

ASCITES

CAUSES

Common causes of ascites	
Extraperitoneal causes	• Cirrhosis • Acute liver failure • Alcoholic hepatitis • Budd-Chiari syndrome • Heart failure • Hypoalbuminemia • Malnutrition • Nephrotic syndrome
Peritoneal causes	• Malignancy (ovarian, pancreatic) • Infection (tuberculosis, fungal)

EVALUATION:

- Diagnosis confirmed via clinical, laboratory, and radiologic evaluation
- Abdominal USG— needed to evaluate for complications (e.g. splenomegaly, HCC)
- Paracentesis is recommended in new-onset ascites to determine etiology
- Liver biopsy is required if diagnosis of cirrhosis is unclear or will alter course of management

ASCITES FLUID CHARACTERISTICS

Ascites fluid characteristics	
Color	• Bloody: Trauma, malignancy, TB (rarely) • Milky: Chylous, pancreatic • Turbid: Possible infection • Straw color: Likely more benign causes
Neutrophils	• $<250/\text{mm}^3$: No peritonitis • $\geq 250/\text{mm}^3$: Peritonitis (secondary or spontaneous bacterial)
Total protein	• $\geq 2.5 \text{ g/dL}$ (high-protein ascites) ◦ CHF, constrictive pericarditis, peritoneal carcinomatosis, TB, Budd-Chiari syndrome, fungal (eg, coccidioidomycosis) • $<2.5 \text{ g/dL}$ (low-protein ascites) ◦ Cirrhosis, nephrotic syndrome
SAAG	• $\geq 1.1 \text{ g/dL}$ (indicates portal hypertension) ◦ Cardiac ascites, cirrhosis, Budd-Chiari syndrome • $<1.1 \text{ g/dL}$ (absence of portal hypertension) ◦ TB, peritoneal carcinomatosis, pancreatic ascites, nephrotic syndrome

CHF = congestive heart failure; SAAG = serum-to-ascites albumin gradient; TB = tuberculosis.

Leads to $\uparrow$ in capillary hydrostatic pressure. $\downarrow$ liver synthetic function is indicated by $\downarrow$ serum albumin which can cause edema in later stages of liver failure and $\uparrow$ PT/INR

$\uparrow$ capillary permeability

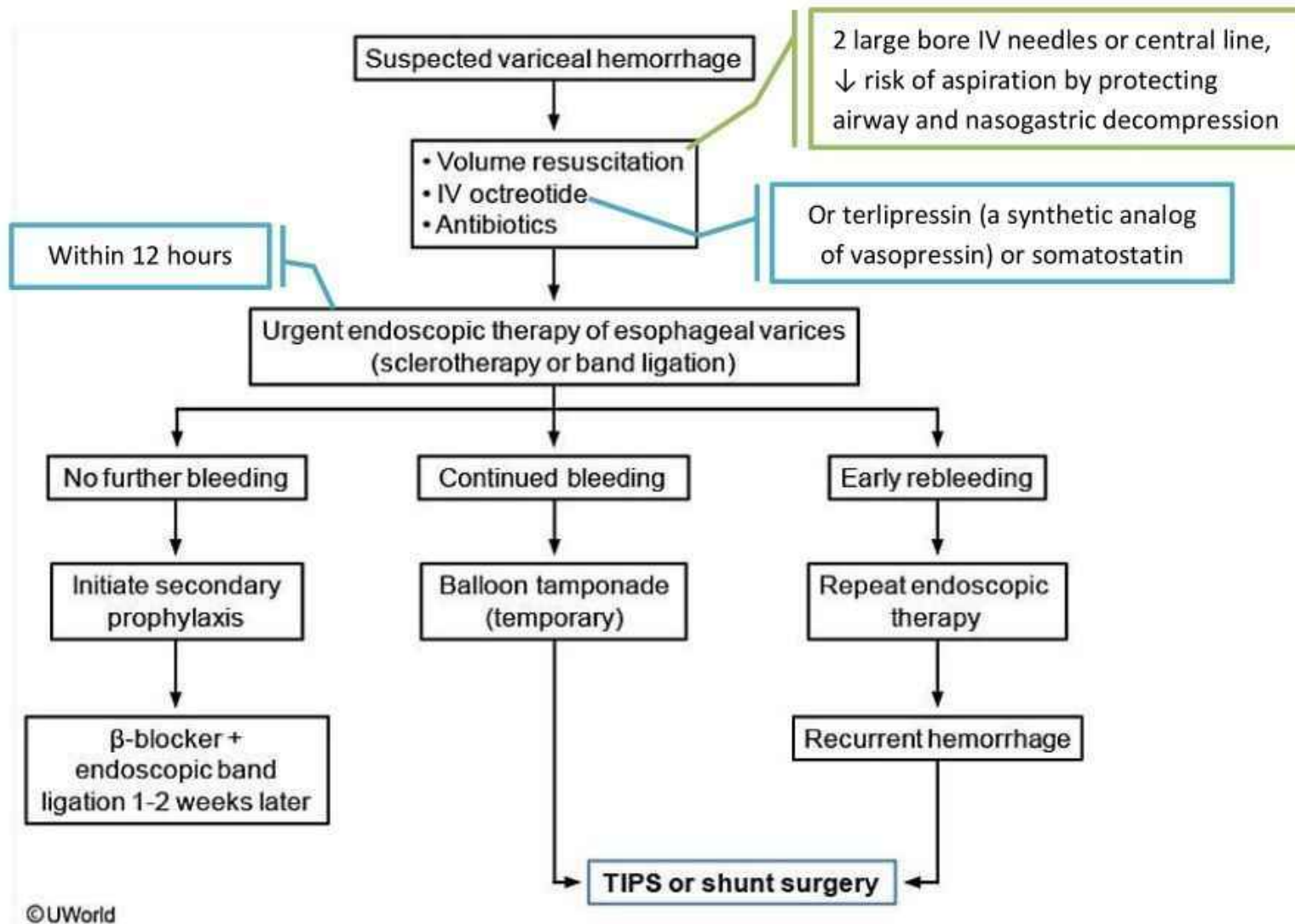
- SAAG = serum albumin – ascites albumin (it is gradient not ratio)

TREATMENT OF ASCITES

1. Start with Na^+ and water restriction (2L/day)
2. Diuretic therapy, if needed $\rightarrow$ start with spironolactone
3. When maximal dose of spironolactone fails to improve $\rightarrow$ add loop diuretic (eg furosemide). Aggressive diuresis ($>1 \text{ L/day}$) $\rightarrow$ not recommended coz of risk of hepatorenal syndrome
4. Does not respond $\rightarrow$ slow tapping of 2-4 L/day of ascitic fluid (with/without albumin infusion) but renal function is regularly monitored $\rightarrow$ less aggressive paracentesis in pts with borderline renal function

HEPATIC HYDROTHORAX

- Pleural effusion due to ascites
- Due to defect in diaphragm
- Right sided more common
- Best option for treatment: liver transplant
- Primary treatment: thoracentesis followed by diuresis and salt restriction $\rightarrow$ no response $\rightarrow$ TIPS $\rightarrow$ TIPS contraindicated $\rightarrow$ pleurodesis
- Surgical portosystemic shunts have more morbidity



- Control of bleeding is established in 80% pts → rebleeding is very common esp. in 6 wks of initial hemorrhage
- Examples of temporary balloon tamponade: Sengstaken- Blackemore, Minnesota, Linton- Nachlas tubes
- Coagulopathy, thrombocytopenia and anemia are common complications and may also need correction
- Blood transfusion will be needed if Hb falls <9g/dl—regularly monitor blood counts
- Fresh frozen plasma for correction of coagulopathy but can cause volume overload
- Platelet transfusion if platelet count <50,000/mm³

SPONTANEOUS BACTERIAL PERITONITIS

Spontaneous bacterial peritonitis	
Clinical presentation	• Temperature ≥ 37.8 C (100 F) • Abdominal pain/tenderness • Altered mental status (abnormal connect-the-numbers test) • Hypotension, hypothermia, paralytic ileus with severe infection
Diagnosis from ascitic fluid	• PMNs $\geq 250/\text{mm}^3$ • Positive culture, often gram-negative organisms (eg, <i>Escherichia coli</i>, <i>Klebsiella</i>) • Protein < 1 g/dL • SAAG ≥ 1.1 g/dL
Treatment	• Empiric antibiotics - third-generation cephalosporins (eg, cefotaxime) • Fluoroquinolones for SBP prophylaxis

Most common features—abdominal pain less prominent than in other causes of peritonitis

Known as Reitan trail test

Paracentesis is the test of choice for dx—these two are main diagnostic criteria—*E. coli* and *Klebsiella* most common followed by streptococcal species

PMN = polymorphonuclear leukocytes; SAAG = serum-ascites albumin gradient; SBP = spontaneous bacterial peritonitis.

- Keep a low threshold of suspicion
- Pts with cirrhosis are normally hypothermic and > 100 F warrants investigation

SOLID LIVER MASSES

Solid liver masses	
Focal nodular hyperplasia	• Associated with anomalous arteries • Arterial flow & central scar on imaging
Hepatic adenoma	• Women on long-term oral contraceptives • Possible hemorrhage or malignant transformation
Regenerative nodules	• Acute or chronic liver injury (eg, cirrhosis)
Hepatocellular carcinoma	• Systemic symptoms • Chronic hepatitis or cirrhosis • Elevated alpha fetoprotein
Liver metastasis	• Single/multiple lesions • Known extrahepatic malignancy

Not associated with OCP use—caused by hyperperfusion from anomalous arteries

Common in young women

Other risk factors: pregnancy and anabolic steroid use.
USG: well-demarcated hyperechoic lesion.
CT: early peripheral enhancement.
Biopsy: not recommended. Rx: excision

HEPATIC ENCEPHALOPATHY

Clinical features of hepatic encephalopathy	
Precipitating factors	• Drugs (sedatives, narcotics) • Hypovolemia (eg, diarrhea, vomiting, diuretics, high-volume paracentesis) • Excessive nitrogen load (gastrointestinal bleeding, constipation, high-protein diet) • Hypokalemia & metabolic alkalosis • Hypoxia & hypoglycemia • Infection (eg, pneumonia, urinary tract infection, spontaneous bacterial peritonitis) • Portosystemic shunting (eg, surgical shunts)
Clinical presentation	
Stage 1	• Hypersomnia, insomnia, or inverted sleep cycle • Slightly impaired cognition • Mild confusion • Tremor, possible asterixis
Stage 2	• Lethargy with slow responses to stimuli • Moderate confusion • Difficulty with writing, slurred speech
Stage 3	• Marked confusion • Sleeping but arousable
Stage 4	• Stupor or coma
Neuromuscular findings in overt encephalopathy include slurred speech, ataxia, bradykinesia, asterixis, hyperactive deep-tendon reflexes with Babinski & clonus, & nystagmus.	

- Pathogenesis: inability of liver to break down ammonia → urea. Other toxins may also be responsible. These neurotoxins stimulate inhibitory (e.g. GABA) and impairment of excitatory (e.g. glutamate) pathways in brain
- Dx: ↑ NH₃ is diagnostic in symptomatic pts with high clinical suspicion → but non-specific and may be ↑ in asymptomatic pts
- EEG may show B/L synchronous ↓ wave frequency and ↑ wave amplitude → non-specific and non-diagnostic

MANAGEMENT OF HEPATIC ENCEPHALOPATHY

Management of hepatic encephalopathy
• Supportive care (eg, volume repletion if needed, restraints if agitated, correct electrolytes) • Adequate nutrition without protein-restricted diet unless patient is unable to tolerate protein intake • Treat precipitating cause (eg, gastrointestinal bleed, hypovolemia, infection, electrolyte abnormalities) Medicines like sedatives • Lower serum ammonia • Lactulose orally or enema if unable to take orally • Rifaximin orally if no improvement in 48 hours with lactulose or unable to take lactulose

- Non-absorbable disaccharides (lactulose, lactitol)—preferred: colonic bacteria → metabolize lactulose to short chain fatty acids (lactic acid, acetic acid) → converts absorbable ammonia to non-absorbable ammonium (an ammonia trap) and also cause catharsis. Antibiotics (like rifaximin) → ↓ ammonia producing bacteria—added to lactulose if pt does not improve in 48 hours or monotherapy in those unable to take lactulose. Catharsis using any laxative may also be beneficial. Neomycin is an alternate therapy in pts unable to take rifaximin.
- Cirrhotic patients—usually malnourished and should not follow a protein-restricted diet. Protein-free diets → cause a negative nitrogen balance → increase mortality. Protein restriction—recommended only in patients unable to tolerate protein intake (ie, recurrent encephalopathy episodes after a high-