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General Medicine

A STUDY ON CLINICAL PROFILE AND OUT-COME OF ACUTE KIDNEY INJURY IN ACUTE GASTROENTERITIS IN A TERTIARY CARE CENTRE

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ABSTRACT In India, acute kidney injury (AKI) due to diarrhea is not uncommon in adults and elderly people. Therefore, understanding of the clinical spec-trum of the disease is needed to devise methods to improve the final out-come due to this problem.

KEYWORDS : Acute Kidney Injury, Acute Tubular Necro-sis,novel Biomarkers For Aki.

INTRODUCTION

Acute kidney injury is a syndrome characterized by rapid (hours to weeks) decline in glomerular filtration rate and retention of ni-trogenous waste products such as blood urea nitrogen and creatinine[1]. It is one of the most commonly encountered in clinical prac-tice[2]. Acute kidney injury occurs in response to a wide range of insults to kidney such as septic shock, drug toxicity[3]. AKI com-plicates approximately 5% of hospital admissions and up to 30% of admissions into intensive care units[4]. As diarrhoea is one of the common causes of AKI in tropics, this study was conducted to evaluate clinical and biochemical aspects and outcome of ARF due to gastroenteritis.

AIMS AND OBJECTIVES

This study was carried out

1. To know the outcome of prompt rehydration and treatment in patient presenting with Acute kidney injury due to Gastroenteritis.
2. To study the clinical profile of patients with AKI following Gastroenteritis.
3. To correlate clinical features and laboratory parameters with outcome of AKI and to identify the poor prognostic factor.
4. To analyze the laboratory parameters in patients with AKI following Gastroenteritis.

MATERIALS AND METHODS

STUDY AREA

The study was conducted in the Dept of General Medicine ASRAM Hospital, ELURU.

STUDY PERIOD

The study was conducted between December 2016 and July 2018 .

DESIGN OF STUDY

Prospective study.

SAMPLE SIZE

The study population included 60 patients of Acute kidney injury due to GE admitted to the hospital.

INCLUSION CRITERIA

1. Presence of clinical manifestation of gastroenteritis
2. All patients of either sex diagnosed as having Acute kidney injury due to Gastroenteritis.

EXCLUSION CRITERIA

All patients diagnosed as Acute kidney injury due to causes other than Gastroenteritis.

CONSENT

Informed consent was obtained from the patients.

STUDY PROTOCOL

Data was collected from sixty patients admitted to this hospital during December 2016 to July 2018.

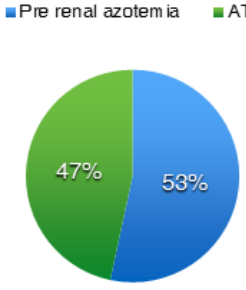
A detailed history and a clinical profile of these patients were recorded.The hydration status at time of admission was recorded as follows:

1. No signs of dehydration
2. Mild dehydration in the form of dryness of mucosa.
3. Moderate dehydration with loss of skin turgor.
4. Severe dehydration with hypotension
5. Fluid overload with pedal and/or pulmonary edema

Renal function tests with electrolytes are done daily and recorded. Other laboratory parameters such as CBC, ESR,Urine examination, Stool Examination,HIV,blood glucose, liver function test are also done.

These patients were given adequate fluid replacement Antibiotics which have gram negative spectrum are started to the patients with ongoing gastroenteritis. Dialysis was done in patients with hyperkalemia, pulmonary edema and severe metabolic acidosis, who did not respond to medical treatment and prophylactically in patients whose creatinine is more than 4 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 analyzed to assess the role of each of these factors as the possible outcome i.e. recovery or death.



RESULTS

Out of 60 patients,
Mean age of presentation 46.5 ± 11.35years.
The mean age of presentation in males was 47.3 ±11.35 and that of females in 45.72 ± 10.23.
Out of 60 patients 38 (63.33 percent) were Males and 22 (36.3 percent) were Females.

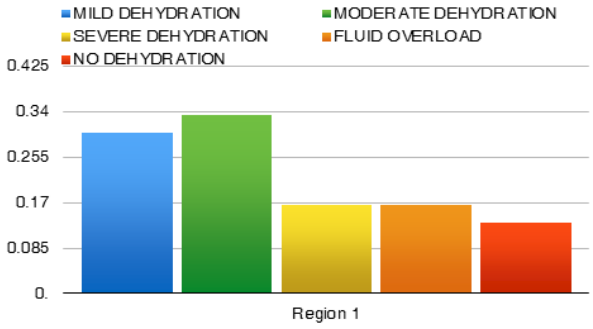
The commonest type of renal failure in our study was Pre Renal Azotemia[53.3%] followed by Acute Tubular Necrosis[46.6%]

Out of 60 patients prospectively studied 56 patients (93.3 percent)survived. All other 4 patients who expired belong to ATN group.(6.7%)

Majority of patients 20 (33.33%) had moderate dehydration. 18(30%) patients had mild dehydration and in 10 (16.6%)patients it was severe.

Fluid overload was observed in 10(16.6%) patients and there was no dehydration in 8 (13.33%)patients. Majority of non survivours be-long to group of moderate dehydration.

Other manifestation at the time of admission
Fever occurred in 9 (15%) patients, followed by abdominal pain in 5 (8%) patients.



Hypotension was observed in 2 patients and were expired.
The mean s.creatinine 5.95 ± 1.62 in survivours and 5.1 ± 2.81 in non survivoirs. It was 3.86 ± 1.82 in prerenal and ATN it was 4.2 ± 2.13 .The peak urea level (80 ± 21.5) in survivors and 76 ± 18.31 in non survivors
26 (41.6%) patients had hyponatremia (<125meq/l).
18 (32.7%) patients had hyponatremia (>150 meq/L).
50 patients (83%) had hypokalemia(<3.5).
Hyperkalemia (>5 meq/dl) occurred in 24 (40%) patients.
Stool Examination showed E.COLI in 20 pts, KLEBSIELLA in 10 pts, E.HISTOLYTICA in 15 pts and negative in 15 pts.
52 pts were managed conservatively and 8 underwent hemodialysis.
out of these 8, 4 survived and 4 died.
3 patients died due to septicemia and 1 patient had Multi organ failure.

DISCUSSION

Acute kidney injury (AKI) is a clinical syndrome that complicates the course and worsens the outcome in a significant number of hospitalised patients.AKI is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and im-pairment (loss of function).The current diagnostic approach of AKI is based on an acute decrease of GFR, as reflected by an acute rise in sCr levels and/or a decline in UO over a given time interval[5]

AKI is *defined* as any of the following:	
1	Increase in sCr ≥0.3 mg/dL (≥26.5 µmol/L) within 48 hours; or
2	Increase in sCr ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
3	Urine volume <0.5 mL/kg/h for 6 hours.

KDIGO explicitly states that a *rolling baseline* can be used over 48-hour and 7-day periods for diagnosis of AKI, while in RIFLE or AKIN it is not clear how this is handled.

Classification of AKI includes pre-renal AKI, acute post-renal obstructive nephropathy and intrinsic acute kidney diseases. Of these, only 'intrinsic'AKI represents true kidney disease, while pre-renal and post-renal AKI are the consequence of extra-renal diseases leading to the decreased glo-merular filtration rate (GFR). If these pre- and/or post-renal conditions persist, they will eventually evolve to renal cellular damage and hence in-trinsic renal disease.[6]Due to the climatic conditions, overcrowding and poor socioeconomic factors, AKI in India differs from the world. Most common causes of AKI in India are acute diarrheal disease, malaria, lep-tospirosis, snakebite, insect stings, intravascular hemolysis due to septi-cemia, chemical poisoning.Overall, these causes constitute 40% AKI in India. [7,8] Complications include hypotension, dyselectrolemia, metabolic acidosis and hyperuricemia, arrhythmias.

The evaluation and initial management of patients with acute kidney in-jury (AKI) should include: (1) an assessment of the contributing causes of the kidney injury, (2) an assessment of the clinical course including comorbidities, (3) a careful assessment of volume status, and (4) the insti-tution of appropriate therapeutic measures designed to reverse or prevent worsening of functional or structural kidney abnormalities.

Early in the course of AKI, optimization of the hemodynamic status and correction of any volume deficit will have a salutary effect on kidney functionDiuretics do not have any significant effect on progression or outcome of AKI [9]Traditional blood (creatinine, blood urea nitrogen) and urine markers of kidney injury (epithelial cells, tubular casts, frac-tional excretion of Na+, urinary concentrating ability, etc.) are insensitive and nonspecific for the diagnosis of AKI.

Efforts to identify biomarkers to assist with the early diagnosis of AKI , such as KIM-1, NGAL, IL-18, Cys-C, clusterin, FABP, and osteopontin. A single biomarker may not be adequate to define AKI [10]

CONCLUSION

1. Gastroenteritis was one of the leading causes of ARF accounting for 24% of all the patients with ARF admitted in our hospital.
2. Among the 60 patients prospectively studied 46.6% of the patients had pre-renal azotemia, 53.3% of the patients had ATN.
3. Male patients were more compared to Females as males are more com-monly exposed to contaminated food and poor sanitary conditions dur-ing their course of occupation.
5. The most common electrolyte abnormality noted at the time of admis-sion was hypokalemia is found in 83% of the patients.
6. Septicemia was the commonest complication occurring in 10 patients. Seven of them expired.
7. Management consisted of fluid replacement, correction of electrolyte abnormalities administration of appropriate antibiotics. Dialysis was done in 21 patients. Haemodialysis was done in 21 patients.
8. ARF following gastroenteritis differs from other causes of ARF by fre-quent occurrence of hypokalemia and has a better prognosis.

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