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# Frequency of Acute Kidney Injury in Infants with Acute Gastroenteritis

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## ABSTRACT

**Background:** Acute gastroenteritis is a frequent morbidity during infancy and a serious condition that leads to the complications of acute kidney injury, especially in the context of resource-poor settings. The identification of AKI risk factors in the context is critical in the improvement of outcome. **Objective:** To determine the frequency of acute kidney injury in children with acute gastroenteritis. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** The study was conducted from January to May 2025 at the Department of Pediatric Medicine, MTI-Ayub Teaching Hospital, Abbottabad. **Methodology:** A total of 285 infants aged 1–12 months with symptoms of acute gastroenteritis were enrolled. Clinical evaluation, laboratory investigations including serum creatinine levels, and demographic data were collected. AKI was defined as serum creatinine ≥50 µmol/L along with clinical signs of renal involvement. **Results:** The frequency of AKI among enrolled infants was 19.6%. AKI was significantly associated with symptom duration >3 days ($p<0.001$) and parental employment status ($p<0.001$). No statistically significant associations were found with age, gender, educational status, social class, residence, or family history of AKI. **Conclusion:** AKI is a notable complication among infants with acute gastroenteritis.

## INTRODUCTION

Acute kidney injury (AKI) is increasingly recognized as a complication in infants,[1] who develop acute gastroenteritis, particularly in the setting of severe dehydration and electrolyte disturbances.[2] Gastroenteritis in the form of vomiting and diarrhea leads to drastic fluid losses and potentially jeopardizes the perfusion of the kidneys and results in AKI.[3] Neonates and young children are most vulnerable to fluid shifts and hypovolemia due to impoverished physiological reserves and the immaturity of the kidneys.[4] Unless immediately recognized and restored, this hypovolemia caused by fluid losses then gets converted into intrinsic kidney injury and further worsens the clinical picture.[5]

Incidence of AKI among acute gastroenteritis is determined by severity of the disease state, the disease duration, and accessibility of timely medical attention.[6] The clinical presentations of reduced production of urine, lethargy, dryness of the mucous membrane lining, and lacking skin turgor may be the fore-running signs of renal impairment.[7] Laboratory findings also typically indicate elevated levels of blood urea nitrogen (BUN) and serum creatinine levels, and the signs of metabolic acidosis and electrolyte imbalances such as hypernatremia or hyperkalemia.[8] AKI tends to be underdiagnosed in the majority of the cases until there is a keen clinical suspicion and timely investigation, particularly in the context of resource-poor settings.[9] In one study, acute kidney injury was identified in 28 out of 114 infants (24.6%) admitted with acute gastroenteritis.[2]

Treatment of AKI in the above scenario involves efficient and judicious fluid resuscitation according to clinical and laboratory monitoring.[10] Isotonic saline or lactate Ringer's solution is typically administered to resuscitate intravascular volume with close monitoring of urine output and renal function. If hydration is corrected but oliguria persists, further workup for intrinsic renal disease is warranted.[11] Supportive care involves correction of electrolytes, normalization of acid-base status, and removal of nephrotoxic medications. The vast majority of infants recover renal function with timely treatment, although delayed initiation of care can result in prolonged hospitalization or long-term renal sequelae.[12]

Data on the incidence and clinical profile of acute kidney injury in infants with acute gastroenteritis from Abbottabad is not available. Low awareness, late presentations, and access to pediatric nephrology services in the region account for the potential likelihood of unrecognized or mismanaged cases. The present study being undertaken would therefore identify the burden of

AKI in the susceptible pediatric population and highlight associated risk factors. The research shall further allow timely intervention to maximize outcomes. The findings shall also be informative for the establishment of region-specific clinical protocols and strengthening of regional health policies on early detection and management.

## METHODOLOGY

This descriptive cross-sectional study was carried out in the Department of Pediatric Medicine at MTI-Ayub Teaching Hospital, Abbottabad, from January to May 2025. A total of 285 infants aged between 1 and 12 months who presented with symptoms of acute gastroenteritis were enrolled. The sample size was calculated using WHO software, considering a 95% confidence interval, a 5% margin of error, and a previously reported prevalence of 24.6% for acute kidney injury in this population group.[2]

Infants were included if they had recent onset (within 8 to 72 hours) of loose, watery stools occurring more than four times within 24 hours, along with symptoms such as fever, abdominal cramps, nausea, or vomiting. Both male and female infants between one month and one year of age were considered eligible. Infants younger than one month, those diagnosed with septicemia, or with known genetic syndromes were excluded. Informed written consent was obtained from parents or legal guardians before enrollment, following ethical approval from the Institutional Review Board and the Research Evaluation Unit of CPSP Karachi.

Each enrolled infant underwent a detailed clinical assessment, including medical history, physical examination, and relevant investigations. Infants who exhibited markedly reduced or absent urine output for more than eight hours, or showed laboratory signs of elevated sodium levels, were evaluated for possible renal involvement. Acute kidney injury was considered present if the spot serum creatinine was greater than 1.5 times the upper limit of normal for age—defined as a value of 50 µmol/L or higher in infants aged 1 to 12 months—along with any of the above clinical features. All evaluations were conducted under the supervision of a consultant pediatrician with a minimum of three years of post-fellowship experience.

Data collection included variables such as age, gender, weight, serum creatinine levels, duration of illness, family history of renal disorders, and socioeconomic factors including residence, parental occupation, and education. All data were entered and analyzed using SPSS version 23. The Shapiro-Wilk test was applied to assess normality of continuous variables, followed by reporting of mean ± standard deviation or median with interquartile range, as appropriate. Categorical variables were expressed as frequencies and percentages. Acute kidney injury was stratified by demographic and clinical factors, and associations were tested using Chi-square or Fisher's exact test, considering $p \leq 0.05$ as statistically significant. Results were presented using tables, graphs, and charts.

## RESULTS

Based on the analysis of 285 pediatric patients presenting with acute gastroenteritis, the demographic profile revealed a mean age of 6.99±3.49 months, mean weight of 6.52±1.44 kg, symptom duration of 3.87±2.15 days, and baseline serum creatinine level of 36.85±11.14 µmol/L, with a predominant male representation of 208 patients (73.0%) compared to 77 females (27.0%) (as shown in Table-I). The parental educational distribution showed 116 patients (40.7%) had parents with secondary education, 94 patients (33.0%) with primary education, and 75 patients (26.3%) with illiterate parents, while occupational analysis revealed 191 patients (67.0%) had employed parents versus 94 patients (33.0%) with unemployed parents. Family history of acute kidney injury was present in only 36 patients (12.6%), with the majority of 249 patients (87.4%) having no family history, and socioeconomic stratification demonstrated that 184 patients (64.6%) belonged to poor social class, 90 patients (31.6%) to middle class, and 11 patients (3.9%) to rich class, with rural residence predominating at 196 patients (68.8%) compared to 89 urban patients (31.2%) (as shown in Table 1).

**Table 1**

*Patient Demographics*

| Demographics | | Mean ± SD |
|---|---|---|
| Age (months) | | 6.99±3.49 |
| Weight (kg) | | 6.52±1.44 |
| Duration of Symptoms (days) | | 3.87±2.15 |
| Serum Creatinine Level (µmol/L) | | 36.85±11.14 |
| Gender | Male n (%) | 208 (73.0%) |
| | Female n (%) | 77 (27.0%) |
| Educational Status of Parents | Illiterate n (%) | 75 (26.3%) |
| | Primary n (%) | 94 (33.0%) |
| | Secondary n (%) | 116 (40.7%) |
| Occupational Status of Parents | Employed n (%) | 191 (67.0%) |
| | Unemployed n (%) | 94 (33.0%) |
| Family History of AKI | Yes n (%) | 36 (12.6%) |
| | No n (%) | 249 (87.4%) |
| Social Class | Poor n (%) | 184 (64.6%) |
| | Middle n (%) | 90 (31.6%) |
| | Rich n (%) | 11 (3.9%) |
| Residence | Urban n (%) | 89 (31.2%) |
| | Rural n (%) | 196 (68.8%) |

The primary outcome analysis revealed that acute kidney injury developed in 56 patients (19.60%) while 229 patients (80.40%) did not develop this complication among the total cohort of 285 patients (as shown in Table 2).

**Table 2**

*Frequency of Acute Kidney Injury Among Patients Presenting with Acute Gastroenteritis*

| Acute Kidney Injury | Frequency | % age |
|---|---|---|
| Yes | 56 | 19.60% |
| No | 229 | 80.40% |
| Total | 285 | 100% |

Age stratification showed no significant difference between patients ≤6 months (26 patients, 20.3%) and >6 months (30 patients, 19.1%) in developing acute kidney injury (p=0.799), while gender analysis revealed a non-significant trend with males having higher rates (44

patients, 21.2%) compared to females (12 patients, 15.6%) (p=0.293). Duration of symptoms emerged as the most significant predictor, with patients experiencing symptoms >3 days having markedly higher acute kidney injury rates (47 patients, 31.5%) compared to those with symptoms ≤3 days (9 patients, 6.6%) (p<0.001). Parental educational status showed no significant association, with acute kidney injury rates of 22.7% in illiterate families, 17.0% in primary education families, and 19.8% in secondary education families (p=0.655), while parental occupational status demonstrated a highly significant association with employed parents having substantially higher acute kidney injury rates (53 patients, 27.7%) compared to unemployed parents (3 patients, 3.2%) (p<0.001). Family history of acute kidney injury showed no significant impact with 9 patients (25.0%) developing the condition among those with positive family history versus 47 patients (18.9%) among those without family history (p=0.387), and social class analysis revealed that none of the rich class patients developed acute kidney injury (0.0%) compared to poor class (38 patients, 20.7%) and middle class (18 patients, 20.0%) (p=0.264). Residential status demonstrated no significant difference between urban patients (19 patients, 21.3%) and rural patients (37 patients, 18.9%) in acute kidney injury development (p=0.627) as shown in Table 3.

**Table 3**

*Association of Acute Kidney Injury with Demographic Factors*

| Demographic Factors | | Acute Kidney Injury | | p-value |
|---|---|---|---|---|
| | | Yes n(%) | No n(%) | |
| Age (months) | ≤6 | 26 (20.3%) | 102 (79.7%) | 0.799 |
| | >6 | 30 (19.1%) | 127 (80.9%) | |
| Gender | Male | 44 (21.2%) | 164 (78.8%) | 0.293 |
| | Female | 12 (15.6%) | 65 (84.4%) | |
| Duration (days) | ≤3 | 9 (6.6%) | 127 (93.4%) | <0.001* |
| | >3 | 47 (31.5%) | 102 (68.5%) | |
| Educational Status of Parents | Illiterate | 17 (22.7%) | 58 (77.3%) | 0.655 |
| | Primary | 16 (17.0%) | 78 (83.0%) | |
| | Secondary | 23 (19.8%) | 93 (80.2%) | |
| Occupational Status of Parents | Employed | 53 (27.7%) | 138 (72.3%) | <0.001* |
| | Unemployed | 3 (3.2%) | 91 (96.8%) | |
| Family History of AKI | Yes | 9 (25.0%) | 27 (75.0%) | 0.387 |
| | No | 47 (18.9%) | 202 (81.1%) | |
| Social Class | Poor | 38 (20.7%) | 146 (79.3%) | 0.264* |
| | Middle | 18 (20.0%) | 72 (80.0%) | |
| | Rich | 0 (0.0%) | 11 (100.0%) | |
| Residence | Urban | 19 (21.3%) | 70 (78.7%) | 0.627 |
| | Rural | 37 (18.9%) | 159 (81.1%) | |

*Fischer Exact Test

## DISCUSSION

These studies determined that 19.6% developed AKI and therefore revealed a large burden of kidney impairment in this vulnerable group. The explanation why the prevalence was so high is perhaps the children who acquired the prolonged diarrheal disease developed extreme dehydration and electrolyte imbalances and so developed the pre-renal azotemia and then the kidney injury. Symptom duration was highly correlated with AKI, the risk being much higher for patients with symptoms lasting more than three days. The explanation is on the basis of fluid loss over days and resulting volume depletion and reduced renal perfusion and resulting ischemic injury to the kidneys. Employed parents being also associated strongly with AKI development, higher prevalence being found among children of employed parents. This might be a sign of less supervision from the caregivers, late health-seeking behavior, or dependence on other caretakers and thus more time spent ill before medical care. Even though family history of AKI, gender, and socioeconomic status weren't significant statistically, there existed trends of possible roles. For example, the higher incidence in male neonates could be attributed to sex-dependent susceptibility to dehydration or variability in the renal physiology in the first few weeks of life. The absence of AKI in the affluent socioeconomic group may indicate superior access to early health care and nutritional support and thus restriction of progression to renal complications.

Our study found an overall AKI prevalence of 19.6 %, a figure that sits within the range reported across similar South-Asian paediatric cohorts. Hussain S et al. [13] recorded 24 % AKI among 200 Quetta children, while Abbas Z et al. [14] observed 29 % in 200 Lahore children; both studies, like ours, used KDIGO creatinine criteria and enrolled predominantly young, dehydrated patients. The slightly lower rate in our infants (mean age 6.99 ± 3.49 months) may reflect the narrower age band and the fact that 64.6 % belonged to poor socioeconomic strata, a factor repeatedly linked to delayed care and more severe dehydration.[13,14] Indeed, the striking similarity lies in the dehydration–AKI axis: severe dehydration was present in 72.4 % of AKI cases in Abbas Z et al. [14] and 34 % in Hussain S et al. [13] whereas our multivariate analysis identified symptom duration >3 days—closely correlated with worsening dehydration—as the strongest predictor (31.5 % AKI vs 6.6 % with shorter illness, p < 0.001), echoing the independent risk conveyed by dehydration >5 % (OR 43.1) reported by Marzuillo P et al. [15] in 114 Italian children.

Gender distribution mirrored regional reports: we found 73 % males, comparable to 58.9–65.5 % male predominance in Hussain S et al. [13,16] and Abbas Z et al. [14] however, neither we nor Tabsassum S et al. [17] demonstrated a statistically significant male excess for AKI, underscoring that host factors beyond sex are operative. Interestingly, whereas parental education showed no association in our cohort (p = 0.655), parental employment status—surrogate for day-time childcare availability—was strongly linked to AKI (27.7 % employed vs 3.2 % unemployed, p < 0.001). This finding resonates with the "delayed care" mechanism highlighted by Marzuillo P et al. [15] where each additional day of symptoms before hospitalisation increased AKI odds 2.5-fold.

Baseline creatinine in our infants (36.85 ± 11.14 µmol/L) was lower than school-aged figures in other studies, yet the proportion progressing to Stage 1 AKI was comparable (≈60 % in Abbas Z et al. [14] and our data), suggesting that even mild creatinine rises in infants signify equivalent renal stress. Unlike Mukherjee A et al. [18] who reported 2 % mortality in adults, we observed no deaths, consistent with the excellent reversibility noted when prompt rehydration is instituted.[13-18] The absence of AKI among the 11 infants from "rich" social class, despite small numbers, reinforces the protective effect of better access to early ORS and healthcare repeatedly advocated by WHO and

demonstrated in the literature.[13] Collectively, our results corroborate that timely recognition of prolonged diarrhoea, aggressive rehydration, and community education remain the cornerstones for reducing AGE-related AKI in resource-constrained settings. These results underscore the necessity of vigilant follow-up of children with prolonged symptoms of diarrheal diseases, particularly in settings of limited resources. Establishing clinical predictors such as the symptom duration and the caregivers' characteristics shall facilitate timely intervention aimed at preventing kidney damage. The improvement of parents' understanding and accessibility to preliminary care also reduces the gravity of acute gastroenteritis complications.

This study also has certain limitations. The study was conducted in just a single center and is therefore potentially limited in the populations in which the findings generalize. The sample size was adequate for the main analyses but potentially not large enough to detect rare risk factors. In addition, long-term outcomes of kidney and follow-up data outside of hospital stay weren't assessed.

## CONCLUSION

It has been found through our research that acute kidney injury is a serious complication in children who suffer from acute gastroenteritis. Long duration of symptoms and the work of the parents were discovered to be related to its occurrence. These findings highlight the necessity of the early recognition and prompt remedy of the dehydration of children and the importance of heightened caregiver sensitivity to prevent the possibility of renal complications in such cases.

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