7/31/26 Morning Report with @CPSolvers

"One life, so many dreams" ***Case Presenter****: Lourenço(@)* ***Case Discussants****: Rabih(@) & Reza(@)*

https://clinicalproblemsolving.com/present-a-case/

Scribing (Lukas):
CC: 62 yo female PCP -> ED referral
HPI: asthenia, apathy, mutism, inability to walk for 6 mo. BI - speaks in short sentences and can walk.

ROS (-): fever, night sweats, arthralgias, myalgias, weight loss, anorexia, chest or abdominal pain, gastrointestinal or urinary symptoms

PMH:
Attempted suicide 10-15 years ago
Secondary encephalomalacia of the left frontotemporal lobes
Cognitive impairment and epilepsy secondary to encephalomalacia
obesity

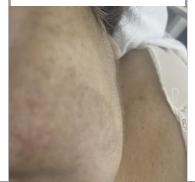
Meds:
Furosemide
Phenobarbital
VPA
Sertraline
Recently suspended: clozapine, risperidone, rivastigmine, clonazepam, quetiapine

Fam Hx: —

Social Hx:
resident of nursing home for 15 y

Health-Related Behaviors:

Allergies: no known allergies



Vitals: T: 36°C **HR**: 69 **BP**: 150/77 **RR**: 16 **Sat**: 98% on RA **BMI**:
Exam: Gen: centrally obese, round face, fine hairs with hyperpigmentation of the skin mainly in the face and upper trunk and upper limbs
HEENT: no LAD **CV**: nl **Pulm**:nl **Abd**: nl
Neuro: GCS 10 (E4V1M5), no speaking nor following commands most of the time. Was able to localize her pain. Pupils were symmetric and reactive to light. Could move all 4 limbs spontaneously. Strength seemed intact. No signs for upper motor neuro or lower motor neuron disease. No rigidity, no spasticity, normal reflexes

Notable Labs & Imaging:
Hb: 16,2 **WBC**: 6k monocyte predominance (1220, 14,3%)
PLT: 137k; **INR**: 1,6 (slightly elevated)
Chemistry: lytes: nl BUN: 35 Cr: 0.55
AST: 43 ALT: 23 ALP: 89 T. Bili: 0.44 GGT: 504 CRP: 48 LDH: 219
Ferritin: 1K **TSAT**: 28.9% Ceruloplasmin: nl (0,31) NH3: 250

EKG: wnl **CXR**: wnl
cCT: chronic microangiopathic demyelination pattern, ecstasy of CSF spaces, no acute injury/intracranial process of expansive nature

Valproate level: nl
Stopped, started on lactulose → ammonia levels went down & AMS improved
TSH: 13,2 **fT4**: 1,04 salivary cortisol 0,1 (normal) **ACTH**: nl (49,5)
ID screen: HCV + serology

Abdominal ultrasound: cholelithiasis, no- distended intrahepatic or extraheptic bile ducts. Normal liver, spleen, pancreas

HepC Serology: 1 mio copies Genotyping: 1A genotype

Fiber Scan: Severe steatosis with F2 fibrosis **Derm consil**: hypertrichosis lanuginosa

Porphyria panel: elev. Uroporphyrine. Pentaporphyrine. heptacarboxyporphyrine. stool positive forisocoproporphyrine, NGS negative

Dx: Hyperammonemia and PCT 2/2 to new onset diagnosis of chronic HepC

Problem Representation: 61 yo female referred from PCP to the ED with asthenia, apathy, mutism, inability to walk for 6 months combined with features of hypercortisolism turned out to be chronic HepC with hyperammonemia and PCT.

Teaching Points (Preethi)
Approach to any neurological problem: is it a new problem unrelated to the existing medical data or it is a build up on the previous neuro-psych PMH?
Usually, in advanced dementia, there will be low BMI. unusual to have a higher BMI in advance cognitive impaired patients but can happen in primary eating/depressive disorders.

Inability to walk→neurological vs psychological?
Neurodegenerative disorders: FTD with hyperorality, vascular dementia with metabolic risk factors
Other: Rapidly progressive dementia, Cushings

Fine hair growth in male distribution pattern: excess androgens.
Hirsutism: male pattern distribution
Hypertrichosis: excess hair in female pattern distribution

Hyperpigmentation: occurs in adrenal insufficiency rarely in cushing's, but can happen in ACTH producing tumors both primary (pituitary adenoma) and ectopic ACTH producing tumors
Continuous use of dexamethasone-> ACTH is suppressed. Usually very sick without steroid supply in the hospital.

Hyperammonemia: think about valproate tox. Check VPA level.
Tx: lactulose, rifaximin

Association of Hepatitis C and Porphyria cutanea tarda. Can present with hyperammonemia iso liver dysfunction. Get Fibroscan to understand the fibrosis of liver. (Get an MRI if you want to evaluate the presence of portosystemic shunting)
Skin changes in PCT/Hep C: can present with Hypertrichosis Lanuginosa- fine, soft, unpigmented lanugo hair.