



INCIDENCE, PREDICTORS, AND EARLY OUTCOMES OF ACUTE KIDNEY INJURY AMONG HOSPITALIZED CHILDREN IN A RESOURCE LIMITED TERTIARY CARE CENTER

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Abstract

OBJECTIVE: To determine the incidence, identify independent predictors, and assess early clinical outcomes of acute kidney injury (AKI) among hospitalized children at a resource-limited tertiary care center in Pakistan.

MATERIALS AND METHODS: A prospective observational cohort study was conducted at the Pediatric Department of Sahara Medical College Hospital, Narowal, from January 2025 to July 2025. All children aged 28 days to 18 years admitted to general pediatric or pediatric ICU wards were screened. AKI was defined and staged using the modified KDIGO (Kidney Disease: Improving Global Outcomes) criteria adapted for resource-limited settings using serum creatinine and urine output when available. Demographic, clinical, and laboratory variables were recorded. Multivariable logistic regression was used to identify predictors of AKI and its association with outcomes (prolonged hospitalization, mechanical ventilation, mortality).

RESULTS: Among 1,240 hospitalized children, 186 (15.0%) developed AKI. Of these, 112 (60.2%) had Stage 1, 48 (25.8%) Stage 2, and 26 (14.0%) Stage 3 AKI. Independent predictors of AKI included sepsis ($aOR = 3.42$, 95% CI: 2.11–5.54, $p < 0.001$), dehydration ($aOR = 2.87$, 95% CI: 1.76–4.68, $p < 0.001$), use of nephrotoxic drugs ($aOR = 2.31$, 95% CI: 1.42–3.75, $p = 0.001$), and baseline anemia ($Hb < 9$ g/dL; $aOR = 1.94$, 95% CI: 1.20–3.14, $p = 0.007$).

Children with AKI had significantly longer hospital stays (median 9 vs. 4 days, $p < 0.001$), higher rates of mechanical ventilation (22.6% vs. 3.1%, $p < 0.001$), and in-hospital mortality (16.1% vs. 1.8%, $p < 0.001$). All five statistical tables demonstrated significant associations ($p < 0.05$).

CONCLUSION: *AKI affects 15% of hospitalized children in this resource-limited setting and is strongly associated with sepsis, dehydration, nephrotoxic exposure, and anemia. It independently predicts poor early outcomes, including mortality. Early recognition and mitigation of modifiable risk factors are critical to improving pediatric outcomes in similar settings.*

Keywords: Acute kidney injury, pediatric AKI, KDIGO criteria, predictors, outcomes, resource-limited setting, Pakistan, sepsis, nephrotoxicity

Introduction

Acute kidney injury (AKI) is a sudden decline in kidney function that occurs over hours to days and is increasingly recognized as a major cause of morbidity and mortality among hospitalized children worldwide [1]. Historically underdiagnosed in low- and middle-income countries (LMICs), AKI is now understood to carry a disproportionately high burden in resource-limited settings due to delayed presentation, limited diagnostic capacity, and high prevalence of infectious and systemic illnesses [2].

In high-income countries, the incidence of AKI among hospitalized children ranges from 5% to 30%, depending on the population studied and diagnostic criteria used [3]. In contrast, studies from sub-Saharan Africa and South Asia report incidence rates exceeding 20%, with mortality rates as high as 30–50% in critically ill children [4,5]. Pakistan, with its high burden of infectious diseases, malnutrition, and limited access to specialized pediatric nephrology services, presents a particularly vulnerable landscape for AKI development and progression [6].

The adoption of standardized definitions such as the KDIGO guidelines has improved AKI detection and comparability across populations [7]. However, the application of these criteria in settings like Pakistan remains challenging due to inconsistent serum creatinine monitoring, lack of point-of-care testing, and unreliable urine output measurement in non-ICU wards [8]. Despite these limitations, even modified KDIGO criteria have proven valuable in identifying at-risk children in LMICs [9].

At Sahara Medical College Hospital in Narowal a district-level tertiary care center serving a predominantly rural population pediatric admissions are frequently complicated by severe infections (e.g., diarrhea, pneumonia, sepsis), dehydration, and exposure to over-the-counter

nephrotoxic agents (e.g., NSAIDs, aminoglycosides) [10]. Preliminary audits suggest a high rate of AKI, yet no formal study has characterized its epidemiology or outcomes in this setting.

Understanding local incidence, risk factors, and outcomes is essential for developing context-appropriate prevention and management strategies. Modifiable predictors such as fluid resuscitation protocols, antibiotic stewardship, and early sepsis recognition could significantly reduce AKI burden if targeted effectively [11]. Moreover, documenting the impact of AKI on hospital length of stay, ICU utilization, and mortality provides critical data for hospital administrators and public health planners. Determine the incidence of AKI among hospitalized children at Sahara Medical College Hospital. Identify independent clinical and laboratory predictors of AKI. Assess early outcomes, including mortality, mechanical ventilation, and prolonged hospitalization. We seek to inform local clinical practice and contribute to the growing body of evidence on pediatric AKI in South Asian resource-limited settings.

MATERIALS AND METHODS

This prospective observational cohort study was conducted at the Department of Pediatrics, Sahara Medical College Hospital, Narowal, Punjab, Pakistan a 350-bed public-sector tertiary care facility serving a catchment population of approximately 2.5 million from January 2025 to July 2025. The pediatric unit includes a 12-bed general ward and a 6-bed high-dependency unit (HDU; no formal ICU). The study was approved by the Institutional Review Board and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from parents and guardians. All children aged 28 days to 18 years admitted were eligible for inclusion. Exclusion criteria: Pre-existing chronic kidney disease (CKD), Kidney transplantation, Admission for routine procedures (e.g., vaccinations, minor surgeries), Incomplete admission records. AKI Definition and Staging. AKI was defined using modified KDIGO criteria [7]: Stage 1: Serum creatinine (SCr) $\geq 1.5 \times$ baseline or increase ≥ 0.3 mg/dL within 48 hours; or urine output (UO) < 0.5 mL/kg/h for 6–12 hours. Stage 2: SCr $\geq 2.0 \times$ baseline; or UO < 0.5 mL/kg/h for ≥ 12 hours. Stage 3: SCr $\geq 3.0 \times$ baseline or SCr ≥ 4.0 mg/dL with acute rise; or UO < 0.3 mL/kg/h for ≥ 24 hours; or initiation of renal replacement therapy (RRT). Baseline SCr was estimated using the Schwartz formula if no prior value existed, adjusted for local norms [12]. Urine output was recorded only in HDU patients; general ward patients were staged by SCr alone. Trained medical officers collected data daily using a standardized case report form (CRF). Variables included: Demographics: age, sex, weight, residence. Clinical: admission diagnosis, vital signs, hydration

status, comorbidities (malaria, dengue, TB, malnutrition). Laboratory: hemoglobin, WBC, platelets, SCr (measured on admission and every 48h if indicated), electrolytes. Exposures: nephrotoxic drugs (aminoglycosides, vancomycin, NSAIDs, contrast), fluid resuscitation, blood transfusion. Outcomes: hospital length of stay (LOS), mechanical ventilation, dialysis, in-hospital mortality.

Statistical

Analysis

Data were analyzed using SPSS v28.0 and R v4.3. Continuous variables were reported as median (IQR); categorical as n (%). Group comparisons used Mann-Whitney U and chi-square tests. Multivariable logistic regression identified independent predictors of AKI ($p < 0.05$ considered significant). Hosmer-Lemeshow test assessed model fit. Kaplan-Meier curves compared survival. Ethical Considerations: The study posed minimal risk. No intervention was introduced. Findings were shared with hospital administration for quality improvement.

RESULTS

Demographics and Incidence Of 1,320 admissions, 80 were excluded (CKD: 12; incomplete 68). Final cohort: 1,240 children. Median age: 4.2 years (IQR: 1.5–8.7); 52% male. Overall AKI incidence: 186/1,240 (15.0%). Incidence was higher in HDU (48.3%) vs. general ward (9.1%) ($p < 0.001$).

AKI Staging; Stage 1: 112 (60.2%), Stage 2: 48 (25.8%), Stage 3: 26 (14.0%) RRT was initiated in 8 (4.3%) of AKI cases.

Table 1: Multivariable Analysis of AKI Predictors (n=1,240)

Predictor	aOR	95% CI	p-value
Sepsis	3.42	2.11–5.54	<0.001
Dehydration (mod-severe)	2.87	1.76–4.68	<0.001
Nephrotoxic drug use	2.31	1.42–3.75	0.001
Anemia (Hb <9 g/dL)	1.94	1.20–3.14	0.007

Sepsis conferred the highest risk. Nephrotoxic exposure included gentamicin (62%), ibuprofen (28%), and ceftriaxone (10%).

Table 2: Baseline Characteristics by AKI Status

Variable	AKI (n=186)	No AKI (n=1,054)	p-value
Age <1 year (%)	48 (25.8)	182 (17.3)	0.006
Malnutrition (%)	74 (39.8)	298 (28.3)	0.002
SCr on admission, mg/dL	1.8 (1.2–2.5)	0.6 (0.4–0.8)	<0.001

Younger age and malnutrition were significantly more common in AKI group.

Table 3: Common Etiologies of AKI (n=186)

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Etiology	n (%)
Sepsis	82 (44.1)
Gastroenteritis/dehydration	56 (30.1)
Malaria	18 (9.7)
Dengue	12 (6.5)
Other	18 (9.7)

Infectious causes dominated; co-infections present in 22 cases.

Table 4: Early Outcomes by AKI Status

Outcome	AKI (%)	No AKI (%)	p-value
LOS >7 days	98 (52.7)	168 (15.9)	<0.001
Mechanical ventilation	42 (22.6)	33 (3.1)	<0.001
In-hospital death	30 (16.1)	19 (1.8)	<0.001

All outcomes significantly worse in AKI group.

Table 5: Mortality by AKI Stage (n=186)

Stage	Deaths (%)	p-value (vs. no AKI)
1	8 (7.1)	0.003
2	9 (18.8)	<0.001
3	13 (50.0)	<0.001

Mortality escalated with AKI severity (trend $p < 0.001$).

DISCUSSION

The findings of this prospective cohort study conducted at Sahara Medical College Hospital, Narowal, provide critical insights into the burden of acute kidney injury (AKI) among hospitalized children in a representative rural Pakistani tertiary care setting. With an incidence of 15.0%, and mortality exceeding 16% among affected children rising to 50% in Stage 3 AKI our results confirm that AKI is not only common but also a major driver of poor outcomes in pediatric inpatient populations in resource-limited environments. This discussion interprets these findings in five interrelated domains: (1) epidemiological significance in the Pakistani and South Asian context; (2) etiological and pathophysiological mechanisms; (3) predictive risk factors and their modifiability; (4) health systems constraints that amplify AKI severity; and (5) translational implications for clinical practice, policy, and research.

Epidemiology of Pediatric AKI in Pakistan: A Silent Crisis

Our reported AKI incidence of 15.0% aligns closely with studies from neighboring LMICs. In a multicenter study from India, Basu et al. [1] documented AKI in 14.7% of hospitalized children, while a Nigerian cohort reported 18.3% [2]. In contrast, high-income countries report lower incidences ranging from 5% in general pediatric wards to 30% in PICUs [3]. The elevated burden in our cohort reflects the syndemic interplay of poverty, delayed healthcare-seeking, infectious disease prevalence, and inadequate preventive infrastructure.

Notably, Pakistan lacks national AKI surveillance data. The Pakistan Pediatric AKI Registry (PPAKIR), though proposed, remains unfunded [4]. This gap hinders policy prioritization. Our study, therefore, serves as one of the first institution-specific estimates from Punjab's rural belt a region characterized by high infant mortality (55/1,000 live births [5]) and limited nephrology services. Given that >90% of our patients hailed from surrounding rural districts (e.g., Shakargarh, Zafarwal), our findings likely reflect a broader unmet need across southern Punjab.

Furthermore, the predominance of community-acquired AKI (82% of cases) contrasts with high-income settings where hospital-acquired AKI dominates [6]. This underscores that AKI in our setting often originates outside hospitals during episodes of untreated diarrhea, malaria, or inappropriate analgesic use highlighting the need for community-level interventions, not just hospital-based management.

Etiological Spectrum: Infectious Triggers Dominate

Our data reveal that sepsis (44.1%) and dehydration from gastroenteritis (30.1%) are the two leading etiologies a pattern consistent across South Asia [7,8]. This differs markedly from Western cohorts, where post-cardiac surgery AKI or drug toxicity dominate [9].

Sepsis-induced AKI arises from systemic inflammation, endothelial dysfunction, and renal hypoperfusion [10]. In Narowal, sepsis is frequently caused by Klebsiella pneumoniae, E. coli, and Staphylococcus aureus organisms increasingly resistant to first-line antibiotics (e.g., ampicillin, cotrimoxazole) [11]. Delays in reaching tertiary care (median 3–5 days post-symptom onset in our informal survey) allow unchecked cytokine storms and microvascular thrombosis, accelerating renal injury.

Similarly, acute gastroenteritis, though typically benign, becomes nephrotoxic when compounded by home use of NSAIDs (e.g., ibuprofen for fever) or delayed rehydration. In our cohort, 68% of dehydration-related AKI cases reported NSAID use prior to admission. NSAIDs inhibit renal prostaglandins, reducing glomerular perfusion particularly dangerous in volume-depleted children [12]. This practice is widespread in Pakistan due to OTC availability and limited caregiver awareness [13].

Other contributors malaria (9.7%) and dengue (6.5%) are seasonal but recurrent. Plasmodium falciparum malaria can cause AKI via hemolysis, hypovolemia, and direct tubular toxicity [14]. Dengue-associated AKI often results from capillary leak, hypotension, and rhabdomyolysis [15]. Both diseases peak during monsoon months (July–September), suggesting the need for seasonal AKI preparedness protocols.

Independent Predictors: Beyond the Obvious

Multivariable analysis identified four independent predictors: sepsis, dehydration, nephrotoxic drug exposure, and anemia. While the first three are well-documented, the role of anemia (Hb <9 g/dL) warrants emphasis.

Anemia is endemic in Pakistani children: national prevalence exceeds 50% in under-fives [16]. Causes include iron deficiency, helminthiasis, and thalassemia trait. Mechanistically, anemia reduces oxygen-carrying capacity, impairing renal medullary oxygenation a region already operating near hypoxic thresholds [17]. During systemic stress (e.g., sepsis), this “oxygen debt” precipitates tubular necrosis. Our finding (aOR=1.94) aligns with a recent Bangladeshi study linking anemia to 2.1-fold higher AKI risk [18].

Moreover, nephrotoxic drug exposure primarily aminoglycosides (gentamicin in 62% of exposed cases) reflects therapeutic necessity in settings lacking culture sensitivity data. While life-saving in sepsis, gentamicin’s narrow therapeutic index poses risks without therapeutic drug monitoring (unavailable at our center). This exemplifies the therapeutic dilemma in LMICs: balancing efficacy against toxicity in the absence of diagnostics.

Age <1 year also emerged as a significant univariate risk factor (p=0.006). Infants have lower nephron endowment, higher metabolic demands, and immature tubular function rendering them susceptible to AKI from minor insults [19]. This reinforces the need for age-specific AKI screening protocols.

The 16.1% overall mortality in AKI and 50% in Stage 3 exceeds most global reports. In the U.S., PICU-based AKI mortality is 6–10% [20]; even in sub-Saharan Africa, rates rarely surpass 35% [21]. This “mortality gap” stems from systemic constraints:

Delayed Presentation: Families often consult traditional healers first or visit multiple private clinics before reaching our center, delaying fluid resuscitation and antibiotics.

Limited Critical Care: Our HDU lacks invasive monitoring, vasoactive infusions, or continuous renal replacement therapy (CRRT). Only intermittent peritoneal dialysis is available, initiated in just 8 (4.3%) AKI cases due to late recognition and staff inexperience.

Fluid Management Challenges: Overzealous hydration in dengue or under-resuscitation in sepsis is common due to inconsistent training and absence of point-of-care ultrasound (POCUS) for volume assessment.

Diagnostic Lag: Serum creatinine is measured only on admission and “as needed,” missing early AKI. Point-of-care creatinine devices remain cost-prohibitive.

These factors create a cascade of failure: late detection → delayed intervention → irreversible injury → death. Table 5’s mortality gradient by AKI stage (7.1% → 18.8% → 50.0%) tragically illustrates this progression.

Contextualizing KDIGO in Resource-Limited Settings

We applied modified KDIGO criteria, omitting urine output in general ward patients due to practical constraints. While this may underestimate AKI incidence by 10–15% [22], it reflects real-world feasibility. Studies show that serum creatinine-only KDIGO retains reasonable sensitivity in LMICs when baseline SCr is estimated via height-based equations [21].

Critically, our baseline SCr estimation used the updated Schwartz formula ($k=0.413$ for SCr in mg/dL) [12], validated in South Asian children [15]. This avoids misclassification bias common in studies using adult reference ranges. AKI in hospitalized children at Sahara Medical College Hospital is common, frequently severe, and strongly linked to preventable or modifiable factors sepsis, dehydration, nephrotoxicity, and anemia. The high mortality reflects not just disease severity but systemic gaps in timely recognition, diagnostics, and critical care. However, these gaps also represent opportunities. With context-adapted protocols, workforce training, and policy support, the burden of pediatric AKI in Narowal and similar settings across Pakistan can be meaningfully reduced. Our data provide the evidence base needed to catalyze this change.

CONCLUSION

Acute kidney injury affects 15% of hospitalized children at Sahara Medical College Hospital, Narowal, and is independently predicted by sepsis, dehydration, nephrotoxic drug exposure, and anemia. AKI is strongly associated with prolonged hospitalization, need for mechanical ventilation, and a 9-fold increase in in-hospital mortality. These findings highlight the urgent need for context-specific AKI prevention protocols, enhanced early recognition tools, and investment in pediatric critical care resources in resource-limited settings across Pakistan.

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