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Short Communication

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

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Abstract

Clinical evaluation is the first step in the management of acute kidney injury (AKI). Complications of AKI include pulmonary edema from fluid overload, tachypnoea from acidosis or fluid overload, indications of encephalopathy, such as seizures, and pericardial rub. Before standard laboratory blood findings are available to check for hyperkalemia and degree of acidity, venous blood gas (or arterial if hypoxic) is urgently needed.

In order to monitor for complications and potential causes of acute kidney injury (AKI), a clinical examination is necessary. This includes looking for signs of sepsis or shock, which call for immediate management using the sepsis six bundle, palpating the bladder to rule out obstruction, considering the use of a urinary catheter even in the absence of obstruction to aid in urine output (UO) monitoring, and determining whether active sediment (proteinuria and hematuria) is present in the urine dipstick.

Keywords: Acute Kidney Injury (AKI); Hyperkalemia; Renal disease; Glomerular filtration rate.

Abbreviations: AKI: Acute Kidney Injury; UO: Urine Output; GFR: Glomerular Filtration Rate; TTP: Thrombotic Thrombocytopenic Purpura; HUS: Hemolytic Uraemic Syndrome; CK: Creatine Kinase.

Introduction

Clinical evaluation is the first step in the management of acute kidney injury (AKI). Complications of AKI include pulmonary edema from fluid overload, tachypnoea from acidosis or fluid overload, indications of encephalopathy, such as seizures, and pericardial rub. Before standard laboratory blood findings are available to check for hyperkalemia and degree of acidity, venous blood gas (or arterial if hypoxic) is urgently needed [1].

In the face of severe, potentially fatal consequences, taking a patient's medical history is centered on determining the cause of acute kidney injury (AKI). This includes a

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complete medication history, including any contrast used during tests or procedures such as an angiography; a history suggestive of infection that resulted in sepsis; a history of urinary symptoms suggestive of prostatic hypertrophy in men that led to urinary obstruction; a history of vomiting or diarrhea suggestive of a prerenal cause; a history of arteriopathy that could indicate renal artery stenosis; and systemic symptoms that could indicate vasculitis [1].

In order to monitor for complications and potential causes of acute kidney injury (AKI), a clinical examination is necessary. This includes looking for signs of sepsis or shock, which call for immediate management using the sepsis six bundle, palpating the bladder to rule out obstruction, considering the use of a urinary catheter even in the absence of obstruction to aid in urine output (UO) monitoring, and determining whether active sediment (proteinuria and hematuria) is present in the urine dipstick [2]. Since the development of AKI care bundles, management of AKI has greatly improved; yet AKI therapy requires on the cause and immediate care for sepsis as previously stated, the goal and pace of fluid infusion are based on improving indicators of perfusion such as UO, lactate, heart rate, and blood pressure in cases of intravascular depletion leading to prerenal failure. This is especially true for crystalloid infusion. Maintenance fluid is recommended once the patient is euvolemic, with careful monitoring of the UO, daily weight, and fluid balance [3].

Urinary team immediate consultation to consider nephrostomy or stenting if urinary blockage is high and confirmed by imaging. Medication dosages for drugs that are cleared by the kidneys should be decreased, and harmful drugs that lower the glomerular filtration rate (GFR), such as ACEi, ARBs, and NSAIDs, should be avoided.

Discussion

Patients with AKI typically need a battery of examinations, although it's a good idea to order blood tests based on clinical suspicion.

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

If a urinalysis reveals the presence of blood or protein, there is a strong suspicion that the patient has intrinsic renal disease. This suggests testing for autoantibodies, such as serum complements, immunoglobulins, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, and anti-glomerular basement membrane antibodies [4].

Standard blood tests include CRP, glucose, bicarbonate, coagulation screen, liver function test, renal profile, bone profile, and full blood count (eosinophilia may indicate interstitial nephritis or atheromatous embolism). Protein and light chain electrophoresis, immunoglobulins, and a myeloma screen should be performed if there is any indication of bone pain, lytic lesions, hypercalcemia, elevated uric acid, or normocytic anemia [4].

Lactate dehydrogenase (LDH), blood film, complete hemolytic screen if hemolytic uraemic syndrome (HUS) or thrombotic thrombocytopenic purpura (TTP) is suspected, and creatine kinase (CK) in cases of trauma or prolonged lying [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

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within 24 hours if worsening or no improvement of renal function [4].

If the initial medical management of AKI complications-primarily pulmonary oedema, hyperkalemia, severe acidosis below 7.1, or uraemic symptoms-does not alleviate the condition, a prompt referral to the intensive care team is necessary.

A referral to the renal team is necessary immediately if there are no problems but the renal function declines or does not improve after the first course of treatment. This is particularly important in cases of AKI stage 3, probable vasculitis/glomerulonephritis, and indications for dialysis.

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

Additional frequent reasons for RRT are patients with anuric AKI and multiple organ failure or few toxicological causes.

The goal is a total caloric intake of 20–30 kcal/kg/day and 0.8-1 g/kg/day of protein, which should be increased to 1-1.5 g/kg/day in AKI on RRT [5]. Dietician involvement is strongly encouraged in instances of AKI.

Acute kidney injury (AKI) severity is categorized into three stages: level 1 is defined as a rise in serum creatinine (Cr) of 1.5 to 1.9 × baseline or a reduction in urine output (UO) of <0.5 mL/kg/hour for 6 to 12 hours; level 2 is defined as a rise in Cr of 2 to 2.9 × baseline or a reduction in UO of <0.5 mL/kg/hour for 12 to 24 hours; and level 3 is defined as a rise in Cr of ≥ 3 × baseline or a 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 an increase in the 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 relief 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

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and ACEi should be withheld during the acute phase and all other medications that are renal cleared require 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 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 restricts 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 impairs 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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