01/4/24 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter:Vijay Balaji(@vijaybramhan) Case Discussants:Rabih (@rabihmgeha) and Mengyu(@zhoumy07)

CC: 18M with worsening dyspnea since 3 days. (Clue- Gene or anatomy?)

HPI: Sudden breathlessness (when trying to climb stairs), progressed over one day, unable to walk even few stairs. Left sided dull aching anterior chest pain, worsens with deep inspiration. Few hours later, he developed lightheadedness (transient - 2-3 minutes), no LOC or involuntary movements. Exertional palpitations ++. No orthopnea, PND or recent fevers.

PMH: Recent Dx of unprovoked intermediate PE (involving left main and right seg arteries) Initial Protein C and APLA workup - negative

Meds: Rivaroxaban (taking appropriately with food)

Fam Hx: Not significant (no prothrombotic states known)

Soc Hx: No addictions

Health-Related Behaviors:

Allergies:

Vitals: T: HR: 128/min **BP:** 104/70 **RR:** 34/min ; SpO2 - 92% RA

Exam:

Gen: No rashes, no loss of weight, no loss of appetite

HEENT: wnl

CV: wnl

Pulm: Crackles left infrascapular areas

Abd: wnl

Neuro: wnl

Extremities/skin: wnl

Notable Labs & Imaging:

Hematology:

WBC: 12.9 Hgb: 13.7 Plt: 110

Chemistry:

Comprehensive metabolic panel - wnl

AST: wnl ALT: wnl Alk-P: wnl

PT - 12.7 seconds; INR - 1.03 ; aPTT - 40 seconds

Lupus anticoagulant (repeat) - positive ; beta 2 glycoprotein - negative; homocysteine -13 (borderline elevated); MTHFR - negative

UA: Normal

Troponin - 100 (high sensitivity) ; pro BNP - 1278

Imaging:

EKG: Sinus tachycardia

CT Chest: New segmental PE in the RUL, RML, RLL branches with left LL infarct

POCUS: IVC thrombosis

CT venogram: Nutcracker phenomena (prominent left gonadal veins and multiple venous collaterals, compression of left common iliac vein b/w the right common iliac vein and L4 vertebral body)

Dx: **Low risk APS with May Thurner Syndrome**

Problem Representation: 18M with sudden SOB and chest pain, recently diagnosed with PE, developed repeat PE despite rivaroxaban. Hypercoagulability workup was initially negative, but later revealed low risk APS (lupus anticoagulant positive) and imaging showed May Thurner.

Teaching Points (Oumaima):

. Young patient: Bad luck - Bad behavior - bad genes - Bad environment

. Keep congenital and genetic causes in the differential

. Young patient with sudden onset dyspnea and chest pain makes an underlying disease process more likely: Scrutinize the history

. Chest pain added to dyspnea makes the probability of pleural and cardiac causes higher "Deeper problems"

.-If the pain is unilateral: pleural or pericardial

.-Non-massive PE gives us more time vs mi, dissection

. Provoked PE: presence of trigger (trauma-surgery- long flight) - severe underlying risk factor is less likely

. Unprovoked PE needs work-up, the work-up has to be agnostic to the PE "look behind the PE-curtain": Detailed history, family history , pregnancy history - Labs: factor V leiden - antiphospholipid antibodies -

. Unprovoked PE often needs lifelong anticoagulation: screen family members - cancer

. Severity of PE (hemodynamic consequences) doesn't influence workup

. Thrombosis: Vessel problem (occlusion) or blood problem

. PE with Rivaroxaban (2Ms Meds or Mimics) observance - drug/drug interaction - malabsorptive syndrome : factor Xa levels can be helpful - Mimic (Filling defect is not always thrombus (Tumor thrombus: Resists anticoagulation) and Ultra Hypercoagulability (Occult cancer - P.Vera - Eosinophilic myocarditis - Compressive syndromes- Behcet)

. Compressive syndromes that may cause thrombosis: May-Thurner Syndrome, 90% of people with May-thurner anatomy don't have the syndrome,

. Repeat APLS labs : Anticardiolipin / anti beta 2 glycoprotein / lupus anticoagulant