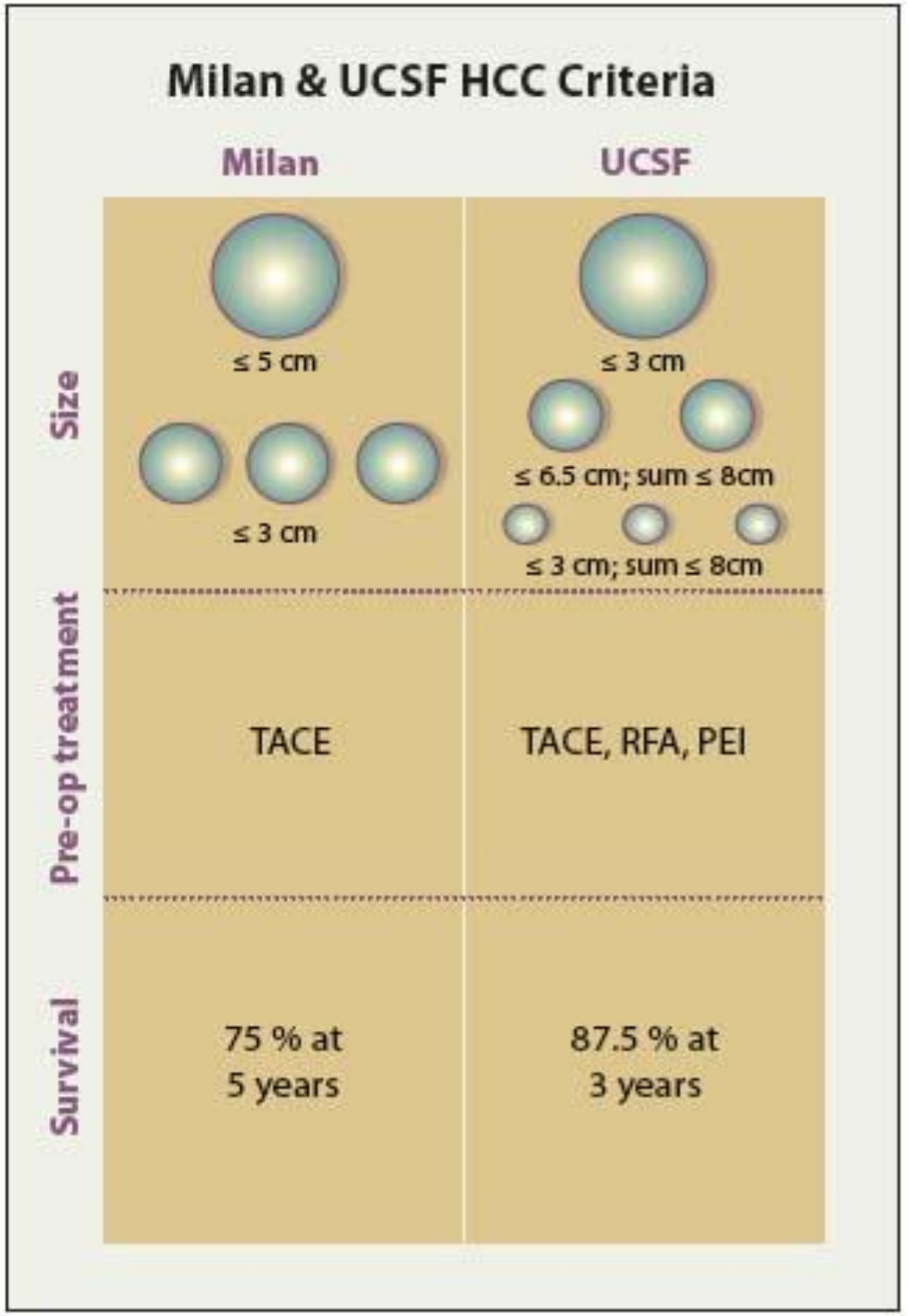


**Figure 2: Comparison of the Milan Criteria and the University of California San Francisco (USCF) Criteria for Orthotopic Liver Transplantation in Patients With Hepatocellular Carcinoma (HCC)**—PEI = percutaneous ethanol injection; RFA = radiofrequency ablation; TACE = transarterial chemoembolization.

Hanish,S., Knechtle, S. (2011). Liver transplantation for the treatment of hepatocellular carcinoma.*Oncology 25(8),* pp.1-2.

# Milan Criteria

- Assigned a MELD priority score of 22.
- Diagnosis based on cross-sectional imaging with the following radiological characteristics diagnostic of HCC:
- contrast enhancement on the late arterial phase with either washout on portal venous phase, pseudocapsule enhancement or growth on serial studies, or consistent biopsy confirming a tissue diagnosis of HCC.
- The tumor must not be amenable to resection and metastatic spread needs to have been excluded by a chest CT and bone scan.



# Milan Criteria

- The tumor dimensions need to be confirmed by an magnetic resonance imaging (MRI) or CT scan interpreted by a radiologist at an OPTN-approved center
- The assigned MELD score currently increases every 3 months consistent with a 10% increase in candidate mortality until the patient is either transplanted or progresses beyond Milan criteria based on serial imaging.

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# OLT Options for Cholangiocarcinoma

20% of pts. with CCA are R0 candidates

Upper Extrahepatic Hilar CCA between cystic duct and the secondary branches

Prior CCA protocol 2yr. Survival rate 50%, 5 yr. <30%

Now survival rates are 90%-1 yr., 88%-3yr., 71%- 5yr.

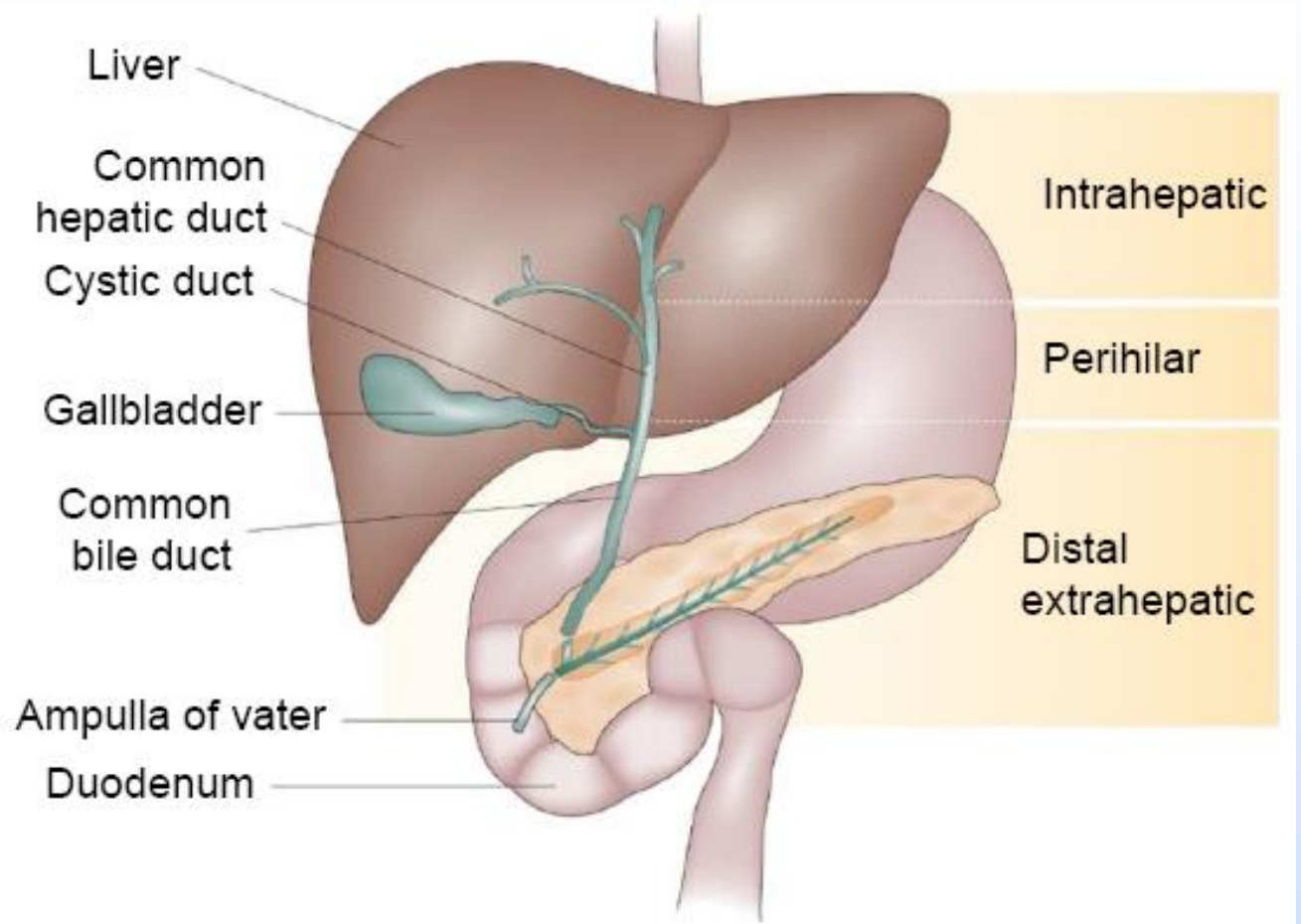


**Figure 1** Types of cholangiocarcinoma.
**Note:** Adapted by permission from Macmillan Publishers Ltd: *Nat Rev Gastroenterol Hepatol*. Blechacz B, Komuta M, Roskams T, Gores GJ. Clinical diagnosis and staging of cholangiocarcinoma. 2011;8(9):512–522. Copyright 2011.[134]

# OLT Options for Cholangiocarcinoma

| Exclusion Criteria for OLT in CCA |
| --- |
| Intrahepatic CCA |
| Evidence of extrahepatic disease |
| Prior radiation or chemotherapy |
| Prior biliary resection or attempted resection |
| Intrahepatic metastases |
| Uncontrolled infection |
| Hx of other malignancy within 5 years |
| Hx of Transperitoneal biopsy (including percutaneous and EUS guided FNA) |



 

# Conditions that Qualify for MELD Exception Points

| | |
|---|---|
| Hepatocellular Carcinoma | T2 lesions (at least 2 cm in diameter, within Milan Criteria |
| Hepatopulmonary Syndrome | PaO2 < 60mmHg on room air |
| Portopulmonary Hypertension | Mean PAP <35 with treatment |
| Familial amyloid polyneuropathy | Confirmed by nucleic acid analysis and histology |
| Primary hyperoxaluria | Combined liver/kidney |
| Cystic fibrosis | FEV1<40% |
| Hilar cholangiocarcinoma | Stage I or II |
| Hepatic Artery Thrombosis | Within 14 days of Tx, not meeting criteria for status 1A |

Carrion, A., Martin,P. (2019). When to refer for liver transplantation. *Am J Gastroenterol* 114(7),114-117.https://doi.org/10.1038/s41395-018-0242-1

# Indications for Transplant Referral

- Acute Liver Failure
  - Occurring without pre-existing liver disease
  - Hospitalized in the ICU with onset of HE within 56 days of the first sign/symptom of liver injury with at least one of the following:
    - Ventilator dependent
    - Requires iHD/CRRT
    - Development of coagulopathy [INR] greater than or equal to 2.0

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# Palliative Care and Cirrhosis

- Discussion of:
  - Goals of care
  - Code status
  - Advanced directives/Healthcare surrogates
- Associated with cost savings (especially in the inpatient setting)
- Early integration can decrease depression and anxiety of caregivers
- Incorporate principles of palliative medicine into practice to assess and address the patient's suffering at every encounter

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# In Summary

- Early identification of risk factors for fibrosis is key to preventing ESLD when possible
- Follow most current EBP for managing complications of portal HTN
- Refer early based on MELD-Na or worsening clinical presentation
- Hepatocellular carcinoma should lead to a transplant evaluation- don’t biopsy if you are unsure!
- Don’t trust the recovery with ALF, refer!
- Don’t forget the significance of early palliative care

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# Questions & Discussion

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