

# The requirement of hemodialysis in patients with acute gastroenteritis-induced acute kidney injury

## Supriyaa Bhakthavatchalam, Devasena Srinivasan, Prithviraj R

General Medicine, Sri Ramachandra Institute of Higher Education and Research (SRIHER), Chennai, India

## Abstract

Acute gastroenteritis is an important cause of preventable acute kidney injury (AKI). Inadequate or delayed restoration of diarrheal losses results in a very high incidence of AKI. Diarrheal illness is a major reason for hospitalization, but data on consequent acute kidney injury are sparse. The objective of the study is to determine the incidence of AKI in infectious and noninfectious diarrheal illness requiring hospitalization and to identify correlates and outcomes of diarrhea-associated AKI. None of the patients had any organism isolated in stool, probably due to prompt initiation of antibiotics/inadequate culture growth. Three out of our 6 cases did not require hemodialysis (HD) and AKI resolved on conservative management alone (fluids, electrolyte management, and antibiotics). Three out of 6 cases had nonresolving AKI and were dependent on renal replacement therapy (RRT) even at 1 month after discharge as they remained oliguric. One recent paper has reported the recovery of renal function after a period of dialysis. Frequent electrolyte abnormalities, risk of (catheter-related/bloodstream) infections, and severity of the primary disease are the chief reasons for the persistently high morbidity. Although, there was no mortality in our study.

**Keywords:** ABG – arterial blood gas, AGE – acute gastroenteritis, AKI – acute kidney injury, BUN – blood urea nitrogen, DM – diabetes mellitus, HD – hemodialysis, HTN – hypertension, LFT – liver function test, RFT – renal function test

### Introduction

Acute diarrheal diseases are an important cause of preventable acute kidney injury (AKI) in India. Inadequate or delayed restoration of diarrheal losses results in a very high incidence of AKI.<sup>[1]</sup>AKI is characterized by abrupt deterioration in kidney function, manifested by an increase in serum creatinine level with or without reduced urine output.<sup>[2]</sup> The spectrum of injury ranges from mild to advanced, sometimes requiring renal replacement therapy (RRT).<sup>[3]</sup>

The present case series focuses on AKI, offset by hypovolemia, secondary to extrarenal losses such as acute gastroenteritis (AGE).

Address for correspondence: Dr. Prithviraj R., Prestige Bella Vista, Tower 20C, Flat 201211, Mount Poonamalle High Road, Iyyapanthangal, Chennai - 600 056, Tamil Nadu, India.

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A study conducted by Carpenter CCJ, Mondal A, Sack RB, *et al.*<sup>[4]</sup> shows that rapid and effective restoration of extracellular fluid (ECF) volume within 4 h can prevent acute renal failure (ARF). Similar results were found in Mahajan *et al.*, wherein it was noted that volume depletion was the most common precipitating factor for ARF, and in Jayakumar *et al.*, it was found that among the medical causes of ARF acute diarrheal disease was the most common. <sup>[5,6]</sup>

Approximately 70% of community-acquired cases of AKI are attributed to prerenal causes. In a study conducted by Kaufman J, Dhakal M, Patel B, Hamburger R. Community-acquired acute renal failure. *American Journal of Kidney Disease*. 1991;17(2):191–198, AGE was the most common cause of AKI followed by ischemic acute tubular necrosis (ATN). In these cases, underlying kidney function may be normal, but decreased renal perfusion associated with intravascular volume depletion (e.g., from vomiting or

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		Table 1: Relevant patient	ant patient details since the	details since the time of admission upto discharge and one month follow up	pto discharge	and one mont	n follow up	
Name/Age/ Gender	Presenting complaints	Relevant past history	Tests on day of admission (only abnormal labs mentioned)	Provisional diagnosis	Organism isolated (stool/ blood/urine)	Requirement of HD (yes/no) if yes, number of HD cycles	Number of days of hospitalization	Condition at discharge & follow up at one month
Mrs Lalitha/60 yrs/Female	4 episodes of vomiting, 10-12 episodes of loose stools	Known case of diabetes and hypertension for 10 years and 2 years, respectively, on regular medication	BUN of 23, creatinine 2.8 mg/dL total count 20,400 cells/cu mm	Acute Gastroenteritis with AKI (prerenal)/ Type 2 Diabetes Mellitus/Systemic Hypertension	NIL	NIL	v	CLINICALLY STABLE, RFT AT FOLLOW UP WAS NORMAL
Mr Selvaraj/75 yrs/Male	loose stools since 3 days, vomiting since 2 days, and reduced urine output for 3 days	Known case of Hypertension since 15 years, known case of Diabetes since 5 years, not on regular medications	BP of 180/90 mmHg, SpO <sub>2</sub> of 88% RA, total count 17,700 cells/ cu mm, creatinine 5.5 mg/dL, s.bicarb 11 mEq/L, s.uric acid 10.1 mg/dL, ABG - pH 7.30 pO <sub>2</sub> 78 mm Hg pCO <sub>2</sub> 40 mm Hg HCO <sub>3</sub> 8 mEq/L	Acute gastroenteritis with AKI (prerenal)/ Flash pulmonary edema (probably secondary to accelerated Hypertension)/Type 2 Diabetes Mellitus	ПХ	YES 8 HD CYCLES	16	Patient had persistent oliguria at time of discharged against medical advice for logistic trasons. On follow up at one month, patient was recieving maintenance HD on alternate days in view of persistent oliguria and non resolving AKI (last creatinine value 3.9 me/dl)
Mr Loganathan/38 years/Male	18-20 episodes of loose stools since 1 day, 3-4 episodes of vomiting since 1 day, nil urine output for more than 12 h	No known co-morbiditics	Total count of 16,000/ cu mm. BUN was 40, screatinine was 5.3 mg/dL, sodium 126 mEq/L, potassium 2.9 mEq/L, chloride 88 mEq/L and bicarbonate 15 mEq/L. Uric acid was 13.5 mg/dL, phosphate 9.5 mg/dL. LFT showed mild transaminits. ABG revealed PH of 7.265, PCO <sub>2</sub> of 29.7, PO <sub>2</sub> of 95.4 and bicarbonate of $13.7$ .	Acute Gastroenteritis with Acute Kidney Injury (prerenal) with Dyselectrolytemia (Hypokalemia, Hyperphosphatemia) and Metabolic Acidosis	Blood c/s grew <i>Peudomonas</i> species on day 15	YES 12 HD CYCLES	22	Patient condition stabilised at time of discharge (remained non oliguric for >3 consecutive days), but required maintenance HD (twice a week) in view of non resolution of AKI (creatinine was 6.2, BUN- 71) . On follow up at one month, patient remained non - oliguric requiring maintenance HD (BUN 70, screatinine 3.5 mg/dl)
Mr.Palani/42 years/male	Vomiting 5 episodes and loose stools 7 episodes per day for the past 1 day.	No comorbidities	Total counts were elevated 16400, among which polymorphs were 92.7 and lymphocytes were 4.8. Renal profile BUN was 23, creatinine was 3.7. SGOT was 78, SGPT- 132. ABG revealed metabolic acidosis. Urine routine showed protein 4+, pus cells 10-12, erythrocytes 5+, nitrites and mvoglobin positive	Acute gastroenteritis Acute kidney injury with post hemodialysis Metabolic acidosis - recovered	Į.N	YES 4 HD CYCLES	5	Patient's your -36, creatinine-3.5 at the time of discharge. Patient required maintanence hemodialysis twice a week post discharge and after one month of follow up aki has been resolved.
Ravichandran, 45 years, male	Loose stools mltiple epsisodes in 1 day post binge alcohol intake for 3 days	No comorbidities chronic alcoholic	Tachycardiand hypotension. electrolytes - sodium- 12 meq/L, potassium-3.9 meq/Lchloride- 93 meq/L, bicarbonate- 14 meq/L. BUN-41, creatinine- 5.7. Uric acid- 10.5, sgot- 142, Urine routine- protein- 4+, erythrocytes- 3+, pus cells 8-10.	Acute gastroenteritis Urinary tract infection ( <i>E. coli</i> ) Acute kidney injury- resolved Dyselectrolytemia- resolved	Urine culture - E. coli	ΪX	×	Clinically stable and normal on follow up.

Contd...

				Table 1: Contd				
Name/Age/ Presenting Gender complaints	Presenting complaints	Relevant past history	Tests on day of admission (only abnormal labs mentioned)	Provisional diagnosis Organism isolated (stool/ blood/urir	Organism Requireme isolated HD (yes/n (stool/ yes, numbe blood/urine) HD cycles	Requirement of Number HD (yes/no) if of days of yes, number of hospitalization HD cycles	Number of days of hospitalization	Condition at discharge & follow up at one month
Kathiravan, 40 Loose stools years, male 10-12 episode day, vomiting 10-12 episode day since 1 da	Loose stools No c 10-12 episodes/ Chro day, vomiting Last 10-12 episodes/ back day since 1 day.	Loose stools No comorbidities. 10-12 episodes/ Chronic alcoholic. day, vomiting Last binge 2 days 10-12 episodes/ back day since 1 day.	Hemoglobin- 19.2, Total count- Acute gastre 18500 cell/cu.m, Neutrophils- with severe 83.6%, lymphocytes- 7.8%, MCV- dehydration. 104.9 FL, Vitamin b12-1515 pg/ Acute kidney mL. BUN- 31, creatinine-4.0, with metabol sodium 129 meq/L, potassium Macrocytosis 3.7 meq/L, chloride 100 meqL, (secondary tt bicarbonate 13 meq/L, uric acid alcoholism) 10.3 mg/dL. ABG - metabolic acidosis. Urine routine - Protein- 3+, Leucocyte- 2+, erythrocytes - 2+, pus cells- 8-10	Acute gastroenteritis with severe dehydration. Acute kidney injury with metabolic acidosis Macrocytosis (secondary to alcoholism)	Nil	ΕX	υ	At the time of discharge BUN- 08, Creatinine- 0.6. On follow up patient was normal.

diarrhea) or decreased arterial pressure (e.g., from sepsis) results in a reduced glomerular filtration rate.

## **Case Description**

The present case series was conducted on 6 patients, in the medical ICU of Sri Ramachandra Institute of Higher Education and Research Centre, Chennai, over a period of 3 months, i.e., 1<sup>st</sup> January 2020–31<sup>st</sup> March 2020. Outcomes in patients were studied who developed AKI secondary to AGE. AKI was diagnosed according to any of the following criteria (1) Increase in serum creatinine (SCr) by X 0.3 mg/dL within 48 h; or increase in SCr to X 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days or (2) Urine volume <0.5 mL/kg/h for 6 h, (3) normal kidneys on ultrasound/ absence of pre-existing renal disease.

Once the diagnosis of AKI was made, the underlying etiology was determined by combining history, examination, and investigative data. Risk factors of AKI such as chronic kidney disease (CKD), hypertension and diabetes mellitus, use of nephrotoxic drugs/ contrast agents administered in the past were ruled out on admission. At the same time, patients were started on appropriate conservative treatment.

Cases were followed up telephonically at 1 month after their discharge to assess recovery for AKI caused by diarrheal illness and/or requirement of HD. It was conferred that 3 out of the 6 AKI cases were requiring maintenance HD at 1 month follow up, following which we terminated our study to prevent superimposing of COVID-19 infection, which could make the purpose of the case series inconclusive.

Table 1 - Title (Relevant Patient Details since the time of admission & discharge & follow up at one month post discharge) it is original patient data taken after patient consent at intensive care unit (ICU) of Sri Ramachandra Institute of Higher Education and Research.<sup>[19]</sup>

## Discussion

ARF, also referred to as AKI, is characterized by sudden kidney function impairment leading to nitrogenous and other waste products retention, which are normally cleared by kidneys.<sup>[2]</sup> The present case series consists of 6 patients, of which all were male, of age range between 38 and 75 years. A similar age and gender distribution are seen in Mahajan *et al.*, and Kumar *et al.*, studies. Diarrhea, nausea, and vomiting were the predominant complaints in all patients and oliguria, anuria, hypotension, and tachycardia were other symptoms observed among a few. Investigations revealed proteinuria noted in four of our cases with no comorbidities, except for two patients with a history of hypertension and diabetes mellitus. Also, the increased neutrophil-lymphocyte ratio (NLR) was highly peculiar in our case as there was no significant infection or another underlying cause was noted. This pattern of NLR elevation<sup>[7]</sup> and proteinuria with AKL<sup>[8]</sup> in few patients were being observed in COVID-19 infection nowadays which gives a suspicion of prevalence during the study period before the announcement of the pandemic.

In India, ARF occurs mainly due to acute diarrheal disease<sup>[1]</sup> similarly in our case food poisoning might be one of the reasons leading to such an entity. The high incidence of ARF is mainly due to delay in reporting to the hospital and delayed or inadequate restoration of diarrheal losses.<sup>[9]</sup> This can be highly related to our case as the patients reported late due to their negligence and were found to have severe uremia, and renal failure was detected. About 40% of cases of AKI in India are caused by acute diarrheal disease, malaria, leptospirosis, snakebite, insect stings, intravascular hemolysis due to septicemia, chemical poisonings such as copper sulphate and vasmol, and pregnancy.<sup>[10,11]</sup> Mehta et al., in their prospective study, related climatic influence on AKI of infectious etiology.<sup>[12]</sup> Basu et al. reported a 41.1% incidence of AKI among the tropical acute febrile illnesses.<sup>[9]</sup> These infections present mostly with fever as a cardinal sign, which was not seen in any of our patients presented in this case series. However, on a microbiological investigation, few organisms isolated were Pseudomonas and E-coli which was evident in 2 of our patients. AKI, in these infections, may be a result of direct invasion of renal parenchyma by microbial agents, tubular necrosis due to hemodynamic disturbances, renal inflammation due to immune response, or iatrogenic renal injury associated with treatment.<sup>[9]</sup> Most studies to date have highlighted the causes of AKI in tropics or individual disease with AKI due to microbial infections, which was insignificant in our cases.

Prakash *et al.*, noted that the main etiological factor for ARF encountered was volume depletion secondary to gastrointestinal fluid loss (35.2%).<sup>[5]</sup> Similar results were found in Mahajan *et al.*, where it was noted that the volume depletion was the most common precipitating factor for ARF<sup>[10]</sup> and in Jayakumar *et al.*, study it was found that among the medical causes of ARF acute diarrheal disease was the most common.<sup>[6]</sup>

It calls for greater awareness about the management of AKI among primary care physicians. In the last few years, various concepts have emerged regarding the prevention and management of AKI.<sup>[13]</sup> The different components of this approach (risk assessment, recognition, response, renal support, and rehabilitation) coincide with various points of contact in the health care system.<sup>[14]</sup> Knowing when to call for help and a timely referral to the nephrologist are important.

## Conclusion

The optimal timing of renal support is still a matter of debate. According to the Kidney Disease: Improving Global Outcomes (KDIGO) review, delayed RRT was associated with increased mortality, longer duration of hospital stays, and dependency on RRT.<sup>[15]</sup>

A holistic approach by the physician by treating the comorbid conditions may not only halt the progression to CKD but will also improve the quality of life.<sup>[16]</sup> AKI being the most common entity in a developing country like India, awareness of such potential renal complications and their prevention and early hospitalization, being the single most cost-effective life-saving measure should be emphasized among people.<sup>[17]</sup> The mortality has been dramatically brought down due to hemodialysis therapy and appropriate medical management,<sup>[18]</sup> and this has been reflected in the above case series.

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### **Conflicts of interest**

There are no conflicts of interest.

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