Massive Ascites Case

**Goals of this session:**
- To review the pathophysiology of ascites and hepatic cirrhosis
- To review the relevant physical findings of cirrhosis
- To place cirrhosis/ascites into a clinical context with a case presentation
- To introduce a symptom-based approach to thinking about pathophys., i.e., differential diagnosis

**Pathophysiology review:**
- Ascites is the collection of fluid in the peritoneal cavity
  - Portal-Hypertension related: Serum-ascites albumin gradient (SAAG) >1.1
    - Sinusoidal: cirrhosis (81%), hepatitis, massive liver mets, HCC
    - Post-sinusoidal: right sided CHF, Budd-Chiari
- Pathophysiology:
  - **“Underfill Theory”:** portal hypertension → transudation of fluid into peritoneum → ↓ plasma volume → renal Na retention
  - **“Overflow Theory”:** hepatorenal reflex → Na retention
  - **Peripheral Vasodilation Theory:** portal htn → systemic vasodilation (due to release of NO?) → ↓ effective arterial volume → renal Na retention
- ↓ Plasma oncotic pressure with decreased albumin
- Cirrhosis is fibrosis and nodular regeneration from hepatocellular injury
- Etiologies: Alcohol, viral hepatitis, autoimmune hepatitis, metabolic diseases, biliary tract diseases
- Portal HTN: Varices, melena, splenomegaly, caput medusae, ascites, testicular atrophy, hemorrhoids
- Liver cell failure: Encephalopathy, scleral icterus, spider nevi, gynecomastia, jaundice, asterixis, anemia, pedal edema, loss of sexual hair, palmar erythema

**CC:** confusion, increasing abdominal girth

**HPI:** 63 y/o female with IHSS, Afib, DMII with chronic ascites secondary to cryptogenic cirrhosis.
- Pt developed ascites 12/03 during hospital admission for afib; denies alcohol abuse, workup negative for HCV/HBV/autoimmune hepatitis, Wilson’s disease and hemochromatosis. Pt. was treated with spironolactone, low Na diet. Colonoscopy revealed AVM in cecum and rectal varices; EGD showed no varices.
- Subsequently patient re-admitted several times for worsening ascites; in March 04 developed SBP (Group B strep) tx with ceftriaxone. 2weeks later ascites worsened & received lg. Volume paracentesis (5L), asterixis noted. 4/7/04 patient readmitted for confusion, lethargy, N/V, worsened ascites. Pt. was hyponatremic (na=119). Pt. diuresed, tx with lactulose & confusion improved.
- On 4/17 pt. became agitated & confused, feeling “unsteady and weak” and went to the Allen Pavillion. She denied HA/CP/SOB/N/V or dizziness. Pt. was hypothermic (96.2), 10lbs over dry weight, Na 122, NH3 193, arouseable to voice but unable to follow commands. 3.5L ascitic fluid was removed, inc. lactulose, SAAG=1.8, mental status improved, tx to CPMC for further workup.

**PMH:** IHSS, PAF, HTN, CAD, NQWMI, DMII,

**PSH:** none

**Medications:** Lasix, Aldactone, Lactulose, Propranolol, Colace, Niferix, Cipro, Pantoprazol, Insulin NPH, ASA 81mg, NKDA

**SH:** denies alcohol, illicit drug or tobacco use

**PE:** T96.4 BP 105/61 P 70 RR20 O2 sat 96% RA
- Gen: thin, alert, NAD, no jaundice
- HEENT: PERRLA, EOMI, anicteric
- Neck: supple, no JVD
- CV: S1, S2 regular rate and rhythm, 3/6 systolic ejection murmur loudest at RUSB
- Lungs: Clear to auscultation bilaterally, no wheezes, rhonchi, rales
- Abd: soft, nontender, **distended with gross ascites, + shifting dullness, + fluid wave**
- Ext: **no edema**
- Neuro: A/O x3, **mild asterixis noted**
Labs: BUN/Cr 26/1.1, INR 1.39, NH3 102, AST/ALT 104/54, Tbili 2.8 (nl<1.5), alkphos 316. Alb 2 (nl >3.4)