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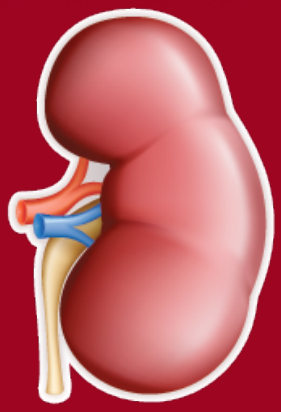
EM CASE OF THE WEEK

BROWARD HEALTH MEDICAL CENTER DEPARTMENT OF EMERGENCY MEDICINE



When a patient comes in with a significantly elevated Creatinine, it is important to know the emergent presentations and their associated management in order to ensure the prevention of life threatening sequele. This month we will explore the facts about Acute Kidney Failure.

EM CASE OF THE WEEK

EM Case of the Week is a monthly "pop quiz" for ED staff. The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

Acute Kidney Injury in the ER

A 46 year old male with a past medical history of hypertension presented to the ED complaining of shortness of breath and increased edema of the legs for the last three weeks. He stated that he went to another local hospital two weeks prior and while there was diagnosed with IgA nephropathy. He has been taking 40 mg of prednisone ever since and states that this is the first time this has happened. His vital signs are T 97.7, HR 97, BP 143/79, RR 22, O2 98RA. What are the first things to assess for in a patient suffering a possible AKI?

A. Mental Status, Urinary Function, Hemoglobin levels

B. Blood Pressure, Signs of Shock, Respiratory Rate.

C. Potassium Levels, EKG, Cardiac Monitor.

D. Abdominal Bruits, Abdominal XRay, CMP.

E. CXR, JVD, Mental Status.

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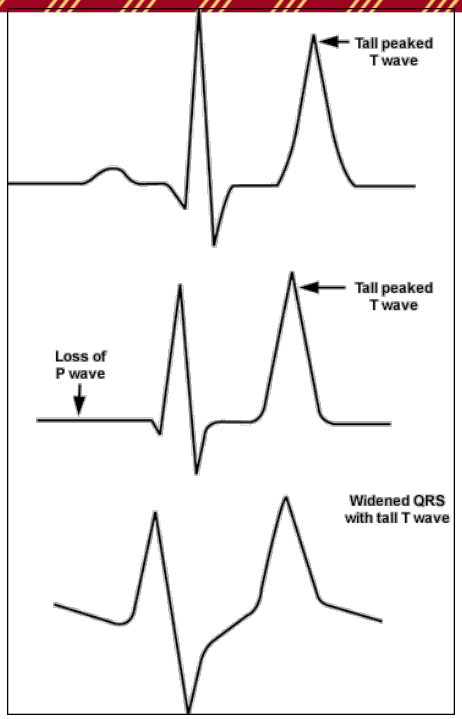

EKG features in Hyperkalemia

Take Home

- Pay attention to electrolyte imbalances hyperphosphatemia, hypermagnesemia, hyponatremia, metabolic acidosis, and especially hyperkalemia
- Assess and treat hypovolemia, respiratory failure and shock first before working up the patient further.

Acute Renal Failure

The correct answer is B. Always make sure your initial assessment includes looking at life threatening complications stemming from AKI including Hypotension, Shock, and Respiratory Failure. Many of the other choices are included in the workup of AKI. Hyperkalemia is also a life threatening condition of AKI but can only be apparent from the chemistry levels. The patient's initial labs included Na 129, Potassium 4.9, Cl 94, CO2 13, AG 21, **BUN 170, Cr 21.7, eGFR 2,** H/H 9.4/27.5, WBC 7.99, Pt 246. Additional work up included a CXR, EKG, CK, UA (since he was still able to urinate), Urine Culture, Phosphorus level and he was given NS fluids. The patient was then admitted to be dialyzed.

Discussion: ***AKI is met by any of the following criteria. 1. Increase in serum creatinine by ≥0.3 mg/dL within 48 hours 2. Increase in serum creatinine to ≥1.5x baseline 3. Urine volume <0.5 mL/kg/h for 6 hours****. AKI can be then broken down into three different causes; pre-renal, intrinsic, and post renal.*

Pre- renal causes include hypovolemia, patients commonly present with symptoms including thirst, decreased urine output, dizziness, and orthostatic hypotension. A patient should be asked about possible volume loss from vomiting, diarrhea, sweating, polyuria, or hemorrhage. Patients with CHF leading to impaired renal perfusion may present with orthopnea.

With intrinsic renal failure, patients can be divided into those with glomerular or tubular etiologies of AKI. Nephritic symptoms of hematuria, edema, and hypertension indicates a glomerular etiology for AKI. While Acute Tubular Necrosis (ATN) should be suspected in any patient presenting after a period of hypotension secondary to cardiac arrest, hemorrhage, sepsis, drug overdose, or surgery.

Post renal failure usually occurs in older men with prostatic obstruction and symptoms of urgency, frequency, and hesitancy. Flank pain and hematuria should raise a concern about renal stones as the source of urinary obstruction

But with any type of cause of AKI, the management is very similar. It is aimed at treating any life threatening features while attempting to prevent any further decrease in renal function to the point of renal replacement therapy/dialysis in the hopes of future kidney recovery. (*cont'd next page)*

Management of AKI After first looking at vitals including BP, RR and O2sat, blood should be drawn immediately to check for hyperkalemia.

▶ **Hyperkalemia** Severe hyperkalemia (>6.5) is a medical emergency because of the risk of life threatening cardiac arrhythmias. It is caused by the reduced potassium excretion of from the damaged kidney(s). You will be able to see EKG changes typical to a patient suffering from hyperkalemia including peaked T waves, flattening of the P waves with prolongation of the QRS complex, sine waves and eventually ventricular fibrillation and asystole. Treatment uses 3 steps. 1. Stablize the cardiac myocytes - A bolus of 10–20 ml of 10% calcium gluconate or chloride is given intravenously over two to five minutes except in patients taking digoxin. They require a slow infusion of calcium so as not to potentiate any cardiotoxicity. 2. Reduce the plasma potassium concentration - B2 agonists and insulin will both allow for an increase in potassium uptake by the cell thereby temporarily reducing the concentration of potassium in the serum. Make sure to also provide glucose when using insulin as to not put the patient in a hypoglycemic state. 3. Remove potassium from the body - This is done either through Ion binding resins like Kayexalate at 15g TID, Loop diuretics or hemodialysis - the definitive way to remove potassium.

▶ **Pulmonary Edema** The H&P in addition to a CXR is necessary to rule out this potential complication of AKIs. But in the ER, if respiratory failure is present, this must be dealt with first, through supplementary oxygen, non-invasive ventilation with Nasal Cannula or Non Rebreather, or intubation and ventilation, depending on the state of the patient. After those measure, pharmacological treatment aimed at treating the fluid overload includes inducing diuresis and reducing the work of a decompensated heart. This is done through IV infusions of nitrates and opioids in addition to using diuretics. Much larger doses of diuretics are required in renal failure. If this does not help offload some of the excess fluid then dialysis would be the definitive choice.

▶ **Metabolic Acidosis** Systemic acidosis impairs cardiac contractility, induces bradycardia, produces vasodilatation, and worsens hyperkalemia. In patients with severe metabolic acidosis (pH < 7.1) and with evidence of cardiovascular compromise, administration of Sodium Bicarb should be considered to maintain blood pH at ~7.2. It should only be given as an isotonic preparation to prevent hyperosmolality. Dialysis is another treatment to fix acidosis in a patient.

▶ **Uremic Pericarditis** Affected patients are often asymptomatic but may be complaining of chest pain. They often have a small to moderate pericardial effusion which can progress to significant hemodynamic compromise. The only therapy is dialysis and pericardiocentesis in case of significant pericardial effusions. Therefore, the presence of a pericardial friction rub on the physical examination and classic ECG changes consistent with pericarditis (Concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6).) are indications for urgent dialysis and an echocardiogram.

▶ **Obstruction relief** If the AKI has a post renal etiology, prompt opening of the urinary tract is necessary. If it is caused by Bladder Outflow Obstruction then passage of a catheter should be considered. Higher obstructions need Urological and Nephrological consults and will include a CT of the Abdomen without contrast in the work up.

Preventative Measures. Patients with AKI should not be given nephrotoxic medications including NSAIDS, ACE inhibitors, aminoglycosides and other antibiotics such as penicillins. The use of contrast dye is also avoided. Tumor lysis syndrome which can precipitate an AKI is prevented using hydration and Allopurinol prior to the start of chemotherapy. In cases of rhabdomyolysis aggressive hydration and the use of mannitol and sodium bicarb are the mainstays of preventing AKI.

THIS WEEK'S CASE WAS WRITTEN BY STANLEY LINDER. STAN IS A 4TH YEAR MEDICAL STUDENT FROM NOVA SOUTHEASTERN UNIVERSITY AND COMPLETED HIS ER ROTATION AT BHMC IN MARCH 2015.