04/07/25 Nephrology VMR with @CPSolvers

"One life, so many dreams" Case Presenter: Yajaira Jimenez (@SunJimenez18) Case Discussants: Alessandra Tomasi (@AleTomasiMD) & Matthew Sparks (@Nephro_Sparks)

scribe(Glen)
CC: 49/M presents with dyspnea and lower extremity edema for 2 weeks and admitted for heart failure

HPI: progressive dyspnea. and admitted for heart failure. Hospital day 2.
Urine op 120 IV BID lasix 500cc 24hrs.Baseline wt 220lb.
Nephrology consulted for worsening hyponatremia and difficulty with diuresis

PMH: NCIM(ef15%) , CKD3a

Meds:
Carvedilol 6.25mg BID
Spironolactone 50 mg OD
Empagliflozin 25 mg OD
Losartan 25mg,
Lasix 60 mg BID

Fam Hx: not significant

Soc Hx: not significant

Health-Related Behaviors:
No smoking, alcohol or drugs use

Allergies:
No known drug allergies

Vitals: **T:afebrile** **BP:** **RR:** **HR**:110 **Sat: 98**
Exam: **Gen: 2+ pitting edema**
Neck:JVD is 14cm **CV:** tachycardia **Pulm:** crackles bilaterally
Abd: normal
Neuro: normal
MSK:extremities warm

Notable Labs & Imaging:
Hematology:
WBC:8 Hgb:12 Plt: 200Hct: MCV:

Chemistry
Na: 125 (130) K:4.1 Cr: 2.1 (bl around 1.2) BUN: 38 Ca: Ph: Mg: Glu: Cl: HCO3: 18 CRP: ESR: LDH: AST & ALT: mildly elevated ALP: Bili:
Lactate 1.5 BNP:2000 serum osmolality: 280 urine osmolality:300 urine sodium: 40
Imaging:
ECG:sinus tachycardia
CXR; pulm congestion with pleural effusions

Urine; no casts or pyuria,
Bladder US: PVR 80
Dobutamine -> Slight improvement in UO and Cr, Na remained low

Day 5, weaned off dobutamine and diuresed with lasix
Weight 190 lb, 2L of UO daily, Cr improved to bl. Na increased back to >134
He is followed by advanced heart failure outpatient with plans to increase GDMT and eventual LVAD as a bridge to transplant.

Dx Cardiorenal syndrome due to HF exacerbation

Problem Representation: 49/M with progressive dyspnea and lower extremity edema for 2 weeks. With history of NCIM and CKD3a. On exam with pitting edema, elevated JVP and crackles B/L. low sodium and high CI. with pul congestion on CXR.Weaned off dobutamine, diuresed and came back to baseline.

Teaching Points (Nikola):
In any patient with CKD, the first step is to identify their **baseline kidney function**—creatinine, eGFR, and urine output—to allow comparison with current values.

Cardiorenal Syndrome (CRS) = AKI due to cardiac failure, pathophysiology: elevated CVP + reduced forward flow (low EF) leads to renal hypoperfusion and AKI. CRS is a common complication in patients with HF. In CRS, on urinalysis, expect a **high (concentrated) urine osmolality (high ADH)**, with a **low urine sodium (<10, high Aldosterone),** but cave: look out for diuretics which can elevate the urine sodium.

In a patient with HF exacerb. and concomitant AKI, CRS is a likely diagnosis however, one should still rule out other potential AKI causes such as obstructive AKI, while also getting an urinalysis to rule out AIN, ATN etc.

The **urine Na** is an effective and objective measure to assess the **effectiveness of diuresis**, a urine sodium of > 70 mmol/L is consistent with adequate diuresis, if it is < 70 mmol/L the dose should be adjusted.

In the acute setting, high-dose bolus diuretics should be initiated, as continuous IV infusion offers no proven advantage, and later transitioned to oral therapy. **DO NOT STOP DIURESIS UNTIL THE PATIENT IS EUVOLEMIC!** (even if Creatinine/Cystatin C is rising or "contraction alkalosis - chloride depletion alkalosis" is occuring, do not be scared by these changes). ***"Give the kidneys a chance to recover!"*** and remember the alternative is dialysis.

What are the goals for diuresis: it should be based on a patients signs/symptoms: reach the dry weight, reduced peripheral edema, prevent respiratory failure requiring intubation.
SGLT2 Inhibitors: on their own do not lead to significant natriuresis/volume loss, however they improve the efficacy (about 20%) of diuresis, when combined with a loop diuretic or a thiazide, keep this in mind as this can be very handy!