

Case Based Discussion; Initial Management of Acute Kidney Injury (AKI)

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Abstract

Acute Kidney Injury (AKI) is a very common clinical presentation, this is a serious yet often avoidable condition. The reported incidence of AKI is between 13-18% of all patients admitted to hospital with older adults being particularly affected. Most important to note is the reported inpatient mortality rates of AKI that vary depending on severity; in the UK this can vary between 25-30%. If medical teams put appropriate measures in place, patients can receive the care and treatment required.

The financial impact of AKI on the NHS is estimated to be between £434 million and £620 million per year, this is reported to be more than the associated cost of breast, lung and skin cancer combined.

Keywords: Diabetes mellitus; Penicillin; Acute kidney injury; Creatinine; Chronic Kidney Disease.

Abbreviations: CKD: Chronic Kidney Disease; KIDGO: Kidney Disease Improving Global Outcome; AKI: Acute Kidney Injury; WCC: White Cell Count.

Introduction

A 71-year-old man; was found on the floor unconscious; by the neighbour. Two days prior, the patient was last observed in garden. From the ambulance, GP notes and the collateral history it was noted that the patient had a past medical history of benign prostatic hypertrophy, type 2 diabetes mellitus and hypertension. Patient medications at the time were Metformin 1 g BD, Sitagliptin 100 mg OD, Ramipril 10 mg ON, Bendroflumethiazide 2.5 mg OD and

Tamsulosin 400 mcg OD. Patient recently started Ibuprofen for back pain, and it was noted that patient was allergic to Penicillin [1].

Medical history was a week history of diarrhoea and vomiting after eating at a Chinese restaurant. Biochemistry showed urea to be 52 and creatinine of 412, creatinine clearance of 22, C-reactive protein (CRP) of 220, White Cell Count (WCC) of 16, neutrophils. CK was 600, urine dipstick showed leucocyte of 3⁺, nitrites positive,

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Blood⁺⁺, Protein ⁺⁺, the patient also had a low-grade temperature. Biochemistry the previous month (requested by the GP) showed urea of 4 and creatinine of 105.

Case study

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Biochemistry the previous month (requested by the GP) showed urea of 4 and creatinine of 105. The patient had a non-contrast CT head and neck and x-ray pelvis to rule out stroke or fractures due to un-witnessed fall and being found unconscious. Patient was alert but confused in A&E.

The patient was admitted into a side room, initial impression being pre-renal AKI

secondary to viral gastroenteritis (confirmed by presence of norovirus in stool) plus urine infection.

A fluid flow chart was commenced upon initiation of crystalloids and a catheter was inserted to monitor urine output, which found a residual volume of 50 mls dark colored urine.

A renal ultrasound was requested to rule out obstruction and/or mass (haematuria) USS reported as within normal limits. A vasculitis screen was requested (active sediment-protein and blood), nephrotoxins stopped and the mean arterial pressure was kept above 65 to maintain good perfusion to kidneys.

The patient was prescribed Ciprofloxacin for three days, as per microbiology advice. On day four, biochemistry showed a creatinine of 130 and urea of 5. Due to the improvement in renal function their medications were recommended. The vasculitis screen was negative.

The patient was discharged home under the care of GP with advice to monitor renal function (risk of diabetic nephropathy and obstructive uropathy).

Evidence states

“30% of patients who died from AKI had predictable and avoidable AKI”. The publication also highlighted “20% of the patients who developed AKI following admission subsequently died” [2,3].

Discussion

Using the Kidney Disease Improving Global Outcome (KIDGO) scale to stage AKI helps provide a universal staging criterion (Shown Table 1).

Stage	Serum Creatinine (SCr)Criteria	Urine Output Criteria
1.	Increase $\geq 26 \mu\text{mol/L}$ within 48 hrs or	$<0.5\text{mL/kg/hr}$ for >6 consecutive hrs
	Increase ≥ 1.5 to $1.9 \times$ reference SCr	
2.	Increase ≥ 2 to $2.9 \times$ reference SCr	$<0.5\text{mL/kg/hr}$ for >12 hrs
3.	Increase $\geq 3 \times$ reference SCr or	$<0.3 \text{ mL/kg/hr}$ for >24 hrs or anuria for 12 hrs
	Increase $\geq 345 \mu\text{mol/L}$ or commenced on renal replacement therapy (RRT) irrespective of stage	

Table 1: KIDGO AKI scale.

Why staging is important?

This scale is used for patients presenting with renal failure and provides medical professionals an easy to use tool to simply identify the level of AKI of their patient. This scale is linked to the possibility of mortality and morbidity with the understanding that a rising creatinine is directly proportional to mortality and morbidity [1-4].

Summary of initial management

1. **Early detection:** high-risk patients should be identified as early as possible. The following list identifies who are classified as high-risk:
 - a. Chronic Kidney Disease (CKD) $\text{eGFR} < 60$.
 - b. Heart failure.
 - c. Liver disease.
 - d. Diabetes.
 - e. History of AKI.
 - f. Dehydration/oliguria.
 - g. Patients taking nephrotoxins.
 - h. Use of iodinated contrast agent within the past 7 days.
 - i. Symptoms or history of urological obstruction.
 - j. Sepsis.

k. Over 65 years of age.

l. Surgical patients-especially intra-peritoneal surgery.

2. **Fluid assessment and therapy:** Adequate fluid therapy requires a number of steps. A volume status assessment is used to assess if the patient is under filled or overloaded by checking heart rate, blood pressure and postural blood pressure, mucous membranes and JVP. Achieve euvolaemia by administration of Crystalloids by boluses and then maintenance is guided by urine output, blood pressure and fluid balance.

3. Investigation

The following investigations should be requested:

- a. Urine dip stick-to look for infection (Nitrites or leucocytes). If proteinuria then protein creatinine ratio and send urine for microscopy (casts) plus autoimmune screen if haematuria+and proteinuria (active sediment) with rash and joint pains.

- b. Bone profile/liver function/CK- to rule out rhabdomyolysis- usually CK in thousands.
 - c. Myeloma screen (normocytic anaemia, raised calcium, bone pains).
 - d. If platelets are low perform microangiopathy screen (blood film, LDH and reticulocytes).
 - e. Ultrasound-within 24 hours if no identified cause of AKI or risk of urinary tract obstruction. This should be requested as urgent if there is suspicion of hydronephrosis.
 - f. Treat sepsis-if present, guided by finding a focus of infection, fever, raised inflammatory markers and lactate.
 - g. Stop nephrotoxins (NSAID/ACEI/ARB/diuretics).
 - h. Avoid hypotension to maintain adequate perfusion to the kidneys.
 - i. Consider PPI (platelet dysfunction and ensure pharmacist input for drug monitoring).
4. **Onward referral:** Nephrology should be made within 24 hours if the likely diagnosis of AKI may need specialist treatment (vasculitis, glomerulonephritis, myeloma). Other reasons for referral to nephrology include AKI with no clear cause, when there is inadequate response to treatment, persistent complications (persistent hyperkalaemia, pulmonary oedema, or acidosis), when the patient is in stage 3

AKI, history of renal transplant or Chronic Kidney Disease (CKD) stage 4 and 5.

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