

Basics of the Management of Acute Renal Failure and its Complications for the General Physician

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Short Communication

Management of Acute Kidney Injury (AKI) starts with clinical assessment, looking for complications of AKI like fluid overload causing Pulmonary oedema, tachypnoea from fluid overload or acidosis), signs of encephalopathy including seizure, and Pericardial rub. Venous blood gas or arterial if hypoxic, is required urgently before routine laboratory blood results are available to assess for hyperkalemia and degree of acidosis [1].

While dealing with serious life-threatening complications, history taking is focused on trying to identify the cause of AKI, full medication history including contrast used for investigations or procedures like angiogram, history suggestive of infection which led to sepsis, history of urinary

symptoms suggestive of prostatic hypertrophy in men leading to urinary obstruction, diarrhea or vomiting suggestive of a prerenal cause, history of arteriopathy suggesting renal artery stenosis, and systemic symptoms suggestive of vasculitis [1].

Clinical examination as mentioned above is essential to monitor for complications as well as causes of AKI, signs of sepsis or shock which require urgent management by using sepsis six bundle, examining for palpable bladder to rule out obstruction and considering urinary catheter even in the absence of obstruction to help with urine output (UO) monitoring and presence or absence of active sediment (proteinuria and haematuria) in urine dipstick [2]. Management of AKI has improved significantly after introduction of AKI care bundles, treatment of AKI depends

on the cause, urgent treatment of sepsis as mentioned before, crystalloid infusion in cases of intravascular depletion leading to prerenal failure, aim and rate of fluid infusion is based on improving signs of perfusion like UO, lactate, heart rate, and blood pressure. Once patient is euvolemic maintenance fluid is advised, with close monitoring of UO, daily weight, and fluid balance [3].

If urinary obstruction is high and confirmed on imaging, urgent consultation with urology team to consider nephrostomy or stenting.

Harmful medications which reduce the glomerular filtration rate (GFR) should be omitted like ACEi, ARB, and NSAIDs) and doses of medications which are renally cleared should be reduced.

Usually, a battery of investigations is required for patients with AKI, but a good practice is to order blood tests according to clinical suspicion.

Blood, urine, sputum or stool culture with imaging of the source of sepsis is indicated in cases where sepsis is likely.

Intrinsic renal disease is strongly suspected if urinalysis is positive for blood and protein and is an indication for auto-antibody testing including, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, antiglomerular basement membrane antibodies, immunoglobulins, and serum complements [4].

Routine blood test including, renal profile, bone profile, liver function test, full blood count (eosinophilia may suggest interstitial nephritis or atheromatous embolism), glucose, bicarbonate, coagulation screen, and CRP.

Myeloma screen (protein and light chain electrophoresis, immunoglobulins) if evidence of

bone pain, lytic lesions, hypercalcemia, raised uric acid, or normocytic anaemia [4].

Creatine kinase (CK) in cases of trauma or long lie, lactate dehydrogenase (LDH), blood film, and full hemolytic screen if hemolytic uraemic syndrome (HUS) or thrombotic thrombocytopenic purpura (TTP) is possible [4]. Venous blood gas as mentioned above, looking for lactate as a sign of sepsis or poor perfusion, as well as K and acidosis.

Urgent renal ultrasound to rule out hydronephrosis, or within 24 hours if worsening or no improvement of renal function [4].

Referral to intensive care team is indicated urgently if AKI complications, mainly pulmonary oedema, hyperkalemia, severe acidosis below 7.1, or uraemic symptoms did not respond to initial medical management.

If no complications but the renal function deteriorates or did not improve to initial treatment, a referral to the renal team is indicated urgently, especially in cases of AKI stage 3, possible vasculitis/glomerulonephritis, and indication for dialysis.

Indication for renal replacement therapy (RRT) includes refractory hyperkalemia of more than 6.5 mmol/L, refractory acidosis below 7.15, refractory pulmonary oedema, and uraemic symptoms including urea more than 27 mmol/L with encephalopathy, seizure, and pericardial effusion.

Other common causes for RRT, are patients with anuric AKI and multiple organ failure or few toxicological causes.

Dietician input is strongly advised in cases of AKI, aim is total energy intake of 20-30 kcal/kg/day, 0.8 to 1 g/kg/day protein which

should be increased to 1 to 1.5 g/kg/day in AKI on RRT [5].

Severity of acute kidney injury (AKI) is staged as stage one, rise in serum creatinine (Cr) of 1.5 to 1.9 x baseline or reduction in Urine output (UO) of <0.5 mL/kg/hour for 6 to 12 hours, stage 2 when the rise in Cr is 2 to 2.9 x baseline or UO of <0.5 mL/kg/hour for 12 to 24 hours, while stage 3 is rise in Cr of ≥ 3 x baseline or reduction in UO of <0.3 mL/kg/hour for ≥ 24 hours or anuria [6].

If there is a delay in treatment, stage 1 can progress rapidly to stage 3 with increase in rate of complications.

Indications for urgent renal replacement therapy (RRT) are hyperkalemia of more than 6.5 with ECG changes and resistant to treatment, pulmonary oedema due to oliguria/anuria, metabolic acidosis of PH <7.1 resistant to treatment, and signs of uraemia including encephalopathy, seizures and pericarditis or pericardial effusion.

As RRT can be delayed, initial treatment of these complications is advised to avoid or bridge to RRT. In pulmonary oedema in a context of oliguric AKI, a trial of high dose furosemide is recommended, and the aim is not to improve the renal profile but to relieve pulmonary oedema, up to 200 mg IV furosemide stat and assess the response clinically and measure the UO hourly, 100 ml/hr or more is considered a positive effect. Strict UO monitoring is essential and AKI is an indication for urinary catheter insertion unless high risk of infection or another contraindication. Failure of furosemide challenge (amount of urine in ml is lower than amount of furosemide given) is a marker of increased severity and mortality if RRT is not initiated [7].

Potassium (K) of 6.5 mEq/L or more with ECG changes can receive rapid acting insulin with Dextrose intravenously (IV) to rapidly reduce K

levels by pushing K into the cells as a temporary measure, and IV calcium to protect the heart muscle when ECG changes are present pending improvement in K levels or RRT.

Medications that worsen the glomerular filtration rate (GFR) like NSAIDs and ACEi should be withheld during the acute phase and all other medications that are renally cleared requires adjustment of doses. Worth mentioning that GFR is not accurate in AKI and creatinine clearance equation should be used instead [8].

Intravenous fluid is usually indicated in AKI unless there is a clear evidence of fluid overload, hypotension is a common cause of prerenal AKI or a consequence of AKI in cases like sepsis.

It is a good practice to avoid contrast unless the procedure or scan required to save life and rule in/out a life-threatening condition like aortic dissection or large pulmonary embolism (PE), in this case a close liaising with the radiology team is required to deliver a small dose of contrast if possible.

Assessment of volume status is essential in patients with AKI, this assessment is done clinically (history and physical examination) with the help of inferior vena cava measure by ultrasound (checking for size and compressibility), as mentioned above, IV fluid is indicated in cases of prerenal AKI and intravenous fluid depletion or furosemide high dose in cases of fluid overload.

The rate and amount of fluid given in patients with prerenal AKI depends on perfusion, in hypotensive patients a fluid challenge is indicated, the aim of IV fluid is to improve the cardiac output and oxygenation as well as the renal blood flow, and the response is measured by signs of improved perfusion like blood pressure, reduction in lactate, and rise in UO.

Delay in prerenal stage treatment may risk the progression into acute tubular necrosis (ATN), which is irreversible. The recommended IV fluid to use in prerenal AKI is buffered crystalloids, Colloid solutions are contraindicated.

Dietician has a role in AKI, it is essential to provide energy, protein, and nutrients to patients with renal failure and help with diet that restrict K and phosphorous [9].

Potassium (K) binders are slow in action and risk few complications while the indication for phosphate binders are levels above 1.8 mmol/L. calcium or non-calcium containing phosphate binders depends on the level of calcium. If the treatment of phosphate did not improve calcium levels and patient has symptomatic hypocalcemia with levels below 1.9 mmol/L then IV calcium treatment is indicated.

Metabolic acidosis below 7.1 is an indication for RRT as mentioned above, unless can be controlled or managed medically, Bicarbonate infusion (aim bicarbonate level of 20 mEq/L or more) can treat acidosis or bridge till RRT is available if the patient is not overloaded or hypernatremia [10].

Worth mentioning that bicarbonate infusion can lower calcium and K, increase carbon dioxide partial pressure and increase intracranial pressure in patients with DKA [11].

While severe acidosis below 7.1 can reduce ventricular contractility, leads to fatal arrhythmias, and impair the response to vasopressors [12]. Strict monitoring UO, daily weights, and fluid balance are essential tools in monitoring patients with AKI [13].

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