Original Research Article

A prospective study of clinical profile of patients with acute kidney injury following acute gastroenteritis

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Abstract

Background: Acute kidney injury (AKI), previously known as acute renal failure (ARF), characterized by sudden impairment of kidney function resulting in retention of nitrogenous and other waste products normally cleared by kidneys. AKI is not a single disease but, rather a heterogenous group of condition that share a common diagnostic features, especially increase in the blood urea nitrogen concentration and/or increase in plasma or serum creatinine concentration, often associated with reduction in urine volume. **Materials and Methods:** This study was carried out at tertiary care Teaching and General Hospital, during the period of October 2016 -June 2018. Consecutive cases presenting with AKI due to GE were enrolled in the study. All patients of either sex diagnosed as having Acute Kidney Injury due to gastroenteritis and presence of clinical manifestations of gastroenteritis were included in the study. Patients were categorized into Pre-Renal group and ATN group. Detailed history and clinical profile were recorded in these patients. Duration of GE and time period elapsed between GE and development of Acute Kidney Injury was recorded. Laboratory parameters such as CBC, renal function tests, serum electrolytes, urine examination and stool examination were done at the time of admission. Clinical and laboratory parameters were analysed to assess the role of each of these factors as possible outcome (Recovery or Death). **Results:** The commonest type of renal failure in our study was acute tubular necrosis 54%, followed by prerenalazotaemia46%. Out of 100 patients 82% survived and 18% expired. 64 patients had diarrhoea of less than 5 days with 43.75% in Prerenal and 56.25% in ATN Group and in 36 patients it was > 5 days with 50% in Prerenal and 50% in ATN group. The range of urine output was from 0 to 1300ml with an average of 415.7± 314.80. 30% had moderate dehydration. The mean interval between onset of GE and development of AKI was 3.14± 2.25 days. The mean peak creatinine was 5.478± 3.58 with 4.503± 3.54 in prerenal and 6.309± 3.43 in ATN group. At admission, the urea levels ranged between 30 to 401 mg/dl with mean of 150.51± 95.68. The mean peak urea level was 166.24± 96.14. **Conclusion:** Sustained hypovolemia is usually a cause of development of ATN in gastroenteritis. All patients who died belonged to ATN group. ATN is associated with poor outcome. But, AKI due to gastroenteritis definitely has lower mortality compared to AKI due to other causes. Some studies implicated age per se as one of the predictors for outcome in AKI. However, it is not possible to conclude whether age, sex and interval between the onset of gastroenteritis and development of AKI are independent predictors in the outcome of AKI from our study as it included only a small number of patients belonging to a restricted age group.

Key words: Acute kidney injury, Sustained hypovolemia, ATN, acute tubular necrosis.

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INTRODUCTION

Acute kidney injury (AKI), previously known as acute renal failure (ARF), characterized by sudden impairment of kidney function resulting in retention of nitrogenous and other waste products normally cleared by kidneys. AKI is not a single disease but, rather a heterogenous group of condition that share a common diagnostic features, especially increase in the blood urea nitrogen concentration and/or increase in plasma or serum creatinine concentration, often associated with reduction in

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urine volume.[1] AKI can range in severity from asymptomatic and transient changes in laboratory parameters of glomerular filtration rate to overwhelming and rapidly fatal derangements in effective circulating volume regulation and electrolyte and acid-base composition of the plasma.[2] AKI complicates 5-7% of acute care hospital admissions and up to 30% of admissions to Intensive Care Unit (ICU). AKI is associated with a markedly increased risk of death in hospitalized individuals, particularly in those admitted to the ICU where in hospital mortality rates may exceed 50%.[3] AKI is one of the most common clinical conditions encountered by physicians and nephrologists throughout the world. Due to the climatic conditions, overcrowding and poor socioeconomic factors, AKI in India differs from the world. There is no clear-cut data on the incidence, causes, and recovery from the disease. Most common causes of AKI in India are acute diarrheal disease, malaria, leptospirosis, snakebite, insect stings, intravascular hemolysis due to septicemia, chemical poisoning such as copper sulfate, vasmol, and pregnancy. Overall, these causes constitute 40% ARF in India.[4]

MATERIALS AND METHODS

This descriptive study was conducted on 100 patients who were diagnosed with Acute Kidney Injury (AKI) following acute gastroenteritis fulfilling inclusion and exclusion criteria, admitted at Tertiary Care Teaching and General Hospital, during the period of October 2016 to June 2018.

Inclusion Criteria

- All patients above 18 years of age diagnosed to have acute kidney injury (AKI) due to acute gastroenteritis.(i.e. Progressive Increase in Serum Creatinine by 0.3mg/dl (26.5 mmol/l) within 48 hours;
- Increase in Serum Creatinine to 1.5 times baseline in <7 days; or decrease in Urine volume 0.5ml/kg/h for 6 hours)

Exclusion Criteria

- All patients diagnosed as acute kidney injury (AKI) due to causes other than gastroenteritis.
- Patients with Chronic Kidney Disease (CKD) presenting with acute gastroenteritis.

Data collection

A detailed history, clinical and laboratory data of these patients at admission and then on daily basis was recorded as per the Performa. The duration of gastroenteritis and the time period elapsed between onset of GE and development of renal failure was recorded. The hydration status at time of admission was recorded. Blood urea, serum creatinine and electrolytes (sodium and potassium) were done daily and recorded. Other laboratory parameters such as CBC, ESR, Urine examination Stool Examination including Hanging drop, HIV, Blood glucose, Total leukocyte count and differential count, erythrocyte sedimentation rate, liver function test (serum bilirubin, total serum protein, serum albumin, SGOT, SGPT, ALP) were also done.

Treatment

These patients were given adequate fluid replacement based on severity of dehydration as a first step in management. The complications such as hyperkalaemia and metabolic acidosis were corrected on emergency basis. Daily input and output fluids chart were maintained, and fluids were given accordingly. Antibiotics which have gram negative spectrum preferably quinolones were started to the patients with ongoing gastroenteritis. Change of antibiotics was done whenever deemed necessary based on thought of complications or reports of culture sensitivity. All the patients had ongoing diarrhoea after admission was treated with Fluoroquinolones. Patients who showed evidence of infection were administered parenteral Cefotaxime or Cefoperazone sodium Dialysis was done in patients with hyperkalaemia, pulmonary oedema and severe metabolic acidosis, who did not respond to medical treatment and prophylactically in patients whose creatinine was more than 8 mg/dl. All patients were followed up till discharge or death and complications that occurred in their hospital stay were recorded. The clinical and laboratory parameters were analysed to assess the role of each of these factors as the possible outcome i.e. recovery or death.

Statistical Analysis

The statistical data was compiled using SPSS for windows (version 16.0). Chi-square test, Small Sample t-tests and ANOVA tests were applied for significance. P value of <0.05 was considered as a significant value.

RESULTS

The commonest type of renal failure in our study was Acute Tubular Necrosis 54% followed by Pre-Renal Azotaemia46%. Out of 100 patients prospectively studied 82 patients (82%) survived. All other 18 patients (18%) who expired belong to ATN group. Out of 100 patients 62 (62 percent) were Males and 38 (38 percent) were Females. The age of these 100 patients ranged from 21 to 99 years with mean age of 48.05± 15.720. The age of presentation was over 40 years in 66 (66%) patients and less than 40 years in the remaining 34 patients (34%). The maximum number of patients belonged to age group between 51 to 60 years of age.

Table 1: Age and Sex Distribution

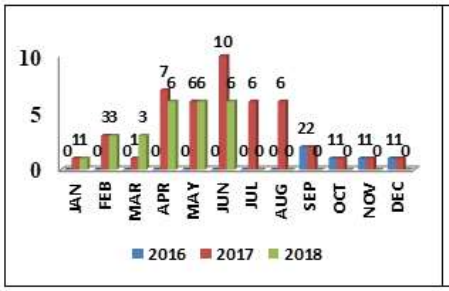
Age Group	Sex		Total
	Male	Female	
18-30	**12**	**3**	**15 (15%)**
31-40	**14**	**5**	**19 (19%)**
41-50	**14**	**9**	**23 (23%)**
51-60	**12**	**14**	**26 (26%)**
>60	**10**	**7**	**17 (17%)**

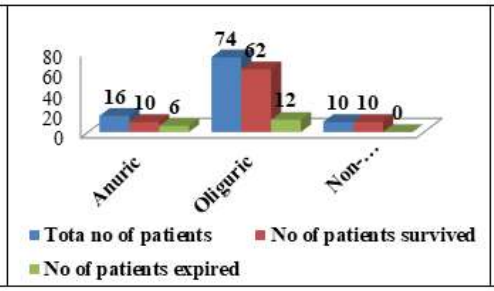
Episodes of Acute Kidney Injury following gastroenteritis were distributed throughout the year. Maximum incidence was recorded in our study was between April and August for 70 patients (70%), highest during month of July 24 %.

Table 2: Mean creatinine levels -baseline, peak and time of discharge

Creatinine (mean)	Overall patients	Pre-Renal	ATN	Survivors	Non-Survivors
Baseline	4.706± 3.32	3.853± 3.21	5.431± 3.25	4.352± 3.29	6.317± 3.00
Peak	5.478± 3.58	4.503± 3.54	6.309± 3.43	4.975± 3.47	7.772± 3.24
At time of discharge	2.329± 1.99	1.691± 1.06	2.872± 2.39	1.585± 0.91	5.717± 2.04

At admission the urea levels ranged between 30 to 401 mg/dl with mean of 150.51± 95.68. The Mean peak urea level was 166.24± 96.14. The mean urea level at the time of discharge was 81.89± 61.92. The mean baseline urea level in survivors was 134.75± 88.79 and in non survivors was 222.32± 95.35.27 patients (27%) had hyponatremia (<125meq/l) at the time of admission. During hospital course 18 more patients developed hyponatremia. Overall 45 (45%) patients had hyponatremia. 28 (28%) patients had hypernatremia (>145 meq/L), 3 at the time of admission and rest during the course of the hospital stay. Serum potassium ranged from 1.9meq/l to 6.2meq/l. The mean potassium at the time of admission was 4.251± 0.744. In survivors it was 4.296± 0.77 where as in non survivors it was 4.044± 0.53. 80 patients (80%) had hypokalaemia out of them 6 had patients had at the time of admission. Hyperkalaemia (>5 meq/dl) was observed in 33 (33%) patients. 23 out of them presented at the time of admission. All non survivors had hypokalaemia.





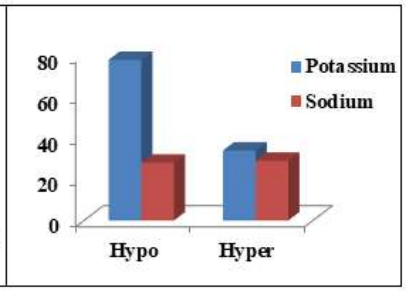


Figure 1: Distribution of Patients According to Season **Figure 2:** Urinary Output In Different Categories **Figure 3:** Electrolyte Imbalance in Patients with AKI due to GE

Figure 1, 64 patients had diarrhoea of less than 5 days prior to development of AKI and in 36 patients it was ≥ 5 days, majority of survivors (52 out of 100) had diarrhoea for less than 5 days.16 patients presented with Anuria. Oliguria was observed in 74 patients. No Oliguria was seen in 10 patients. All the patients who died belonged to anuria and oliguria group.

Figure 2, Majority of patients 30 (30%) had moderate dehydration. 26 (26%) patients had mild dehydration and in 13 (13%) patients it was severe. Fluid overload was observed in 18 (18%) patients and there was no dehydration in 13 (13%) patients. Majority of non survivors belong to group of severe dehydration. Other manifestations at the time of admission included: Fever in 16 (16%) patients, followed by abdominal pain in 8 (8%) patients, altered sensorium 4 (4%), paralytic ileus in 2 (2%) patients. The mean interval between onset of GE and development of AKI was 3.14± 2.25 days. It was 3.09± 2.234in survivors and 3.39± 2.404in non survivors. In pre-renal it was 3.30± 2.269, and ATN was 3.00± 2.257.Baseline creatinine or creatinine at the time of admission ranged from 1.2 to 20 mg/dl with average of 4.706± 3.32. It was 3.853± 3.21 in pre-renal and in ATN group it was 5.431± 3.25. The mean peak creatinine was 5.478± 3.58 with 4.503± 3.54 in prerenal and 6.309± 3.43 in ATN group. The Mean creatinine at the time of discharge was 2.329± 1.99. The mean peak was 5.717± 2.04 in non survivors.

Figure 3, Anaemia (Hb <10 gm %) was seen in 24 patients and 8 of the patients expired. None of the patients was positive for HIV. Hanging drop examination for V cholera was negative in all patients. No specimen showed either cyst of ova. Pneumonia was seen in 10 (10%) patients, pulmonary oedema in 4(4%) patients. Pleural effusion was observed in 8 (8%) patients.20 patients of ATN group improved with conservative management. 34 patients of ATN group required haemodialysis. All the 46 patients of Pre-renal group responded to conservative management and none of the patients required haemodialysis. Out of 82 patients who survived 66 patients improved with conservative treatment, whereas 16 patients required haemodialysis. Allthe 18 patients who died, died in spite of haemodialysis. Out of 18 patients, 12 died due to septicaemia and 6 patients due to Multi organ failure and they all belonged to ATN group.

DISCUSSION

The most common presentation in ourstudy was oliguria (74%) followed by Anuria (16%). Anderson et al9reported that compared to oliguric renal failure, non-oliguric renal failure is characterized by less fluid overload and lower mortality (25% v/s 50%). In our study patients with non-oliguric renal failure were only 10 and all of them survived. The fact that 6 of 16 anuric patients v/s 12 of 74 oliguric patients died indicates that the urinary output can be considered as one of the prognostic factors for AKI due to gastroenteritis.30% patients were in moderate dehydration and 13% were severely dehydrated.[5] out of 13 expired in severe dehydration group and 4 of 30 patients expired in moderate dehydration group. There was no mortality in 7 patients who had severe dehydration and 26 patients who had moderate dehydration; hence the severity of dehydration alone is not a determinant factor of outcome in AKI due to gastroenteritis in our study. Persistent hypotension is associated with poor outcome of AKI was observed in our study. All patients with hypotension due to hypovolemia recovered. 12 patients died due to hypotension due to septic shock. Hence septic shock per se was one of the main predictors of outcome of AKI due to GE. The interval between the onset of gastroenteritis and development of AKI was nearly equal in pre-renal and ATN groups and no statistical difference was seen. Study of more number of patients is required to know whether the interval affects the outcome. The baseline level of creatinine in survivors was almost equal to that of non-survivors. It reflects the severity of renal failure per se and it is not a contributory factor for outcome. The baseline and peak creatinine values of ATN group were higher than that of pre-renal group. It reflects higher morbidity in patients with higher creatinine level. This is true with study of Kaufman et al. The peak urea level was also not associated with higher mortality.[6] Hyponatremia is a common complication of AKI due to absolute or relative increase in free water intake and loss of sodium from the body through vomiting and diarrhoea. It was 27% in our study compared to 19% in previous study by Anderson et a1.[7] Hypernatremia occurred in 25 patients (25%) during hospital course probably of iatrogenic rather than AKI per se. Hypokalaemia is a minor and unusual complication during recovery phase of ATN. But in our study 80% of the patients had hypokalaemia and in 11% of the patients it was severe. All patients who expired were associated with hypokalaemia. Hyperkalaemia, a common complication of ATN was seen in 75% of patients in the study conducted by Minuty et al whereas it was only 33% in our study.[8] It can be concluded that hypokalaemiais a major complication than Hyperkalaemia in patients of AKI due to gastroenteritis. Infectious complications are leading causes of death.[9] In study conducted by Frankel et al it was 30%. In our study most sites of infection were UTI (22%), lungs (10%) and septicaemia was observed in 28% patients. Cardiovascular complications were 43% in Minuty et al study, whereas in our study it was very less (only one patient-pericarditis). Neurological complications were 38% and Pulmonary complications were 28% in Anderson et al study. In our study it was 12% and 22% respectively. Presence of oliguria, sustained hypotension, infection was associated with significantly higher mortality despite the aetiology.[10] In our study oliguria, sustained hypotension and coma were significantly associated with higher mortality. Overall mortality in our study was 18%, most of them belonged to ATN group. It is similar to previous studies conducted by Kaufman et al and Liano et al. The original disease was the cause of death followed by shock and infections in Liano et al study whereas septicaemia was the main cause of death in Beaman et al study group. In our study the sepsis was the cause in 12 cases.

CONCLUSION

Sustained hypovolemia is usually a cause of development of ATN in gastroenteritis. All patients who died belonged to ATN group. ATN is associated with poor outcome. But, AKI due to gastroenteritis definitely has lower mortality compared to AKI due to other causes. Some studies implicated age per se as one of the predictors for outcome in AKI. However, it is not possible to conclude whether age, sex and interval between the onset of gastroenteritis and development of AKI are independent predictors in the outcome of AKI from our study as it included only a small number of patients belonging to a restricted age group.

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