EM CASES SUMMARY

Episode 151 AKI Part 2 – ED Management

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Management of AKI in the ED: "Fluids & Foley" fixes most AKI

The majority of AKI can be fixed by "a bag of LR and a urethral catheter"… tincture of time may be all that's necessary in many other cases. Somewhere between 70-90% of AKI is pre-renal or post-renal in etiology, so that fluid resuscitation and removal of the obstruction (i.e. a urethral catheter) will probably resolve 70-90% of AKI. However, a more nuanced treatment algorithm should be considered in complex cases.

5 step approach to AKI in the ED

Step 1: Rule out the 2 immediate life-threats

1. Hyperkalemia – get ECG, electrolytes off the blood gas
2. Severe acidosis – get blood gas

Step 2: Assess for adequate perfusion – are they in shock?
Use your history, physical examination and POCUS to assess for perfusion and treat shock (hemorrhagic, vasodilatory, cardiogenic shock etc.) accordingly.
*the patient in shock with acute heart failure, pulmonary edema and AKI is especially challenging, and may require norepinephrine to support the blood pressure and dobutamine to help improve cardiac forward flow, in addition to usual acute heart failure management; early consultation with an intensivist is recommended.

Step 3: Assess for both pulmonary and peripheral edema
Assess JVP and lungs with POCUS for pulmonary edema, look and palpate for peripheral edema (including pre-tibial edema, sacral edema)
If there is no evidence of pulmonary or peripheral edema, give a fluid challenge.
AKI with adequate perfusion, with pulmonary edema (with or without peripheral edema)

1. Give furosemide 1 mg/kg IV (or 1.5 mg/kg IV if on furosemide already)
2. Think about pulmonary renal syndromes other than CHF (such as anti-GBM disease, ANCA associated vasculitis, circulating immune complex syndromes like lupus), and look for clinical clues (inflammatory arthritis, purpura, Raynaud's, mononeuritis multiplex, uveitis or Sicca syndrome ?)

AKI with adequate perfusion, with peripheral edema but not pulmonary edema

1. Give furosemide 1 mg/kg IV (or 1.5 mg/kg IV if on furosemide already)
2. If no improvement in renal function think about hypovolemia ("pre renal") despite peripheral edema

o Low serum albumin – treat underlying cause, and consider hepatorenal syndrome which may require IV albumin
o Venous insufficiency and/or lymphedema – give crystalloid, consider compression therapy
o Drug induced edema – give crystalloid, reassess offending drug
o Severe myxedema – give L-thyroxine and monitor

Step 4: The golden rules of AKI workup

1. Measure a **post-void residual (PVR)** with bladder scan or urethral catheter
2. Get a **urine dip** to look for blood and protein suggestive of **nephritic syndrome**
3. Monitor **urine output** ideally with a urethral catheter
4. **Avoid nephrotoxins** (NSAIDs, ACEi, ARBs, gentamicin etc)

Step 5: Consider imaging for a small subset of post-renal AKI
Radiology department imaging should be reserved for those patients who:

- Do not improve with fluid challenge (making pre-renal less likely),
- Have a normal urine dip (making intra-renal less likely),
- Have a post-void residual <100mL (making BPH less likely)
- Have obvious bilateral hydronephrosis on POCUS

These patients warrant further imaging as they might have a rare post-renal bilateral ureteric obstruction cause of AKI such as obstructive metastatic cancer, lymphoma or a kidney stone with a solitary kidney.

Consider a nephrology or internal medicine consult if any of:

- Inadequate urine output after 4 hours management
- Unexplained blood and protein in urine required workup for intrinsic renal disease
- Creatinine rises despite initial management
- Worsening pulmonary status despite initial management

IV crystalloid of choice, timing and volume in patients with AKI

Fluid of choice and timing in AKI

Our experts recommend a **balanced fluid** for resuscitation such as Ringer's Lactate (RL) based on SMART and SALT-ED trials, even though the benefit in AKI patients was based on sub-group analysis. SMART and a recent study looking at starting balanced fluids in the ED vs ICU suggests a mortality benefit if balanced

solutions such as RL are started early as opposed to waiting until they are in the ICU to switch over. RL is slightly acidotic, less so than NS, so monitoring pH is important regardless of which crystalloid is given. RL is less like to lead to hyperkalemia compared to NS based on evidence from renal transplantation studies where anephric patients given NS were more likely to become hyperkalemic; remember that an acidotic environment leads to potassium shifts which may lead to life-threatening hyperkalemia.

Patients with end-stage liver cirrhosis or post massive paracentesis should be volume resuscitated with albumin rather than crystalloid.

Volume of fluid in AKI resuscitation

With the goal in mind of improving cardiac output and tissue oxygenation whilst achieving acceptable urine output and mean arterial pressure, our experts recommend small, frequent crystalloid boluses of 3mL/kg with frequent clinical assessments incorporating POCUS for evidence of volume overload. In the patient in NSR on a ventilator, consider using pulse pressure variation to help guide fluid resuscitation.

AKI dialysis indications and timing

In general patients with the following conditions should be considered for immediate dialysis: severe electrolyte derangements, specifically hyperkalemia, that are resulting in hemodynamic instability or arrhythmia and are refractory to pharmacologic treatment, life-threatening fluid overload leading to respiratory or cardiac failure, uremia (presenting as pericarditis or altered mental status), severe metabolic acidosis (pH <7.1), symptomatic ethylene glycol ingestion and severe rhabdomyolysis.

AEIOU mnemonic for indications for emergent dialysis

Acidemia – pH<7.1 despite medical management
Electrolyte abnormalities – hyperkalemia refractory to medical management
Ingestion – nephrotoxic drug ingestion amenable to dialysis
Overload – volume overload resulting in respiratory failure
Uremia with bleeding, pericarditis or encephalopathy

Timing of dialysis in non-emergent AKI: STARRT-AKI trial

STARRT-AKI is the largest trial to date comparing immediate/early dialysis with delayed dialysis in AKI patients. It is a multinational RCT of 3019 critically ill patients with AKI (mostly with sepsis), comparing an accelerated strategy (median 6hrs to initiation of dialysis) vs standard strategy dialysis (median 31hrs to initiation of dialysis) with a primary outcome of death from any cause at 90 days.

Primary outcome of death similar in both groups: Death at 90 days occurred in 43.9% in the accelerated strategy and 43.7% in the standard strategy (RR, 1.00; 95% CI 0.93-1.09)

Adverse events more common in early dialysis group: Adverse events occurred in 23% in the accelerated strategy and 16.5% in

the standard strategy group (RR, 1.40;95% CI, 1.21 to 1.62), most commonly adverse hypophosphatemia and hypotension.

Bottom line: starting dialysis early (6hrs) for patients with AKI and no true emergency indications compared to 31hrs, has no mortality benefit and may incur some harm.

Are piperacillin and vancomycin contraindicated in patients with severe AKI?

The most common reason to be admitted to an ICU with AKI is septic shock. Piperacillin-tazobactam and vancomycin are commonly used in patients with septic shock. Some observational data suggest that these antibiotics may be nephrotoxic when combined, however no causation has been demonstrated and newer formulations are thought to contain fewer nephrotoxic impurities. Our experts do not withhold these antibiotics in AKI patients when otherwise indicated, however other antibiotic options should be considered in consultation with ICU or ID in patients with severe AKI.

Aminoglycosides and amphotericin should generally be avoided in patients with AKI.

Is vasopressin preferred over norepinephrine for patients with AKI and septic shock?

There is mixed evidence for preferential use of vasopressin over norepinephrine as the initial vasopressor of choice in AKI patients with septic shock.

VANCS ll trial: no outcome difference with vasopressin compared to norepinephrine, regardless of renal status
VANISH trial: no difference in developing AKI but less dialysis in vasopressin group

VASST: slower progression toward renal failure in vasopressin group compared to norepinephrine group in secondary analysis

A 2019 metaanalysis of trials concluded that "Vasopressin therapy in septic shock had no effect on 28-day mortality although the confidence intervals are wide. It appears safe but with a different side effect profile from norepinephrine. The finding on reduced [dialysis] should be interpreted cautiously. Future trials should focus on long-term outcomes in select patient groups as well as incorporating cost effectiveness analyses regarding possible reduced [dialysis] use."

Bottom Line: Our experts continue to use norepinephrine as their first line vasopressor in patients with AKI and septic shock.

Is there a role for giving sodium bicarb in AKI patients?

IV sodium bicarbonate is a reasonable therapy in AKI patients with severe metabolic acidosis as a temporizing measure, but

should not preclude the search for, and reversal of the underlying cause.

BICAR-ICU was an RCT of 389 ICU patients with severe metabolic acidemia (pH ≤7·20, PaCO ≤45 mm Hg, and bicarb ≤20 mmol/L), a total Sequential Organ Failure Assessment score of 4 or more or an arterial lactate concentration of 2 mmol/L or more who were randomized to receiving 4.2% sodium bicarbonate to maintain a pH>7.3 or crystalloid.

Although there was no significant difference for the primary outcome (mortality at day 28 and the presence of organ failure at day 7), bicarbonate showed a trend toward decreased need for dialysis, and in the subgroup of patients with AKI bicarbonate decreased mortality and vasopressor requirements. NNT=6 for requiring dialysis in the AKI subgroup.

Bottom line: our experts recommend giving bicarb in patients with AKI and refractory severe acidosis

Take Home Points for AKI ED Management

- First and foremost, rule out immediate life threats – hyperkalemia and severe acidosis
- Most patients with AKI simply require "fluids and a foley", however a more nuanced treatment algorithm should be considered in complex cases
- Have respect for new severe hypertension in the setting of AKI as these patients may have an intrarenal cause that requires urgent BP control and internal medicine consultation for further workup
- Get help from ICU for patients with AKI, pulmonary edema and cardiogenic shock as these patients are challenging to manage
- Not all patients with AKI require imaging; bilateral ureteric obstruction is a rare cause of AKI
- Avoid nephrotoxins in patients with AKI whenever possible (NSAIDs, ACEi/ARBs, gentamicin, amphotericin)
- The resuscitation fluid of choice in AKI patients is Ringer's Lactate in small boluses with frequent assessments of volume status
- Use AEIOU mnemonic for emergency indications for dialysis; for other AKI patients it is generally safe to delay dialysis for 1-2 days
- IV bicarb is reasonable in AKI patients with refractory severe metabolic acidosis

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