



9/12/24 Morning Report with @CPSolvers



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CC: 42 y/o M presents with 2 weeks of chest pain.

HPI:

Chest pain was characterized as constant, substernal, and radiating to the R shoulder. His pain acutely worsened over the last day along with the development of new onset SOB. His chest pain improved with yard work, but he denied worsening with deep breaths, cough, positional changes or exertion.

PMH:
HIV
H/o Toxoplasmosis w/ CNS involvement & epilepsy
Factor V deficiency
Cardiac arrest with anoxic infarction of b/l thalami

Surgical Hx:
Percutaneous gastrostomy placement

Meds:
Dapagloflosin
Dolutegravir-Rilpivirine
Lacosamide
Levetiracetam
Levothyroxine
Metformin

Fam Hx:
DM - mother

Health-Related Behaviors:
Vaping daily
Marijuana daily
Drinks pints of hard liquor once per month

Allergies:
NKA

Vitals: T: 98.2 F HR: 58 BP: 104/79 RR: 19

Exam:

Gen: No acute distress

HEENT: normal

CV: Bradycardic, regular rate, no murmurs, rubs, or gallops, normal S1 S2, no peripheral edema, no JVP

Pulm: Clear to auscultation b/l

Abd: No abd tenderness, normal bowel sounds

Notable Labs & Imaging:

Hematology:

WBC:11.3 (neut: 86.1%. Lymph 10.7%. Mono: 3.2%. Abs & relative eosin: 0%) Hgb: 12.7Plt: 314

Chemistry:

Na:130 K:4.5 Cl: HCO3:18 BUN: 21 Cr:2.045(baseline ?)glucose:125 Ca: Mag:
AST: ALT: Alk-P: Albumin: wnl lipid panel: wnl
Troponin 7.75 Ck MB 43.3 BNP 2857
TSH 4.04 ESR 120. CRP>31.7
CD4: 447

Imaging:

EKG: 78 bpm, regular rhythm, LAD, Low voltage QRS, ST elevations in V1-V4, II, III, AvF
CXR:
Echocardiogram: LVEF 25-30%, RV systolic function mod-severely reduced

Toxocara Ab, Strongyloides Ab, Quantiferon Gold, Lyme Ab, N. Gonorrhea & Chlamydia negative
Blood cultures x2 for 5 days negative

Coronary angiogram: RH: Low filling pressures, reduced CO, elevated SVR, improved with IVF & Dobutamine

Cardiac MRI: Extensive pericardial & mid-myocardial enhancement, decreased ventricular function, LVEF 30% RVEF 16%

Cardiac biopsy: Myocardial infiltration by lymphoplasmacytic cells, multinucleated giant cells, eosinophils; no necrosis.

Dx: Eosinophilic Myocarditis

Problem Representation: 42 y/o M presented with 2 weeks of chest pain, 1 day of SOB, elevated troponin and diffuse ST elevations. Reduced LV function was noted. Cardiac biopsy showed lymphocytic infiltration, multinucleated giant cells & eosinophils, confirming eosinophilic myocarditis.

Teaching Points:

Don't miss diagnosis in approach to Chest pain: 4+2+2 framework

4: cardiac -> ACS, takotsubo, tamponade, dissection 2:pulmonary -> PE, pneumothorax
2:esophageal -> impaction and perforation

First pass to check these out -> ECG, cxr, cardiac biomarkers

What does the subacute presentation tell us?

Unusual presentation of something usual,
Coping mechanisms by the patient kicking in making them take longer to come to the hospital
High reserve in patient -> pericarditis without initial effusion in patients with higher cardiac reserve
Check for how the pain evolves -> the point of pain is to protect you from dysfunction so if there is no dysfunction the problem is probably with the nerves

Risk evaluation of chest pain

Most patients with chest pain have a benign cause, we use physical examination and rule out tests to identify those with highest risk first. In those that we suspect a sinister cause but can't find an immediate cause the places it can hide itself include: Microscopic arteries of the heart, Mediastinum, Subclinical CV process -> Large effusion not yet developed

Putting the initial data together

Troponin stays elevated longer, while ck-mb goes down much sooner which is why its used for testing for re-infarction -> all point to heart muscle damage -> Heart muscle damage can be caused by Takotsubo, myocarditis, missed infarction
Low voltage QRS -> Are things in the way of voltage (effusion, obesity, COPD, pneumothorax) or are the voltages diseased (infarcted, cellular infiltration, inflammation) -> Pericarditis? -> Diffuse ST elevation in all leads except AVR which has ST depression and LOW BNP -> compression of the heart muscle doesn't provoke its secretion
Elevated vascular resistance -> hypotension is from the heart, the patient is well diuresed but the output is not getting better

Inflammation of the heart

Most common cause is idiopathic -> Drugs (clozapine, immunotherapy) and recent vaccination vs infectious vs autoimmune -> What infections cause myocarditis? -> chagas disease, Lyme disease (usually conduction disease), viral infection, etc. > which are known to do it reliably? 2Cs -> Covid (post covid multisystem inflammatory disease), coxsackie hand foot mouth disease, 2Ts -> toxocara, chagas disease, toxoplasmosis
Deciding when to do Biopsy -> the vast majority of patients have idiopathic myocarditis and biopsy is not productive -> patients with fulminant myocarditis are a subgroup in which we are comfortable exposing them to the risk because of the severity, Deciding on empiric corticosteroids -> if you're doing the biopsy its not recommended