Original Research Article

Clinical profile of patients presented with acute kidney injury

Amin BK¹, Parmar SJ², Talsaniya KA^{3*}, Gangadia YS⁴, Mori MB⁴, Dhanani KV⁴

¹Professor, ²Associate Professor, ³Assistant Professor, ⁴Resident Doctor Department of General Medicine, B J Medical College, Ahmedabad, Gujarat, India ^{*}Corresponding author email: **krunal.talsaniya@gmail.com**

	e	Medicine, Vol. 6, Issue 3, March, 2019. M, All Rights Reserved.			
	Available online at <u>http://iaimjournal.com/</u>				
John L	ISSN: 2394-0026 (P) ISSN: 2394-0034 (O)				
IAIM	Received on: 08-02-2019	Accepted on: 15-02-2019			
TAIM	Source of support: Nil Conflict of interest: None decla				
How to cite this article: Amin BK, Parmar SJ, Talsaniya KA, Gangadia YS, Mori MB, Dhanani KV.					
Clinical profile	Clinical profile of patients presented with acute kidney injury. IAIM, 2019; 6(3): 44-49.				

Abstract

Background: Acute kidney injury (AKI) is defined as abrupt and rapid decline in renal filtration function. We have studied cases of AKI in which there was no primary renal involvement.

Materials and methods: A hospital based retrospective study of 50 indoor patients was carried out at B J Medical college and Civil Hospital, Ahmedabad to study the cases of acute kidney injury in patients with no primary kidney involvement and their incidence with relation to age, gender, comorbidities, substance abuse and treatment modalities.

Results: Total 50 patients of acute kidney injury were analyzed. Peak incidence was found in fourth decade with overall male: female ratio 2:1. The most common etiology was acute gastroenteritis 24% (n=12) followed by infections 40% (n=20). Most of the patients 28% (n=14) who presented with renal failure on admission, resolved with proper fluid resuscitation and antibiotics. Hemodialysis was required in only 6% (n=3) of the patients.

Conclusion: Acute gastroenteritis was the most common etiology of acute kidney injury in our study with median age of 45 years with male preponderance.

Key words

Acute kidney injury, Clinical profile, Renal involvement.

Introduction

Acute kidney injury (AKI) is defined by the impairment of kidney filtration and excretory function over days to weeks, resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. The causes of AKI have traditionally been divided into three broad categories: prerenal azotemia, intrinsic

renal parenchymal disease, and post renal obstruction [1].

According to Kidney Disease Improving Global Outcome (KDIGO) classification

Stages	Serum creatinine	
Stage 1 AKI	1.5-1.9 times the baseline	
Stage 2 AKI	2-2.9 times the baseline	
Stage 3 AKI	3 times the baseline	

According to the Acute Kidney Injury Network (AKIN) criteria, AKI is defined as abrupt (within 48 hours) increase in serum creatinine of 0.3mg/dl or increase in serum creatinine by 1.5 times or oliguria of < 0.5 ml/kg/h for > 6 hours [2, 3]. Risk Injury Failure Loss and End stage renal disease (RIFLE) criteria are defined based on increase in serum creatinine / GFR and decrease in urine output. The AKIN criteria differ from the RIFLE criteria in several ways. The RIFLE criteria are defined as changes within 7 days while AKIN criteria suggest using 48 hours. AKIN criteria avoid using glomerular filtration rate as a marker in AKI. In pre renal causes of AKI underlying kidney function may be normal, but decreased renal perfusion associated with intravascular volume depletion (e.g., from vomiting or diarrhea) or decreased arterial pressure (e.g., from heart failure or sepsis) results in a reduced glomerular filtration rate. Autoregulatory mechanisms often can compensate for some degree of reduced renal perfusion in an attempt to maintain the glomerular filtration rate. In patients with preexisting chronic kidney disease, however, these mechanisms are impaired, and the susceptibility to develop acute-or-chronic renal failure is higher [4].

Aim

Our aim was to study the cases of acute kidney injury in patients with no primary kidney involvement and their incidence with relation to age, gender, etiology, comorbidities, substance abuse, treatment modalities and outcome.

Materials and methods

A single centric, retrospective, hospital based study was conducted among indoor patients at B College and Civil Hospital J Medical Ahmedabad, Ahmedabad. The patients willing to participate in the study were enrolled with proper counseling. Out of the 110 indoor patients enrolled with acute kidney injury, 50 patients had no primary kidney involvement which was included in our study. The cases selected were subjected to detailed physical and systemic examination and then investigated for various parameters. Hematological laboratory and biochemical investigations were performed and special investigations such as eGFR, iPTH and USG KUB were performed on patients as and when required to rule out chronic kidney disease.

Inclusion criteria

- Age >12 years
- All patients presented to the hospital with signs and symptoms of dehydration, decreased urine output, nausea, vomiting, bilateral pedal edema or breathlessness.

Exclusion criteria

- Critically ill patients.
- Pregnant females.
- All the patients with primary renal disease or chronic kidney disease.
- Patients who did not consented for the study.

Results and Discussion

50 patients were included in our study of which 64% (n=32) were male and 36% (n=18) female. Maximum number of patients were in the age group of 41-50 years 24% (n=12) followed by 61-70 years with 22% (n=11) of the patients. Least common age group was 13-20 years 4% (n=2) as per **Table - 1**.

The highest incidence of acute kidney injury with acute gastroenteritis 48% (n=24), followed by infective causes 40% (n=18), hepatorenal syndrome 10% (n=5) and snake bite 10% (n=2). 1 snake bite patient presented with septicaemia on admission (**Table – 2**).

Age group (years)	Male (n=32)	Female (n=18)	Total	Percentage (n=50)
13-20	2	0	2	4%
21-30	5	1	6	12%
31-40	6	3	9	18%
41-50	4	8	12	24%
51-60	6	0	6	12%
61-70	6	5	11	22%
71-80	3	1	4	8%

Table - 1: Age and sex distribution of the patients.

Table - 2: Etiology of Acute kidney Injury.

Etiology	Total	Percentage
		(n=50)
Acute gastroenteritis	24	48%
Infective causes	18	36%
Hepatorenal syndrome	5	12%
Snake bite	2	10%
Multiple myeloma	1	2%

<u>**Table - 3:**</u> Incidence of Acute kidney injury due to Infective causes.

Infective causes	Total	Percentage
		(n=18)
Malaria	6	33.33%
UTI	5	27.77%
H1N1	3	16.66%
LRTI	2	11.11%
Viral hepatitis	1	5.55%
Dengue	1	5.55%

<u>**Table - 4a</u>:** Serum Creatinine value on admission.</u>

Serum Creatinine	Total	Percentage
(mg/dl)		(n=50)
1.6-3	19	38%
3.1-4.5	11	22%
4.6-6	8	16%
6.1-7.5	6	12%
9.1-10.5	4	8%
10.6-12	2	4%

The most common infective cause was malaria 33.33% (n=6), followed by urinary tract infection 27.77% (n=4) and H1N1 16.66% (n=3). 33.33% (n=6) of the patients with infective causes had septicemia on admission (**Table – 3**).

Table - 4b: Trend of Serum Creatinine values in	l
patients during hospital stay.	

Serum Creatinine (mg/dl)	Number	of
	patients	
1^{st} value >1.5 day 1^{st}	50	
2^{nd} value >1.5 day 3^{rd}	48	
3^{rd} value >1.5 day 6^{th}	18	
$4^{\text{th}} \text{ value } > 1.5 \text{ day } 9^{\text{th}}$	7	
5 th value >1.5 day 12^{th}	2	
$6^{\text{th}} \text{ value } > 1.5 \text{ day } 15^{\text{th}}$	Nil	

In our study, at the time of admission 72% (n=36) patients had creatinine <5mg/dl, 22% (n=11) were having creatinine between 5- 10, and 6% (n=3) were having creatinine more than 10. It was observed that by 3rd day, out of 50 patients, 96% (n=48) had creatinine more than 1.5, by 6th day, 36% (n=18) had raised creatinine level and by 9th day, only 14% (n=7) patients had raised creatinine (**Table – 4a, 4b**).

According to KDIGO classification, on admission, 28% (n=14) patients were having stage 3 kidney injury, 40% (n=20) were having stage 2 kidney injury. On discharge all the patients were having normal creatinine values. 3 patients of stage 3 kidney injury required hemodialysis during hospital stay, out of which 2 patients had HRS and 1 had H1N1 (**Table – 4c**).

24 patients presented with AGE of which 50% (n=12) presented with severe kidney injury but due to proper fluid management they required shorter (5 days) duration of hospital stay. The longest duration of hospital stay (12 days) was with H1N1 followed by hepatorenal syndrome (10 days). This shows that average duration of

hospital stay is related with etiology. The patients with substance abuse or co-morbidities had longer duration of hospital stay (7 days) then other patients (**Table – 5**).

74% (n=37) of our patients responded to fluids and antibiotics, 20% (n=10) required vasopressor support along with them, while 6% (n=3) required hemodialysis (**Table – 6**).

Staging of Acute Kidney	Number of	Average Serum	Average Serum
Injury according to	patients (n=50)	creatinine of patients	creatinine of patients
KDIGO classification		on admission (mg/dl)	on discharge (mg/dl)
Stage 1 AKI	16	2.41	0.90
Stage 2 AKI	20	3.82	1.39
Stage 3 AKI	14 11	5.24	1.46
	3	8.24	1.6

Table - 4c: Compa	arison of average serum	creatinine values in	patients on admission	and discharge.

Table - 5: Etiolo	ogy, Severity and A	verage duration	of hospital s	stay of patients.

	lology, Seventy and Average	Stage 1	Stage 2	Stage 3	Average hospital
		AKI	AKI	AKI	stay (days)
Etiology	Acute gastroenteritis	4	12	8	5
	Malaria	2	4	Nil	6
	UTI	2	3	Nil	4
	H1N1	Nil	2	1	12
	LRTI	2	Nil	Nil	3
	Snake bite	Nil	1	1	8
	Viral Hepatitis	Nil	1	Nil	7
	Dengue	Nil	1	Nil	6
	Hepatorenal syndrome	Nil	3	2	10
Co-	Hypertension	2	2	1	7
morbidities	Diabetes mellitus	3	2	Nil	6
	Tuberculosis	Nil	Nil	1	6
Substance	Smoking	4	4	2	7
abuse	Tobacco	4	4	2	5
	Alcohol	2	4	4	8
Average Dur	ation of hospital stay (days)	3	5	8	

*Staging of Acute Kidney Injury according to KDIGO classification

Table - 6: Treatment I	Modality of the patients	•
------------------------	--------------------------	---

Treatment Modalities	Total	Percentage (n=50)
Fluids and antibiotics	37	74%
Vasopressor +fluids+ antibiotics	10	20%
Hemodialysis	3	6%

Clinical presentation varies with the cause and severity of renal injury, and associated diseases. Most patients with mild to moderate acute kidney injury are asymptomatic and are identified on laboratory testing. Patients with severe cases of acute kidney injury, however, may be symptomatic and present with listlessness, confusion, fatigue, anorexia, nausea, vomiting, weight gain, or edema [5].

The definition of acute kidney injury indicates that a rise in creatinine has occurred within 48

hours, although in the outpatient setting, it may be hard to ascertain when the rise actually happened in a defined period. A patient history and physical examination, with an emphasis on assessing the patient's volume status, are crucial for determining the cause of acute kidney injury [6]. The history should identify use of nephrotoxic medications or systemic illnesses that might cause poor renal perfusion or directly impair renal function.

AKI is a common complication occurring in patients with cirrhosis, with reported frequencies of up to 20%-50% in patients with cirrhosis admitted to hospital for complications of the disease [7, 8]. HRS is a unique form of AKI that develops in patients with advanced cirrhosis and is mainly related to a marked renal vasoconstriction secondary to the systemic circulatory impairment, characteristic of patients with advanced cirrhosis [9]. There was no past history of NSAIDS among any patients in our study.

Hypertension and diabetes mellitus 2 are known to cause chronic renal impairment but we excluded renal involvement in them by doing iPTH, USG KUB and fundus examination of the patients. Renal biopsy is the gold standard for ruling out renal involvement but was not done as patients in our study had not consented for the same.

If fluid resuscitation is required because of intravascular volume depletion, isotonic solutions (e.g., normal saline) are preferred over hyperoncotic solutions (e.g., dextrans, hydroxyethyl starch, albumin). A reasonable goal is a mean arterial pressure greater than 65 mm Hg, which may require the use of vasopressors in patients with persistent hypotension [11, 12].

Conclusion

In our study, AKI was more common in age group of 41-50 years with male preponderance; male: female ratio 2:1 with most common cause as acute gastroenteritis followed by infections. Patients with acute kidney injury generally should be hospitalized unless the condition is mild and clearly resulting from an easily reversible cause. H1N1 and hepatorenal syndrome patients had longer duration of hospital stay as compared to others which suggest that duration of illness is dependent on etiology of acute kidney injury.

The key to management is assuring adequate renal perfusion by achieving and maintaining hemodynamic stability and avoiding hypovolemia. Most of the patients 74% (n=37) in our study responded to fluids and antibiotics, vasopressor agents were required along with them in 20% (n=10) of the patients and dialysis was required in 6% (n=3) of the patients. Patients with acute kidney injury are more likely to develop chronic kidney disease in the future and are also at higher risk of end-stage renal disease and premature death.

Abbreviation

KDIGO:	Kidney	Disease	Improving	Global		
Outcome						
AKI: Acute Kidney Injury						
AKIN: Acute Kidney Injury Network						
RIFLE: Risk Injury Failure Loss and End stage						
renal disease						
AGE: Acute Gastroenteritis						
NSAIDS:	Non-St	eroidal	Anti Inflan	nmatory		
Drugs						
eGFR: Estimated Glomerular Filtration Rate						
iPTH: Intact Parathyroid Hormone						
HRS: Hepatorenal Syndrome						
KUB: Kidney Ureter Bladder						
D (

References

- Harrison's Principles of Internal Medicine, 20th edition, McGraw-Hill, 2018, Volume 2, p. 2099.
- Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int., 2012; 2(Suppl 1): 1-138.

- Angeli P, Ginès P, Wong F, et al. Diagnosis and management of acute kidney injury in patients with cirrhosis: revised consensus recommendations of the International Club of Ascites. J Hepatol., 2015; 62: 968-974.
- Christensen PK, Hansen HP, Parving HH. Impaired autoregulation of GFR in hypertensive non-insulin dependent diabetic patients. Kidney Int., 2007; 52(5): 1369–1374.
- 5. Meyer TW, Hostetter TH. Uremia. N Engl J Med., 2007; 357(13): 1316–1325.
- Holley JL. Clinical approach to the diagnosis of acute renal failure. In: Greenberg A, Cheung AK, eds. Primer on Kidney Diseases. 5th edition, Philadelphia, Pa.: National Kidney Foundation; 2009.
- Garcia-Tsao G, Parikh CR, Viola A. Acute kidney injury in cirrhosis. Hepatology, 2010; 48: 2064-2077.
- 8. Huelin P, Piano S, Solà E, et al. Validation of a staging system for acute

kidney injury in patients with cirrhosis and association with acute-on-chronic liver failure. Clin Gastroenterol Hepatol., 2017; 15: 438-445.

- Ginès P, Schrier RW. Renal failure in cirrhosis. N Engl J Med., 2009; 361: 1279-1290.
- Schortgen F, Lacherade JC, Bruneel F, et al. Effects of hydroxyethylstarch and gelatin on renal function in severe sepsis: a multicentre randomised study. Lancet, 2001; 357(9260): 911–916.
- 11. Brochard L, Abroug F, Brenner M, et al. An Official ATS/ERS/ESICM/SCCM/SRLF Statement: Prevention and Management

of Acute Renal Failure in the ICU Patient: an international consensus conference in intensive care medicine. Am J Respir Crit Care Med., 2010; 181(10): 1128–1155.