1/15/26 Morning Report with @CPSolvers

"One life, so many dreams" ***Case Presenter****: Fahed* ***Case Discussants****: Rabih (@rabihmgeha) & Kirtan (@KirtanPatolia)*

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

Scribing (Gillian)

CC: 58 yo female progressive breathlessness and progressive bilateral limb swelling for 3 mo.

HPI: breathlessness insidious in onset class with rapid progression I→IV
PND, orthopnea, no chest pain, syncope, fatigue prior
Painless symmetric swelling in upper and lower limb

ROS:
Positive for PND, orthopnea.
Negative for constitutional symptoms (fever, anorexia), decreased urine output, trauma, local inflammation

PMH: bipolar affective disorder (30yr), Hypothyroidism (presumed secondary to lithium)

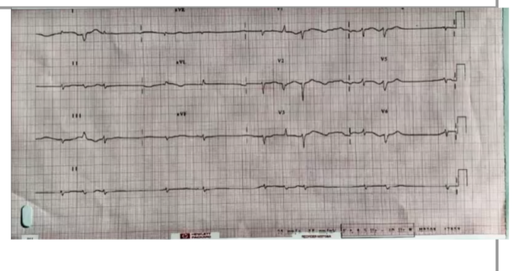
Meds: lithium, olanzapine, escitalopram, levothyroxine

Fam Hx: unremarkable

Vitals: T: afebrile **HR**: 80 **BP**: 110/70 **RR**: 16 **Sat**: **BMI**:

Exam: Gen: conscious, oriented x3, not dyspneic at rest
CV: irregularly irregular w/ variable volume, no JVD, no added sounds, no pericardial rub
Pulm: normal vesicular breath sounds, fine basal crackles bilaterally
Abd: unremarkable with exception for ascites
Neuro: nl **Extremities/skin**: bilateral pitting edema of UE and LE

Notable Labs & Imaging:

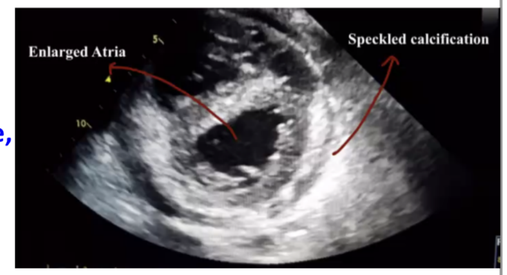


Hematology:
WBC: nl Hgb: 12.2 Plt:6.3 (630k) MCV:

Chemistry:
Na: nl K: nl Cl: nl HCO3: nl Cr: 1.6 BUN: 106
AST: nl ALT: nl Albumin: 1.9
TSH: elevated, FT3 and FT4: mildly decrease; Anti-TPO: elevated at 1589
ANA profile wnl, complement wnl; Blood lithium: 0.6 (nl)

Imaging: UA: trace 1+ protein
EKG: irregularly irregular rhythm w/ ventricular rate of 80. Low voltage QRS, sinus pauses, multiple PVCs. No discernible P waves
CXR: cardiomegaly, no pleural disease, normal lung fields
Echo: severe PAH, RA and RV dilation, LV EF 55%; Repeated 1 yr later: enlarged atrium & speckled calcification, atrial standstill
HRCT: bl pleural, pericardial effusions, moderate ascites, no signs of PE / ILD – parenchyma looked ok
Left due to financial constraints, represented 1 yr later
Fat pad biopsy confirmed amyloidosis

Dx: Amyloidosis w/ restrictive cardiomyopathy,
SA Node dysfunction and type 2 cardiorenal syndrome,
Severe PAH 2nd to restrictive cardiomyopathy.



Problem Representation: 58 yo female w/ bipolar disorder treated w/ lithium for 30 yrs presents w/ breathlessness and bl upper & lower extremity edema. She was found to have afib w/ low voltage QRS and severe PAH w/ preserved LVEF, low albumin, and anti-TPO + hypothyroidism. Fat pad biopsy confirms amyloidosis

Teaching Points (Eyron)

Edema in Heart Failure
-Swelling in HF related to how much above baseline the venous pressure is elevated compared to the absorptive capacity of that part of the body
-The pressure to push fluid out increases more distal from the heart (e.g., toes) in addition to gravitational forces
-Lymphatics, Venous Pumps, and use of the limb = increased resorption
-Seen less in the upper extremities due to constant motion and venous pump in the arms and decreased amount of pressure needed to absorb fluid in the arms
-Venous hypertension is proportional to the amount the body has to fight against gravity

SVC Syndrome
-Suspect in patients disproportionate swelling of the upper extremities
-Thoracic duct drains lymphatics of all the body
-Impairment of drainage at the level of the mediastinum can result in swelling

Variable Volume
-Could be due to inability to pump properly - consider pericardial issue secondary to mediastinal mass vs. lymphadenopathy
-Consider Cardiomyopathy impairing contractility

Low QRS Voltage
Diffuse myocardial infiltration vs. pericardial disease
Concomitant conduction system disease and UE edema suggests venous HTN - myocardial > pericardial issue

Anasarca
Consider structural issues (venous valves, heart, kidney, liver) vs.
Substance (albumin, thyroid hormone, exogenous medications such as amlodipine, gabapentin)

Hypoalbuminemia, pulmonary HTN, and hypothyroidism
-Pulmonary HTN can be a consequence of chronic thyroid disease
-Hypothyroidism can lower albumin by decreased hepatic protein synthesis increasing capillary leak
How can we tie everything together?
-Albumin losing process → thyroglobulin loss + hypothyroidism → pulmonary HTN

Protein Losing Enteropathy
-Can be a consequence of severe elevated right sided heart pressures (pulmonary HTN) via compression of gastric lymphatics leading to protein loss