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A study of 50 patients with acute kidney injury due to gastroenteritis

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Abstract: Background: With an estimated 13.3 million cases each year, acute kidney injury (AKI) become a problem for world health. India has a high prevalence of AKI following volume depletion from gastrointestinal fluid loss. Due to poor socioeconomic situations, limited access to care, lack of awareness of personal cleanliness, crowding, and climatic factors that encourage the spread of infection, diarrheal illnesses are widespread in India. AKI following gastroenteritis is probably caused by a lack of medical facilities in rural areas and a delay in treating dehydration. Therefore, in order to come up with solutions to this issue, it is necessary to comprehend the disease's clinical spectrum.
Materials and Methods: This is a prospective observational study conducted on 50 patients with AKI due to Acute Gastroenteritis admitted to Narayana medical college & hospital, Nellore, Andhra Pradesh, over a period of 1 year. The diagnosis of acute kidney injury was used when there was evidence of kidney injury in some clinical settings without any kidney disease history. The term acute kidney injury was used when there was a rise in Serum creatinine $\geq 44\mu mol/L(\geq 0.5mg/dL)$ and the history of decreased urine output of less than $0.5ml/kg/hr$ for more than 6hrs. The criteria used for AKI in the study was Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease (RIFLE) criteria (given by Acute Dialysis Quality Initiative Group 2004) and is as follows.
Results: Pre Renal Azotemia, which occurred in 58% of cases, was followed by Acute Tubular Necrosis, which occurred in 42% of cases in this study. The pre-renal group's mean age was $49.3 + 5.66$ years, while the ATN group's was $48.6 + 7.40$ years. The mean age of those who survived was 46.73 4.75, while that of those who did not survive was 65 6.034. On admission, Baseline creatinine with a mean of $3.032 + 0.37mg/dl$. It was $2.70 + 0.29$ and 3.48 ± 0.77 inpre-renal and Acute Tubular Necrosis (ATN) groups. The mean peak creatine was $4.73 + 0.48mg/dl$. It was 4.13+ 0.59 in pre-renal and 5.56$\pm$ 0.66 in ATN groups, respectively. The Mean creatinine at the time of discharge $2.87 \pm 0.39mg/dl$. The mean peak creatinine was 2.42 ± 0.33 in pre-renal & 3.48+0.42 in ATN groups (In survivors) and 4.11$\pm$ 0.79 in non-survivors.
Conclusion: Replacement of lost fluids, correction of electrolyte imbalances, and delivery of the proper antibiotics made up the course of treatment. Due to the frequent incidence of hypokalemia, ARF brought on by gastroenteritis differs from other ARF and has a better prognosis. An significant electrolyte disruption in AKI brought on by gastroenteritis is hypokalaemia. It was determined that the primary factor leading to death in AKI caused by gastroenteritis is septicemia.

Keywords: Acute kidney injury; Gastroenteritis; Creatinine.

1. Introduction

With an estimated 13.3 million cases each year, acute kidney injury (AKI) become a problem for world health. In underdeveloped nations, there are 11.3 million cases every year. Globally, it results in 1.7 million deaths annually; 1.4 million of those deaths occur in low- and middle-income nations. AKI affects 15 to 20% of hospitalised individuals in wealthy nations, whereas it affects 25 to 30% of those in underdeveloped nations [1].

Data on the prevalence, causation, and prognosis of the condition are not entirely known. Acute diarrheal illness, malaria, leptospirosis, snakebites, insect stings, intravascular hemolysis brought on by septicemia, and chemical poisonings (copper sulphate, Vasmol, and pregnancy) are the most typical causes of AKI in India. They make up 40% of all Aki in India [2–4].

In the literature, the range of renal failure in the adult population and the variables indicating a bad outcome are not clearly characterised. Planning efforts to prevent AKI and give priority to scarce and expensive therapy modalities is made easier when risk factors and poor prognostic markers are identified in these patients, especially in developing nations.

Different regions of India have different etiologies, courses, and outcomes. According to [5], the volume depletion caused by gastrointestinal fluid loss (35.2%) was the primary etiological factor for acute renal failure (ARF) that was seen. Similar findings were made by Mahajan *et al.*, [6], who reported that volume depletion was the most frequent cause of ARF, and by Jayakumar *et al.*, who discovered that acute diarrheal illness was the most frequent medical cause of ARF [5,6].

India has a high prevalence of AKI following volume depletion from gastrointestinal fluid loss. Due to poor socioeconomic situations, limited access to care, a lack of awareness of personal cleanliness, crowding, and climatic factors that encourage the spread of infection, diarrheal illnesses are widespread in India. AKI following gastroenteritis is probably caused by a lack of medical facilities in rural areas and a delay in treating dehydration.

One of the main causes of AKI in the tropics is decreased intravascular volume in patients with viral gastroenteritis, bacillary dysentery, cholera, and food poisoning [7]. This study was carried out to investigate the clinical and biochemical correlation and outcome of AKI related to gastroenteritis because diarrhoea is one of the common causes of AKI in tropical regions.

Hence, an understanding of the disease's clinical spectrum is needed to devise methods to improve the outcome due to this problem.

2. Materials and Methods

This is a prospective observational study conducted on 50 patients with AKI due to Acute Gastroenteritis admitted to Narayana medical college & hospital, Nellore, Andhra Pradesh, from Novmeber 2021 to October 2022.

2.1. Inclusion Criteria

1. All patients with AKI due to Gastroenteritis
2. All patients of either sex and age above 18 years
3. The serum creatinine level must rise gradually over 48 hours to $0.3mg/dl(26.5mmol/l)$, reach 1.5 times baseline in 7 days, or decline by 0.5 $ml/kg/h$ for 6 hours.

2.2. Exclusion Criteria

1. AKI brought on by conditions other than gastroenteritis is not included.
2. Patients with acute gastroenteritis who also have chronic kidney disease (CKD).

The diagnosis of acute kidney injury was used when there was evidence of kidney injury in some clinical settings without any kidney disease history. The term acute kidney injury was used when there was a rise in Serum creatinine $\geq 44\mu mol/L(\geq 0.5mg/dL)$ and the history of decreased urine output of less than $0.5ml/kg/hr$ for more than 6 hrs.

The criteria used for AKI in the study was RIFLE criteria (given by Acute Dialysis Quality Initiative Group 2004) and is as follows.

There are many causes of acute kidney injury, of which we used acute gastroenteritis in this study.

- Acute Gastroenteritis- History of Passage of abnormally liquid or unformed stools at an increased frequency stool weight $> 200g/d < 2$ weeks in duration.

3. Diagnosis of Azotemia

Laboratory evaluation for azotemia includes Blood Urea Nitrogen BUN/Cr, urinary sodium (Na), urea, urine osmolality (Ur Osmo), urinalysis (UA).

3.1. BUN greater than 21 mg/dL

Significant findings for prerenal azotemia

- BUN: Cr ratio greater than 20:1
- Fractional excretion of sodium (FeNa) less than 1, fractional excretion of urea (FeUr) less than 35%
- Urine osmolality 500 $mOsm/kg$
- UA can show hyaline casts [5]

3.2. Diagnosis of ATI or acute tubular necrosis (ATN)

ATI was synonymous with acute tubular necrosis (ATN). However, frank tubular epithelial necrosis is only 1 histologic pattern observed in clinical ATI and may reflect particular etiologies and/or severity of injury. The diverse pathologic changes are often dynamic and may differ by the timing of kidney tissue procurement and etiology.

3.3. Data collection

The length of the gastroenteritis and the interval between the beginning of GE and the development of renal failure were noted. At the time of admission, the patient's level of hydration was noted. Daily tests and records were made for blood urea, serum creatinine, and electrolytes. Other laboratory tests were performed, including the complete blood count (CBC), the erythrocyte sedimentation rate (ESR), the human immunodeficiency virus (HIV), the blood glucose level, the total leukocyte count and differential count, the erythrocyte sedimentation rate, and the liver function test.

4. Statistics

Frequency and percentage were used to summarise categorical variables. Mean, SD, and Median were used to represent continuous variables. For all 2 X 2 tables, the chi-square test and Fisher's Exact test were used to determine the association between categorical independent and dependent variables. Small counts rendered the p-value of the Chi-Square test invalid. A p-value of 0.05 or lower indicates statistical significance in all two-sided tests of significance. Version 22.0 of the Statistical Package for the Social Sciences was used to analyse the data.

5. Results

There were 50 Patients with Acute Kidney Injury due to gastroenteritis were included in the study.

Table 1. Age group

Age group	Number			Percentage
	Male	Female	Total	
<24	4	1	5	10.0
25-34	8	2	10	20.0
35-44	5	3	8	16.0
45-54	2	4	6	12.0
55-64	6	4	10	20.0
>65	7	4	11	22.0
Total	32	18	50	100%

They were between the ages of 20 and 70, with a mean age of 49.02 years. The maximum incidence was seen in the age group $>$ 65 years, followed by 25 to 34 years & 55-64 years, respectively in Table 1.

The mean age of the pre-renal group was 49.3 $\pm$ 5.66 years, and in the ATN group, it was 48.6 $\pm$ 7.40. The mean age in survivors was 46.73 $\pm$ 4.75, and that of non-survivors was 65 $\pm$ 6.034. Pre Renal Azotemia,

Table 2. Mean Age of pre-renal ATN patients and in survivors vs non-survivors

Category	Mean Age (Years)	P-value
Males	47.12 ± 6.03	0.156(ns)
Females	52.38 ± 5.21	
Pre Renal Azothemia (29)	49.3 ± 5.66	0.16(ns)
ATN (21)	48.6 ± 7.40	
Survivors	46.73 ± 4.75	0.21(ns)
Non-Survivors	65 ± 6.034	

which was the most common kind of renal failure in this study, was followed by Acute Tubular Necrosis, 42% in Table 2.

Table 3. Patients and dehydration levels and outcome

Severity	No	Survivours	Non-survivors
Mild	17	17	0
Moderate	20	19	1
Severe	13	6	7
Fluid Overload	3	1	2
No dehydration	-	-	-

In Table 3, 20 (40%) of patients had moderate dehydration. 17 patients had mild dehydration, 13 patients had severe dehydration, andfluid Overload was observed in 3 patients. The majority of non-survivours belong to the group of moderate and severe dehydration.

Table 4. Time between the beginning of GE and the emergence of ARF

Category	The interval between onset of GE and the development of ARF
Pre Renal	4±0.23
ATN	5.19 ± 0.68
Survivors	3.71±0.40
Non-Survivors	5.75±0.88

The mean interval between the onset of GE and the development of ARF was 3.0 ± 2.9 days. It was 3.71 + 0.40 days in survivors and 5.75 + 0.88 days in non-survivors. In the pre-renal group, it was 4 + 0.23, and ATN was 5.19 + 0.68 days in Table 4.

In Table 5, On admission, Baseline creatinine with a mean of $3.032 + 0.37 mg/dl$. It was 2.70 + 0.29 and 3.48±0.77 inpre-renal and ATN groups. Themean peakcreatinewas $4.73 + 0.48 mg/dl$. It was 4.13 + 0.59 in pre-renal and 5.56± 0.66 in ATNgroups, respectively. The Mean creatinine at the time of discharge 2.87 ± 0.39 mg/dl. The mean peak creatinine was 2.42 ± 0.33 in pre-renal & 3.48 + 0.42 in ATN groups (In survivors) and 4.11± 0.79 in non-survivors.

In Table 6, At admission, the urea levels ranged between 69 to 251 mg/dl with a mean of 120.44 ± 18.13 mg/dl. The peak meanurea level was observed as 153.76 ± 12.29 mg/dl. At the time of discharge, the urea levels ranged between 35 to 181, with a mean of 86.28 ± 9.07mg/dl. The mean peak urea levels in survivors were175.0 ± 65.5, and in non-survivors were 201.75 ± 13.92 mg/dl.

6. Discussion

The glomerular filtration rate rapidly decreases and nitrogenous waste products like blood urea nitrogen and creatinine are retained in the body when someone has AKI, a syndrome. One of the frequent and serious syndromes seen in clinical practise is AKI. Gastroenteritis is an inflammation of the small intestine and stomach that causes diarrhoea, vomiting, and nausea. In India, diarrheal illnesses constitute a significant public health issue.

Furthermore, Pre-Renal Azotaemia 58%, followed by acute Tubular Necrosis in 42%.This is not similar to a study by Shah *et al.*, [8], that reported Acute Tubular Necrosis in 54% followed by Pre-Renal Azotaemia 46%.

Table 5. Mean creatinine concentrations at baseline, peak, and discharge

Creatinine (Mean)	Overall Patients (0n admission)	Pre-renal	ATN	Non-survivors
Baseline	3.03 ± 0.37	2.70 ± 0.29	3.48±0.77	4.7±1.25
Peak	4.73 ± 0.48	4.13±0.59	5.56±0.66	7.36±0.52
At the time of Discharge	2.87 ± 0.39	2.42±0.33	3.48±0.42	4.11±0.79

Table 6. Mean blood urea levels at time of discharge, baseline, and peak

Blood Urea	Overall Patients (0n admission)	Pre-renal AKI	ATN	Non-survivors
Baseline	120.44±18.13	126.41 ± 15.52	112.19±24.94	177.75+56.36
Peak	153.76±12.29	139.10 ± 14.72	174 ± 17.62	201.75+13.92
At the time of Discharge	86.28± 9.07	78.93+13.44	96.42+12.97	133+16.50

Moreover, 42% of patients were in the age group of $55- > 65$ years, 36% of patients were in the age group of 25-44 years. This differs from studies of Feest *et al.*, [9], where the Majority of the people were more than 60 yrs, and 48% of ARF cases occurred in patients over 65 yrs in a study by Liaño *et al.*, [10]. This implies the occurrence of AKI more in younger age groups, also observed in other studies. This is mostly related to factors encountered in the environment and exposure due to work-related activities [11].

A study by Pajai *et al.*, [12] demonstrated the major complications noted were hypotension in 32 patients (60.4%), sepsis in 29 patients (54.7%), and multiorgan failure in 9 patients (16.9%), encephalopathy in 4 patients (7.5%), and ARDS 3 patents (5.66%). Inbanathan *et al.*, [13] recorded hypovolemic shock (61%), anemia (19%), pulmonary edema (14%) metabolic encephalopathy (7%) as complications.

Twenty patients in the ATN group saw improvements after receiving conservative care. The ATN group's 34 patients required hemodialysis. None of the 46 patients in the Pre-renal group required hemodialysis since they all responded to conservative treatment. Sixty-six of the 82 patients who lived made improvements with conservative therapy, while sixteen needed hemodialysis.

A study by Shah *et al.*, [8] reported at admission. The mean urea concentration was 150.51 mg/dl with a range of 30 to 401 mg/dl. A mean peak urea level of 166.24 96.14 was recorded. At the time of discharge, the mean urea level was 81.89 61.92. The mean baseline urea level in survivors was 134.75 88.79, while it was 222.32 95.35 in non-survivors. Hyponatremia (145 meq/L) was present in 27 patients (27%) of whom 3 were admitted and the rest were hospitalized.

In Mc Murray's study, 74% of ATN patients had infections. In their study, the urinary tract was the site of infection most frequently (20%), and 7 patients (14%) had septicemia. The incidence of oliguric renal failure reported by Anderson *et al.*, [14] was similar (9%) in this case. In our study, UTI (%) and the lungs were the most commonly infected areas, and 11 patients developed septicemia.

In our study, the most common complication was septicemia was observed. Cardiovascular complications were 43% in Minuth *et al.*, 13 study [15]. Neurological complications were 38%, and Pulmonary complications were 28% in Anderson *et al.*, [14] 12 study.

Oliguria, persistent hypotension, and coma were all found to be substantially linked with greater mortality in our study. In our study, mortality was 16% overall, with the ATN group accounting for the majority of deaths. It is considerably lower than in earlier research. In the prerenal and intrinsic groups of ARF, it was 10.3% and 23.8%, respectively; in a research by Kaufman *et al.*, [16] 60% mortality in the ATN group and 45% overall mortality were reported.

In the pre-renal and ATN groups, the time from the onset of gastroenteritis and the development of AKI was practically comparable, and no statistical difference was found. Age alone has been linked in certain studies to the outcome of AKI, however other investigations have found that age alone is not a predictor of AKI. Given that our study only included a small number of patients who fell within a specific age range, it is not reasonable to draw the conclusion that age and sex are independent predictors of the outcome of AKI.

In our study, there were 15 patients with non-oliguric renal failure (30%), and none of them passed away. The fact that 10 of the 42 oliguric and anuric patients died compared to 10 of the 15 non-oliguria patients suggests that the urine output may be one of the predictive indicators for AKI brought on by gastroenteritis.

7. Limitation

This is a single-center study, and the limited number of patients. Despite the limited sample size, however, the post hoc power of our study is acceptable. Another limitation was that it was not possible to use the KDIGO urinary output criteria, which could have determined underestimation of AKI prevalence.

8. Conclusion

One of the main causes of AKI, gastroenteritis, accounted for 16% of all patient deaths who had AKI in our study. The 50 patients had an ATN diagnosis in 42% of cases and pre-renal azotemia in 58% of cases. 30% of the patients weren't oliguric at admission, 20% had anuria, and 50% of the patients had oliguric symptoms. The most frequent complication, which affected 8 patients, was septicemia. All of them passed away. Due to the frequent incidence of hypokalemia, ARF brought on by gastroenteritis differs from other ARF and has a better prognosis. An significant electrolyte disruption in AKI brought on by gastroenteritis is hypokalaemia. It was determined that the primary factor leading to death in AKI caused by gastroenteritis is septicemia.

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