http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v8i12.43

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

A Prospective Study on Aki Clinical Profile and Outcome Secondary to Acute Gastroentiritis

Authors
Dr P. Vijay Kumar[1], Dr T. Sai Phanindra[2], Dr B Gowri Sankar[3], M. Ranjini[4]
[1,2,4]Post Graduate Dept of General Medicine GEMS Medical College
[3]Assistant Professor, Department of General Medicine, GEMS Medical College

Abstract
AKI secondary to infectious and non-infectious diseases is a common condition in India. In that AKI secondary to diarrheal diseases are also not uncommon. Therefore it is important to know its clinical spectrum to devise out methods to improve final outcome of the disease.

Introduction

AKI refers to abrupt (hours to days) decline in renal functions, resulting in accumulation of nitrogenous waste products like urea and creatinine; alteration of body homeostasis in the form of extracellular volume and electrolytes dysregulation[1]. It can be seen in up to 7% of hospital admissions and 30% ICU admissions. AKI results due to wide variety of causes, like infectious diseases, poisonings, trauma, chemical toxins etc. In India AKI due to community acquired diseases is more common[2].As diarrhea is one of the common cause of AKI in tropics, this study was conducted to evaluate clinical, biochemical aspects and outcomes of AKI due to gastroenteritis.

Aims and Objectives

1. To study the clinical presentation of patients with AKI secondary to gastroenteritis.
2. To correlate clinical presentation with outcome of AKI.
3. To analyse laboratory parameters in patients with AKI secondary to acute gastroenteritis.
4. To know the outcome of prompt fluid replacement in AKI.

Materials and Methods

Study Area- the study was conducted in Dept. of General Medicine Great Eastern Medical School and Hospital, Ragolu, Srikakulam.

Study Design- Prospective Study.

Inclusion Criteria- all patients with AKI clinical presentation and diagnosis secondary to Gastroenteritis.

Exclusion Criteria- Patients diagnosed with AKI secondary to other than Acute Gastroenteritis.

Consent- Informed consent was obtained from the patients.

Study Protocol- data was collected from 50 patients admitted to this hospital during December 2018- February 2020.

Detailed history and clinical presentation were noted.

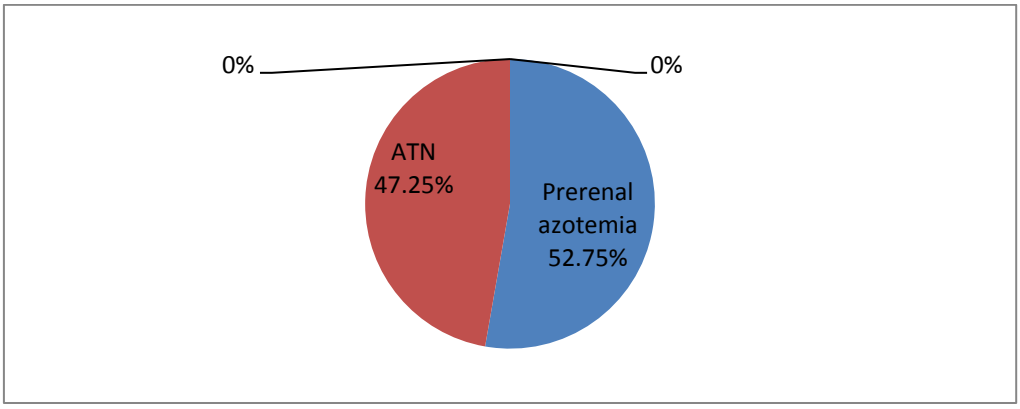
1. On clinical examination at the time of admission hydration status of the patients noted as no dehydration; mild dehydration(dryness of mucosa); moderate dehydration (loss of skin turgor); severe dehydration (with evidence of hypotension); fluid overload state (pedal/pulmonary edema).
2. Laboratory tests-CBC, ESR, random blood sugar, serum creatinine, blood urea, urine routine examination, serum electrolytes, stool examination, viral markers, liver function tests, arterial blood gas analysis.
3. Radiological tests-chest x- ray, USG abdomen.
4. Patients were given appropriate treatment based on their clinical conditions like fluid management, broad spectrum antibiotics, conservative medical management; supportive care like hemodialysis.

Results

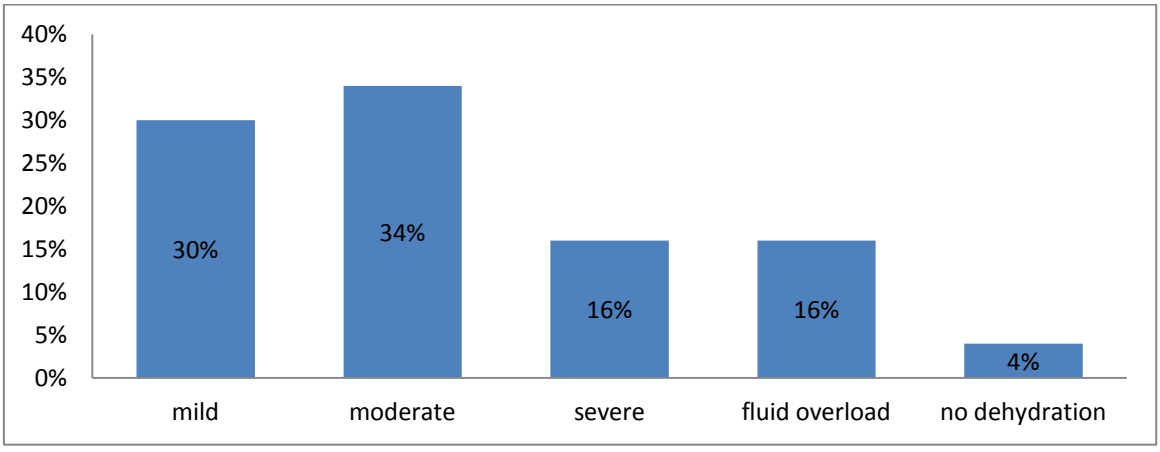
Out of 50 patients -32patients were males, 18 were females.

Mean age of presentation 45.5 ± 11.5 years. Out of 50 patients 47 patients were survived, 3 patients were expired.

The commonest type of renal failure in our study was prerenal azotemia (52.75%) followed by acute tubular necrosis (47.25%).



Out of 50 patients hydration status –no dehydration in 2(4%) patients, mild dehydration in 15(30%) patients, moderate dehydration in 17(34%) patients, severe dehydration in 8(16%)patients, fluid overload state in 8(16%) patients. 80%(40) of patients had some amount of dehydration



Based on fluid status in the body at the time of admission

Electrolyte abnormality-Out of 50 Hyponatremia-21(42%), Hypernatremia-16(32%),Hyperkalemia-12(24%), Hypokalemia-38(76%) patients. Mean sr.creatinine 5.9 ± 1.6 in survivors and 5.1 ± 2.8 in non survivors. Blood urea levels 60 ± 21.5 in survivors and 76 ± 18.5 in non survivors. Out of 50 patients in our study majority belongs to pre renal azotemia group 52.75% followed by acute tubular necrosis 47.25%. 3 patients who expired in this study group belongs to ATN group.

Out of 50- 44pts were managed conservatively and 6 underwent hemodialysis, out of these 3pts died, in that 1 pts had multiorgan failure and 2 died due to sepsis.

Discussion

AKI (formally known as ARF) is a common clinical syndrome that complicates the clinical outcome in many hospitalized patients. There are many diagnostic criteria proposed for early detection in order to prevent AKI induced complications, like RIFLE(2002); AKIN (2005); KDIGO-2012(Kidney Disease Improving Global Outcomes) is the most recent work group combined both RIFLE and AKIN criteria and defines AKI as follows:

1. Increase in serum creatinine by 0.3mg/dL or more within 48 hours or
2. Increase in serum creatinine to 1.5 times baseline or more within the last 7 days or
3. Urine output less than 0.5 mL/kg/h for 6 hours.

KDIGO staging for severity of AKI[3,4]

Stage	Serum Creatinine	Urine Output
1	1.5-1.9 times baselineor ≥0.3 mg/dL increase	< 0.5 mL/kg/h for 6 h
2	2-2.9 times baseline	< 0.5 mL/kg/h for 6 h
3	3 times baseline or Increase in serum creatinine to ≥4 mg/dL or Initiation of renal replacement therapy	< 0.3 mL/kg/h for 24 h or Anuria for ≥12 h

AKI noted in hospitalized diarrheal illness approximately accounts for 1 in 10 persons[5]. A 5-fold increase in mortality is seen. In India mean age of presentation was 37.1 years[6]. Elderly individuals; Underlying comorbid conditions like CKD and hypertension were associated with the increased risk of AKI in both infectious and non-infectious diarrheal illness. Whereas episodes of infectious diarrhea related AKI are often acute in onset, self-limited, and reversible.AKI was most common in elderly but younger age group experience worse outcomes. Delay in diagnosis plays a detrimental role.

Aki is categorized into pre renal causes, intrinsic renal causes, post renal causes[7].AKI in tropics is mostly CA-AKI rather than HA-AKI due to climatic conditions and population[8]. Tropical causes includes due to infections like malaria, leptospirosis, typhoid, hemorrhagic fevers, infective diarrhea, HIV; trauma, plant toxins, snake bite, chemical toxins, etc. complications include hypotension, dyselectrolemia, metabolic acidosis, hyperuricemia and arrhythmias, and death.

Evaluation and management of AKI includes (1) assessment of clinical course and careful assessment of volume status, (2)assessment of contributing cause and initiate treatment, (3)institution of appropriate therapeutic measures to reverse or prevent worsening of functional or structural kidney condition by giving prompt fluid therapy, vasopressin if needed, antibiotics, supportive treatment.

Drugslike diuretics and RAAS blockers (ACE inhibitors, ARBs) impairs body's natural renal auto-regulatory response in early volume depletion state, resulting in a sharp decline in filtration fraction and GFR. Some broad spectrum antibiotic use could lead to acute interstitial nephritis[9]. Diuretics may be needed during the

oliguric phase of ATN if significant volume overload is present. National Institute for Health and Care Excellence (NICE) suggests temporary cessation of ACE inhibitors or ARB therapy could be considered in acute illness. Furosemide is frequently used drug to facilitate fluid and electrolyte management of acute kidney injury in many institutions, yet in terms of benefits and adverse effects in treatment of AKI remains uncertain. Some Meta analysis states that diuretics do not have any significant effect on progression or outcome of AKI. Our data support the need for further randomized trials in this aspect as it is most commonly instituted treatment in view of cost effectiveness treatment of AKI. One particular challenge is judging the severity of the illness versus the benefits of continuation of therapy.

Biomarkers for AKI - S.Cr, is the gold-standard marker for renal function but levels are affected by age, sex, and body mass as well as dietary factors and volume status. urine microscopy is also insensitive and non specific. So need for usage of noval biomarkers like **Serum and urinary cystatin C, Serum and urinary NGAL, Urinary KIM-1,Urinary IL-18,Urinary L-FABP, NAG** in early detection is present[10]. They are not much in clinical use due to less availability and cost.

Conclusion

1) Gastroenteritis is one of the leading cause of ARF in hospital setting.
2) Gender predominance is usually male sex.
3) Pre renal azotemia is common than ATN due to GE in our study it corresponds to 52.75% pre renal azotemia and 47.25% ATN.
4) Most common electrolyte abnormality in our study is hypokalemia accounting for 76%.
5) Septicemia was the commonest complication seen in 10 patients out of it 2 were expired.
6) Management consists of fluid replacement therapy, electrolyte correction, appropriate antibiotics, supportive care like HD.
7) ARF followed by acute GE usually is a preventable cause of death as it has good prognosis, so at most attention should be given for early detection and management.

References

1. Palevsky PM. Endpoints for clinical trials of acute kidney injury. Nephron. 2018;140(2):111-5.
2. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. Saudi Journal of Kidney Diseases and Transplantation. 2018 Jul 1;29(4):956.
3. Machado MN, Nakazone MA, Maia LN. Lesão renal aguda baseada nos critérios KDIGO (Kidney Disease: Improving Global Outcomes) empacientes com creatininaséricaelevadasubmetidos à cirurgiacardíaca. Brazilian Journal of Cardiovascular Surgery. 2014 Sep;29(3):299-307.
4. Wiersema R, Jukarainen S, Eck RJ, Kaufmann T, Koeze J, Keus F, Pettilä V, van der Horst IC, Vaara ST. Different applications of the KDIGO criteria for AKI lead to different incidences in critically ill patients: a post hoc analysis from the prospective observational SICS-II study. Critical Care. 2020 Dec;24:1-8.
5. Mathew AJ, George J. Acute kidney injury in the tropics. Annals of Saudi medicine. 2011 Sep;31(5):451-6.
6. Jayakumar M, Ram Prabahar M, Fernando EM, Manorajan R, Venkatraman R, Balaraman V. Epidemiologic trend changes in acute renal failure—a tertiary center experience from South India. Renal failure. 2006 Jan 1;28(5):405-10.
7. Moresco RN, Bochi GV, Stein CS, De Carvalho JA, Cembranel BM, Bollick YS. Urinary kidney injury molecule-1 in renal

disease. Clinica Chimica Acta. 2018 Dec 1;487:15-21.

8. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. Saudi Journal of Kidney Diseases and Transplantation. 2018 Jul 1;29(4):956.
9. Lameire NH, Bagga A, Cruz D, De Maeseneer J, Endre Z, Kellum JA, Liu KD, Mehta RL, Pannu N, Van Biesen W, Vanholder R. Acute kidney injury: an increasing global concern. The Lancet. 2013 Jul 13;382(9887):170-9.
10. Schmid M, Dalela D, Tahbaz R, Langetepe J, Randazzo M, Dahlem R, Fisch M, Trinh QD, Chun FK. Novel biomarkers of acute kidney injury: Evaluation and evidence in urologic surgery. World journal of nephrology. 2015 May 6;4(2):160.