



9/17/24 Morning Report with @CPSolvers



"One life, so many dreams" Case Presenter: Dr. John Black (@) Case Discussant: Dr. Ravi (@rav7ks) & Kirtan (@KirtanPatolia)

CC: 40yrs man referred for high blood pressure

HPI:

He has several weeks of non productive cough, SOB . difficulty on exertion, no fever ,no systemic symptoms.

ROS : No visual changes , headache , GI or Gu symptoms.

→ Given IV hydralazine , carvedilol , nifedipine

PMH: High Blood pressure

Meds: No medication

Fam Hx: Several member with HBP, Heart Dx

Soc Hx: No tobacco , alcohol. Cocaine, marijuana occasionally No surgery No travel hx

Allergies: Denied

Vitals: 204/130, HR : 98, RR : 18 , O2 : 92%

Exam:

Gen: calm , relax

HEENT: Non relevant

CV: Regular, tachycardic

Pulm: Normal

Abd: Non tender, distended, no hepatomegaly

Extremities/skin: No edema

Notable Labs & Imaging:

Hematology:

WBC:10.7 Hgb: 10.3 Plt: 141

Chemistry:

Na:nl K: 3.4 Cl:102 HCO3: 25 BUN: 29 Cr: 3.09 AST: nl ALT:

Alk-P:nl Troponin: 68 BNP 3k

UA: 1+ blood , 2+ protein ,+ cocaine ,

B12: 3.10 B9 : 3

Fibrinogen : 468 , INR : 1.2

Hep B,C neg , ADAMTS13 : nl

ANCA NEG, ANA nl

Renin: 3.5 aldosterone 9

Imaging:

EKG: sinus tach, LVH

CT: severe bronchial wall thickening , moderate pulmonary edema , small bilateral effusion, GGO

Echocardiogram: LVEF 40- 44%, diastolic dysfunction ,

pericardial effusion without tamponade ,

Renal US: No RAS

Dx: Hypertensive emergency with HF ,TMA induced by cocaine

Problem Representation: 40 y/o man w/ history of HTN and occasional cocaine and marijuana use presents with severe HTN, non-productive cough, and exertional SOB. He has elevated troponin and BNP levels, renal dysfunction, anemia, and ground glass opacities on CT

Teaching Points: (Kuchal)

% High Blood pressure: it's important to confirm if they are asymptomatic/ through ROS: High SBP (+ Chest pain/ SOB) (+AMS) (+Abd pain) (+back pain) (+ depression. R/o Clonidine treatment) / PMH to see if its acute or chronic or urgency or emergency , NS. EKG: to r/o End organ damage(others end organs are Eye, Kidney etc. Target 140/90 to 130/80 mmHg. SE status also influences the BP, Social History like substance use: cocaine, ETOH, Heroin/ withdrawal from these drugs, Levamisole with cocaine use causes Cutaneous vasculitis and drugs which could be used to treat it. # Essential HTN: Polygenic, affected multiple factors. Kidney disease: Parenchymal issue, Glomerular disease (nephritic/nephrotic syndrome), Vascular. Micro issues related to the Kidney. # HTN: age of onset is important to take into consideration.

Cough: ?due to drug, Acute Flash Edema (SCAPE). ?Unrelated like Pneumonia etc.
Management reasoning of High BP: Treatment (i) one ACE inhibitor/ ARBs. HCTZ/ Aldactone/ Labetalol. (ii) Labs to look out for end organ effect due to High BP or the other way around. Relevant History to r/o secondary causes(iii) secondary High BP: ?Vascular, ?Familial(channelopathy), ?Co existing Renal Disease/ Cardiac Disease/Endo, like Adrenal (11 Beta Hydroxylase or 17 Beta Hydroxylase deficiency/ Hyperthyroidism. Due to Herbal supplements patient might be taking. Metabolic syndrome/OSA.
POCUS. UA. TSH. Imaging of Abdomen to r/o kidney problems and RAS; ANCA. Also see if High BP is causing the renal problem or if pre existing renal pathology is causing High BP.
High BP precipitates Arteriosclerosis vs Atherosclerosis# High BP: shear stress can ppt Hemolytic anemia.
High BNP: Cardiac complication of High BP, causing Pulmonary Edema precipitating Septal thickening on CT (Interlobular Septae: contains the Pulmonary Vein and Lymphatics).
High BP: hyperkalemia, Acidosis in AKI. But if patient is instead having Alkalosis, and hypokalemia r/o Endocrine issue, channelopathy precipitating the High BP.
Thrombotic Microangiopathy: Coagulation factors normal. Dd from DIC. - Important drugs can cause TMA. Fentanyl, Heroin, Levamisole (drugs also cause ANCA negative vasculitis). HUS/Complement mediated™, Scleroderma. APS both cause Normal complements. IGA vasculitis.Endocarditis?? (Fever, leukocytosis). Hypertensive TMA (r/o all other causes)??
Glucocorticoid Remediable Hyperaldosteronism;- Renin/Aldo might be normal at the time of the attack, and labs have to repeated at a later time.