03/24/25 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: (Andrew@ASanchez_PS) Case Discussants: (Alec @ABRezMed) and (Austin@RezidentMD)

CC: A 60 year old female presented after a fall having history of MASH cirrhosis decompensated by ascites and hepatic encephalopathy with AMS. She also complained of bilateral thigh pain and back pain.

HPI: Husband described that the patient had "diarrhea" (slightly increased loose stool) so has decreased frequency of lactulose and eating less.

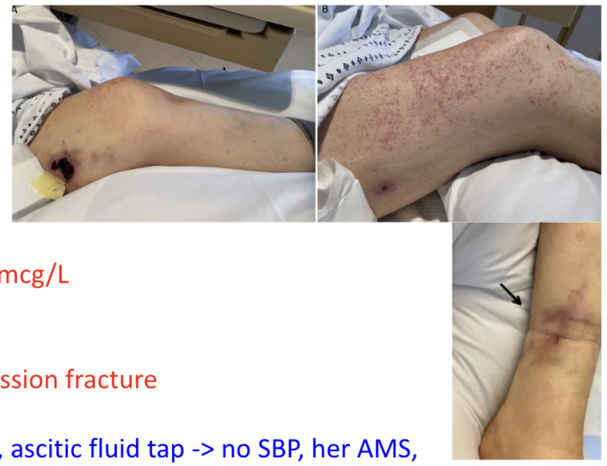
PMH: Type 2 DM
Gastric bypass history

Meds:
Apixaban (Hx of portal vein thrombosis)
Spironolactone
Pantoprazole
Nadolol
furosemide

Vitals: all nl
Exam: Gen: Disoriented and inattentive
Abd: mildly distended abdomen + ascites
Neuro: asterixis
MSK: no peripheral edema, very tender to light touch on thighs, no lesions

Notable Labs & Imaging:
Hematology:
WBC: 7.3 Hgb: 12.6 Plt: 252

Chemistry
Cr: 1.9 (0.7) BUN: 24 Ph: 3.3
AST: 55 ALT: 37 ALP: 137 Bili: 2.2
Albumin: 2.3 INR: 1.7 ammonia: 52 mcg/L



Imaging:
Head CT: nl **Back CT:** new L1 compression fracture

The patient received fluids, lactulose, ascitic fluid tap -> no SBP, her AMS, Renal function improved -> reported that her thighs are painful and developed non-palpable petechiae and subcutaneous nodules + retiform purpura forming an eschar.

Bilateral hip x rays:soft tissue vascular calcifications
Coags: no DIC. Urinalysis: normal
B2 glycoprotein and cardiolipin tests negative. Cryoglobulin, APLS workup (anti-CL, anti-B2GP) negative, low antithrombin activity, low protein C activity, protein S normal.

Despite 2 weeks of Rx -> septic shock -> CT 7 cm abscess underlying one of the thigh lesions -> no improvement despite ABx and drainage.
Biopsy: small vessel calcifications. PTH 244 mcg/dl, 1,25-vit D high. Vit C borderline low.

Dx: non-uremic calciphylaxis.

Problem Representation: A 60 yo female with decompensated MASH cirrhosis and Hx of gastric bypass presented with AMS, thigh and back pain after suffering a fall. Labs showed AKI. Developed retiform purpura.

Teaching Points (SEEME):

Encephalopathy:
We can see for the tiggers present and medication history is also important. History of cirrhosis can be linked to hepatic encephalopathy. Hypovolemia and kidney injury can also contribute.

Diarrhea:
might be related to lactulose or a separate entity. Chronic illness can cause a lot of things and may contribute to the fall. Viral illness can also contribute to diarrhea.

Pain:
might be linked to muscles, nerves,bones or underlying vasculitis. Also imaging and neurological exam can be really helpful.We can always look for restriction to dermatomes if present. Rhabdomyolysis and compartment syndrome may develop after the fall.

Hypovolemia ,hyperammonemia and hyperbilirubinemia:
We can stop the diuretics. Cirrhosis can contribute to hyperammonemia. Increased bilirubin in someone with cirrhosis is highly indicative of infection.

Approach to SBP:
It has high mortality so ascites if present, we should always check for SBP by tap and ascites and should be treated timely.

Approach to rash: We can think about vasculitis (inflammation)or any blood stream problem, thrombus,eschar formation may be related to poor perfusion. We can consider platelet defect or vitamin C deficiency. We have to think about a local process or a systemic process. Patients with APLS may also develop purpura. Cholesterol emboli can also contribute to purpura.Age can cause calcifications on X-ray. **Calciphylaxis** may cause painful skin lesions and is a calcific arteriolopathy. Liver disease is an important risk factor for non-uremic calciphylaxis. Located in areas of rich adipose tissues. Diabetes is also a risk factor.