02/01/24 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: David Serantes(@davserantes) ***Case Discussants***: Rabih (@rabihmgeha) and Andrew(@ASanchez_PS)

CC: 64 F came to ER after one day of disorientation, incoherent speech and abnormal behavior.

HPI: Family mentions fall in the shower, but did not sustain trauma. No fever, vomiting, chest pain, abdominal pain.

Vitals: T: 37ºC **CHR:** 84/min **BP:** 135/63 mm Hg **RR:** 16/ min; SPO2 - 95% RA
Exam:
Gen: Not in acute distress
CV-Pulm: Normal
Abd: Soft, non distended, non tender
Neuro: Sleepy, confused, disoriented in time and space. Language was coherent, understood simple orders, but not complex tasks. Strength in limbs were normal
Extremities/skin: Normal

PMH:
Mild COPD
Hypothyroidism

Cholangiocarcinoma Stage IV (2020 - resected)- received adjuvant chemotherapy with capecitabine in May 2022, developed recurrence - hilar liver mass noted. She received chemotherapy and radiotherapy and in Dec 2022 - there was tumor progression. She was started on FolFox - last cycle - 3 days prior to presentation

Meds:
Levothyroxine
Lorazepam
FolFox

Fam Hx:
None

Soc Hx: Lives with her son, independent

Health-Related Behaviors:
Past smoker since 3 years
Does not drink/ use drugs

Allergies:
None

Notable Labs & Imaging:
Hematology:
WBC: 9.66 (85% neutrophils) Hgb: 11 Plt: 120 MCV - 96
INR - 1.1 ; APTT - 28 seconds
Chemistry:
Na:140; K: 4.0; Cl:105; HCO3: 22; BUN: 44; Cr:1.35; Glucose - 274; Ca:9.4
AST:38 ALT:35 Alk-P: 200 (baseline) ; GGT - 116 (baseline) Bili - 1.4;
Albumin: 3.2 CRP - 1 mg/dL ; VBG -pH - 7.4 ; pCO2 - 34 ; UA - normal
Labs (2 days prior- she DID NOT have AMS at the time of this test): TSH - 169 ; FT4 - 0.36 ; CA19-9: 300 (increased)
Hydrocortisone and T4 was started; patient's level of consciousness improved -> alert and oriented -> TSH - 6.5 ; FT4 - 1.0 (4 days after T4 Rx)
3 months later -> similar presentation with disorientation, incoherent speech (labs - normal at the time) CT head - Normal
Admitted to the ICU -> EEG at the time: Diffuse slowness ; The patient recovered within 48 hours; Ammonia during ICU stay- 196 (<50 = normal)

Imaging:
EKG (on current admission): Normal
CXR (on current admission): Normal
Head CT (on current admission) - Normal (with and without contrast)
CT chest, abdomen pelvis (current hospitalization): Pulmonary nodules disappeared; marked morphological appearance in the liver with pseudocirrhotic changes.

Dx: Hepatic encephalopathy

Problem Representation: 64 F with PMH of cholangiocarcinoma on FolFox chemotherapy and prior radiotherapy presented with disorientation, incoherent speech and abnormal behavior, and notable hyperammonemia on labs.

Teaching Points (Anmolpreet):
I] AMS: reduction in the consciousness level; Emergent causes: **SCAN**:-
- Fingerstick blood glucose to r/o hypoglycemia
- CT Head to rule out any acute intracranial pathology,
- ABG/VBG:- acidosis/alkalosis;
- N:Narcotics- narcan overdose

II] MINT causes for AMS:- T:Toxic causes
Metabolic: electrolyte abnormalities: uremia, hyperammonemia
Infection: CNS/ extra- CNS⟶ meningitis, encephalitis, brain abscess
Neurological: (just like AKI) **pre brain causes**: decrease in blood flow from shock/ hypoxemia; increased blood flow to brain in hypertensive emergencies, PRES,
Intra brain causes:- a lot of fluid around brain- edema, PACNS, Catatonia
post brain causes:- cerebral venous sinus thrombosis
III] Important to know if the problem is restricted to brain or systemic like metastasis ; and if it is a focal cortical problem/ diffuse cortical problem
IV] If the patient seems to have good consciousness level, then we need to think of focal intrinsic neurological problem more than AMS.
V] H/O Cytotoxic chemotherapy : makes us think of cell counts especially thrombocytopenia which can cause brain haemorrhage; therefore a brain CT is recommended.
VI] Overdose of Lorazepam is possible & especially in cases of COPD;can lead to hypercapnia;
Important investigations to be done: BMP, VBG
VII] BUN,Cr:- uremic encephalopathy can be present with underwhelming BUN,Cr and vice versa too, high values don't necessarily cause uremic encephalopathy
VIII] Hepatic encephalopathy: 3 types:- Type A, Type B, Type C;
Type A:- acute liver injury;
Type B: high ammonia with normal liver enzymes:- bypassing the urea cycle or inherent dysfunction with no existing liver disease present.
Also, drugs like 5-FU and Valproate inhibit urea cycle and can cause Type B HE
Type C: cirrhosis
Hyperammonemia can help us with diagnosing Type A and Type B hepatic encephalopathy; and not much with Type C (caused due to cirrhosis)
IX] Sudden extrinsic AMS which is MIST negative: urinary retention, constipation
X] Mild respiratory alkalosis: could possibly indicate hepatic dysfunction
XI] Radiographic evidence of liver dysfunction with underwhelming labs: we need ammonia levels which showed **hyperammonemia;** pseudocirrhotic changes: imaging findings of cirrhosis that occurs with hepatic metastases and is most common following chemotherapeutic rx of certain cancers
XII] Rx:- Address nutritional deficiency with Zn, replace 5-FU with some other drug, introduce ammonia scavenging drugs; look for portal vein thrombus;
important to check for ammonia levels; as this pt also had recurrence of similar episode