01/13/25 Nephrology VMR with @CPSolvers

"One life, so many dreams" Case Presenter: Kuchal Agadi () Case Discussants: Douglas Farrell (@), Samira Farouk (@ssfarouk)

CC: **Hypotension** w/ cold, clammy skin

HPI: 27 M w/multiple comorbidities. Speech disturbances from **medulloblastoma**. **Lightheadedness**, couldn't feel his pulse. Ambulance: no IV access possible.

PMH:
Hypothyroidism
Turcot syndrome (FAP + Medulloblastoma)

Soc Hx: living independently in nursing home,
walks w/ a walker

Vitals: T: 35°C BP: 96/53 mmHg **RR: HR: 103 bpm**
Exam: Gen: poorly nourished, awake, alert, blinks in response,
HEENT: loss of buccal fat and temporal wasting present
CV: clear, **Pulm:** clear, **Abd:** soft
Neuro: aphasic, speech disturbance 2/2 medulloblastoma; cooperative, no FND
MSK: Loss of muscle mass w/ temporal wasting, Tinea corporis

Notable Labs & Imaging:
Hematology:
WBC: 12 Hgb: 11.4 Plt: 278

Chemistry
Na: 134 K: **8** Cr: **6.3** (baseline 1.3), GFR **17.8,** BUN: 84 Ph: **9.3** Cl: 103
Bicarb: 8, Lactic acid 8.7, Anion Gap: 23, LDH 324
Alk-Phos: 70, AST 26, ALT 19, Uric acid 7.3 CK 370
UA: yellow-hazy appearance, **1.005**, **large amount of blood**, Nitrite and leukocyte esterase negative, RBC 0-3, 1+ mucus
Nephrology consulted => **emergent HD**

Imaging:
EKG: Sinus Tachycardia w/ RBBB, L atrial enlarged, QTc 530
Echo: EF 60%, **RV reduced function,** trace MR, mild pulmonary regurgitation, LV diastolic function nl

CK -> 1583
Blood culture: Gram + cocci in clusters (Staph hominis)
Started on Cefepime + Vancomycin + Flagyl.
Urine output: 2L -> 2.5 L -> 2.7 L

Dx: ATN secondary to sepsis and rhabdomyolysis.

Problem Representation: A poorly nourished 27yM w/ a PMH of Turcot syndrome and hypothyroidism p/w hypotension, hypothermia, sinus tachycardia and QTC prolongation, severe hyperkalemia of 8, lactic acidosis, high phosphorus and LDH, hematuria w/ no RBC and AKI (Cr 6.3 w/ a bl of 1.3).

Teaching Points:

TREND YOUR UA LIKE YOU TREND YOUR TROPONIN

Approach to hypotension (low blood pressure):
- Look for signs of end organ damage
- Bedside evaluation to get a sense of the underlying physiology for example examine knee caps and asses vessels (cold vs warm)
- Assess urine output

Causes of low blood pressure:
sepsis , cirrhosis(hepatorenal syndrome), adrenal insufficiency , cardiorenal syndrome

Management of hyperkalemia: there's a low threshold for giving 1 gram of Ca gluconate to stabilize the cardiac muscle , there are 2 buckets of managing it 1 is temporizing 2 getting rid off
Causes of hypothermia: sepsis , hypothyroidism, primary adrenal insufficiency
Sepsis can cause kidney injury by 1/ low perfusion 2/ toxic cytokines
Hyperkalemia happens either due to increased production or decrease excretion , and for it to be cleared we need 1/ GFR 2/distal Na delivery 3/ Aldosterone
Approach to AKI (0.3 increase in Cr over a 24 h period): prerenal(hemodynamic aki?)>intrarenal>postrenal
*Get a UA look at RBC ,proteins , and casts * look under microscope*
CK greater than **5000** is concerning for kidney damage.
AKI +low Hb + low plt= think TMA
Cr limitation : 1/ affected by muscle mass 2/ affected by excretory capacity only rises after it reached its limit
High AGMA " GOLD MARK":
Glycols,oxoproline by product of acetaminophen , lactate, D-lactate by microbes as in SIBO patients ,methanol , aspirin ,renal failure , ketoacidosis .
When suspecting an intoxication calculate the osmolal gap , when there's a discrepancy of 15-20% this highly suggest it.
Indications of dialysis : AEIOU (acidosis,electrolyte imbalance,intoxication,overload,uremia) all when failed medical therapy except for uremia has a more broad approach to it as it doesn't necessarily depend on BUN as there are other uremic toxins contributing to it .
Uremia is a clinical diagnosis
Loop diuretics are not nephrotoxic , **UNLESS** your patient is hypovolemic
Dr Farouk's AEIOU (ACS of nephrology):
AKI etiology, evaluate based on drug dosing, optimize volume status , Urinalysis
Hemodialysis prerequisites : access , correct water hookup, second take
Glomerulonephritis associated with infections:
IgA nephropathy , post strep infection
3 phases of ATN : initiation (rise in Cr)> maintenance phase (stable)> recovery phase (fall in Cr and increase in U/O) be aware pg the post ATN diuresis phase the urine is usually hypotonic as the nephrons have not yet recovered .