05/20/24 Cardiology VMR with @CPSolvers

"One life, so many dreams" Case Presenter: Dr. Maulin Shah(@shahmaulinb) ***Case Discussants****: Dr. Michelle Kittleson (@MKittlesonMD)*

CC: SOB and dry cough

HPI: 19 F with no PMH presented with 2 months of dry cough. Initially, she felt tickle in the throat which triggered cough and she also endorsed a sense of SOB. She was presumed as CAP by PCP but didn't have improvement with 5 days Tx. She endorsed morning HA in frontal area for several months, associated with vision change and tinnitus. She had joint pain which didn't respond to OTC medicine.

ROS: endorsed decreased appetite, nocturia, exertional fatigue, weight loss; denies fever, chills, chest pain, abd pain.

PMH:
Seasonal allergies
rhinitis

Meds:
None

Fam Hx:
DM2 on father
Health siblings

Soc Hx: College student no sex history

Health-Related Behaviors:
Denies tobacco, alcohol, drugs, no recent travel or sick contact

Vitals: T: 98.2 **HR:102 BP:172/106 RR:** 26 **SaO2:**100%@RA Wt:98pl BMI 18.5
Exam:
Gen: mild distressed
HEENT: JVP 12cm
CV: tachycardic, no murmur
Pulm: decreased sounds in the base lung, crackles in bl up to mild lung
Abd/Neuro: nl
Extremities/skin: no edema, small exorciations in RLE and 2 lesions on LLE. Hyper-extension of elbow and wrist joints

Notable Labs & Imaging:
Hematology:
WBC:11.5 (neutrophil dominant) Hgb:9.9 (11.2 one year)Plt: 454 (562)
Chemistry:
Na: 136 K: 3.1 Cr:0.8 AST: 66 ALT: 53
ESR:55 CRP: 12.7 Troponin 0.11 BNP:2734 Lac:0.7
AM Aldosterone: 12 Renin:26.12 Ratio: slightly low
AM cortisol: 581 (high)
ANA:1:40 Neg: SSA/SSB, smith Ab, RF, Smith Ab, Scl-7 Ab; normal complement
UA: nl STI: neg; Urine toxin: neg; Viral panel: neg
Imaging:
EKG: sinus rhythm, rate 93, PR 114, QRS 84, QTc 544, large QRS in V2 and V3
CXR: mild cardiomegaly with pulmonary edema, small bl pleural effusion
Echocardiogram:LVEF 15%, global hypokinesis, multiply left apical mural thrombi, IVC dilated and less than 50% collapsible with elevated RA pressure
Cardiac MRI: LEF 27%, minimal LGE, minimal pericardial edema, diffuse pericardial inflammation
Left cath: no evidence of coronary disease
MRA Chest: mild enhancement of mid-abnormal aorta and the proximal renal arteries; mild diffuse mural thickening of the distal arch through the abd aorta. Stenosis of renal arteries, distal thoracic aorta and origin of SMA.
Dx: Med-large vessel vasculitis
The pt received prednisone with improved EF and Sx

Problem Representation: 19F with no PMH presented with 2 months of SOB and dry cough, along with headache, joint pain and weight loss. She was found to have hypertension, tachycardia, heart failure and diffuse vessels change.

Teaching Points (Anmol): ***know the right questions to ask! :)*** → Dr. Kittleson
I] Reasoning out! : SOB is something that can bring to ER, and the dry cough as a result of that. Morning headache makes us think of increased ICP 2/2 space occupying lesion but too soon to conclude that. Differentials for SOB:- ***(organise D/D by etio and pathophys)***
1. Cardiovascular :- arteries (dissection, anomalous), electrical (arrhythmia- palpitations), pericardium, myocardium
2. Pulmonary: parenchyma, pleura, vasculature
3. Psychiatric: anxiety, stress;
4. Deconditioning/ Acid-Base disorders
5. Neurologic :- muscle weakness (diaphragm involvement)

II] Hypertension in a young: needs to be evaluated, cannot be only due to stress response; **Collagen vascular ds** comes to mind→ look for *abdominal bruit*(not very specific) 2/2 renal artery stenosis; the connective tissue affected can explain the fragility of skin or vascular phenomena of brain leading to morning headache. Ask for history of cocaine abuse. Pheochromocytoma is also one imp differential for secondary causes of hypertension. Skin findings → angiokeratomas seen in infiltrative cardiomyopathy

III] High AM cortisol:- could indicate a Cushingoid picture: pituitary→mineralocorticoid excess→ hypokalemia, hypertension→ HF; Next steps: consult Endocrinologist, ACTH, look for moon facies, skin striae, free urinary cortisol, dexa suppression test ***(know who to consult)***

IV] High BNP:- hypertensive cardiomyopathy could have led to increase in troponin & BNP .

V] Hyperacute/acute HF: young people can compensate for a long time, so seemingly acute ppt could be chronic; correct way to divide it would be on the basis of ECHO→ HFrEF and HFpEF!
Old pt: **Ischemic cardiomyopathy** more chances; **Non ischemic causes :** endocrine, toxins

VI] EF of 15% producing such high blood pressures: we believe some cortisol-driven process→ hypertension→ driving the whole process→ hypertensive cardiomyopathy

VII] Cardiac MRI: we mainly look for e/o amyloidosis, HOCM, myocarditis, pericardium

VIII] Types of myocarditis important to be ruled out : giant cell myocarditis (biopsy-needed), lymphocytic myocarditis, viral myocarditis ; **Diagnosis and management:-** biopsy only in 2 conditions: 1. cardiogenic shock 2/2 giant cell myocarditis 2. New onset HF (wks-months)

IX] Suspected Vascular involvement→ + high inflammatory markers→ vasculitis→ aorta & renal art.→ medium-large vessel vasculitis→ <50 years→ Takayasu arteritis? → hypertension is the main driver because of RAS leading to cardiomyopathy and HF→ **Rx: normalising BP**