Original Research Paper

Volume - 12 | Issue - 08 | August - 2022 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Nephrology

# CLINICAL PROFILE OF ACUTE GASTROENETERITIS PATIENTS HAVING ACUTE KIDNEY INJURY AND OUTCOME

**Dar maqsood ahmad** — Senior Resident, Department of General Medicine, Sher-i-Kashmir Institute of Medical sciences, Srinagar(Jammu and Kashmir) India.

**Nath Mohd Yousuf** — Senior Resident, Department of General Medicine, Sher-i-Kashmir Institute of Medical sciences, Srinagar(Jammu and Kashmir) India

**Wani Firdous Dawood*** — Senior Resident, Department of General Medicine, Sher-i-Kashmir Institute of Medical sciences, Srinagar(Jammu and Kashmir) India*Corresponding Author

**ABSTRACT** **Background:** In India, acute kidney injury (AKI) due to diarrhea is not uncommon in adults and elderly people. Therefore, understanding of the clinical spectrum of the disease is needed to devise methods to improve the final outcome due to this problem.**Materials and Methods:** We studied 160 patients of acute gastroenteritis admitted to medical wards who met the inclusion and exclusion criteria after obtaining the informed consent out of which 60 patients who had AKI were taken as cases and 100 patients who did not have AKI were taken as controls. The clinical and laboratory data were collected at admission and then on daily basis. All patients were followed up during the hospital stay and outcome of the patient was recorded. **Results:** Majority of patients were of the age group of 45-64 years (53%) followed by >65 year age group (26.7%).In our study the majority of patients were females (63.3%). Majority of the patients presented with loose stools (100%), vomiting (78.4%). Fever was present in 15% of patients and other symptoms like pain abdomen, dysuria was seen in 28.3% of patients. Majority of the patients (86.7%) had tachycardia and hypotension on presentation and 78.3% had oliguria. The common comorbid conditions noted in the present study are hypertension(56.7%) and diabetes mellitus 40%. Hemodialysis was required in 8.3% of patients in the present study. The rest 91.7% were managed conservatively and had complete recovery. At admission most of the patients were in the injury group (80%) as per RIFLE Criteria for AKI, 13.3% were in the failure and 6.7% in the risk group. On follow up after one month 28% were in the risk group and on further follow up of these patients at three and six months, 28% and 8.5% were in the risk group respectively which is statistically significant. 95% of patients recovered there renal function and only 5% (3 patients) progressed to chronic kidney disease .Mortality rate in our study was 0% in both control and cases group. **Conclusion:** AKI following acute gastroenteritis is not uncommon in developing country like ours. General improvement in standard of living, early use of oral rehydration therapy and fluid therapy to correct dehydration, and creating awareness in primary care physicians about the high incidence of AKI following gastroenteritis will solve this problem. Early detection and referral of these patients will bring down mortality in these patients. Hemodialysis plays an important role in improving the prognosis.

**KEYWORDS :** Acute kidney injury, Gastroenteritis, Hemodialysis

## INTRODUCTION

Acute kidney injury (AKI), previously known as acute renal failure is characterized by sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI is not a single disease but, rather, a designation for a heterogeneous group of conditions that share common diagnostic features, especially increase in the blood urea nitrogen (BUN) concentration and / or an increase in the plasma or serum creatinine concentration, often associated with a reduction in urinary volume. 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. AKI (Acute Kidney Injury) is reversible when recognized and managed early.[8] Delay in diagnosis of ARF may lead to increased morbidity and mortility[7].Acute kidney injury can be divided into three categories: Prerenal causes (kidney hypo perfusion leading to lower GFR), intrinsic kidney disease, and post renal causes (obstructive uropathy). Prerenal causes are the most common etiology of acute kidney insults and injury, accounting for 40-80% of cases.[16]

Decreased renal perfusion can occur in several ways, such as a decrease in intravascular volume, a change in vascular resistance, or low cardiac output. Causes of volume depletion include hemorrhage, GI losses, dehydration, excessive diuresis, extravascular space sequestration, pancreatitis, burns, trauma, and peritonitis.[16] AKI is generally detected by an increase in the serum creatinine and/or a decrease in urine output. The magnitude of the increase in creatinine and/or decrease in urine output that is required to establish a diagnosis of AKI has been the focus of multiple expert consensus groups. The purpose of establishing a precise definition of AKI is to allow better interpretation of epidemiologic and clinical studies and to identify potential therapies. In addition, it is now recognized that even small increases in serum creatinine (>0.3 mg/dl) can have important prognostic implications and are clinically relevant. [26]

The Acute Dialysis Quality initiative established a multilayered definition of AKI called the RIFLE criteria. In this AKI is stratified into five stages, based on severity and duration of renal injury: Risk, Injury, Failure, Loss, and End stage disease. RIFLE defined AKI is associated with significantly reduced survival (with increasing stage leading to greater risk of death).[17] More recently, AKIN(an international network of AKI experts) modified RI FLE to incorporate small changes in serum creatinine occurring within a 48h period and to remove changes in GFR as diagnostic criteria[18].

AKI, classified by either of these criteria, may identify slightly different patients: RIFLE may not detect approximately 10% of AKIN identified cases and AKIN may miss approximately 25% RIFLE cases.[19]

KDIGO (Kidney disease improving global outcomes) have recently produced a definition that incorporates the key elements of both, and this criteria now form the basis of, and have been validated in, hundreds of epidemiological studies and several clinical trials.[19]

AKI complicates 5 to 7 % of acute care hospital admissions and up to 30% of admissions to the intensive care unit, particularly in the setting of diarrheal illnesses, infectious diseases like malaria and leptospirosis, and natural disasters such as earthquakes.[1] The development of AKI is associated with a significantly increased risk of in hospital and long term mortality, longer length of stay, and increased costs. Prerenal azotemia, with the exception of cardiorenal and hepatorenal syndromes and postrenal azotemia carry a better prognosis than most cases of intrinsic AKI. The kidneys may recover even after severe, dialysis requiring AKI. Survivors of an episode of AKI requiring temporary dialysis, however, are at extremely high risk for progressive CKD and upto 10% may develop ESRD .Patients with AKI are more likely to die prematurely after they leave the hospital even if their kidney function has recovered.[1]

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.[25]

All patients who present with AKI must be carefully evaluated both for reversible causes, such as hypotension, volume depletion, or obstruction, and for the presence of complications such as hyperkalemia, metabolic acidosis, and volume overload. The initial evaluation of the patient with AKI is directed at determining the cause, removing any active insults, minimizing new injury, and identifying the complications that may require immediate attention.

The major complications of AKI include volume overload, hyperkalemia, metabolic acidosis, hypocalcaemia, and hyperphosphatemia. With severe forms, mental status changes may be present. Hyperuricemia and hypermagnesemia may also occur. The initial assessment therefore should include the careful evaluation of volume status and measurement of serum electrolytes, particularly potassium and bicarbonate, and serum phosphate, calcium, and albumin. Also check serum uric acid, magnesium, and a complete blood count. As these patients are often unstable and critically ill, we recommend careful attention to serial measurement of these values as well as meticulous measurement of fluid balance.[27]

Measures to correct underlying causes of acute kidney injury (AKI) should begin at the earliest indication of renal dysfunction. Serum creatinine does not rise to abnormal levels until a large proportion of the renal mass is damaged, because the relationship between the glomerular filtration rate (GFR) and the serum creatinine level is not linear, especially early in disease. Indeed, the rise of serum creatinine may not be evident before 50% of the GFR is lost.[45]

It cannot be overstated that the current treatment for AKI is mainly supportive in nature; no therapeutic modalities to date have shown efficacy in treating the condition. Therapeutic agents (eg, dopamine, nesiritide, fenoldopam, mannitol) are not indicated in the management of AKI and may be harmful for the patient.[46]

Maintenance of volume homeostasis and correction of biochemical abnormalities remain the primary goals of treatment and may include the following measures:

- Correction of fluid overload with furosemide
- Correction of severe acidosis with bicarbonate administration, which can be important as a bridge to dialysis
- Correction of hyperkalemia
- Correction of hematologic abnormalities (eg, anemia, uremic platelet dysfunction) with measures such as transfusions and administration of desmopressin or estrogens[28]

Acute gastroenteritis is one of the common cause of prerenal acute kidney injury. The clinical manifestations of acute gastroenteritis can include diarrhea, vomiting, fever, anorexia, and abdominal cramps. Vomiting followed by diarrhea may be the initial presentation or vice versa.[20,21,22]

Acute gastroenteritis leads to volume depletion and pre renal azotemia which refers to rise in Scr or BUN concentration due to inadequate renal plasma flow and intra glomerular hydrostatic pressure to support normal glomerular filtration. Prolonged periods of pre renal azotemia may lead to ischemic injury often termed acute tubular necrosis (ATN). Diarrhea sometimes is defined as a stool weight of more than 200-300 g/ 24hr, quantification of stool weight is necessary only in some patients with chronic diarrhea. In most cases, the physician's working definition of diarrhea is increased stool frequency (more than three bowel movements per day) or liquidity of feces.[23]

The following definitions have been suggested according to the duration of symptoms:

- Acute – 14 days or fewer in duration
- Persistent diarrhea – more than 14 but fewer than 30 days in duration
- Chronic – more than 30 days in duration

Diarrhea, acute in onset and persisting for < 2 weeks is most commonly caused by infectious agents, bacterial toxins (either preformed or produced in the gut), or medications. Community outbreaks (including nursing homes, schools, and cruise ships) suggest a viral etiology or a common food source. Similar recent illnesses in family members suggest an infectious origin. Ingestion of improperly stored or prepared food implicates food poisoning. Pregnant women have an increased risk of developing listeriosis. Day care attendance or exposure to unpurified water (camping, swimming) may result in infection with Giardia or Cryptosporidium. Recent travel to any endemic area suggests travelers' diarrhea. Antibiotic administration within the preceding several weeks increases the likelihood of clostridium difficile colitis.[23]

The nature of the diarrhea helps distinguish among different infectious causes.

1) Non inflammatory diarrhea- watery, nonbloody diarrhea associated with periumbilical cramps, bloating, nausea, or vomiting suggests a small bowel source caused by either a toxin producing bacterium (ETEC ,staphylococcus aureus, bacillius cereus, clostridium perfringens) or other agents (viruses, Gardia) that disrupt normal absorption and secretory process in the small intestine. Prominent vomiting suggests viral enteritis or S aureus food poisoning. Because tissue invasion does not occur, fecal leucocytes are not present. The isolation rate of bacterial pathogens from stool cultures in patients with acute non inflammatory diarrhea is under 3%.[23]

2) Inflammatory diarrhea- the presence of fever and bloody diarrhea (dysentry) indicates colonic tissue damage caused by invasion (shigellosis, salmonellosis, campylobacter or yersinia infection, amebiasis) or a toxin (C difficle, shiga-toxin-producing E coli). Because these organisms involve predominantly colon, the diarrhea is small in volume(< 1 l/d) and associated with left lower quadrant cramps, urgency, and tenesmus. Fecal leucocytes usually are present in infections with invasive organisms.

The isolation rate of bacterial pathogens from stool cultures in patients with acute non inflammatory diarrhea is under 3%. In case of inflammatory diarrhea the rate of positive bacterial cultures is 60%-75%.[23]

Viral gastrointestinal infections remain a significant cause of morbidity and mortality worldwide.[24] Viral infections usually are characterized by low grade fever and watery diarrhea without blood.[21] The important viruses which give viral gastroenteritis are- Rotavirus, Noroviruses, Enteric adenoviruses, Caliciviruses, Astroviruses, Enteroviruses. Among them Rotavirus remains the leading cause and the burden is most severe in the very young and developing countries.[24] Stool for polymerase chain reaction is the method of choice for diagnosing rotavirus. It can also be used for the diagnosis of other viruses.[24]

Characteristics of the history that suggest a viral etiology of acute gastroenteritis include: an intermediate incubation period (24 to 60 hours), a short infection duration (12 to 60 hours), and a high frequency of vomiting[29]. However, these epidemiologic criteria for differentiating between outbreaks caused by norovirus and bacterial pathogens may not be as useful for individual patient assessment[30]. As an example, one observational study showed few differences in the clinical presentation between adults with acute gastroenteritis due to viral and bacterial pathogens[31]. The duration of the diarrhea may differ among viral and bacterial acute gastroenteritis. Norovirus infection usually lasts a median of two days, rotavirus infection three to eight days, and Campylobacter and Salmonella two to seven days[32]. Viral gastroenteritis does not typically cause bloody diarrhea

Common findings on physical examination of patients with acute viral gastroenteritis include mild diffuse abdominal tenderness on palpation; the abdomen is soft, but there may be voluntary guarding. Fever (38.3 to 38.9°C [101 to 102°F]) occurs in approximately one-half of patients[33].

While relatively uncommon, it is important to identify signs of moderate to severe dehydration, including dry mucous membranes, decreased skin turgor, tachycardia, hypotension, or altered mental status. These were present in fewer than 10 percent of patients presenting to the emergency department with acute gastroenteritis in one study[31].

The management of patients with acute diarrhea begins with general measures such as fluid repletion and nutrition maintenance, with adjustments in diet if necessary[35]. Patients who have bothersome symptoms may benefit from symptomatic pharmacologic therapy[37]. Antibiotic therapy is not indicated in most cases since the illness is usually self-limited. Nevertheless, empiric and specific antibiotic therapy may be appropriate in certain situations, mainly in patients with severe disease, with symptoms and signs suggestive of invasive bacterial infection, or at high risk for complications[36]

Acute viral gastroenteritis is usually self-limited and is treated with supportive measures (fluid repletion and unrestricted nutrition). No specific antiviral agents are available.

For adults presenting with acute viral gastroenteritis without signs of volume depletion, adequate volume can be maintained with sport drinks and broths34. For adults presenting with mild to moderate hypovolemia, oral rehydration solutions may be superior to sports drinks in maintaining electrolyte balance along with hydration. Patients with severe dehydration require intravenous fluids.

Antiemetics and antimotility agents are used sometimes for excessive vomiting or excessive fluid loss from diarrhea, respectively. In known viral gastroenteritis epidemics, antibiotics are not indicated[36]. Empiric antibiotics may have a limited role in the management of acute gastroenteritis, when it is unclear if the etiology is viral or bacterial[38].

## MATERIALS AND METHODS :

This was a prospective, observational study carried out in the Department of Emergency Medicine in our tertiary care hospital. All the patients who presented with acute gastroenteritis in medical emergency were taken into the study with following inclusion and exclusion criteria:

### Inclusion criteria:

1) All patients above 18 years of age presenting with acute gastroenteritis for more than 24 hours.
2) Patients with progressive elevation in serum creatinine > 0.3 mg/dl or more within 48 hours or increase in serum creatinine to 1.5 times baseline or more within last 7 days or reduction in urine output to < 0.5 ml/kg/hr for longer than 6 hours.

### Exclusion criteria:

1) Patients with chronic renal insufficiency (medical records, history)
2) Patients who are initially considered as AKD but subsequently found to be suffering from long standing renal disease.
3) Patients having malignancy, HIV and immunosuppression.

### Method of study

Data was collected using a proforma meeting the objectives of the study. Detailed history and necessary investigations were undertaken. The purpose of study was explained to the patient and informed consent obtained. Patients are selected for study who satisfy all inclusion and exclusion criteria. Patients with progressive elevation of serum creatinine >0.3 mg/dl or 50% higher than baseline within a 24-48 hours period or reduction in urine output to <0.5 ml/kg/hr for longer than 6 hours. The clinical and lab data was collected at admission and then on daily basis. Data recorded include patient characteristics, primary comorbid medical conditions, dialysis requirement, total duration of hospital stay and final outcome.

## RESULTS & OBSERVATION

### Statistical methods

Descriptive statistics was applied for continuous variable as Mean, Standard deviation (mean $\pm_{SD}$) and for categorical variables as frequency distribution and percentage. Student's unpaired t – test for continuous variables and Chi Square test for categorical variables was used to see the significance difference between the groups. $P < 0.05$ will be treated as significant.

Total of 160 patients were studied with 60 patients as cases and 100 as controls. For convenience controls were designated as Group A and cases as Group B .The majority of patients were of the age group of 45-64 years (53%) followed by >65 year age group (26.7%). No patient was noted in the age group of 18-24 years(fig1,1a). In our study the majority of patients were females (63.3%). Majority of the patients presented with vomiting (78.4%), loose stools (100%).Fever was present in 15% of patients and other symptoms like pain abdomen, dysuria was seen in 28.3% of patients(fig2,2a) Majority of the patients (86.7%) had tachycardia and hypotension on presentation and 78.3% had oliguria. (Fig3,3a). Majority of the patients on presentation had urea in the range of 40-60 (61.7%) and creatinine in the range of 1.5-2 (73.3%)(fig 4,4a). Urinalysis revealed albuminuria in 13.3% and glycosuria in 16.7% of patients(fig 6,6a) Urine microscopy showed pus cells in the range of 0-5 in 85% of patients, RBC'S <3 in 90% of patients and casts were present in 20% of patients(fig 7,7a). Most of the patients (66.7%) had raised echopattern on presentation (fig 8,8a). Most of the patients had hypertension (56.7%) as comorbid illness. Diabetes mellitus was present in 40% of patients (fig 9,9a). Majority of the patients were managed conservatively (91.7%). Hemodialysis were required in 8.3% of patients (fig 10,10a). Majority of the patients (85%) were discharged within 2-5 days of hospital admission. At discharge most of the patients were having creatinine in the range of 1.5-2 (73.3%), but on following there creatinine after 4 weeks, 12 weeks and 24 weeks majority of patients had recovered there renal functions (fig 11,11a). 95% of patients recovered there renal function and only 5% (3 patients) progressed to chronic kidney disease. There was 0% mortality in our study patients. At admission most of the patients were in the injury group (80%), 13.3% were in the failure and 6.7% in the risk group. On follow up after one month 28% were in the risk group and on further follow up of these patients at three and six months, 28% and 8.5% were in the risk group respectively which is statistically significant(fig 12,12a). There was no mortality in both control and case groups. The mean ages of controls and cases were 58.3±3.12 and 62.9±2.3 respectively. Among cases 38(63%) were females and 22(36.7%) were males. There were 56(56%) females and 44(44%) males in the control group. Among controls 46% of patients presented with vomiting within 24hrs and in cases 71.7% of patients presented with vomiting for more than 48hrs. Similarly among controls 63% of patients presented with loose stools with in 24hrs and in cases 88.3% of patients presented with loose stools for more than 48hrs which makes it statistically significant data (fig13,13a). In the control group 39% of patients had tachycardia on presentation and among the cases 86.7% had tachycardia and hypotension on presentation and 78.3% had oliguria( fig14,14a). The average urea at presentation were 34.5±0.18 and 77.4±0.61 among controls and cases respectively .Similarly the average creatinine were 0.8±0.48 and 1.9±0.78 among controls and cases respectively(fig 15 ,15a). Both these values show statistically significant difference between the two groups. In control group all the patients were managed conservatively and in cases 8.3% underwent hemodialysis fig 16, 16a). Among the controls all the patients (100%) were discharged within 48 hours and in the cases 85% of patients were discharged between 2-5 days.(fig 17,fig 17a)

- **Age distribution of study patients fig 1 & fig 1a**

**Table 1: Age distribution of study patients**

| Age (years) | No | %age |
|---|---|---|
| 18-24 | 0 | 0.0 |
| 25-44 | 12 | 20.0 |
| 45-64 | 32 | 53.3 |
| >65 | 16 | 26.7 |
| Total | 60 | 100.0 |
| Mean±SD | 62.9±2.3 | |

**Fig1**

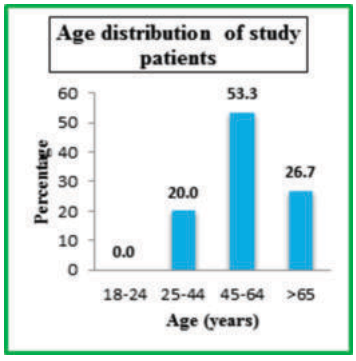


**Fig 1a**

**Table 2: Showing clinical presentation of study patients**

| Clinical Presentation | | No | %age |
|---|---|---|---|
| Vommiting | <24 hrs | 0 | 0.0 |
| | 24-48 hrs | 4 | 6.7 |
| | >48 hrs | 43 | 71.7 |
| Loose Stools | <24 hrs | 0 | 0.0 |
| | 24-48 hrs | 7 | 11.7 |
| | >48 hrs | 53 | 88.3 |
| Fever | Yes | 9 | 15.0 |
| | No | 51 | 85.0 |
| Others | Yes | 13 | 21.7 |
| | No | 47 | 78.3 |

**Fig 2**
**Symptoms of study patients fig 2 & fig 2a**

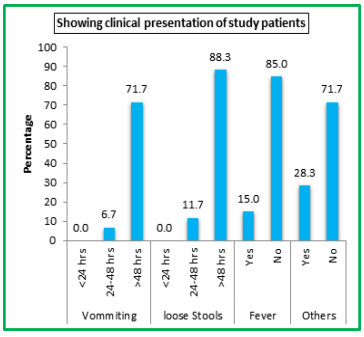


**fig 2a**
**Clinical signs of study patients fig 3 & fig 3a**

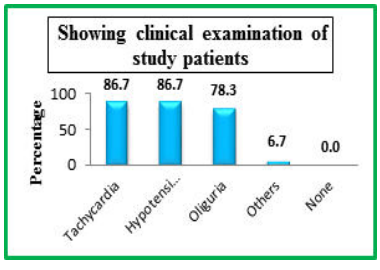


**Fig 3**
**Fig 3a**

**Table 3: Showing clinical examination of study patients**

| Clinical Examination | No | %age |
|---|---|---|
| Tachycardia | 52.0 | 86.7 |
| Hypotension | 52.0 | 86.7 |
| Oliguria | 47.0 | 78.3 |
| Others | 4.0 | 6.7 |

**Kidney function of study group at presentatationfig 4 & fig 4a**

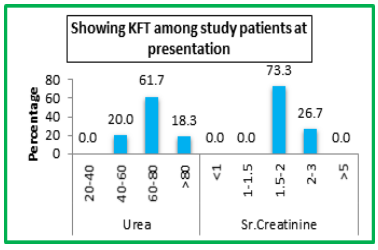


**Fig 4**
**Fig 4a**

**Table 4: Showing KFT among study patients at presentation**

| Clinical Presentation | | No | %age |
|---|---|---|---|
| Urea | 20-40 | 0.0 | 0.0 |
| | 40-60 | 12.0 | 20.0 |
| | 60-80 | 37.0 | 61.7 |
| | >80 | 11.0 | 18.3 |
| Mean±SD | 77.4±0.61 | | |
| Sr.Creatinine | <1 | 0.0 | 0.0 |
| | 1-1.5 | 3.0 | 5.0 |
| | 1.5-2 | 41.0 | 68.3 |
| | 2-3 | 16.0 | 26.7 |
| | >3 | 0.0 | 0.0 |
| Mean±SD | 1.9±0.78 | | |

**Lab parameters at presentation fig 5 & fig 5a**

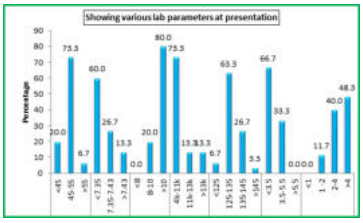


**Fig 5**
**Fig5a**

**Table 5: Showing various lab parameters at presentation**

| Clinical | Presentation | No | %age |
|---|---|---|---|
| Hematocrit | <45 | 12.0 | 20.0 |
| | 45-55 | 44.0 | 73.3 |
| | >55 | 4.0 | 6.7 |
| Ph | <7.35 | 36.0 | 60.0 |
| | 7.35-7.45 | 16.0 | 26.7 |
| | >7.45 | 8.0 | 13.3 |
| Hb | <8 | 0.0 | 0.0 |
| | 8-10 | 12.0 | 20.0 |
| | >10 | 48.0 | 80.0 |
| TLC | 4k-11k | 44.0 | 73.3 |
| | 11k-13k | 8.0 | 13.3 |
| | >13k | 8.0 | 13.3 |
| Na | <120 | 2.0 | 3.3 |
| | 120-130 | 12.0 | 20.0 |
| | 130-140 | 42.0 | 70.0 |
| | >140 | 4.0 | 6.7 |
| Pottassium | <3.5 | 40.0 | 66.7 |
| | 3.5-5.5 | 20.0 | 33.3 |
| | >5.5 | 0.0 | 0.0 |
| Sr.Lactate | <1 | 0.0 | 0.0 |
| | 1-2 | 7 | 11.7 |
| | 2-4 | 24.0 | 40.0 |
| | >4 | 29.0 | 48.3 |

**Urinalysis at presentation fig 6 & fig 6 a**

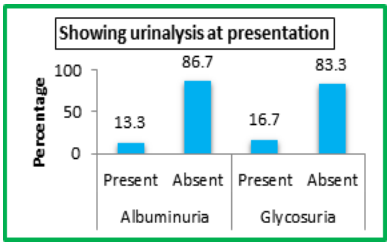


**Fig6**
**fig 6a**

**Table 6: Showing urinalysis at presentation**

| Urinalysis | | No | %age |
|---|---|---|---|
| Albuminuria | Present | 8.0 | 13.3 |
| | Absent | 52.0 | 86.7 |
| Glycosuria | Present | 10.0 | 16.7 |
| | Absent | 50.0 | 83.3 |

**Urine microscopy at presentation fig 7 & fig 7a**

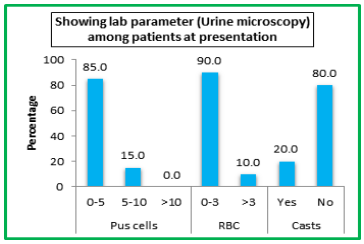


**Fig 7**
**fig 7a**

**Table 7: Showing lab parameter (microscopy) among patients at presentation**

| Microscopy | | No. | %age |
|---|---|---|---|
| Pus cells | 0-5 | 51.0 | 85.0 |
| | 5-10 | 9.0 | 15.0 |
| | >10 | 0.0 | 0.0 |
| RBC | 0-3 | 54.0 | 90.0 |
| | >3 | 6.0 | 10.0 |
| Casts | Yes | 12.0 | 20.0 |
| | No | 48.0 | 80.0 |

**Usg findings at presentation fig 8 & fig 8 a**

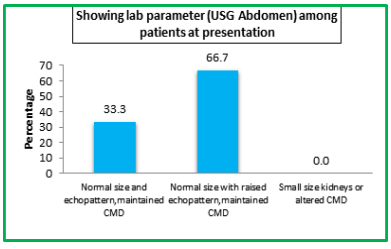


**Fig 8**
**Fig 8a**

**Table 8: Showing lab parameter (USG Abdomen) among patients at presentation**

| USG Abdomen | No. | %age |
|---|---|---|
| Normal size and echopattern,maintained CMD | 18.0 | 30.0 |
| Normal size with raised echopattern,maintained CMD | 42.0 | 70.0 |
| Small size kidneys or altered CMD | 0.0 | 0.0 |
| Total | 60.0 | 100.0 |

**Various comorbidities present among patients fig 9 & fig 9a**

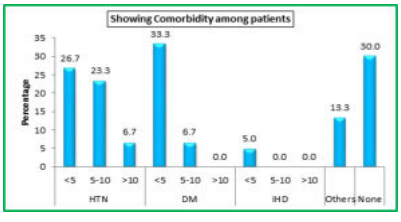


**Fig 9**
**Fig 9a**

**Table 9: Showing Comorbidity among patients**

| Comorbid condition (years) | | No | %age |
|---|---|---|---|
| HTN | <5 | 16.0 | 26.7 |
| | 5-10 | 20.0 | 23.3 |
| | >10 | 4.0 | 6.7 |
| DM | <5 | 20.0 | 33.3 |
| | 5-10 | 4.0 | 6.7 |
| | >10 | 0.0 | 0.0 |
| IHD | <5 | 3.0 | 5.0 |
| | 5-10 | 0.0 | 0.0 |
| | >10 | 0.0 | 0.0 |
| others | | 8.0 | 13.3 |
| None | | 18.0 | 30.0 |

**Mangement modalities of patients in hospital fig 10 & fig 10a**

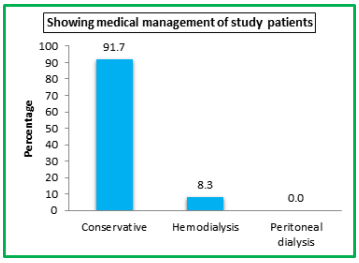


**Fig 10**
**Fig 10a**

**Table 10: Showing medical management of study patients**

| Management | No | %age |
|---|---|---|
| Conservative | 55.0 | 91.7 |
| Hemodialysis | 5.0 | 8.3 |
| Peritoneal dialysis | 0.0 | 0.0 |
| Total | 60.0 | 100.0 |

**Renal function recovery in study patients fig 11 & fig 11a**

**Fig 11**
**Fig 11a**

**Table 11: Showing recovery of renal function of study patients as per creatinine**

| Recovery of Renal function | | No | %age |
|---|---|---|---|
| Sr.creatinine at D/C | <1 | 0.0 | 0.0 |
| | 1-1.5 | 16.0 | 26.7 |
| | 1.5-2 | 44.0 | 73.3 |
| | 2-3 | 0.0 | 0.0 |
| Sr.Creatinine at one month | <1 | 7.0 | 11.7 |
| | 1-1.5 | 53.0 | 88.3 |
| | 1.5-2 | 0.0 | 0.0 |
| | 2-3 | 0.0 | 0.0 |
| Sr.Creatinine at three months | <1 | 9.0 | 15.0 |
| | 1-1.5 | 51.0 | 85.0 |
| | 1.5-2 | 0.0 | 0.0 |
| | 2-3 | 0.0 | 0.0 |
| Sr.Creatinine at six months | <1 | 9.0 | 15.0 |
| | 1-1.5 | 51.0 | 85.0 |
| | 1.5-2 | 0.0 | 0.0 |
| | 2-3 | 0.0 | 0.0 |

**RIFLE on admission ,one month ,3 month, 6 month fig 12 & fig 12a**

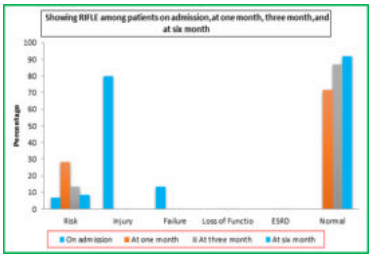


**Fig 12**
**Fig12a**

**Table 12: Showing RIFLE among patients on admission, at one month, three month and at six month**

| RIFLE | On admission (n=60) | | At one month (n=60) | | At three month (n=60) | | At six month (n=60) | | P value |
|---|---|---|---|---|---|---|---|---|---|
| | No | %age | No. | %age | No. | %age | No. | %age | <0.001* |
| Risk | 4.0 | 6.7 | 17.0 | 28.3 | 8.0 | 13.3 | 5.0 | 8.3 | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Injury | 48.0 | 80.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Failure | 8.0 | 13.3 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Loss of Functio | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| ESRD | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Normal | 0.0 | 0.0 | 43.0 | 71.7 | 52.0 | 86.7 | 55.0 | 91.7 |
| Total | 60 | 100.0 | 60 | 100.0 | 60 | 100.0 | 60 | 100.0 |

**Outcome of patients among cases vs controls fig 13 & fig 13a**

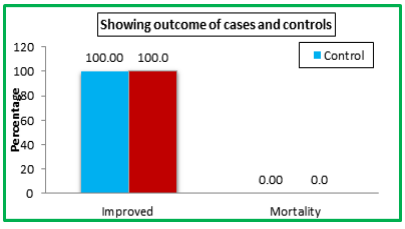


**Fig 13**
**fig 13a**

**Table 13: Showing outcome of patients among controls and cases**

| Outcome | Control (n=100) | | Cases (n=60) | |
|---|---|---|---|---|
| | No | %age | No. | %age |
| Improved | 100.0 | 100.0 | 60.0 | 100.0 |
| Mortality | 0.0 | 0.0 | 0.0 | 0.0 |
| Total | 100.0 | 100.0 | 60.0 | 100.0 |

**Clinical presentation of control and cases fig 14 & 14a**

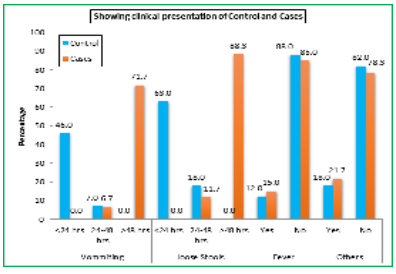


**Fig 14**
**fig 14a**

**Table 14: Showing clinical presentation of Control and Cases**

| Clinical Presentation | | Control | | Cases | | P value |
|---|---|---|---|---|---|---|
| | | No | %age | No | %age | |
| Vommiting | <24 hrs | 46.0 | 46.0 | 0.0 | 0.0 | <0.001* |
| | 24-48 hrs | 7.0 | 7.0 | 4.0 | 6.7 | |
| | >48 hrs | 0.0 | 0.0 | 43.0 | 71.7 | |
| Loose Stools | <24 hrs | 63.0 | 63.0 | 0.0 | 0.0 | <0.001* |
| | 24-48 hrs | 18.0 | 18.0 | 7.0 | 11.7 | |
| | >48 hrs | 0.0 | 0.0 | 53.0 | 88.3 | |
| Fever | Yes | 12.0 | 12.0 | 9.0 | 15.0 | - |
| | No | 88.0 | 88.0 | 51.0 | 85.0 | |
| Others | Yes | 18.0 | 18.0 | 13.0 | 21.7 | 0.064 |
| | No | 82.0 | 82.0 | 47.0 | 78.3 | |

**Clinical signs among cases and controls fig 15 & fig 15a**

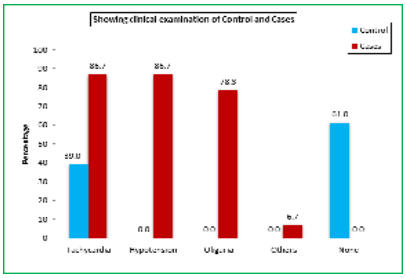


**Fig 15**
**fig15a**

**Table 15: Showing clinical examination of Control and Cases**

| Clinical Examination | Control | | Cases | | P value |
|---|---|---|---|---|---|
| | No | %age | No | %age | <0.001* |
| Tachycardia | 39 | 39.0 | 52.0 | 86.7 | |
| Hypotension | 0 | 0.0 | 52.0 | 86.7 | |
| Oliguria | 0 | 0.0 | 47.0 | 78.3 | |
| Others | 0 | 0.0 | 4.0 | 6.7 | |
| None | 61 | 61.0 | 0.0 | 0.0 | |

**Kidney function at presentation among cases and controls fig 16 & fig 16A**

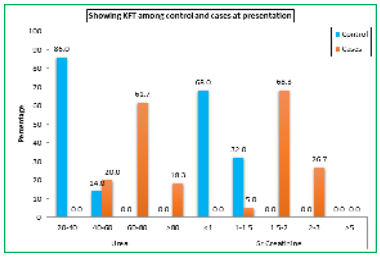


**Fig 15**
**fig15a**

**Table 16: Showing KFT among control and cases at presentation**

| Clinical Presentation | | Control | | Cases | | P value |
|---|---|---|---|---|---|---|
| | | No | %age | No | %age | |
| Urea | 20-40 | 86.0 | 86.0 | 0.0 | 0.0 | <0.001* |
| | 40-60 | 14.0 | 14.0 | 12.0 | 20.0 | |
| | 60-80 | 0.0 | 0.0 | 37.0 | 61.7 | |
| | >80 | 0.0 | 0.0 | 11.0 | 18.3 | |
| Mean±SD | | 34.5±0.18 | | 77.4±0.61 | | |
| Sr.Creatinine | <1 | 68.0 | 68.0 | 0.0 | 0.0 | <0.001* |
| | 1-1.5 | 32.0 | 32.0 | 3.0 | 5.0 | |
| | 1.5-2 | 0.0 | 0.0 | 41.0 | 68.3 | |
| | 2-3 | 0.0 | 0.0 | 16.0 | 26.7 | |
| | >5 | 0.0 | 0.0 | 0.0 | 0.0 | |
| Mean±SD | | 0.8±0.48 | | 1.9±0.78 | | |

**Medical management among cases and controls fig 17 & fig 17a**

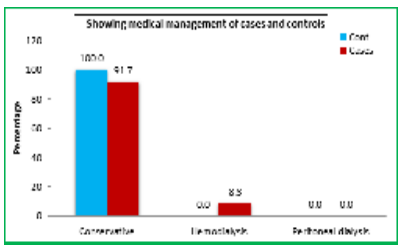


**Fig 17**
**Fig 17 a**

**Table 17: Showing medical management of cases and controls**

| Management | Control | | Cases | | Pvalue |
|---|---|---|---|---|---|
| | No | %age | No | %age | <0.001* |
| Conservative | 100.0 | 100.0 | 55.0 | 91.7 | |
| Hemodialysis | 0.0 | 0.0 | 5.0 | 8.3 | |
| Peritoneal dialysis | 0.0 | 0.0 | 0.0 | 0.0 | |
| Total | 100 | 100.0 | 60.0 | 100.0 | |

**Discussion :**

Acute diarrheal disease is a leading cause of illness globally. It is estimated that three to five billion cases of gastroenteritis resulting in 1.4 million deaths occur globally on an annual basis47,48 and those in the developing world being primarily affected.49 As acute gastroenteritis through volume depletion causes renal failure, failure to correct hypovolemia in time in acute gastroenteritis leads to acute tubular necrosis and renal failure. If hypovolemia persists, it results in tubular injury and irreversible damage leading to the need of renal

replacement therapy.44 If volume depletion is corrected before development of tubular injury, chances of acute renal failure (ARF) can be minimized. Studies had been done which showed that acute gastroenteritis is the one of the common cause of acute renal dysfunction.40,44The spectrum of renal failure in the adult population and the factors predicting poor outcome is not well defined in literature. Identification of risk factors and poor prognostic markers in these patients help in planning strategies to prevent AKI and to prioritize the utilization of sparse and expensive therapeutic modalities, especially in developing countries like ours. There is very little data in the incidence of AKI in India due to the lack of central registry. The etiology, course, and outcome differ in various parts of India.

The present study consisted of 160 patients above 18 years of age. We divided the patients into age groups like (18-24yrs, 25-44yrs, 45-64yrs and >65yrs) according to the standard survey classifications. Most of the patients in the case group (Group B) fall in the age group of 45-64yrs of age, with the mean age of 62.9yrs. Similarly most of the patients in the control group (Group A) fall in the age group of 45-64yrs of age, with mean age of 56.3yrs. There was a female preponderance in both the groups with 56% females in the control group (Group A) and 63.3% in the case group (Group B). Our results are consistent with the study carried out by Mahajan et al and Kumar et al.50,51 Sex distribution was different in Jayakumar et al52 study with males being 70.5%.This is probably due to varied etiology of the study, we have considered only one etiology that is gastroenteritis. The clinical features observed in our study was vomiting which was present in 53% of patients in control group and 78.4% in case group. Loose stools which was present in 81% of patients in control group and 100% of patients in case group. It was observed from the study that most patients in control group presented with in 24 hrs of their symptoms. However patients from the case group presented to the hospital for more than 48 hrs of their symptoms. This presentation to the hospital or healthcare facility has clinical significance as the patients who present early to the health care facility have low chances of developing acute kidney injury as compared to patients who present late, more dehydrated and thus high chances of AKI. Same results were observed by Baig MMI et al44, PK Chhetri et al40 Chirag et al.53The other clinical features which were observed in our study was fever in 12% of patients in control group and 15% of patients in study group. Pain abdomen in 11% in patients in control group and 9% in case group. Dysuria in 7% of patients in control group and 4% of patients in case group. Tachycardia and hypotension was present in 86% of patients in case group and only tachycardia in 39% of patients in control group. Oliguria was present in 78.3% of patients in case group and none had oliguria in control group. Other clinical features like drowsiness (altered sensorium) were present in 6.7% of patients in case group. The observations in Prakash et al54 study apart from vomiting,diarrhea were oliguria in 47%, anuria in 27%, CNS manifestations in 27%, bleeding diathesis in 10.3%, edema 12.9% and pulmonary edema in 4.2%.the disparity in clinical features among the two studies may be due to varied etiology considered by Prakash et al.54Most of the patients in our study on presentation had urea in the range of 60-80 mg/dl with mean of 77.4 in the case group and 20-40 mg/dl with mean of 37.5 in the control group. Creatinine was in the range of 1.5-2.0 with mean of 1.9mg/dl in the case group and <1mg/dl with mean of 0.8 mg/dl in the control group. These results were consistent with the study carried by the Pereira et al,55 J.Inbanathan et al.56The other complications noted in our patients (case group) were raised hematocrit in 80% of patients, acidosis with pH <7.35 in 60% of patients. Hyponatremia in 70% of patients with severe hyponatremia in 6.7% of patients. Hypokalemia was noted in 66.7% of patients and lactic acidosis in 88.3% of patients at presentation. In our study patients (case group) albuminuria was noted in13.3% of patients and glycosuria in 16.7% of patients. Our results are consistent with the study carried out by J.Inbanathan et al56 who studied total of 100 patients and detected albuminuria in 13% of patients and glycosuria in 14.9% of patients. Microscopic examination of urine revealed pyuria in 15% of patients, RBC in 10% of patients and casts in 20% of patients. However on following these patients none had features of intrinsic renal disease like glomerulonephritis and moreover these findings can be normally seen in severe prolonged pre renal AKI and also in viral infections. Moreover most of the casts where acellular ( hyaline, granular). In our study patients (group B) 70% of patients had raised renal echo pattern on USG and 30% of patients had normal USG. These results were in consistent with the study conducted by J.Inbanathan et al.56Most of the patients in our study (group B) presented in theinjury stage 80% according to RIFLE staging of the acute kidney injury.13.3% patients presented in failure stage and 6.7% presented in risk stage. These results are consistent with the study conducted by Neha Balkunde et al,57 where 16.4% patients are in Risk group, 74.3% in Injury and 9.3% in Failure group. Comorbid conditions noted in our patients where Hypertension in 56.7%, diabetes mellitus 40%, Ischemic heart disease 5%, COPD 5%, hypothyroidism 8.3% of patients. These results were consistent with the study done by J.Inbanathan et al.56 However in Prakash et al54 study comorbidity was seen in 52% of patients; hypertension (34.7%), diabetes mellitus (28.3%) and coronary artery disease (30.4%). The difference was probably because the study was done in ICU setting only .In our study all the patients from group A were managed conservatively. However in Group B 91.7% of patients were managed conservatively and 8.7% of patient's required renal replacement therapy in the form of hemodialysis. The requirement of hemodialysis in these patients were because of rapidly deteriorating renal function, uremic symptoms, volume overload and rapid worsening of other lab parameters like acidosis, hyperkalemia, anuria. The requirement of hemodialysis in our study was similar to the study conducted by Neha balkunde et al.57 Contrary to our study dialysis requirement in Jayakumar et al52 was 30% and in the program to improve care in acute renal disease (PICARD) study was 34%. This was probably due to indications for dialysis considered, difference in study population and probably because PICARD study was undertaken in critically ill patients with varied etiology considered.58 In our study most of the patients in group A were discharged with in 24 hrs and most of the patients in group B were discharged within 5 days with mean duration of hospital stay in group A of 0.8 days and in group B of 3.7 days which is statistically significant ($p<0.0001$). Our results were consistent with the study carried out by Archana deshpande et al,57 J. Inbanathan et al.56 However the median length of hospital stay was 25 days in PICARD study. This was probably because PICARD study was undertaken in critically ill patients and varied etiology considered. In our study there was no mortality (0%). In J.Inbanathan et al56 study, the mortality was 4%. The most significant factor for mortality was the time interval from the onset of ADD to the diagnosis of ARF. The difference in mortality rate was probably due to increasing percentage of patients needed RRT (30%) in this study and varied indications for dialysis. The other prognostic factors which affect the mortality are multiorgan failure, circulatory failure, and vasopressor support. In our study we followed the group B patients i.e those patients who developed AKI after ADD for a period of six months with serial monitoring of their renal functions at one month, three month and at six months. We used RIFLE staging and GFR (MDRD equation) to study the follow up.After one month most of the patients had recovered their renal functions except 28.3% which were still in the Risk stage of Rifle staging. Further follow up of these patients at 3 months of their discharge revealed that 13.3% are still in the Risk group and among them 5% (3 patients) had GFR below 60 ml/min/1.73m2 and were categorised as having CKD. One among these three patients had received hemodialysis during the hospital stay. On further follow up at 6 months only 8.3% of patients were in the Risk group and 5% out of this 8.3% were in CKD. Studies performed by Pereria et al9 and Chawla et al10 showed that the older age and the severity of AKI predicted the progression to CKD. The most severe AKI patients evaluated by the RIFLE or AKIN criteria presented lower rate of recovery of renal function at the time of hospital discharge and higher progression to CKD. Thakur et al studied patients with diabetes and risk of progression to CKD after ATN episodes. It was observed that an episode of AKI in diabetic patients compared with diabetic patients without ATN was associated with progression to CKD.

## Conclusion:

As severity and duration of dehydration increases, risk of AKI increases. Also as the stage of renal injury advances, duration of hospital stay increases and clinical outcome become poorer. Out of many causes of AKI, Acute diarrheal diseases are important cause of preventable AKI in India. So, early referral and adequate replacement of fluid and electrolyte can improve outcome in these patients.

## REFERENCES:


1. Sushrut S Waiker, Joseph V. Bonventre:Acute kidney injury. Harrison's principles of internal medicine19e;2:1799-1811.
2. Jayakumar M,Ram Prabakar H, Edwin Fernando M, et al: Epidemiological trend changes in ARF: A teritary centre experience from south india. Renal failure 2006;28:405 - 10.
3. Muthusethupathi MA, Shiva Kumar S: Acute renal failure in South India. J Assoc Physicians India 1987;35:504-7
4. Liano F,Pascual J: Epidemiology of Acute renal failure: a prospective, multicentre,

community based study, Madrid, Spain. Acute Renal Failure Study Group 2000;294:813-818.
5. Jorge Cerda,Norbert Lameire, Paul Eggers, Neesh Pannu, Sigehiko Uchino, Haiyan Wang,Arvind Bagga and Adeera Levin: Epidemiology of acute kidney injury. Clin J Am Soc Nephrol 2006;1:43-51
6. J Prakash,K Tripati,Malhothra v,om Kumar,Srivastsa: Acute renal failure in Eastern India. J Assoc Physicans India 1994;28:327-332
7. JorresAJ:Acute renal failure : pathogenesis, diagnosis and conservative treatment.Minerva Med2009;93:85-93.
8. Morgera S, Kraft AK ,Siebert G, Luft FC, Neumayer HH: Long term outcomes in acute renal failure patients treated with continous renal replacement therapies . Am J Kidney disease 2002;40:275-9.Kelly KJ, Molitoris BA.
9. Pereira MB, ZanettaDM,AbdulkaderRC:The renal importance of pre existing comorbidities on long term mortality after acute kidney injury. PLoS One 2012;7:e47746.DOI: http://dx.doi.org/10.1371/journal. pone.0047746
10. Chawla LS, Amdur RL,AmodeoS,Kimmel PL,Palant CE:The severity of AKI predicts progression to chronic kidney disease.Kidney Int2011;79:1361-9.PMID:21430640DOI: http://dx.doi.org/10.1038/ki.2011.42
11. Brito GA,Babli AL, Abrao JM , Ponce D: Long term outcome of patients followed by nephrologists after an ATN episode .Int J Nephrol 2012; 2012:361528.PMID:23227335 DOI: http://dx.doi.org/10.1155/2012/ 361528
12. Macedo E, Zanetta DM, Abdulkader RC: Long term follow up of patients after AKI:patterns of renal functional recovery. PLoS One 2012;7:e36388.DOI: http://dx.doi.org/10.1371/journal.pone.0036388
13. Amdur RL, Chawla LS, Amodeo S Kimmel PL, Palant CE: Outcomes following diagnosis of ARF in U.S. veterans:focus on ATN. Kidney Int 2009; 76:1089-97.PMID:19741590 DOI: http://dx.doi.org/10.1038/ki. 2009.332
14. Wu VC, Huang TM , Lai CF, Shaio CC, Lin YF, Chu TS , et al: Acute on chronic kidney injury at hospital discharge is associated with long term mortality. Kidney Int2011; 80:1222-30.PMID:21832983 DOI: http://dx. doi.org/10.1038/ki.2011.259
15. Thakar CV,Christianson A,Himmelfarb J,Leonard AC: AKI episodes and CKD risk in diabetes mellitus. Clin J Am Soc Nephrol 2011;6:2567-72. DOI: http://dx.doi.org/10.2215/CJN.01120211
16. Suzanne Watnick, Tonja Dirkx:Acute kidney injury. CMDT 2015;893-95.
17. R. Bellomo, et al. Critical Care, vol. 8, no. 4, pp. R204-R212, 2004.
18. R. L. Mehta, et al. Critical Care, vol. 11, article R31, 2007.
19. Simon Steddon, Neil Ashman, et al:Acute kidney injury. Oxford Handbook of Nephrology and Hypertension 2e; 88-92.
20. King CK, Glass R, Bresse JS, Duggan C; Centers for Disease Control and Prevention. Managing acute gastroenteritis among children: oral Rehydration, maintenance, and nutritional therapy. MMWR Recomm Rep. 2003;52(RR-16):1-16.
21. Elliott EJ. Acute gastroenteritis in children. BMJ. 2007;334(7583):35-40.
22. Committee on Nutrition. Oral therapy for acute diarrhea. In: Kleinman RE, ed. Pediatric Nutrition Handbook. 6th ed. Elik Grove Village,IL: American Academy of pediatrics;2009:651-659.
23. Kenneth R. McQuaid:Diarrhea. CMDT 2015;571-574.
24. Benjamin Clark and Mike McKendrick:A review of viral Gastroenteritis. Current Opinion in Infectious Diseases 2004,17:461-469
25. Ahuja MM. Progress in Clinical Medicine in India. 3 rd ed. New Delhi: Arnold-Heinemann Publishers; 1979. p. 312-26.
26. Chertow GM, Burdick E, Honour M, et al. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. J Am Soc Nephrol 2005; 16:3365
27. RENAL Replacement Therapy Study Investigators, Bellomo R, Cass A, etal. An observational study fluid balance and patient outcomes in t Randomized Evaluation of Normal vs. Augmented Level of Replacement Therapy trial. Crit Care Med 2012; 40:1753.
28. Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 2004 Jul. 114(1):5-14.
29. Kaplan JE, Feldman R, Campbell DS, et al. The frequency of a NorwalkLike pattern of illness in outbreaks of acute gastroenteritis. Am J Public Health1982; 72:1329.
30. Lopman BA, Reacher MH, Vipond IB, et al. Clinical manifestation of norovirus gastroenteritis in health care settings. Clin Infect Dis 2004; 39:318.
31. Bresee JS, Marcus R, Venezia RA, et al. The etiology of severe acute gastroenteritis among adults visiting emergency departments in theUnited States. J Infect Dis 2012; 205:1374.
32. Jones R, Rubin G. Acute diarrhoea in adults. BMJ 2009; 338:b1877.
33. Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention. Updated norovirus outbreak management and disease prevention guidelines. MMWR Recomm Rep 2011; 60:1.
34. Riddle MS, DuPont HL, Connor BA. ACG Clinical Guideline: Diagnosis, Treatment, and Prevention of Acute Diarrheal Infections in Adults. Am J Gastroenterol 2016; 111:602.
35. Avery ME, Snyder JD. Oral therapy for acute diarrhea. The underused simple solution. N Engl J Med 1990; 323:891.
36. Santosham M, Burns B, Nadkarni V, et al. Oral rehydration therapy for acute diarrhea in ambulatory children in the United States: a double-blind Comparison of four different solutions. Pediatrics 1985; 76:159.
37. Khan WA, Seas C, Dhar U, et al. Treatment of shigellosis: V. Comparison Of azithromycin and ciprofloxacin. A double-blind, randomized, controlled trial. Ann Intern Med 1997; 126:697.
38. Shane AL, Mody RK, Crump JA, et al. 2017 Infectious Diseases Society Of America Clinical Practice Guidelines for the Diagnosis and Management of Infectious Diarrhea. Clin Infect Dis 2017; 65:e45.
39. Garzotto F, Piccinni P, Cruz D, Gramaticopolo S, Dal Santo M, Aneloni G, Kim JC, Rocco M, Alessandri E, Giunta F, Michetti V. RIFLE-based data collection/management system applied to a prospective cohort multicenter Italian study on the epidemiology of acute kidney injury in the intensive care unit. Blood purification. 2011;31(1-3): 159-171.
40. Chhetri PK, Manandhar DN, Pahari LR, Chhetri SS, Chaudhary SK. Acute renal failure in Nepal medical college teaching hospital. Nepal Med Coll J 2008;10(2):132-135.
41. Chijioke A, Makusidi AM. Severe Acutekidney Injury in Adult Nigerians From University of ILORIN Teaching Hospital, Ilorin, Kwara State. BOMJ 2011;8:1.
42. Gentian P. Stroni, Majlinda M. Dhimolea, Pëllumb S. Pipero, Dhimiter V. Kraja, Suela Y. Sallavaci, and Silva F. Bino2Balkan Med J. 2014 Sep;31(3): 196–201.
43. Zollner-Schwetz and Krause R. Therapy of acute gastroenteritis: role of antibiotics. Clin Microbiol Infect 2015; 21: 744–749.
44. Baig MMI N,Randhawa FA. Renal involvement in acutegastroenteritis; our population at presentation and its outcome with follow up for 6 months. Professional Med J. 2012 Dec;19(6):905-908.
45. Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest. 2004 Jul. 114(1):5-14.
46. Lauschke A, Teichgräber UK, Frei U, Eckardt KU. Low-dose' dop worsens renal perfusion in patients with acute renal failure. Kidney I 2006 May. 69(9):1669-74.
47. Elliott, Elizabeth Jane. "Acute gastroenteritis in children." BMJ: British Medical Journal 2007 Jan 4;334(7583): 35-40.
48. Lozano, R., Naghavi M, Foreman K, Lim S, Shibuya K, AboyansV, Abraham J,Adair T,Aggaewal R, Ahn SY,AlMazroa MA. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010.The Lancet 2013 Jan 4;380(9859): 2095-128.
49. Webber R,editor. Communicable disease epidemiology and control: a global perspective. Cabi,3rd ed. 2009.p79.
50. Mahajan S, Tiwari S, Bhowmik D, Agarwal SK, Tiwari SC, Dash SC. Factors affecting the outcome of acute renal failure among the elderly population in India: A hospital based study. Int Urol Nephrol 2006;38:391-6.
51. Kumar SS, Paramananthan R, Muthusethupathi MA. Acute renal failure due to acute diarrhoeal diseases. J Assoc Physicians India 1990; 38:164-6.
52. Jayakumar M, Prabahar MR, Fernando EM, Manorajan R, Venkatraman R, Balaraman V. Epidemiologic trend changes in acute renal failure – A tertiary center experience from South India. Ren Fail 2006;28:405-10.
53. Chirag J Patel, Amit Desai, Vidur Joshi, Binit Jhaveri: Clinical Profile and Management of Patients Having Acute Gastroenteritis Induced Acute Kidney. Injury JMSCR Vol05,Issue06,Page 23492-23496||June17.
54. Prakash J, Tripathi K, Malhotra V, Kumar O, Srivastava PK. Acute renal failure in Eastern India. Nephrol Dial Transplant 1995;10:2009-12.
55. Pereira MB, ZanettaDM,AbdulkaderRC:The renal importance of pre existing comorbidities on long term mortality after acute kidney injury. PLoS One 2012;7:e47746.DOI: http://dx.doi.org/10.1371/journal. pone.0047746.
56. Inbanathan J, Lavanya BU: Clinical Profi le of Renal Involvement in Acute Gastroenteritis Patients. Int J Sci Stud 2016;4(8):48-52.
57. Neha Balkunde,Archana Deshpande: Study of Acute Kidney Injury in Acute Gastroenteritis. Vidarbha Journal of Internal Medicine 2017; 23:175-10.
58. Mehta RL, Pascual MT, Soroko S, Savage BR, Himmelfarb J, Ikizler TA, et al: Spectrum of acute renal failure in the Intensive Care Unit: The PICARD experience. Kidney Int 2004;66:1613-21.
59. G.A.Brito, A. L. Balbi, J. M. G. Abrão, and D: Ponce. Long-Term Outcome of Patients Followed by Nephrologists after an Acute Tubular Necrosis Episode. International Journal of Nephrology Volume 2012, Article ID 361528, 7 pages
60. Luana Pôncio, Andre Luis Balbi, Érica Pires da Rocha, Dayana Bitencourt Dias,Daniela Ponce: The long-term outcome after acute kidney injury: a narrative review. J. Bras. Nefrol. vol.37 no.1 São Paulo Jan./Mar. 2015.
61. Ponce D, Dias DB, Nascimento GR, Silveira LV, Balbi AL: Long term outcome of severe acute kidney injury survivors followed by nephrologists in a developing country. Nephrology (Carlton). 2016 Apr;21(4):327-34.