# CIRRHOSIS

KOGATAM.LIVINGSTONE

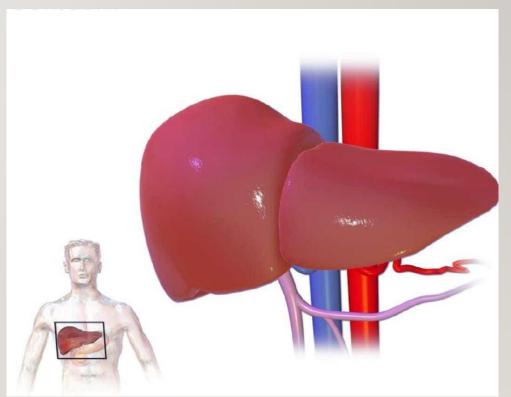
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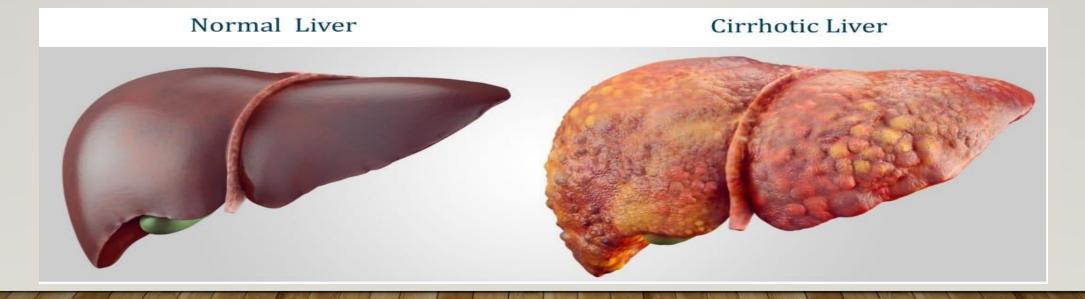
# **CIRRHOSIS**

- End stage liver disease (irreversible)
- Result from many causes of chronic liver disease:
  - Viral Hepatitis (especially B and C)
  - Alcoholic liver disease
  - Non-alcoholic fatty liver disease





- Liver tissue replaced by **fibrosis** and **nodules**
- Smooth liver surface replaced by nodules
- In advanced cirrhosis, liver becomes shrunken



# **CLINICAL FEATURES**

• Hyperammonemia

• Asterixis, confusion, coma



#### HYPERAMMONEMIA TREATMENT

- Low protein diet
- Lactulose
  - Synthetic disaccharide (laxative)
  - Colon breakdown by bacteria to fatty acids
  - Lowers colonic pH; favors formation of NH4+ over NH3
  - NH4 + not absorbed  $\rightarrow$  trapped in colon
  - Result: *Jplasma ammonia concentrations*

# CIRRHOSIS CLINICAL FEATURES

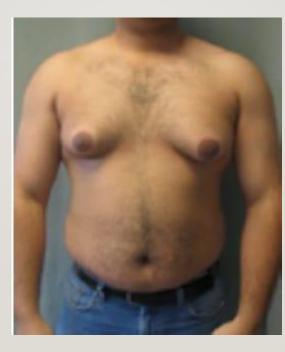
#### • Jaundice

- Loss of bilirubin metabolism
- Hypoglycemia
  - Loss of gluconeogenesis
- Coagulopathy
  - Loss of clotting factors
  - Elevated PT/PTT
- Hypoalbuminemia
  - May cause low oncotic pressure
  - Contributes to ascites, edema



#### • Elevated estrogen

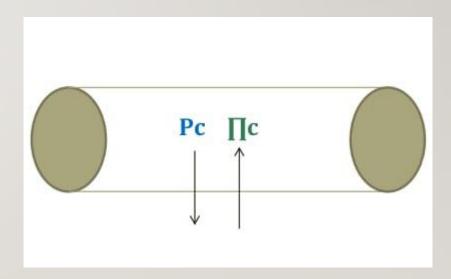
- Normally removed by liver
- Gynecomastia in men
- Testicular atrophy
- Spider angiomata
- Palmar erythema





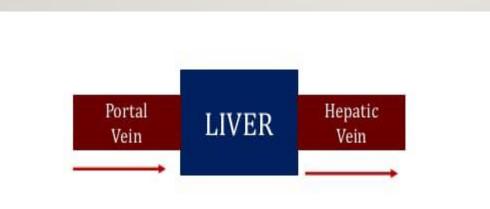
# **CAPILLARY FLUID SHIFTS**

- Capillary hydrostatic pressure (Pc)
  - Drives fluid out of capillaries into tissues
- Capillary **oncotic** pressure ( $\Box c$ )
  - Proteins (albumin) pull water into capillaries
  - Resists movement of fluid out of capillaries

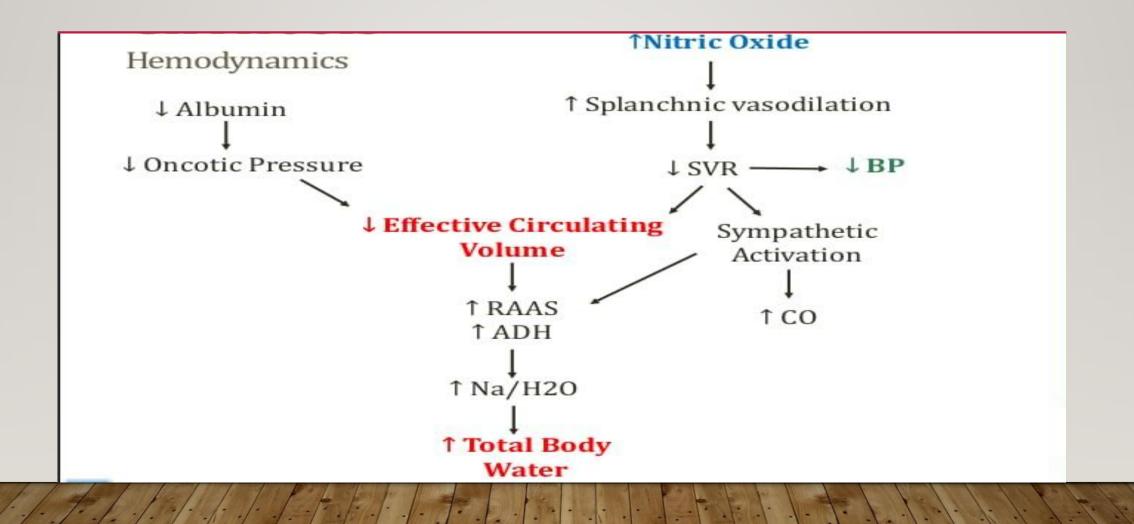


# **PORTAL HYPERTENSION**

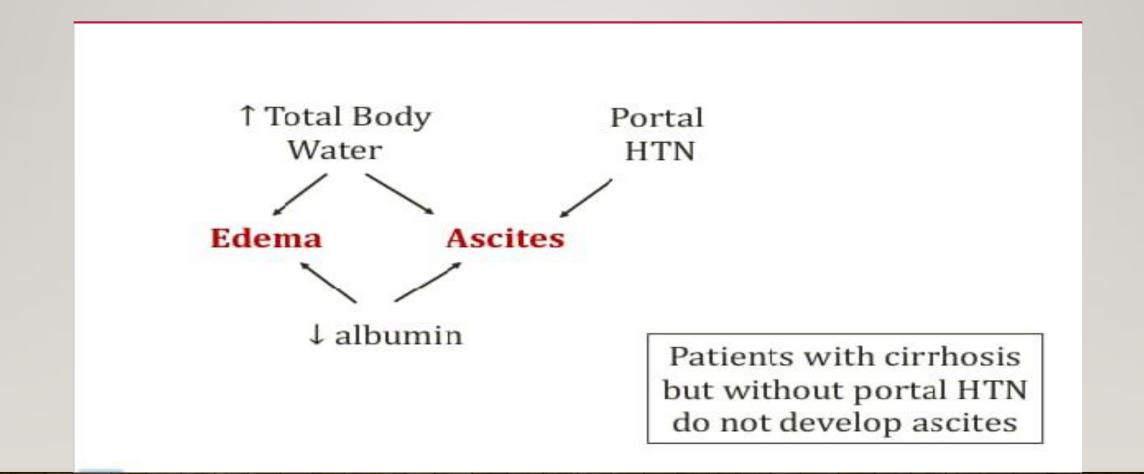
- Blood flows portal vein  $\rightarrow$  liver  $\rightarrow$  hepatic vein
- Cirrhosis  $\rightarrow$  obstructed flow through liver
- High pressure in portal vein ("hypertension")



### **CIRRHOSIS**



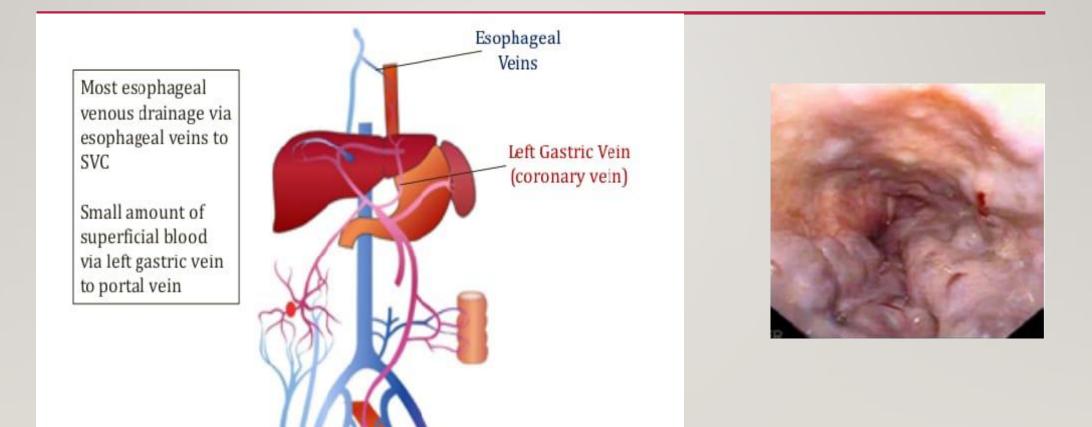
### **ASCITES AND EDEMA**



# VENOUS ANASTAMOSES

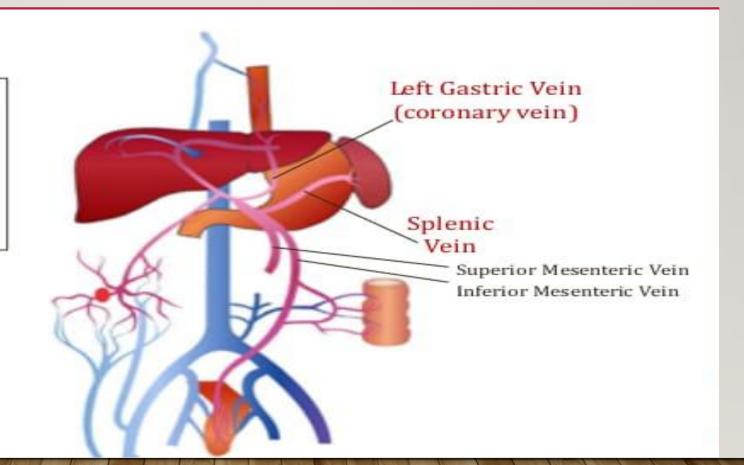
- High portal pressure opens "venous collaterals"
- Connection between portal-systemic veins
- Normally small, collapsed vessels
- Engorge in portal hypertension
- Key collaterals:
  - Umbilicus physical exam finding: "caput medusa"
  - Esophagus upper gastrointestinal bleeding
  - **Stomach** upper gastrointestinal bleeding
  - **Rectum** hemorrhoids which may also bleed

### **ESOPHAGEAL VARICES**

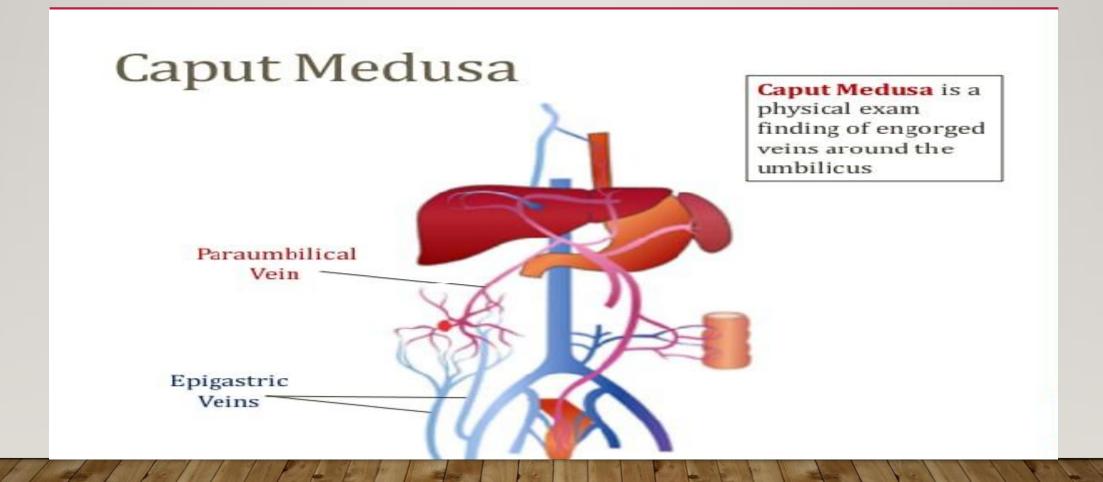


#### **GASTRIC VARICES**

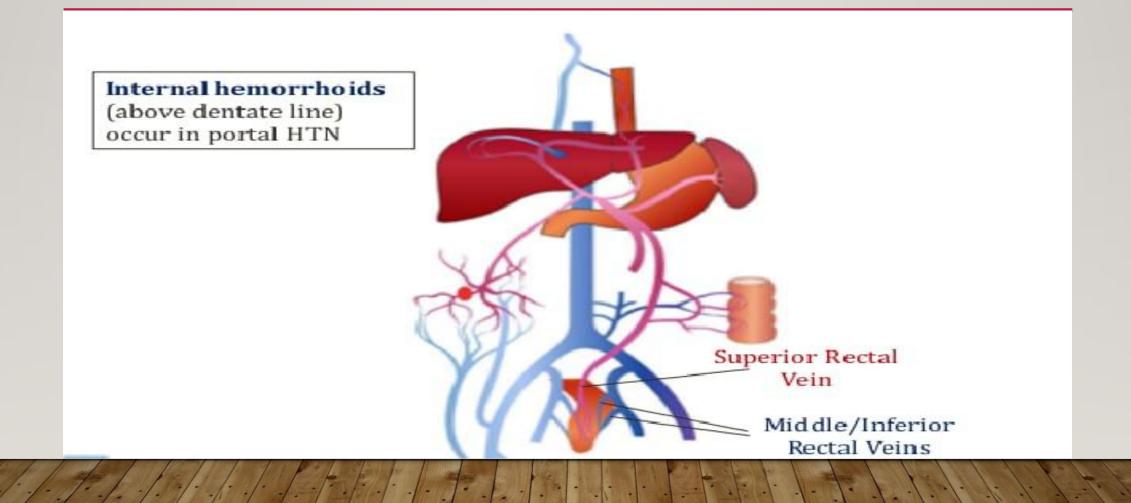
Short gastric veins drain blood from stomach fundus to left gastric vein and splenic vein (both part of portal system)



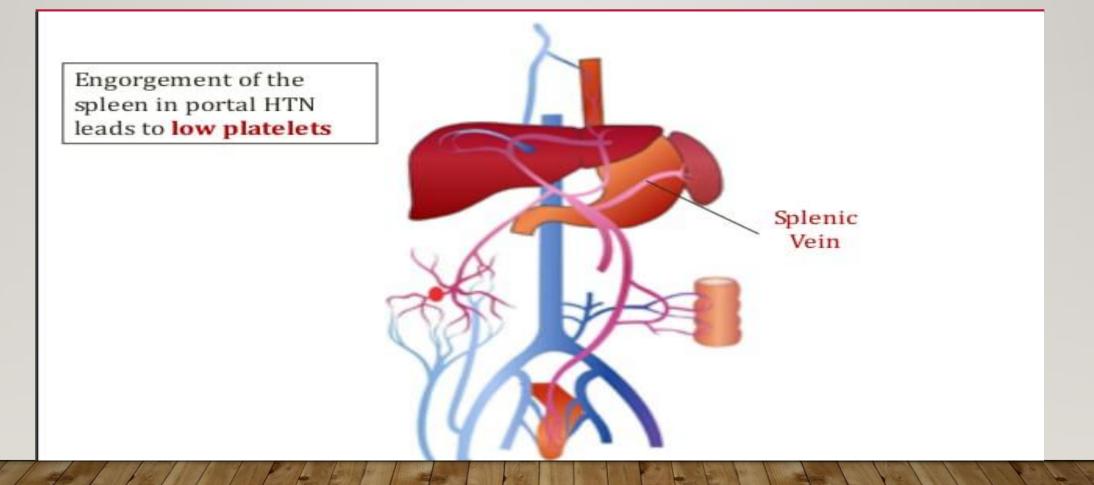
### **CAPUT MEDUSA**



#### **INTERNAL HEMORRHOIDS**



#### **HYPERSPLENISM**



# **PORTAL VEIN THROMBOSIS**

- Rare cause of portal hypertension
- Acute onset abdominal pain
- Splenomegaly (palpable spleen one exam)
- May result in gastric varices with **bleeding**
- Liver biopsy will be normal

# **ASCITES**

• Accumulation of fluid in **peritoneal cavity** 

• In liver disease, from portal hypertension +/- low albumin



# SAAG SERUM ASCITES ALBUMIN GRADIENT

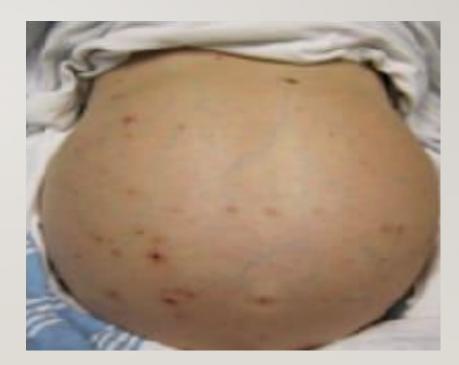
- Test of ascitic fluid
- Two reasons for new/worsening ascites
  - Portal hypertension
  - Malignancy (leaky vasculature)
- Sample of ascitic fluid via paracentesis
- Serum albumin ascites albumin = SAAG

# **SAAG** SERUM ASCITES ALBUMIN GRADIENT

- SAAG >1.1 g/dL
  - Large difference between serum and ascites albumin
  - High pressure driving fluid (not albumin) into peritoneum
  - Seen in portal hypertension
- SAAG <1.1 g/DI</li>
- Albumin levels similar between serum and ascites
- Leaky vasculature leading to fluid/albumin into peritoneum
- Seen in malignant ascites (malignant cells in peritoneal cavity)

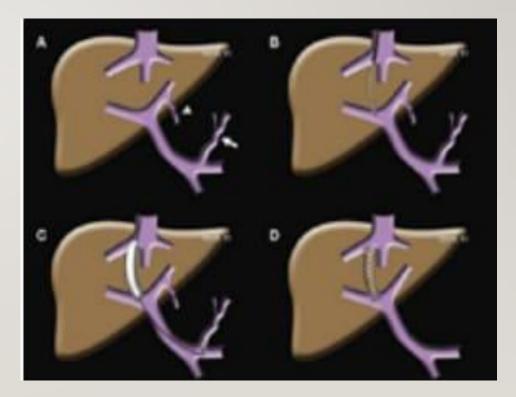
# **ASCITES TREATMENT**

- Sodium restriction
- Spironolactone (drug of choice)
  - Potassium-sparing diuretic
  - Blocks aldosterone distal tubule
  - Most effective drug for ascites
- Loop diuretics (2nd line)
- Large volume **paracentesis**
- TIPS



### TIPS TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT

- Transjugular Intrahepatic Portosystemic Shunt
- Treatment of portal hypertension
- Creation of channel in liver
- Connects portal vein to hepatic vein



# **SBP** SPONTANEOUS BACTERIAL PERITONITIS

- Ascitic fluid infection
- Bacteria in gut gain entry into ascitic fluid
- Usually E. coli and Klebsiella; rarely strep/staph
- Fever, abdominal pain/tenderness
- ↑ ascitic absolute PMNs (≥250 cells/mm3)
- Common treatment:
  - 3rd generation cephalosporin (cefotaxime)
  - Gram positive and gram negative coverage
  - Achieves good levels in ascitic fluid

### MELD SCORE MODEL FOR END-STAGE LIVER DISEASE

- Scoring system for chronic liver disease or cirrhosis
- Estimates 3-month mortality from liver disease
- Point system using:
  - Bilirubin level
  - Creatinine level
  - INR
- >40 = 71% mortality
- <9 = 2% mortality

# **CHILD-PUGH CLASSIFICATION**

- Five variables to predict risk/survival
  - Points for encephalopathy, ascites, bilirubin, albumin, PT
- Score ranges from 5 to 15
  - 5 or 6: Child-Pugh class A cirrhosis
  - 7 to 9: Child-Pugh class B cirrhosis
  - 10 to 15: Child-Pugh class C cirrhosis (worst)

# **CIRRHOSIS** DIAGNOSIS

- Gold standard is liver biopsy
  - Not required if diagnosis is clear from history
  - Done only when biopsy will change management
- Imaging (ultrasound, CT, MRI)
  - May show small, nodular liver
  - Not sensitive or specific for diagnosis
  - More helpful for detection of hepatocellular carcinoma
- Clinical diagnosis (common)
  - Presence of ascites
  - Low platelet count
  - Spider angiomata

# **STELLATE CELLS**

- Perisinusoidal cell
- Storage site for retinoids (vitamin A metabolites)
- Activated in liver disease
- Secrete TGF- $\beta$
- Proliferate and produce fibrous tissue
- Major contributor to cirrhosis

