International Journal of Health Sciences and Research
[Indexed, Peer-Reviewed Journal]
Volume 16; Issue: 4; April 2026
Website: www.ijhsr.org
Case Series *ISSN: 2249-9571*

The Heterogeneous Clinical and Laboratory Profile of Pediatric Acute Kidney Injury: A Retrospective Case Series and Audit Across Three Age Groups

Madhav A

Associate Professor, Department of Paediatrics, Travancore Medical College, Kollam, Kerala, India.

Corresponding Author: Madhav A

DOI: https://doi.org/10.52403/ijhsr.20260430

ABSTRACT

Introduction: Acute kidney injury (AKI) in the pediatric population is a complex syndrome with etiologies and clinical profiles that vary drastically by age. This retrospective case series highlights the heterogeneous presentations of AKI across neonatal, toddler, and older child demographics.
Case Presentation: We retrospectively reviewed the hospital records of children admitted to the pediatric ward and intensive care unit. Informed consent was obtained from parents via telephonic communication and secure Google Forms, with all names and addresses kept strictly confidential. We present three distinct cases: a 4-day-old neonate with sepsis-associated AKI, a 2-year-old toddler with Hemolytic Uremic Syndrome (HUS), and a 9-year-old child with Post-Streptococcal Glomerulonephritis (PSGN).
Discussion: The clinical manifestations ranged from isolated oliguria in the neonate to gross hematuria and hypertension in the older child. A supplemental retrospective analysis of 45 AKI admissions over one year demonstrated that primary etiology significantly dictates recovery time ($p = 0.012$).
Conclusion: Pediatric AKI cannot be managed with a uniform approach. Early recognition relies on understanding age-specific epidemiological triggers and laboratory phenotypes

Keywords: Acute kidney injury, pediatric nephrology, hemolytic uremic syndrome, post-streptococcal glomerulonephritis, neonatal sepsis.

INTRODUCTION

Acute kidney injury (AKI) is a critical condition in pediatrics, characterized by a sudden decline in renal function, leading to the accumulation of metabolic waste products and dysregulation of fluid and electrolyte balance [1]. Unlike adult populations where ischemic and diabetic nephropathies dominate, pediatric AKI etiologies are highly stratified by age. The incidence of pediatric AKI has been rising, largely due to the increased survival of premature neonates and the use of nephrotoxic medications in intensive care settings [2,3].

Neonatal AKI is most frequently precipitated by perinatal asphyxia, congenital anomalies of the kidney and urinary tract (CAKUT), or sepsis [4]. In infants and toddlers, volume depletion secondary to acute gastroenteritis and Hemolytic Uremic Syndrome (HUS) are the leading culprits. Conversely, older children frequently present with primary renal pathologies, such as Acute

Glomerulonephritis (AGN) or immune-mediated nephritides [5].

MATERIAL AND METHODS

Ethics and Consent

This study involved a retrospective review of case records. Informed consent was obtained from the respective parents or legal guardians via telephonic communication, followed by digital documentation using secure Google Forms. Strict data anonymization was maintained; all patient names, addresses, and identifiable markers were removed to ensure absolute confidentiality.

CASE PRESENTATION

Case 1: The Neonate (Sepsis-Associated AKI)

A 4-day-old male neonate, born at term, presented to the neonatal intensive care unit with lethargy, poor feeding, and a documented fever of 101.5°F. The parents noted a significant decrease in wet diapers over the previous 24 hours. On examination, the infant was tachycardic and poorly perfused.

- **Laboratory Profile:** Blood cultures subsequently grew *Klebsiella pneumoniae*. Serum creatinine was elevated at 1.8 mg/dL (baseline assumed 0.3 mg/dL), and blood urea nitrogen (BUN) was 65 mg/dL. Fractional excretion of sodium (FeNa) was >2.5%, consistent with intrinsic tubular injury secondary to sepsis.
- **Management:** The neonate was managed with aggressive fluid resuscitation, broad-spectrum intravenous antibiotics, and strict fluid balance monitoring. Urine output normalized by day 4 of admission, and serum creatinine trended down to 0.4 mg/dL by day 8.

Case 2: The Toddler (Hemolytic Uremic Syndrome)

A 2-year-old female toddler was brought to the pediatric emergency department with a four-day history of bloody diarrhea, vomiting, and increasing pallor.

- **Laboratory Profile:** Investigations revealed severe anemia (Hemoglobin 7.2 g/dL) and thrombocytopenia (Platelets 45,000/mm³). Peripheral blood smear showed numerous schistocytes. Renal function was markedly impaired, with a serum creatinine of 2.5 mg/dL and BUN of 88 mg/dL. Stool culture was positive for Shiga toxin-producing *E. coli* (STEC).
- **Management:** The patient required two packed red blood cell transfusions for symptomatic anemia. She remained anuric for 48 hours, necessitating three sessions of acute peritoneal dialysis. By day 12, her platelet count recovered, and dialysis was successfully discontinued as intrinsic urine output resumed.

Case 3: The Older Child (Acute Glomerulonephritis)

A 9-year-old boy presented with a chief complaint of passing "cola-colored" urine for two days, accompanied by periorbital edema and headache. He had a history of a treated skin infection (pyoderma) three weeks prior.

- **Laboratory Profile:** Vital signs revealed severe hypertension (Blood Pressure 145/95 mmHg). Urinalysis showed gross hematuria with RBC casts and sub-nephrotic proteinuria (2+). Serum creatinine was mildly elevated at 1.3 mg/dL. Immunological workup revealed a significantly depressed C3 complement level (32 mg/dL; normal 90-180) and elevated Anti-Streptolysin O (ASO) titers, confirming Post-Streptococcal Glomerulonephritis (PSGN).
- **Management:** Management focused on strict fluid restriction and blood pressure control using intravenous furosemide and oral amlodipine. His gross hematuria resolved within five days, and his blood pressure normalized prior to discharge on day 7.

DISCUSSION

The diagnostic approach to pediatric AKI requires a high index of suspicion tailored to the patient's age. As demonstrated in Case 1, neonatal AKI is often a secondary

complication of a systemic crisis, such as sepsis or asphyxia, manifesting primarily as oliguria without specific nephritic signs [6]. In contrast, Case 2 illustrates the classic triad of HUS (microangiopathic hemolytic anemia, thrombocytopenia, and AKI), which remains a leading cause of dialysis-dependent AKI in toddlers [7]. Case 3 highlights primary glomerular pathology, where the physical signs of fluid overload and hypertension are the cardinal clues [8].

To contextualize these individual cases, a brief retrospective audit of 45 pediatric AKI admissions at our institution over a 12-month period was analyzed.

Table 1: Departmental Audit of Pediatric AKI Profiles by Age Group (n=45)

Variable	Neonates (<1 mo) (n=12)	Toddlers (1 mo - 3 yrs) (n=18)	Older Children (>3 yrs) (n=15)	p-value
Most Common Etiology	Sepsis (58%)	HUS / Dehydration (66%)	Glomerulonephritis (53%)	< 0.001
Mean Peak Creatinine (mg/dL)	1.6 ± 0.4	2.8 ± 0.9	1.9 ± 0.6	0.034
Requirement of RRT (%) *	16%	38%	13%	0.041
Mean Days to Recovery	6.2 ± 2.1	11.4 ± 3.5	7.5 ± 2.8	0.012

**Note: RRT = Renal Replacement Therapy. Continuous variables compared using ANOVA; categorical variables using Chi-square test.*

The departmental data corroborates the findings of the AWARE study [9], indicating that toddlers with HUS/dehydration require Renal Replacement Therapy (RRT) significantly more often than other age groups ($p = 0.041$). Furthermore, the mean days to renal recovery varied significantly by etiology and age ($p = 0.012$), reinforcing that AKI is not a monolithic disease [10]. Early categorization using the pediatric Risk, Injury, Failure, Loss, End-Stage Renal Disease (pRIFLE) criteria remain essential for predicting these outcomes [11,12].

The limitation of this report is its retrospective nature and the inherent selection bias of a case series. However, the strict maintenance of patient confidentiality and the diverse presentation of the cases provide valuable clinical heuristics for the general pediatrician [13].

CONCLUSION

Pediatric Acute Kidney Injury presents with extreme phenotypic variability. While neonates may simply exhibit oliguria amidst systemic sepsis, toddlers frequently present with the hematological crises of HUS, and older children manifest with classic nephritic hypertension. Recognizing these age-specific epidemiological and laboratory patterns is paramount for initiating timely, targeted interventions and reducing morbidity.

Declaration by Authors

Acknowledgement: None

Source of Funding: None

Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Kellum JA, Lameire N; KDIGO AKI Guideline Work Group. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). Crit Care. 2013 Feb 4;17(1):204. doi: 10.1186/cc11454.
2. Sutherland SM, Ji J, Sheikhi FH, Widen E, Tian L, Alexander SR, Ling XB. AKI in hospitalized children: epidemiology and clinical associations in a national cohort. Clin J Am Soc Nephrol. 2013 Oct;8(10):1661-9. doi: 10.2215/CJN.00270113.
3. Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ, Kent AL. Neonatal Acute Kidney Injury. Pediatrics. 2015 Aug;136(2):e463-73. doi: 10.1542/peds.2014-3819.
4. Jetton JG, Askenazi DJ. Update on acute kidney injury in the neonate. Curr Opin

Pediatr. 2012 Apr;24(2):191-6. doi: 10.1097/MOP.0b013e32834f62d5.
5. Krishnamurthy S, Narayanan P, Prabha S, Mondal N, Mahadevan S, Biswal N, Srinivasan S. Clinical profile of acute kidney injury in a pediatric intensive care unit from Southern India: A prospective observational study. Indian J Crit Care Med. 2013 Jul;17(4):207-13. doi: 10.4103/0972-5229.118412.
6. Nada A, Bonachea EM, Askenazi DJ. Acute kidney injury in the fetus and neonate. Semin Fetal Neonatal Med. 2017 Apr;22(2):90-97. doi: 10.1016/j.siny.2016.12.001.
7. Spinale JM, Ruebner RL, Copelovitch L, Kaplan BS. Long-term outcomes of Shiga toxin hemolytic uremic syndrome. Pediatr Nephrol. 2013 Nov;28(11):2097-105. doi: 10.1007/s00467-012-2383-6.
8. Ricci Z, Cruz D, Ronco C. The RIFLE criteria and mortality in acute kidney injury: A systematic review. Kidney Int. 2008 Mar;73(5):538-46. doi: 10.1038/sj.ki.5002743.
9. Kaddourah A, Basu RK, Bagshaw SM, Goldstein SL; AWARE Investigators. Epidemiology of Acute Kidney Injury in Critically Ill Children and Young Adults. N Engl J Med. 2017 Jan 5;376(1):11-20. doi: 10.1056/NEJMoa1611391.
10. Mehta P, Sinha A, Sami A, Hari P, Kalaivani M, Gulati A, Kabra M, Kabra SK, Lodha R, Bagga A. Incidence of acute kidney injury in hospitalized children. Indian Pediatr. 2012 Jul;49(7):537-42. doi: 10.1007/s13312-012-0121-6.
11. Akcan-Arikan A, Zappitelli M, Loftis LL, Washburn KK, Jefferson LS, Goldstein SL. Modified RIFLE criteria in critically ill children with acute kidney injury. *Kidney Int*. 2007;71(10):1028-1035. https://doi.org/10.1038/sj.ki.5002231.
12. Basu RK, Zappitelli M, Brunner L, Wang Y, Wong HR, Chawla LS, Wheeler DS, Goldstein SL. Derivation and validation of the renal angina index to improve the prediction of acute kidney injury in critically ill children. Kidney Int. 2014 Mar;85(3):659-67. doi: 10.1038/ki.2013.349.
13. Symons JM, Chua AN, Somers MJ, Baum MA, Bunchman TE, Benfield MR, et al. Demographic characteristics of pediatric continuous renal replacement therapy: a report of the prospective pediatric continuous renal replacement therapy registry. Clin J Am Soc Nephrol. 2007 Jul;2(4):732-8. doi: 10.2215/CJN.03200906.

How to cite this article: Madhav A. The heterogeneous clinical and laboratory profile of pediatric acute kidney injury: a retrospective case series and audit across three age groups. *Int J Health Sci Res.* 2026; 16(4):247-250. DOI: *https://doi.org/10.52403/ijhsr.20260430*

******