



# 04/04/25 Morning Report with @CPSolvers



"One life, so many dreams" Case Presenter: Elena (@) Case Discussants: Rabih (@rabihmgeha) and Ibrahim (@IbrahimOmer\_)

<p><b>Scribing (Lera)</b>  <b>CC:</b> ED received a call from the HD unit due to <b>AMS and somnolence</b> in a patient.</p> <p><b>HPI:</b> Presented to ED. On admission seems <b>sleepy, opens eyes</b> to non-painful stimuli, able to answer Qs without concern for dysarthria or aphasia. <b>Disoriented to place and time</b>, but slow response. Recalls name and date of birth, can't recall their age.</p> <p>Regularly attends HD, <b>has been on for 2h before presentation.</b></p>	<p><b>Vitals:</b> T: afebrile <b>BP:</b> 105/65 <b>RR:</b> nl <b>HR:</b> 93 <b>Sat:</b> nl  <b>Exam:</b> <b>Gen:</b> somnolent  <b>HEENT, Neck:</b> unremarkable  <b>CV, Pulm, Abd:</b> unremarkable <b>Neuro:</b> strength, reflexes, sensation, CNS nl. No meningismus, <b>asterixis present.</b></p> <p><b>Notable Labs &amp; Imaging:</b>  <b>Hematology:</b>  WBC: 6 with nl diff <b>Hgb:</b> 9 (below bl of 10) <b>Pit:</b> 84 <b>MCV:</b> 93  <b>CT head:</b> no hemorrhage / ischemia, stenosis of left ACI.</p>	<p><b>Problem Representation:</b> Patient with Hx of cirrhosis s/p ileal conduit on current TAPT transferred to ED after 2h HD session with acute onset AMS. Found to be hypotensive with asterixis, later developed UGIB. Ammonia levels were significantly elevated.</p>	
<p><b>PMH:</b>  CAD (T2DM, HTN, HLD) w/ stenting 1 week ago  CKD G4A3 ( DM), Afib</p> <p><b>Cirrhosis CHILD C, MELD3 23 2/2 MASLD</b>  Decompensation 2 y ago</p> <p>LN mts urothelial carcinoma of bladder (ileal conduit 5 y ago)</p> <p><b>Meds:</b> <b>Warfarin, aspirin, clopidogrel,</b> atorvastatin  Torasemide, Spironolactone, PPI, phosphate binder, rifaximin, lactulose  Insulin glargine</p>	<p><b>Fam Hx:</b> not relevant</p> <p><b>Soc Hx:</b> not relevant</p> <p><b>Health-Related Behaviors:</b> 70 pack/year smoking Hx</p> <p><b>Allergies:</b> not relevant</p>	<p><b>Chemistry</b>  Na: nl K: nl Cr: 1.7 (bl) BUN: nl Ca: nl Ph: nl Mg: nl Glu: nl  AST: nl ALT: nl <b>ALP and GGT:</b> slightly elevated <b>Bili:</b> nl  CK and LDH nl <b>Albumin:</b> 3.2 <b>Total protein:</b> 6  <b>INR:</b> 2.8 <b>PTT:</b> 40 <b>Fibrinogen:</b> 5.2 <b>NH3:</b> pending</p> <p><b>EEG:</b> abnormal basic activity, delta range and sometimes theta. Intermittent generalised triphasic waves <b>c/w encephalopathy.</b> <b>LP:</b> not done due to INR.  <b>US abdomen:</b> small liver w/ nodularity, IVC and PV enlarged, no signs of thrombosis or ascites.</p> <p>-&gt; <i>empiric Tx with ceftriaxone, amoxicillin, vancomycin, acyclovir, thiamine, lactulose and rifaximin. Transported to ICU. <b>Melena noted, required 1 unit transfusion 2/2 Hgb drop to 7. Started on high dose PPI, PPSB, vit K.</b></i></p> <p><b>NH3:</b> 116 (high) <b>BcX:</b> negative <b>EGD:</b> esophageal varices, no active bleeding, no cherry spots / esophagitis or gastritis.</p> <p>-&gt; <i>Encephalopathy resolved with treatment.</i></p> <p><b>Dx:</b> <b>hepatic encephalopathy in context of warfarin-related UGIB (likely predisposition with ileal conduit) with non-decompensated cirrhosis.</b></p>	<p><b>Teaching Points (Julia):</b> <b>Slow deterioration : MIST</b>  <u>rapid</u> deterioration: Dissect a case: (rapid/reversible) causes vs. (severe)consequences of chief complaint  -&gt; <b>SCAN</b> Sugar CT scan (hypertensive encephalopathy, intracranial hemorrhage, PRES) ABG Airway Naloxone</p> <p><b>Asterixis:</b> (measure CO2) in ESRD <b>PLUS Hypotension</b>  Encephalopathic despite dialysis: ? YES: conditions because of dialysis: dialysis disequilibrium syndrome NO: Toxic metabolic consequence, shock</p> <p><b>Hyperacute encephalopathy :</b> ESRD (dialysis) -&gt; cirrhosis??  <b>Decompensated cirrhosis: liver:</b> infections w/ parenchymal dysfunction, increase in <b>portal pressure:(reduced blood supply)</b> a) portal <b>resistance</b> (thrombosis) or <b>reduced flow</b> in splanchnic arteries (vasodilation: Sepsis, bleeding, HRS)  <b>AMS 2/2 cirrhosis ?? Diagnostic criteria :</b> (1) Cirrhosis AND (2) decline in synthetic function (INR, albumin, Bili) AND (3) portal pressure increased (ascitis ?)  Alternative cause: <b>Ileum conduit</b> NAGMA, impaired ammonia clearance -&gt; encephalopathy (MRI, Hgb)</p> <p>melena -&gt; upper GI bleed, endoscopy (-); bleeding not result of portal hypertension (no - warfarin related bleed (stent)  <b>Ammoniaogenesis: cirrhosis type c</b> &gt;&gt;ALF (typ a), <b>type b</b> hepatic encephalopathy without cirrhosis, e.g. ileal conduit, Ammonium metabolism  Protein metabolism of blood high BUN/Cr ratio =&gt; Hyperammonemia (in vulnerable host)</p>