Acute kidney injury in Sri Lanka: A comprehensive approach to recognition, management, and prevention

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Abstract

Acute kidney injury (AKI) poses a significant health challenge in Sri Lanka, affecting both community and hospitalised populations with distinct epidemiological patterns. This review provides evidence-based guidance on recognising, managing, and preventing AKI, emphasising the unique disease patterns and resource considerations relevant to local practice. Since community-acquired causes account for 80% of AKI cases in low- and middle-income countries, understanding tropical infections, environmental toxins, and socioeconomic factors is crucial for effective management.

Key words: acute kidney injury, AKI, Sri Lanka, tropical infections, environmental toxins

Introduction

Acute kidney injury (AKI) is characterised by a sudden decline in kidney function that occurs over hours to days, with or without structural kidney damage. Despite advances in prevention, diagnosis, and treatment, AKI affects approximately 13.3 million people each year, resulting in 1.7 million deaths globally.[1,2] The condition poses particular challenges in Sri Lanka due to the high prevalence of tropical infections, environmental toxins, and limited resources.

AKI can occur in community or hospital settings, affecting about 20% of admitted patients with a fourfold increase in mortality risk.[3] The International Society of Nephrology (ISN) 0 by 25 Global Snapshot study[4] demonstrated that 80% of AKI cases in low- and lower-middle-income countries were community-acquired, emphasising the importance of understanding local disease patterns and developing effective prevention strategies.

Causes of AKI

The most common causes of AKI are dehydration, sepsis, drugs, toxins, and pyelonephritis. Poor socio-economic status, environmental, and occupational factors significantly contribute to the development of AKI within the community. A study conducted in an intensive care unit (ICU) at a tertiary care hospital in Sri Lanka reported an AKI incidence of 60.2%. The highest incidence of AKI was seen in patients admitted for cardiac causes, followed by sepsis, respiratory, and hepatic causes. Thirty-seven per cent (37%) of these patients were in AKI Network stage 3 and required RRT[6]. Another study involving 333 patients managed outside the ICU in a tertiary care centre found that the most common cause of AKI was systemic infections (59.5%). Nephrotoxic medications (33.6%), dehydration (24.6%), and pyelonephritis (10.8%) were other prevalent causes of AKI[6].

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Table 1. Common causes for AKI in Sri Lanka

Community acquired	*Hospital acquired*
Tropical infections ▪ Leptospirosis ▪ Dengue ▪ Typhus ▪ Hanta virus Other Infections ▪ Pyelonephritis	Sepsis
Diarrhoeal illnesses	Hypotension and Dehydration
Hypovolemia/Dehydration	Nephrotoxic medications ▪ Vancomycin ▪ Aminoglycosides ▪ Amphotericin ▪ Acyclovir ▪ Chemotherapy agents
Snake bite ▪ Hump-nosed viper ▪ Russell's viper ▪ Saw-scaled viper (rarely)	Contrast agents (Iodine and Gadolinium)
Environmental toxins ▪ Plants – Toxic mushrooms ▪ Chemicals – Prinso (combination of potassium permanganate and oxalic acid), brake oil	Post-surgical complications
Nephrotoxic medications ▪ NSAIDS	
Acute glomerulonephritis (AGN) Acute interstitial nephritis (AIN)	

High-risk patients

High-risk patients for AKI include the elderly (over 65 years), those with pre-existing CKD, diabetes, hypertension, heart failure, and liver disease. Patients who have dehydration, sepsis, and who are undergoing major surgeries are also at high risk for AKI. Some medications, such as NSAIDs, aminoglycosides, vancomycin, ACEI, angiotensin receptor blockers, empagliflozin, and dapagliflozin, can cause AKI in susceptible patients (hypotensive, dehydrated). These patients have a high risk of AKI and require regular assessment of urine output and serum creatinine.

Diagnosis

The diagnosis of AKI relies primarily on the Kidney Disease Improving Global Outcomes (KDIGO) criteria, which utilise changes in serum creatinine levels and urine output[7] (Table 2). However, diagnostic delays remain a significant problem in Sri Lankan healthcare settings. A study at the National Hospital of Sri Lanka found that among 226 patients with AKI, the diagnosis was missed in 29% of cases and delayed by 48 hours in another 17% cases[5]. These delays are due to limited awareness among healthcare providers, late patient presentation to healthcare facilities, and

resource constraints in monitoring systems. In addition to serum creatinine, all patients with AKI should urgently undergo urinalysis and abdominal ultrasound.

Novel Biomarkers

While serum creatinine remains the standard diagnostic marker, novel biomarkers such as KIM-1, IL-18, neutrophil gelatinase-associated lipocalin (NGAL), and Nephro Check (TIMP-2 and IGFBP7) have received FDA approval for specific clinical applications. However, their routine use in resource-limited settings requires further evaluation of cost-effectiveness and clinical utility[8].

Prevention of AKI

The incidence of AKI is likely to rise due to various factors. These include an ageing population, the increasing prevalence of diabetes mellitus, hypertension, and other non-communicable diseases, climate change, rising environmental pollution, and exposure to toxic substances. Most data on AKI originates from high-income countries. In contrast, information from low- and lower-middle-income countries remains limited due to under-reporting and poor recording systems, leading to inadequate surveillance of AKI[8]. Preventing AKI is vital, particularly for low-income countries that lack resources and infrastructure to manage AKI and its acute and long-term complications.

Community-level preventive strategies:

- Public education on early recognition and prevention of dehydration, leptospirosis, and snake bites (e.g., adequate hydration to correct volume depletion, prevention of leptospirosis by using protective gloves, boots, and taking pre- or / and post-chemoprophylaxis in risk exposure)
- Avoiding prescription of NSAIDs in sick, hypo-volemic, hypotensive patients.
- Instruct patients not to take ACEI/ARB, metformin, or SGLT2 inhibitors during acute illness (sick day rule).

Hospital-level preventive strategies:

- Identification of high-risk patients (elderly, comor-bidities, major surgery)
- Monitor renal function and urine output closely in high-risk patients.

Table 2. KDIGO criteria[7] for diagnosis and staging of AKI

The KDIGO criteria define acute kidney injury based on increases in serum creatinine or a decrease in urine output. AKI is diagnosed if any of the following occur:

- An increase in Serum Creatinine by 0.3mg/dL (26 mol/l) or more within 48 hours.
- An increase in Serum Creatinine to 1.5 times the baseline level, which is known or presumed to have occurred within the prior seven days.
- A urine volume of less than 0.5 mL/kg/h for six hours.

Stage	*Serum creatinine*	*Urine output*
1	1.5-1.9 times baseline OR ≥0.3 mg/dl (≥26.5 mmol/l) increase	<0.5 ml/kg/h for 6-12 hours
2	2.0-2.9 times baseline	<0.5 ml/kg/h for ≥12 hours
3	3.0 times baseline OR increase in serum creatinine to ≥4.0 mg/dl (≥353.6 mmol/l) OR Initiation of renal replacement therapy	<0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours

- Optimise volume status and blood pressure and withdraw nephrotoxic medications before surgery and contrast studies.
- Correct hypovolemia or hypotension promptly.
- Minimise nephrotoxic exposure and ensure hydration before and after administering high-risk medications (e.g., vancomycin, aminoglycosides, acyclovir, and chemotherapy drugs).
- Monitor drug levels of vancomycin and aminoglycosides.

Initial management

If a patient has an elevated serum creatinine that has not been previously documented, it should be considered AKI unless proven otherwise (use the KDIGO diagnostic and staging criteria). Distinguishing between hypovolemia and fluid overload is vital as the treatments are opposite. Fluid resuscitation in AKI aims to restore intravascular volume and renal perfusion, primarily with crystalloids such as normal saline.

Low blood pressure, increased heart rate, cold extremities, dry mouth, reduced skin turgor, and prolonged capillary refill time indicate dehydration. Elevated jugular venous pressure, oedema (puffy eyelids, peripheral oedema), lung crepitations, and tachypnoea indicate fluid overload. In the meantime, careful fluid intake and monitoring of urine output, along with advanced tools such as bedside ultrasound (IVC, lungs) and dynamic measures (pulse pressure variation), are crucial for targeted treatment.

Identifying and removing the cause of AKI, along with prompt treatment, are essential. After initiating treatment, the patient should be reassessed for improvements in hydration, blood pressure, and urine output, as well as signs of fluid overload. If fluid overload signs develop, fluid intake should be restricted, generally to 1-1.5 litres over 24 hours. Severe fluid overload may necessitate single or multiple doses of intravenous furosemide (1 mg/kg).

When managing blood pressure, it is crucial to consider comorbidities, such as pre-existing hypertension and diabetes, particularly in elderly patients, in whom higher mean arterial pressure may be necessary to ensure adequate renal perfusion. Antihypertensive medications should be discontinued if blood pressure is already low or if a decline is expected due to sepsis or other organ dysfunction.

Summary of initial management

Assessment of volume status and optimisation of fluid balance:

- Careful assessment of intravascular volume status
- Correct hypovolemia with suitable fluid resuscitation (crystalloid solutions)
- Prevent fluid overload in patients with adequate or excessive volume
- Monitor fluid balance and urine output closely
- Restrict fluids (usually 1-1.5 litres per 24 hours) in cases of fluid overload
- Judicious use of diuretics (furosemide 1 mg/kg) for fluid overload

Blood pressure management

- Maintenance of adequate mean arterial pressure for kidney perfusion (According to the patient's age and comorbidities)
- Consideration of pre-existing hypertension, particularly in elderly patients
- Discontinuation of antihypertensive medications if hypotension is present or anticipated. (You may discontinue them even if blood pressure is normal)

Cause identification and treatment

- Prompt identification and treatment of underlying causes
- Withdrawal of nephrotoxic medications
- Treatment of infections and sepsis
- Management of obstruction

Management of metabolic complications

- Monitoring and correction of electrolyte imbalances
- Management of metabolic acidosis
- Attention to hyperkalaemia and its cardiac effects
- Optimisation of nutrition

Kidney Replacement Therapy (KRT)

Timely initiation of KRT is crucial if kidney function does not improve and the patient develops life-threatening complications of AKI (Box 1). The evidence primarily supports an indication-based KRT strategy, as early initiation does not improve survival and carries

greater risks. However, KRT facilities vary across hospitals, and these differences directly affect outcomes.[7,8] Therefore, when KRT services are unavailable on-site, the patient must be transferred to another hospital for further management as early as possible. It is vital to seek a nephrologist's advice early to minimise unnecessary delays and to arrange KRT during regular working hours rather than as an emergency. Stabilising the patient medically before transfer for KRT is essential.

Modes and timing of KRT

Modes of KRT depend on patients' hemodynamic status. Stable patients can undergo intermittent haemodialysis, whereas unstable patients should receive continuous renal replacement therapy (CRRT). Alternatively, acute peritoneal dialysis can be considered in haemodynamically unstable patients with a flexible single cuff CAPD catheter. Clinicians should focus on identifying specific complications necessitating KRT, rather than rushing to start it based solely on AKI staging. In uncertain situations, it is advisable to consult a nephrologist to decide on KRT.

Indications for KRT in AKI

- Medically refractory fluid overload
- Medically refractory hyperkalaemia
- Medically refractory metabolic acidosis
- Uraemic encephalopathy
- Uraemic pericarditis
- Drug toxicity/ poisoning with dialysable substances (Eg, lithium, ethylene glycol, ethanol, metformin)

Follow up

Most patients with AKI recover completely, but some may progress to acute kidney disease (AKD) and CKD. Therefore, it is essential to schedule a follow-up visit for all patients after hospital discharge to assess the recovery of kidney function. Additionally, this visit should be used to optimise blood pressure management and other comorbidities, review medication lists, and reinforce the patient's understanding of risk factors for AKI recurrence and its prevention.

Outcomes and prognosis

Factors linked to poor outcomes include delayed diagnosis, inadequate initial management, limited access to KRT, hypotension, and the presence of multiple comorbidities. Early recognition and appropriate management can significantly improve patient outcomes and reduce the risk of progression to CKD.

Challenges in the Sri Lankan context

Resource limitations and gaps in healthcare delivery hinder the management of AKI in Sri Lanka.[8] Access to KRT remains limited outside major centres, compounded by a shortage of specialised nephrology services and inadequate diagnostic capabilities in peripheral hospitals.[8] Weak referral networks, a lack of standardised clinical protocols, and insufficient training among frontline healthcare providers further hinder effective care. To address these issues, quality improvement initiatives are essential, including the development of AKI registries, the implementation of quality indicators, and the regular auditing of clinical practice. Capacity-building efforts, such as training programs for healthcare workers, the implementation of clinical decision support tools, and the enhancement of laboratory services, are also critical to improving early detection, timely management, and overall patient outcomes in AKI.

Conclusion

AKI is an increasing health burden in Sri Lanka, affecting both community and hospital populations. Tropical infections such as leptospirosis and dengue, along with sepsis, dehydration, and the use of nephrotoxic drugs, continue to be significant contributors. Limited resources, delayed diagnosis, and gaps in healthcare providers' awareness compound the challenge. Early recognition through simple tools, such as monitoring serum creatinine and urine output, along with prompt correction of hypovolemia and withdrawal of nephrotoxins, can significantly improve outcomes. Timely access to kidney replacement therapy when needed is lifesaving but remains limited to major centres, underscoring the need to enhance referral networks. Community-level preventive measures, such as public education, infection control, and the avoidance of inappropriate drug use, are equally crucial. Post-discharge follow-up should be prioritised to promote kidney recovery, manage blood pressure and comorbidities effectively, and prevent recurrence. Strengthening surveillance systems and capacity building across all levels of the health system are vital to reducing the burden of AKI in Sri Lanka and improving long-term patient outcomes.

Key learning points

- Identification of high-risk patients, with regular monitoring of urine output and serum creatinine, is essential for early diagnosis of AKI.
- Reduced urine output is often the earliest indication of AKI and should prompt immediate intervention even before the rise of serum creatinine is observed.
- Urine analysis is crucial in the initial assessment, and urinary tract obstruction should be excluded by imaging.
- Identifying and correcting the underlying cause of AKI is fundamental in the management.
- Optimal care includes appropriate fluid and electrolyte management, timely initiation of KRT, and effective management of complications.
- Early referral to a nephrologist is recommended in appropriate cases.

Conflict of interests

The authors declare no conflict of interests.

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