03/12/25 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: Maryana (@maryanamribeiro) Case Discussants: Dr. Rich Snyder (@) and Mark Heslin (@Mark_Heslin)

Scribing (Yaz)

CC: 65 yo M with **Bilateral Lower Limb Edema** (L>R) and Redness

HPI: Presented to the ED with 2 days of leg swelling and tenderness. He was in usual state of health until 2 days ago, he first noted that his legs were both feeling numb, then his leg started having more swelling, redness and pain when he walked.

PMH:
HTN
Dyslipidemia
HFpEF
CKD stage IIIA
Hypothyroidism
PAD (L>R)

Meds:
Aspirin
Atorvastatin
Hydrochloro
Lisinopril
Levothyroxine

Fam Hx:

Soc Hx:
active tobacco use (1 pack per day)
Alcohol use
Marijuana use (intermittently)

Allergies:
none

Vitals: T: 36.9 (98.4 F) BP: 169/63 **RR:** 16 **HR:**55 **Sat: 99**
Exam: Gen: AOx3
HEENT: EOMI, PERRL, moist mucous membranes
CV: RRR, No murmurs or gallops, no JVD
Pulm: Clear to auscultation bilaterally, no crackles, no wheezes
Abd: Non tender, non distended, normal bowel sounds no organomegaly.
Neuro: CN-12 intact, 5/5 strength
MSK: BLE +2 (L>R) up to the mid-shin, warm and mild erythema of left shin/mid-calf, tenderness to palpation on LLE
Skin: Intact without any skin breaks, onychomycosis on both feet

Notable Labs & Imaging:

Hematology:
WBC: 16.7 (Neutrophilic)
Hgb: 11.3 Plt: 367

Chemistry
Na:135 K: 3.5 Cr: **1.6** (baseline 1.5) BUN: 36
Glu:90 Cl:94 CO2: 31 Anion GAP: 10
Albumin: **2.6** Total Protein: 6.2
BNP: 300
UA: +3 Protein
Urine protein: 1177 mg/dl
Urine creatinine: 184 mg/dl

Patient was initiated on cefazolin but edema persisted

TTE: LV hypertrophy, LV systolic function normal, LV EF = 60-65%, RV systolic function normal. Normal size IVC with normal collapse consistent with RA pressure of 3 mmHg (nl)

Additional labs:
LDL: 174, **HDL:** 17, **TSH:** nl, **SPEP:** no monoclonal gammopathy, K/L ratio normal ASO normal, ANA negative, C3C4 nl, ANCA (-), GBM(-), cryoglobulin screen is negative
PLA2R Ab strongly positive, patient was discharged with outpatient follow up > biopsy showed membranous glomerulonephritis
Dx. Primary Membranous Nephropathy

Problem Representation: 65 yo M with PMH of HTN, Dyslipidemia, HFpEF, CKD3a, hypothyroidism, presents with 2 days of BLE, redness and pain. UA with +3 protein, Urine protein of 1177 mg/dl and urine creatinine 184 mg/dl,

Teaching Points (SEEME):

Approach to edema:
If **unilateral** - we can think about cellulitis and venous thrombosis. We can ask about any history of a recent surgery affecting lymphatics or venous system.
If **bilateral** : We can think about liver, heart and kidney. Amyloidosis is worth considering, we can look for signs of heart failure, get basic metabolic panel and urinalysis.
We can ask about any hematuria history as well as extensive examination is important (look for claudication, trauma)
-30- 59 eGFR is seen in stage 3 CKD.
- Neuropathy can be indicative of a systemic disease. Acute neuropathy can be indicative of Guillain Barre syndrome,AIDP, any toxin affecting nerves.
- When patients have CKD, hypertension control is very important. Hypertension can cause hypertensive nephrosclerosis.
Focal segmental glomerulosclerosis is seen in young african american males, related to ApoL1 gene.
-Hypertension can cause CKD and CKD can also contribute to hypertension. Tubulointerstitial disease can be seen with HTN.
Occupation, ancestry, medication history is important in cases of kidney disease.
-Amyloidosis can cause HFpEF as well as neuropathy. Transthyretin amyloidosis and AL amyloidosis are worth considering when heart and kidney are affected together.
-Marijuana can inhibit CYP34. Tacrolimus is a medication having a common interaction with marijuana.
Nephrotic syndrome: Always look for periorbital edema. Three liters of fluid in interstitial space can cause edema. Nephrotic syndrome, AKI and CKD can cause salt and water retention. Hereditary hypoalbuminemia can cause edema by upregulation of receptors of distal tubule of kidney (Na retention). When there is no edema, no JVD, heart is less likely cause of edema. Hyperlipidemia is also seen as liver causes increase production of lipids, loss of immunoglobulin, loss of anticoagulant proteins can cause increased risk of DVT in nephrotic syndrome.
-Wide pulse pressure indicates stiff vessels. When patient has bradycardia, TSH levels can be checked.
- Elevated bicarbonate : 40 or higher can be seen in metabolic alkalosis and get also be linked to drugs such as thiazide.
-Elevated protein can be seen in MM and amyloidosis.
-Hypoalbuminemia is seen in malabsorption, celiac disease, liver disease, renal losses.
- Cefazolin can be used for cellulitis.
- Biatrial enlargement is seen on Echo in Amyloidosis.
- approach to urinalysis: 24 hour urine protein electrophoresis is important. For resistant hypertension, aldosterone is also monitored over 24 hours. Elevated protein/ creatinine ratio, hypoalbuminemia, vitamin D deficiency is seen in nephrotic syndrome.
Approach to types of nephrotic syndrome:
Minimal change disease is seen in children. Young age african americans have FSGS, above age of 65 amyloidosis and minimal change disease can be seen. Chronic inflammatory disease can cause secondary amyloidosis. Amyloidosis and MM can be seen as a overlapping syndrome. Minimal change disease has more severe anasarca and can be seen with lymphoma. Lithium and Nsaids can cause minimal change disease and membranous nephropathy. Membranous nephrotic syndrome is seen with lung cancer, SLE, Hepatitis and syphilis. HIV causes FGFS and can cause immune complex deposition in the kidney. Large kidneys are seen in MM and Amyloidosis.
Membranous nephropathy is greatly linked with venous thrombosis. Kidney biopsy can help us diagnose the type of nephrotic syndrome. Anti-THSD7A antibody can be see in membranous nephropathy. Calcineurin inhibitors , diuretics, statins and certain antibodies can be used to treat membranous nephropathy. Loop diuretics are effective even in cases of Low GFR.