10/19/23 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: Promise Lee (@promiseflee) Case Discussants: Rabih Geha (@rabihmgeha) and Gerardo Luna (@gerarlunap)

CC: 25F presented with R side facial droop, aphasia and weakness.

HPI: Patient started 2 weeks ago with SOB, intermittent headache and changes in behavior.

She was normal 2 hours before but she presented with R side facial droop and R side paralysis. No history of trauma.

PMH: none

Meds: none

Fam Hx: young family members with CKD on dialysis

Soc Hx: none

Health-Related Behaviors: no smoking, no alcohol use, no drugs, no history of recent travel or triggers

Allergies: none

Vitals: T: afebrile **HR:** 80´s **BP:** 180/127 **SpO2:** 93% RA
HEENT: Left pupil constricted
CV: Regular rate and rhythm
Pulm: Crackles
Neuro: R side paralysis, R side facial droop and R side weakness

Notable Labs & Imaging:
Hematology:
HB: 8.8. Rest normal
Chemistry & other labs:
Cr: 8.7, electrolytes abnormal, BUN elevated
Trop 36.4, BNP: elevated
Normetanephrine: 683 (H), 24h metanephrines: 830 (H)
Aldosterone: 32 (H), Renin: 2.8, aldosterone:renin ratio 12, TSH normal
UA: 1+ heme, 3+ protein, 8 WBC, 9 RBC. 24h urine protein: 2500
ANA 1:80, cytoplasmic, mitotic intracellular
All viral testing -ve. Sjogren, RA -ve.

Imaging:
CXR: enlarged cardiomediastinal silhouette with diffuse GGO and increased interstitial markings suggestive of fluid overload with pulmonary edema.
CT: focal intraparenchymal hematoma 5x2x3 cms in left basal ganglia and external capsule, edema and local mass effect. Left to right midline shift.
TTE: 43% EF. LA and LV moderately dilated. Global hypokinesis, mod-severe LV hypertrophy. RV normal.
Renal USG: atrophy on right side more than left, no hydronephrosis
Intracranial angiogram: normal
CT angiogram: opacity on lung upper lobe, mediastinal lymph nodes
New TTE: 35% EF

Kidney biopsy: diffuse global glomerulosclerosis, chronic tubulointerstitial disease, no evidence of complement mediated GN

Diagnosis: to be determined…

Problem Representation:
A 25 yo F presented with a 2 week history of R sided weakness, facial droop, and aphasia. Laboratories showed anemia, electrolyte abnormalities, cardiac and renal dysfunction. Imaging revealed cerebral hematoma, pulmonary edema and cardiac abnormalities. The case is still under diagnostic work up.

Teaching Points (Bettina):
- **Peripheral vs. central facial palsy:** Check for other cortical or subcortical signs, risk factors depending on age
  - Bell's palsy: Unilateral, acute in onset, isolated (no systemic, no other neuro sx)
- **Aphasia:** Language impairment (problem with what the person is saying)
  - Versus dysarthria (problem with how to say something, more diffuse)
  - Check comprehension (follows commands), fluency, writing, reading, repetition
- Time course can help differentiate etiology
  - A hyperacute syndrome superimposed upon a subacute syndrome may mean that she has mass-forming disease (abscess, cancer) that either bled, caused a seizure, caused obstruction through hydrocephalus, or vascular + mass effect
  - Causes of stroke: Atherosclerosis, antiphospholipid syndrome/SLE, cardioembolic disease, multiple emboli (which can cause headache)
  - **Kernohan syndrome:** Lesion causing mass effect on the contralateral midbrain against the tentorium cerebelli
  - Causes of intraparenchymal hemorrhage: microvascular disease, anticoagulation, trauma, AVMs, angiopathy, hypertension
    - Hypertension can be a physiologic response to an ischemic event (check if it may be long standing)
      - To determine chronicity of HTN, look for retinal changes (nicking, exudates), kidney damage (creatinine), LVH on EKG
      - Current debate on whether isolated HTN can cause kidney disease
      - Secondary HTN: Renal, adrenal, coarctation, OSA
- CKD: Intrinsic (HTN, DM, GN, calcineurin inhibitor) vs. postrenal causes (interstitial nephropathy due to HTN), familial (usually if noninflammatory), Sickle cell disease, Fabry disease (angiokeratomas, peripheral neuropathy), gammopathies (electrophoresis)
- Increased NE: Paragangliomas vs. pheochromocytoma vs. acute illness
- Intrinsic lymph node disease vs. lymphatic congestion
- In patients with kidney disease, when they have extrarenal manifestations, they occur for 2 reasons: they may have consequences of the body's adaptations to renal failure (HTN, hyperkalemia, acidosis, encephalopathy) causing issues elsewhere but not specific to the underlying cause of kidney disease; or you can have a systemic disease affecting the rest of the body that can point to the exact etiology
- A bias is only if you refuse to update your thinking given more data :)