# 7/3/26 Morning Report with @CPSolvers

*"One life, so many dreams"* **Case Presenter**: Alexander (Alex) Winkler(@) **Case Discussants**: Rabih(@) & Rahul(@)

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

Scribing (Lukas)

**CC**: 73 yo woman with suspected dehydration

**HPI**: reported feeling tired and fatigued that afternoon. Had some episodes of stomach pain so she lied down. Very sleepy with difficulty to wake her up. 1x prior episodes of non bloody emesis. EMS was called and brought into the ED

**ROS**:

**PMH**:
HTN
Recurrent stomach pain (chronic gastritis)

**Meds**:
Pantoprazol
Bisoprolol
candesartan

**Fam Hx**: -

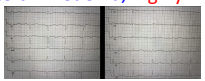
**Social Hx:**
Avoids medical system, poor therapeutic adherence
Baseline: fully independent
Lives with husband

**Health-Related Behaviors:**
No smoking
no alcohol or other substance abuse

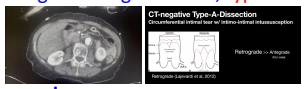
**Allergies**: -

**Prehospital:**
**Vitals: T**: wnl **HR**: 85 **BP**: 110 **RR**: **Sat**: **BMI**:
**Exam: Gen**: non verbal, non responsive
**HEENT**: **CV**: **Pulm**: **Abd**:
**Neuro**: tried to defend herself, closed her eyes and slept again, chewing like movements - new onset; arms crossed over chest
**Extremities/skin**:
**Course:** 500 cc ringer lactate - slight improvement to stimuli

**Hospital:** vitals stable on arrival
**Neuro**: concerned for nonconvulsive status epilepticus - planned administering levetiracetam
Soporous patient, roving eye movements and Gaze preference to the left without forced gaze shift; corneal reflexes were intact, visual fields on threatening gestures seems normal. No facial motor deficit. No nystagmus. Patient wasn't able to communicate at all or follow any commands. Strong motor response in all four extremities in a symmetric fashion. No tongue bite, Babinski downgoing bilaterally
**ABG: pH 7.18, pCO2 68, HCO3 16, Glu 209 Lactate : 9 Hb 14,6**
Keppra drip administered -> SBP 60s
**Physical**: Unchanged eyes widely open after provocation, eye contact for split second. Stable peaceful position with folded hands. Unable to follow commands. Pupils equal and symmetric in size, slightly delayed light reaction
**Skin** - No Hives, skin - cool, prominent mottling of chest, no bilateral LL edema, highly positive JVD



**ECG:** wnl
**Echo:** large pericardial effusion/tamponade not hemodynamically relevant, reduced EF, Aortic ectasia/insufficiency (ascending), improved atrial filling after large infusion, type B dissection below the renal arteries




**Dx: obstructive shock due to type B aortic dissection and CT-negative Type-A-dissection with pericardial tamponade**

**Problem Representation**: 73 yo woman with HTN and chronic gastritis p/w AMS, abdominal pain and highly elevated JVD without LL edema and large pericardial effusion with reduced EF to the ED quickly developing shock. While CT was only showing clear aortic dissection below the renal arteries there was suspected CT-negative Type-A-dissection associated to the pericardial tamponade.

**Teaching Points (Rahul's fans)**

**Approach to AMS + emesis** - ABCDs, Identify tempo (hyperacute vs acute) and set the scene - what changed recently, check glucose, assess hemo stability, acquire more history
**Differentiate hyperacute encephalopathy** -> focal vs diffuse -> Stroke (CT angio) vs substances (Sugar, opioids) {consider UDS} - negative -> EEG for status and LP
**Approach to facial movements** - 2/2 seizure vs neurological deficit -> Work getting CT to r/o Stroke.
**Suspecting stroke/Seizure ->** Ability to overcome through stimulation -> likely indicates substance. One consideration -> bacterial meningitis
**Approach to anion gap metabolic acidosis in this context with elevated lactate** -> elevated glucose -> get BOHbutyrate + creatinine + alcohol levels
**Approach to hypotension** -> hypovolemia, blood loss, hypoperfusion, sepsis (reqs. Blood cultures -> start broad spectrum Abx (?Vanc cefepime + 2x BCx)
Hypotension 2/2 levetiracetam is known
**Lactic acidosis** -> poor o2 delivery to tissues vs seizures vs *type B lactic acidosis {( no ischemia or hypoxemia) mitochondrial toxicities (most likely) , hepatic dz ( hypoglycemia), excessive catecholamine stimulation (cocaine/methamphetamine use, albuterol, pheochromocytoma)}*
**JVD** - > distension usually takes time -> s/o central obstruction -> pressure related or volume related vs obstructive physiology ( tamponade, PTX, Massive PE) -> POCUS + d-dimer + BNP + Cardiac troponins
**Approach to central obstruction** -> POCUS + CT scan -> may require circulatory support
**Low voltage on EKG ->** consider if patient has high BMI
**Pericardial tamponade** -> Cardiology opinion -> drain -> identify characteristics of fluid -> transudate, exudate (pus is important), blood -> support pressures; Tamponade + Prolonged QT -> check all electrolytes + TSH
***Encephalopathy with response to stimulation*** *-> consider hypoperfusion -> global leading to poor response to pressors + cerebral perfusion is low 2/2 carotid or vertebral issue*
***Blood in the pericardium*** *-> rupture to -> atria - iatrogenic; ventricle - ischemia (free wall rupture); aorta - new dissection or aortopathy*
*CT- (-) type A dissection -> intussusception -> Retrograde usually but rarely antegrade*
*AA tear -> propels down -> to infrarenal aorta (no lumen b/n intima and media)*
***"Try to *unify as many findings as possible with one core mechanism*, and only accept multiple diagnoses when a single, mechanistically coherent explanation truly fails."***