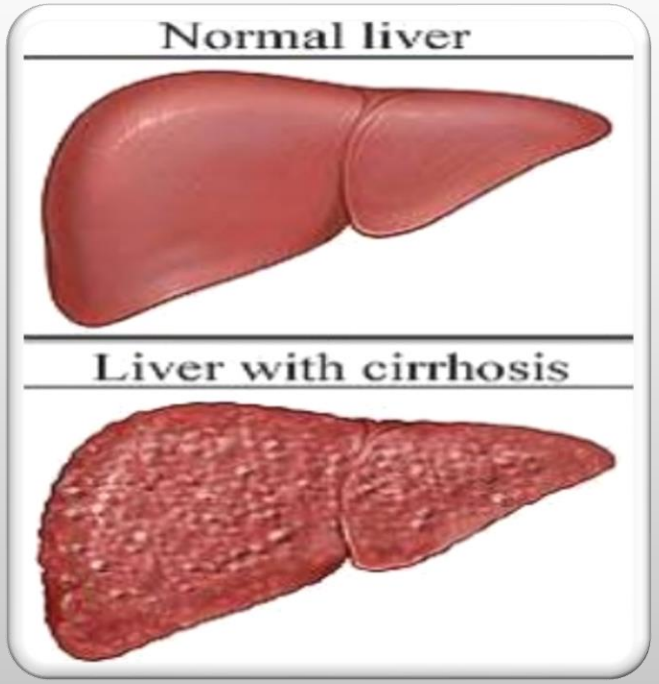
MANAGEMENT OF CHRONIC LIVER DISEASE



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LECTURE OUTCOMES

BY THE END OF THIS LECTURE YOU SHOULD BE ABLE TO:

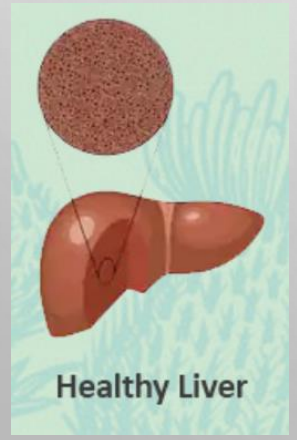
1. Describe the Pathophysiology of chronic liver disease.
2. List the common causes of chronic liver disease.
3. Name the different symptoms of chronic liver disease based on the four different pathological processes.
4. Explain the mechanism of ascites in chronic liver disease.
5. List the common findings and studies needed to diagnose chronic liver disease based on H&P, Labs, Imaging and procedures.
6. Differentiate between the common classifications used in Cirrhosis.
7. Differentiate between compensated and decompensated cirrhosis and the significance of each in the prognosis of liver disease.
8. List the main points in management of chronic liver disease.
9. Explain how to treat common symptoms in chronic liver disease.
10. Name the common complications of chronic liver disease and how to treat and prevent each of them.
11. Describe what a TIPS procedure is and list the common indications and possible complications that might result from it.

OUTLINES

- Pathophysiology of chronic liver disease
- Common causes of chronic liver disease
- Presenting symptoms
- Diagnosis of chronic liver disease
- Commonly used Classifications
- Course of Cirrhosis
- Management of patients with CLD
- Chronic liver disease complications
- Prevention of complications
- TIPS

PATHOPHYSIOLOGY

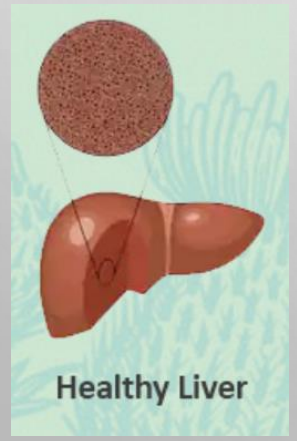
Normal Liver → Acute inflammation → Regeneration → Complete recovery

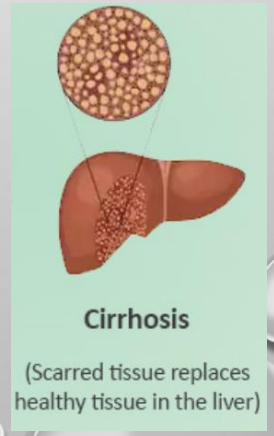


PATHOPHYSIOLOGY

Normal Liver → Acute inflammation → Regeneration → Complete recovery

Normal Liver → Recurrent /Chronic Inflammation → Necrosis → Fibrosis → Scarring → Cirrhosis





ETIOLOGY OF CHRONIC LIVER DISEASE

Q1. All of the following can cause cirrhosis except:

(can select more than one answer)

A. Hepatitis B
B. Wilson’s disease
C. Autoimmune hepatitis
D. Alcoholic hepatitis
E. Hepatitis E
F. Heart failure

ETIOLOGY

Infections	Hepatitis B, Hepatitis C, Schistosomiasis
Toxins	Alcohol, Herbal
Metabolic	Hemochromatosis, Wilson's, Alpha-1 Antitrypsin def, Amyloidosis, NASH
Autoimmune	Autoimmune Hepatitis, PSC, PBC
Vascular	Budd-Chiari Syndrome
Cardiac	Heart Failure (Congestive hepatopathy)

SYMPTOMS OF CHRONIC LIVER DISEASE

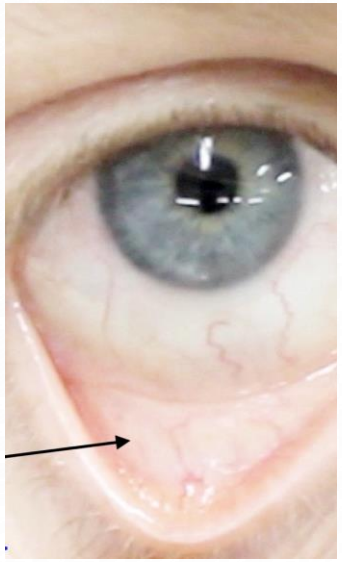
Symptoms secondary to LIVER DAMAGE	Symptoms secondary to PORTAL HYPERTENSION

Hepatic insufficiency

High Estrogen

Portal hypertension effect

Hypersplenism



SYMPTOMS OF CHRONIC LIVER DISEASE

Symptoms secondary to LIVER DAMAGE	Symptoms secondary to PORTAL HYPERTENSION
Jaundice	
Encephalopathy	
Ascites/Ankle edema	
Anemia	
Bleeding tendency	

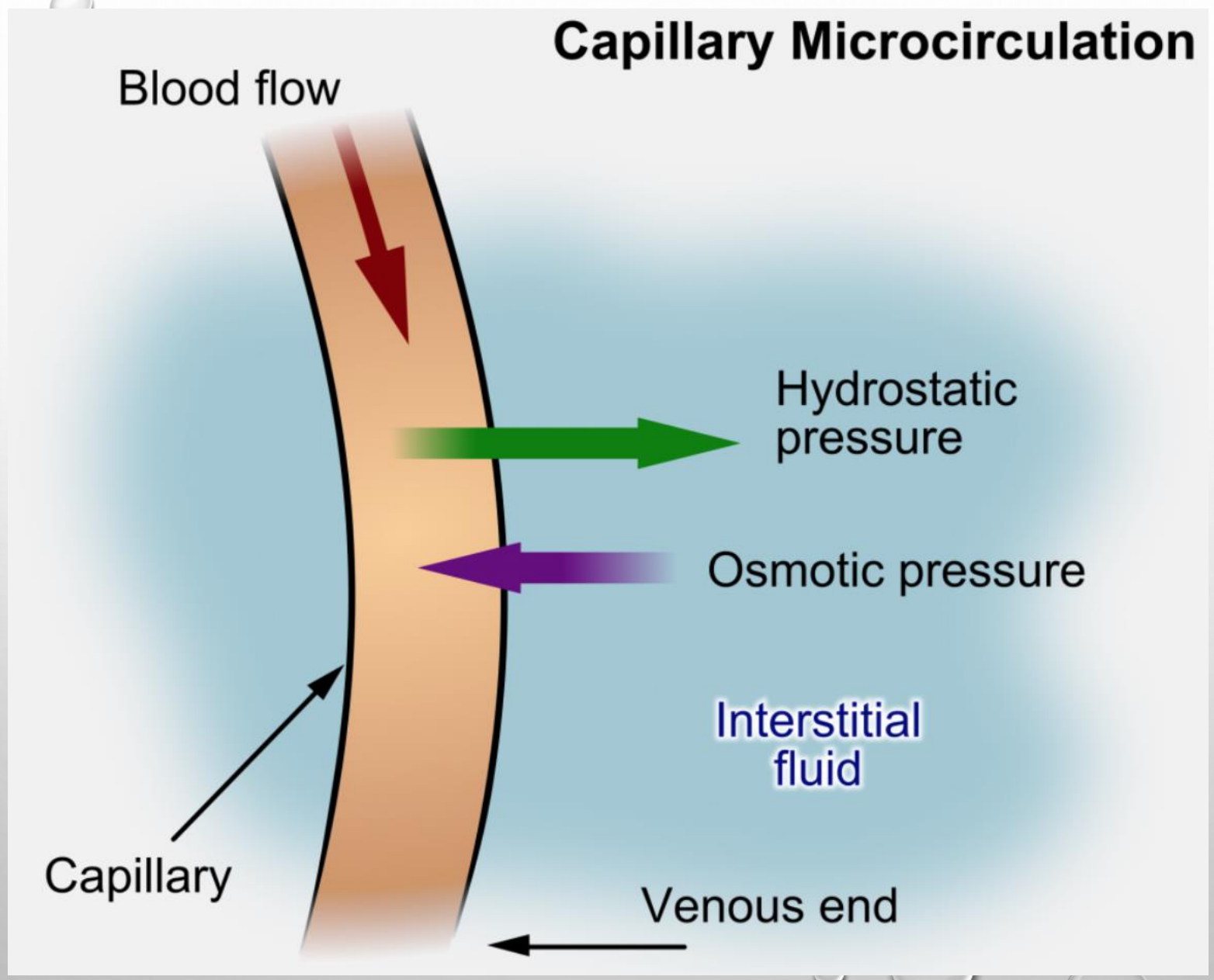
Hepatic insufficiency

High Estrogen

Portal hypertension effect

Hypersplenism

What causes ascites in chronic liver disease?


Capillary Microcirculation
Blood flow
Hydrostatic pressure
Osmotic pressure
Interstitial fluid
Capillary
Venous end

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SYMPTOMS OF CHRONIC LIVER DISEASE

Symptoms secondary to LIVER DAMAGE	Symptoms secondary to PORTAL HYPERTENSION
Jaundice	
Encephalopathy	
Ascites/Ankle edema	
Anemia	
Bleeding tendency	
Spider nevi	
Gynecomastia (males)	
Testicular atrophy (males)	
Palmar erythema	

Hepatic insufficiency

High Estrogen

Portal hypertension effect

Hypersplenism

SYMPTOMS OF CHRONIC LIVER DISEASE

Symptoms secondary to LIV	Symptoms secondary to ERTENSION
	edusa
E	egaly
Asc	e edema
	Varices
Bl	arices
	rices
Gyn	
Testicu	
Palmar erythema	

Hepatic insufficiency

High Estrogen

Portal hypertension effect

Hypersplenism

SYMPTOMS OF CHRONIC LIVER DISEASE

Symptoms secondary to LIVER DAMAGE	Symptoms secondary to PORTAL HYPERTENSION
Jaundice	Caput Medusa
Encephalopathy	Splenomegaly
Ascites/Ankle edema	Ascites/ankle edema
Anemia	Esophageal Varices
Bleeding tendency	Gastric Varices
Spider nevi	Rectal Varices
Gynecomastia (males)	Anemia
Testicular atrophy (males)	Leukopenia
Palmar erythema	Thrombocytopenia

Hepatic insufficiency

High Estrogen

Portal hypertension effect

Hypersplenism

Q2. Which of the following is not a sign of portal hypertension? **(can select more than one answer)**
A. Esophageal Varices
B. Caput Medusa
C. Spider nevi
D. Jaundice
E. Ascites
F. Splenomegaly

DIAGNOSIS

H&P	
Labs	
Imaging	
Procedures	

DIAGNOSIS

H&P	Symptoms suggestive of chronic liver disease Signs suggestive of chronic liver disease
Labs	
Imaging	
Procedures	

DIAGNOSIS

H&P	Symptoms suggestive of chronic liver disease Signs suggestive of chronic liver disease
Labs	Low platelets Low albumin High INR
Imaging	
Procedures	

DIAGNOSIS

H&P	Symptoms suggestive of chronic liver disease Signs suggestive of chronic liver disease
Labs	Low platelets Low albumin High INR
Imaging	Cirrhotic liver (nodular, shrunken) Splenomegaly
Procedures	

DIAGNOSIS

H&P	Symptoms suggestive of chronic liver disease Signs suggestive of chronic liver disease
Labs	Low platelets Low albumin High INR
Imaging	Cirrhotic liver (nodular, shrunken) Splenomegaly
Procedures	Liver biopsy Fibroscan

ASCITES

- ANY ASCITES NEEDS TAPPING TO DETERMINE THE CAUSE OF ASCITES. HOW?

SAAG

SERUM ALBUMIN ASCITES GRADIENT

>/=1.1 g/dl PORTAL HYPERTENSION	<1.1 g/dl NON PORTAL HYPERTENSION

SAAG

SERUM ALBUMIN ASCITES GRADIENT

>/=1.1 g/dl PORTAL HYPERTENSION	<1.1 g/dl NON PORTAL HYPERTENSION
Chronic Liver disease	Nephrotic syndrome
Budd-Chiari Syndrome	Peritoneal Tuberculosis
Congestive heart failure	Pancreatitis
	Peritoneal carcinomatosis (non-hepatic malignancy)

Protein >/=2.5 g/dl	Protein <2.5 g/dl	Protein >2.5 g/dl	Protein <2.5g/dl

SAAG

SERUM ALBUMIN ASCITES GRADIENT

>/=1.1 g/dl PORTAL HYPERTENSION	<1.1 g/dl NON PORTAL HYPERTENSION
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Protein >/=2.5 g/dl	Protein <2.5 g/dl	Protein >2.5 g/dl	Protein <2.5g/dl
Congestive heart failure	Chronic Liver disease	Pancreatitis TB	Nephrotic Syndrome
Budd-Chiari Syndrome		Cancinomatosis	

COMMON CLASSIFICATIONS USED

1. **CHILD- PUGH CLASSIFICATION :**

- BILIRUBIN, ALBUMIN, PT, ASCITES AND ENCEPHAOPATHY.
- CHILD A (MILD COMPENSATED)
- CHILD B AND CHILD C (DECOMPENSATED)

2. **MELD SCORE (MODEL FOR END STAGE LIVER DISEASE)**

- PROGNOSIS/ LIVER TRANSPLANT

COURSE OF CIRRHOSIS

Compensated Cirrhosis

Decompensated Cirrhosis

COURSE OF CIRRHOSIS

Compensated Cirrhosis

Asymptomatic

Usually incidental finding

Decompensated Cirrhosis

Cirrhosis with symptoms

(Any of the following):

1. Ascites
2. Bleeding Varices
3. Hepatic Encephalopathy
4. Jaundice

MANAGEMENT

MANAGEMENT

1. **Determine the etiology of chronic liver disease/cirrhosis and treat in order to slow or reverse the progression of liver disease.**

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2. **Prevent superimposed insult to the liver (Vaccinations, hepatotoxic medications, Alcohol).**

MANAGEMENT

1. Determine the etiology of chronic liver disease/cirrhosis and treat in order to slow or reverse the progression of liver disease.

2. Prevent superimposed insult to the liver (Vaccinations, hepatotoxic medications, Alcohol).

3. **Treat symptoms.**

TREATMENT OF ASCITES

1. Salt Restriction (<2g/day)
2. Diuretics (Furosemide and Spironolactone).
3. Paracenthesis +/- albumin (When to give Albumin?)
4. TIPS

MANAGEMENT

1. Determine the etiology of chronic liver disease/cirrhosis and treat in order to slow or reverse the progression of liver disease.

2. Prevent superimposed insult to the liver (Vaccinations, hepatotoxic medications, Alcohol).

3. Treat symptoms.

4. **Prevent and treat complications.**

COMPLICATIONS OF CIRRHOSIS

1. Hepatic Encephalopathy
2. Spontaneous bacterial peritonitis
3. Bleeding Varices
4. Hepatorenal syndrome
5. Hepatopulmonary syndrome
6. Hepatocellular carcinoma

1. HEPATIC ENCEPHALOPATHY

- Reversible neuropsychiatric symptoms in the presence of liver disease.
- 4 Grades

1. HEPATIC ENCEPHALOPATHY

- **Treat encephalopathy**

- **Treat underlying cause**

1. Dehydration
2. Electrolytes imbalance
3. Infection
4. Bleeding
5. Constipation
6. Medications causing confusion/hepatic encephalopathy.
7. Poor compliance with medications.

2. SPONTANEOUS BACTERIAL PERITONITIS

1. <u>How to diagnose?</u>

Ascitic fluid: WBC >500 and Neutrophils >250

Usually one microbe (gram negatives)- E-coli or klebsiella

If multiple organisms think or secondary peritonitis.

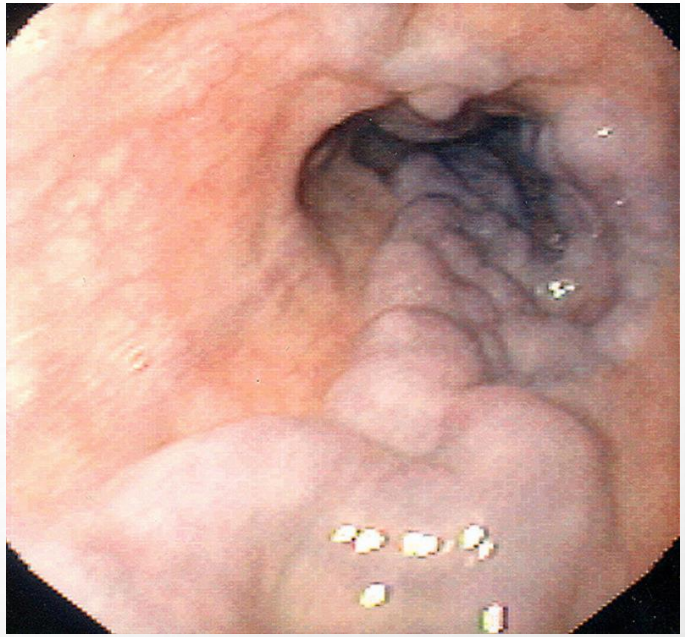
2. <u>Antibiotics</u>

3. <u>Albumin?</u>

1.5g/kg on day 1 and 1g/kg day 3 >> Prevent HRS

3. BLEEDING VARICES

1. Stabilization +/- Intubation.
2. Transfuse as needed
3. Antibiotics
4. Octreotide drip (why?)
5. Endoscopy with intervention
6. Sengstaken–Blakemore tube
7. TIPS



4. HEPATORENAL SYNDROME

- Systemic vasodilation, renal vasoconstriction and decrease flow.
- Diagnosis?

- **Types:**

1. Type 1 (bad!, rapid worsening over 2 weeks).
2. Type 2 (gradual stable worsening)

- **Treatment:**

a) Midodrine (Alpha 1 agonist = systemic vasoconstriction)
b) Octreotide (somatostatin analogue prevent splanchnic vasodilation)
c) Albumin
d) Liver transplant

5. HEPATOPULMONARY SYNDROME

- Pulmonary complication of portal hypertension
- Pulmonary vasodilatation that leads to gas exchange abnormalities and hypoxemia
- Shortness of breath on exertion to severe hypoxemia requiring oxygen.

- **Diagnosis:**

1. Exclusion.
2. PA02 <80 mmHg
3. or alveolar arterial oxygen gradient A-a gradient >15 mmHg on ABG.
4. ECHO or perfusion lung scan showing intrapulmonary vasodilatation.

- **Treatment:**

1. limited.
2. Liver transplantation

5. HEPATOCELLULAR CARCINOMA

- As per size, number and stage

- **Therapy options:**

1. Transplant
2. Surgery
3. TACE/TARE
4. Oral chemotherapy (Sorafinib)
5. Palliative

COMPLICATIONS OF CIRRHOSIS

1
2
3
4

Aim Is Prevention/Early Detection !!!!!

Hepatic Encephalopathy	
SBP	
Bleeding Varices	
Hepatorenal syndrome	
HCC	

Hepatic Encephalopathy	• Avoid Precipitating Factors • Prophylactic Lactulose/Rifaximin
SBP	
Bleeding Varices	
Hepatorenal syndrome	
HCC	

Hepatic Encephalopathy	• Avoid Precipitating Factors • Prophylactic Lactulose/Rifaximin
SBP	• Control Ascites • Prophylactic Antibiotics if indicated
Bleeding Varices	
Hepatorenal syndrome	
HCC	

Hepatic Encephalopathy	• Avoid Precipitating Factors • Prophylactic Lactulose/Rifaximin
SBP	• Control Ascites • Prophylactic Antibiotics if indicated
Bleeding Varices	• Screening Upper endoscopy followed by appropriate surveillance • Prophylactic Banding Vs B.blockers
Hepatorenal syndrome	
HCC	

Hepatic Encephalopathy	• Avoid Precipitating Factors • Prophylactic Lactulose/Rifaximin
SBP	• Control Ascites • Prophylactic Antibiotics if indicated
Bleeding Varices	• Screening Upper endoscopy followed by appropriate surveillance • Prophylactic Banding Vs B.blockers
Hepatorenal syndrome	• Avoid factors that might precipitate or worsen kidney function (Nephrotoxic medication, contract, dehydration, Excessive diuretics)
HCC	

Hepatic Encephalopathy	• Avoid Precipitating Factors • Prophylactic Lactulose/Rifaximin
SBP	• Control Ascites • Prophylactic Antibiotics if indicated
Bleeding Varices	• Screening Upper endoscopy followed by appropriate surveillance • Prophylactic Banding Vs B.blockers
Hepatorenal syndrome	• Avoid factors that might precipitate or worsen kidney function (Nephrotoxic medication, contract, dehydration, Excessive diuretics)
HCC	• Screening liver ultrasound Q6 months +/- AFP

REFER FOR LIVER TRANSPLANT

MANAGEMENT

1. Determine the etiology of chronic liver disease/cirrhosis and treat in order to slow or reverse the progression of liver disease.

2. Prevent superimposed insult to the liver (Vaccinations, hepatotoxic medications, Alcohol).

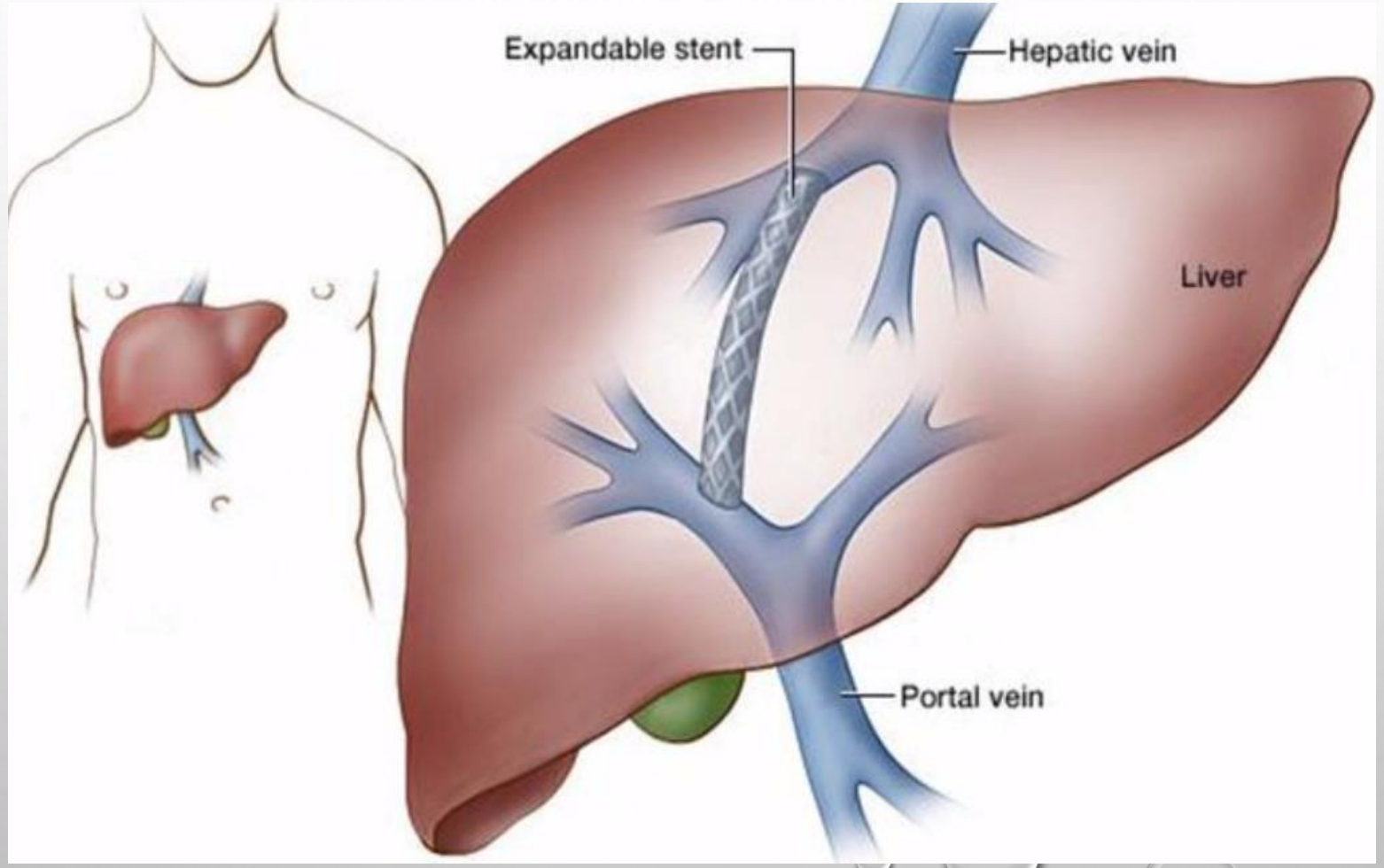
3. Treat symptoms.

4. Prevent and treat complications.

5. **Refer to liver transplant when appropriate.**

TIPS

TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT



TIPS

INDICATIONS:

1. Refractory Ascites
2. Uncontrolled variceal bleeding (Esophageal, Gastric)

COMPLICATIONS:

1. Bleeding
2. Fever
3. Hepatic Encephalopathy.
4. Worsening renal function.

Pearls for your exam

- A liver biopsy is not always needed to make the diagnosis of cirrhosis.
- Upper endoscopy should be done in any patient with cirrhosis to screen for varices.
- First-line therapy for patients with cirrhosis admitted with GI hemorrhage consists of resuscitation (avoiding over transfusion and keeping hemoglobin ~7-8), antibiotics and infusion of a splanchnic vasoconstrictor (Octreotide).
- Ascites is the most common decompensating event in cirrhosis. Most will respond to diuretics (spironolactone alone or in combination with furosemide).
- SBP is diagnosed with an ascites absolute neutrophil count (not total WBC count) greater than 250/mm3. Diagnostic paracentesis should be performed in any patient with cirrhosis that deteriorates.

- When to use antibiotic prophylaxis in patients with cirrhosis?

1) Admitted with GI hemorrhage (short-term prophylaxis)
2) Patients who have recovered from an episode of SBP.

- Patients with cirrhosis are predisposed to develop acute kidney injury. At diagnosis, discontinue diuretics, vasodilators and lactulose, panculture, and expand intravascular volume with albumin.

- When to use albumin in cirrhotic patients?

1) Large volume paracentesis (>5L)
2) SBP
3) AKI

- Hepatic encephalopathy is usually precipitant-induced. The key is to identify and treat the precipitant.

- A patient with cirrhosis should be referred to a transplant center at their first decompensation regardless of MELD score.

REFERENCES

- NETTER'S GASTROENTEROLOGY, 2ND EDITION, MARTIN FLOCH.
- EUROPEAN ASSOCIATION FOR THE STUDY OF LIVER CLINICAL PRACTICE GUIDELINES FOR THE MANAGEMENT OF PATIENTS WITH DECOMPENSATED CIRRHOSIS. JOURNAL OF HEPATOLOGY 2018 VOL. 69 J 406–460.
- AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASE (AASLD) PRACTICE GUIDELINES IN MANAGEMENT OF ASCITES AND HEPATIC ENCEPHALOPATHY. 2013-2014.
- CIRRHOSIS: DIAGNOSIS, MANAGEMENT, AND PREVENTION, AM FAM PHYSICIAN. 2011 DEC 15;84(12):1353-1359.
- 2019 AGA- DDSEP-9

When patients with chronic liver disease present with ascites which of the following is true?

A. Diagnostic paracentesis should always be done at presentation and at any recurrence.
B. Therapeutic paracentesis is a safe and effective therapy in tense and symptomatic ascites with no need for albumin.
C. When using diuretics, a vigorous diuretic response of more than 1L negative balance daily should be the aim.
D. Variceal hemorrhage is more common.
E. A white count of more than 250 indicates peritonitis.

A 38 year-old alcoholic presents with new-onset ascites. Diagnostic paracentesis reveals a cloudy fluid with total protein of 1.5 g/dl, SAAG of 1.6 g/dl, triglyceride 450 mg/dl, and cell count 350 WBC (9% PMN). The most likely etiology for his ascites is:

a. alcoholic cirrhosis
b. abdominal lymphoma
c. alcoholic cardiomyopathy
d. peritoneal tuberculosis

A 32 year-old man with cirrhosis due to autoimmune hepatitis is admitted to the hospital with spontaneous bacterial peritonitis. He is hemodynamically stable, on no diuretics, and does not take NSAIDS.
He is treated with broad-spectrum IV antibiotics, IV fluids, and IV albumin on admission. At the time of admission, his serum creatinine is 1.2. On day 5 of his hospitalization, his serum creatinine is 4.1, and he is anuric. He is diagnosed with hepatorenal syndrome. Which of the following is true?

A. He has type II hepatorenal syndrome and should be evaluated for a combined liver/kidney transplant.

B. He has type I hepatorenal syndrome, and should be treated with daily IV albumin, octreotide, and midodrine, as a bridge to a liver transplant

C. After a liver transplant, his renal function is unlikely to fully recover.

D. Pharmaceutical management involves vasodilator therapy, with the goal of improving renal perfusion.

A 63-year-old man with a history of hepatitis C is seen in your office for evaluation of new diagnosis of cirrhosis. He has had longstanding hepatitis C for 30 years and recently achieved cure with 12 weeks of direct acting antiviral therapy. He was referred to you after seeing a hematologist for thrombocytopenia. He is asymptomatic. His exam is notable for spider angiomata, a non-distended abdomen, and palmar erythema. His most recent labs include a total bilirubin of 1.2 mg/dL, AST 24 U/L, ALT 22 U/L, albumin of 3.6 g/dL, INR 1.0, and a platelet count of 145,000/µL.

Which of the following would be included in this patient's management?

a) Liver biopsy
b) Abdominal ultrasound
c) Vitamin E
d) Diuretics
e) Weight loss

A 53-year-old woman with longstanding alcohol use presents to your office for management of new diagnosis of cirrhosis. She continues to drink two beers daily. She has no complaints of abdominal pain and has no other significant medical history. She is taking thiamine and folic acid. Her vital signs are stable and she does not have ascites but has palmar erythema. Her labs include AST 300 U/L, ALT 150 U/L, total bilirubin of 1.2 mg/dL, albumin of 3.0 g/dL, INR 1.2, and a platelet count of 125,000/µL. She had a recent ultrasound that was negative for liver cancer. What is the next best step in management?

a) Observation
b) Beta-blocker
c) Upper endoscopy
d) Computerized tomography
e) Liver biopsy

What is the most likely mechanism responsible for beta-blockers decreasing risk of variceal bleed?

a) Splanchnic vasodilatation
b) Reduction in portal pressure
c) Rise in cardiac output
d) Enhancement of bacterial translocation

A 55-year-old man with NASH cirrhosis presents to the endoscopy suite for variceal screening. He has longstanding asthma controlled with inhaled corticosteroids. Vitals include T 98.0o F, BP of 88/60 mmHg, HR 70 beats per minute. Abdomen is soft without ascites and he has no wheezing on auscultation. Labs include AST 60 U/L, ALT 40 U/L, total bilirubin of 2.1 mg/dL, albumin of 3.2 g/dL, INR 1.2, and a platelet count of 60,000/µL. You perform an upper endoscopy and there are two columns of large varices without stigmata of recent bleed.

What is the next best step in management?

a) Endoscopic Band ligation
b) Endoscopic Band ligation and start nadolol
c) Start nadolol
d) Referral for Transjugular Intrahepatic Portosystemic Shunt (TIPS)
e) Observation

A 62-year-old man with newly diagnosed hepatitis C cirrhosis is admitted with melena. He is on no medications. Vitals include T 98.0o F, BP of 100/52 mmHg, HR 90 beats per minute. He is alert and oriented, but his abdomen is distended with large ascites. Labs include AST 60 U/L, ALT 40 U/L, total bilirubin of 3.1 mg/dL, albumin of 2.7 g/dL, sodium 135 mEq/L, creatinine of 0.5 mg/dL, hemoglobin of 8.5 g/dL, INR 1.5, and a platelet count of 59,000/µL. Which of the following is included in the management of this patient?

a) Admission into a regular room
b) Early endoscopy within 36 hours
c) Initiation of vasodilating agent
d) Blood transfusion
e) Antibiotics

A 50-year-old man with new onset ascites presents to the emergency room with abdominal pain and discomfort. He has a history of heavy alcohol use for thirty years. He was abstinent for two years after completing an alcohol relapse prevention program but returned to drinking and currently drinks three beers daily. He has no other medical history. Vitals include T 98.7o F, BP of 122/64 mmHg, HR 86 beats per minute. His abdomen is distended with large ascites and he has 2+ pitting edema up to his knees bilaterally. Labs include AST 220 U/L, ALT 100 U/L, total bilirubin of 1.5 mg/dL, albumin of 3.7 g/dL, sodium 142mEq/L, creatinine of 0.5 mg/dL, and a platelet count of 159,000/µL. He had a diagnostic paracentesis, which revealed ascites polymorphonuclear count of 100/mm3, albumin 2.0 g/dL, total protein 1.6 g/dL. In addition to alcohol cessation, what is the next best step in management?

a) Start furosemide and spironolactone
b) Peritoneovenous shunt
c) Placement of peritoneal catheter
d) TIPS placement
e) Referral for liver transplantation

You are called while consulting on the liver service for a newly diagnosed patient with hepatitis C cirrhosis who was admitted for lower extremity swelling. The team started oral diuretics and is asking for an outpatient appointment. He has no complaints and feels better with initiation of diuretics. His abdomen is distended with large ascites and he has 1+ pitting edema up to his knees bilaterally. Labs include AST 50 U/L, ALT 22 U/L, total bilirubin of 2.5 mg/dL, albumin of 3.4 g/dL, sodium 144mEq/L, potassium of 3.2 mEq/L creatinine of 0.5 mg/dL, INR of 1.5, and a platelet count of 159,000/µL. His ultrasound reveals large ascites but does not show hepatocellular carcinoma or new thrombosis.

What is the next best step in management prior to discharge?

a) Endoscopy for variceal screening
b) Diagnostic paracentesis
c) Liver transplant evaluation
d) MRI abdomen
e) Prophylactic antibiotics

A 57-year-old man from China presents to the emergency room with new onset ascites. He has had decreased appetite and lost ten pounds in the last two months. He has a history of heavy alcohol use and hypertension. He has not seen a physician in over twenty years. Vitals include T 98.8 F, BP of 90/60 mmHg, HR 89 beats per minute. His abdomen is distended with large ascites and he has 2+ pitting edema up to his knees bilaterally. Labs include AST 60 U/L, ALT 30 U/L, total bilirubin of 1.2 mg/dL, albumin of 3.2 g/dL, sodium 138 mEq/L, creatinine of 0.8 mg/dL, and a platelet count of 162,000/μL. He had an ultrasound, which was limited, but did not show any masses or thromboses. He had a paracentesis, which revealed fluid polymorphonuclear count of 100/mm3, albumin 2.0 g/dL, and total protein 3.6 g/dL. What is the most likely etiology of his ascites?

a) Alcoholic cirrhosis
b) Budd Chiari syndrome
c) Cardiac ascites
d) Tuberculous peritonitis
e) Malignant ascites

A 54-year-old man with hepatitis B cirrhosis is admitted to the hospital with worsening ascites. He was recently discharged with an increase in diuretics to furosemide 40 mg and spironolactone 100mg daily. Vital signs include heart rate 102 beats per minute, blood pressure 90/52 mmHg, and oxygen saturation is 98 percent on room
air. He is alert and oriented without evidence of asterixis. His abdomen is significantly distended with fluid wave, and he has 2+ pitting edema to the knees bilaterally. Labs include AST 100 U/L, ALT 120 U/L, total bilirubin of 2.4 mg/dL, albumin of three g/dL, sodium of 135 mEq/L, potassium of 3.5 mEq/L, BUN of 30 and a creatinine of 1.5mg/dL (previously 0.8 on discharge) and a platelet count of 100,000/µL.

In addition to culture workup and urine studies, what is the next best step in management?

a) Continue diuretics
b) Continue lactulose
c) Volume expand with albumin
d) Start midodrine
e) Start octreotide

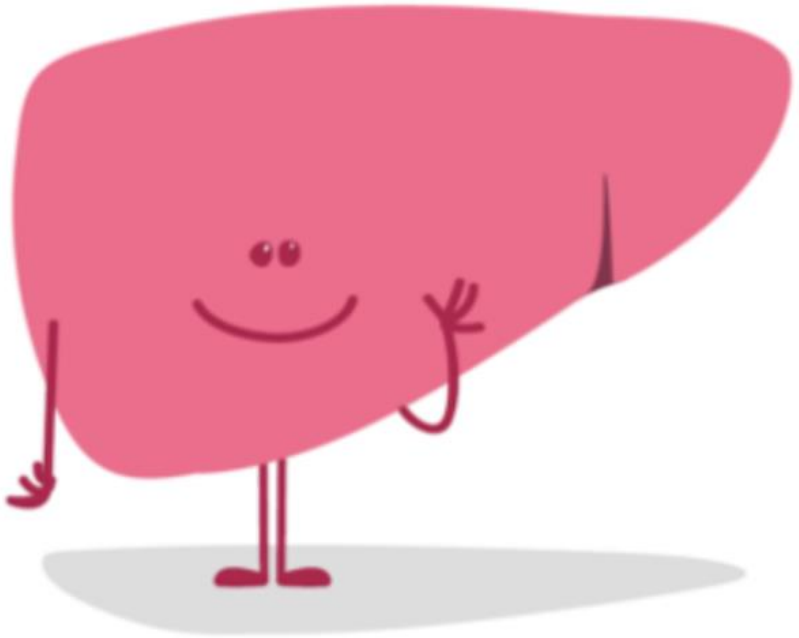
A 50-year-old man is admitted with ascites and abdominal tenderness. He has HCV cirrhosis. A paracentesis is performed. Seven L of ascitic fluid are removed. Results of the cell count in ascitic fluid show an absolute neutrophil count of 750 cells/mm3. You decide to begin therapy for spontaneous bacterial peritonitis.

What is the next best treatment regimen?

a) IV antibiotics and IV albumin
b) IV Antibiotics
c) PO antibiotic
d) Wait for results of ascitic fluid culture
e) Pentoxyfylline to prevent AKI

A 50-year-old man is admitted with ascites and abdominal tenderness. He has HCV cirrhosis. A paracentesis is performed. Seven L of ascitic fluid are removed. Which of the following should be pursued next?

a) Albumin IV
b) Pantoprazole IV
c) Midodrine PO
d) TIPS



THANK YOU