8/23/24 Morning Report with @CPSolvers

***"One life, so many dreams" Case Presenter**: Kara Lau(@ytk_lau) **Case Discussants**: Rabih Geha (@rabihmgeha) and Reza Manesh (@DxRxEdu)*

CC: Malaise for 2 weeks and intractable N/V for 2 days

HPI: 48 yo F brought to the ED by ambulance w/ worsening malaise and mild to recurrent N/V for 2 weeks, Worsening symptoms last night.
Vitals in the triage room: BP: 118/54 T: 36°C, HR 89/min, RR 19, O2Sat 99% on RA.
No abd pain, fevers, chills, chest pain.

Initial labs:
WBC:7 Hb 16.1 plat 182
Na 137, K **3.3**, Cl **112**, pH **7.14**
pCO2 **23**, Anion-Gap: **20**, Glucose: **236,** BUN 28, Crea 2, AST 402, ALT 730, AP 337, Alk Phos **637,** Bili 1.9, Lipase 363, Trop neg, HbA1C **10.3**,
VBG: **7.14** Lactate 2.9 , ß-HB 4.8!
UA: >1000 Glc, > Ketones
Patient later developed LFTs > 1000 and dark urine.

PMH:
DM (20yo,unclear type, t/w Insulin)
Meds:
Lantus 24 Units
Atorvastatin

Health-Related Behaviors:
No recent travel or sick contacts

Vitals: T: HR: 119 **BP:** 163/91 **RR:**20 **O2Sat:** 95% RA **BMI:** 28
Gen: Alert and oriented 4x, **HEENT:** icteric sclera, **CV:** rrr, no murmurs, **Pulm:** lungs clear, **Abd:** distended, soft, non tendon, dull to percussion, fluid wave, **Neuro:** no asterixis, **Extremities/skin:** bilateral +2 pitting edema

Notable Labs:
Plt: **82,** INR 1.7, Cr: 5,53, AST ALT >600 , AP 800s, Lipase >7k, Ferritin >13k
Lact 1,1 Trop 0,41
Follow up:
CBC: stable ß-HB 0.44 after treatment, VBG 7.22, pCO2 37, Crea: **6.84,** Bicarb 11, AG 18, S-Osm **302**, Neg Salicylates, neg Utox
Anuric, LFTs continues >1600
Ascites TAP: no exsudation, no evidence of SBP
UA muddy brown casts -> dialysis.
LFTs declined after dialysis. Neg Hepatitis panel, HIV, autoimmune workup (ANA low, anti- Smooth muscle low), CMV, EBV and Adenovirus PCR neg.
Normal ceruloplasmin and copper, sIL2R high
Hemochromatosis panel: H63D heterozygosity

Imaging:
MRI: moderate ascites and small bilateral pleural effusions. No evidence of biliary obstruction or cholecystitis.
USG Abd: possible hemangioma noted involving the right hepatic lobe on previous study of 2022 not visualized on today's study.
Bilateral non obstructive nephrolithiasis, spleen and gallbladder unremarkable. Echo: normal. IVC small.
Dopper US: no evidence of clot in liver vasculature
MRI: Mild to moderate wall thickening/edema of the visualized small bowel loops. Small ascites.

Dx: Multiorgan failure secondary to DKA

Problem Representation: 48 yoF w/ a PMH of DM on Insulin p/w 2 weeks of malaise and acute N/V, AGMA (DKA) and NAGMA, severe acute liver injury, AKI 2/2 prerenal leading to ATN, hyperferritinemia, acute pancreatitis.

Teaching Points (Anmolpreet):
I] Tracking in triage room : were they able to ambulate/ not? Vital signs?
II] Nausea/vomiting: triggering of vagus nerve in a location.
1. *Localising the nausea/vomiting*: brain (nervous system exam), heart (ECG) and upper GI tract.
2. *Deficiency of substance*: Adrenal Insufficiency,
Excess of substance: beta hcg, cannabinoid hyperemesis syndrome
III] pH:low→**Anion Gap Metabolic Acidosis:** Renal insufficiency, ketones, lactate, toxins/meds; presence of ketones in urine and uncontrolled DM makes us think of DKA in this patient; now we are concerned about the trigger for DKA. ***Treating DKA***: fluids, potassium, insulin
Triple acidosis in patient: AGMA(ketones), NAGMA(vomiting, diarrhea), Respiratory acidosis
IV] Elevated ALP: in this case signals bile duct injury; whether intrahepatic or extrahepatic ducts are involved is reflected on by imaging. Pre-test probability can be guessed from transaminases level (liver-intrahepatic) and lipase levels (extrahepatic)
V] Causes of Acute Pancreatitis: 1. Alcohol, 2. Gallstones→ only extrahepatic ds that can cause elevated transaminases(because of backward mechanical pressure), 3. **Hypertriglyceridemia**
VI] Elevated transaminases~1000 (Severe acute liver injury): *ischemic liver ds* (thrombosis of portal vein-pt need not be hypotensive), ***hepatitis virus***(and herpes virus), ***toxins***(tylenol), ***stones ;*** In severe acute liver injury, we need to figure out if the patient is having acute liver failure (encephalopathy, asterixis) or does not eventually develop it.
VII] Ascites: tap especially to look for SBP and portal hypertension. Portal hypertension comes as a result of chronic fibrotic liver ds; so presence of ascites with no apparent cause other than portal htn makes us think if the pt had chronic liver ds
DDx: acute decompensated right HF, Budd-Chiari syndrome→ BNP, TSH, free T4
Normal heart tests rules out post hepatic causes of portal htn, we think about intrahepatic/ prehepatic→ so we think about any infiltrative lesion of liver (occupying space)
VIII]Persistent anion gap even post fluids makes us think if DKA was actually causing it since it improves with fluids; so we are concerned about toxins. Also it makes us think if renal insufficiency/uremia is also contributing to metabolic acidosis
IX] Real-world case with multiple comorbidities and problems→ with a simple diagnosis of DKA, hypovolemia causing multiple organ failure.