MAK HILL Publications Research Journal of Medical Sciences

Research Article
doi: 10.59218/makrjms.2023.1097.1100

Spectrum of Acute Kidney Injury

[1]Ajaykumar M. Rathod, [2]Gayatri H. Bamaniya, [3]Divya A. Patel, [4]Divyang Makawana, [5]Breej Karmata and [6]Keyuri Parmar

[1,3,4-6]Smt SCL Hospital, Smt NHL Municipal Medical College, Ahmedabad, Gujarat, India

[2]SVP Hospital, Smt NHL Municipal Medical College, Ahmedabad, Gujarat, India

ABSTRACT

Acute kidney injury (AKI) is linked with considerable mortality and morbidity in India. It is a spectrum ranging from a mild form to severe form requiring renal replacement therapy (RRT). Detection of AKI in the early stage of disease and aggressive management of underlying cause will reduce mortality. Detailed history, clinical examination and laboratory investigation were carried out in all patients. All patients were subjected to urine analysis, hemogram, blood biochemistry (which included urea, creatinine, electrolytes, calcium and phosphorus). USG was done to rule out CKD and obstructive causes. The outcome was assessed and all the parameters were compared with outcome. Abput 48% of patients were in the 4 and 5th decades with the age of 49.78±13.63 years. Oliguria (68%) was the most common clinical symptoms of AKI then fever (63%) and vomiting (56%). Sepsis (35%) was the most common cause of AKI followed by acute gastroenteritis (11%), respectively. The AKIN criteria such as AKIN 1, AKIN 2 and AKIN 3 stages were found in 18, 30 and 52% patients, respectively. About (27%) patients required hemodialysis support while remaining (69%) patients were treated conservatively. About (79%) of patients had recovery. Moreover, the overall in hospital mortality rate was (21%). The progression of Oliguric AKI, Higher AKIN stage, Sepsis lead I increased hospital mortality. Patients with co-morbid condition like HTN and DM2 were more prone to AKI. Patients with etiology of sepsis had higher risk of progression of AKI stage 3, oliguria and mortality.

OPEN ACCESS

Key Words
Acute kidney injury, serum creatinine

Corresponding Author
Divyang Makawana,
Smt SCL Hospital, Smt NHL Municipal Medical College, Ahmedabad, Gujarat, India

Author Designation
[1]Associate Professor
[2,4]Assistant Professor
[3,5,6]Junior Resident

Received: 1 August 2023
Accepted: 21 August 2023
Published: 31 August 2023

Citation: Ajaykumar M. Rathod, Gayatri H. Bamaniya, Divya A. Patel, Divyang Makawana, Breej Karmata and Keyuri Parmar, 2023. Spectrum of Acute Kidney Injury. Res. J. Med. Sci., 17: 1097-1100, doi: 10.59218/makrjms.2023.1097.1100

INTRODUCTION

Acute kidney injury depicts the abrupt decline in renal function mostly occurs over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes and acid base homeostasis[1,2]. During the past decades acute loss of kidney function previously referred to as acute renal failure has been modified to acute kidney injury with increased recognition of importance of relatively small changes in renal function on both short and long term clinical outcomes[3].

The aetiology, course and outcome of AKI differ in various parts of India[4,5]. The RIFLE classification defines three grades of severity and two clinical outcomes of acute kidney injury. The kidneys being relatively unique among other organs of the body in its ability to recover from almost complete loss of function, AKI may develop in a wide variety of settings including ambulatory, outpatients, hospitalized and particularly critically ill patients. AKI is associated with substantial morbidity and mortality[2].

Although recovery of renal function occurs in majority of patients surviving an episode of AKI, many patients remain dialysis dependant or are left with severe renal impairment[6]. More recently it has been recognized that even patients who have complete or near complete recovery of renal function are at increased risk of CKD and that superimposition of AKI on CKD is associated acceleration in the rate of progression to ESRD[7]. The risk of AKI is contributed by the acute insult and background morbidity[8]. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction and parenchymal kidney disease[9]. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease, nephrotoxic medication also contribute to insult[10].

MATERIALS AND METHODS

Detailed history, clinical examination and laboratory investigation were carried out in all patients. Following important data were recorded date when AKI was detected. Also, exposure to nephrotoxic drugs prior to or during hospital stay, co-morbid conditions and base line serum creatinine were noted if available. All patients were subjected to urine analysis, hemogram, blood biochemistry (which included urea, creatinine, electrolytes, calcium and phosphorus). USG was done to rule out CKD and obstructive causes. Renal replacement therapy (RRT) was done in patients with symptomatic uraemia, serum creatinine >4 mg dL^{-1}, fluid overload, severe metabolic acidosis and hyperkalemia unresponsive to conservative measures. The modalities of RRT were intermittent haemodialysis (HD). The outcome was assessed and all the parameters were compared with outcome.

Inclusion criteria:

- Patients above the age of 18 years
- Critically ill patients in ICU
- Evidence of target organ damage, either clinically or on laboratory findings

Exclusion criteria:

- Age <18 years
- Pre-existing renal disease
- Small contracted kidneys in USGS
- Obstructive uropathy

In the present study, 48% of patients were in the 4th and 5th decades with the mean age of 49.78±13.63 years. This age decades group also happen to be more common with HTN and DM.

In the present study, the sex ratio is 1. 38:1 as co-morbidities and risk factors makes male more prone than female to AKI.

As observed, worldwide most common cause for nephropathy happens to DM our study also reflects the same.

The clinical features observed in our study were oliguria (68%), Fever (63%). Clinical features depend on the underlying condition and its severity. Most severe symptoms was associated with acute gastroenteritis and sepsis.

The most common cause of AKI was acute sepsis (35%), followed by acute gastroenteritis (11%). While, Drug induced AKI associated with NSAIDS.

In the present study, it was observed that cast increased in drug induced AKI majorly while, pus cells were increased in cases of UTI (Sepsis) majorly. Sugar increased in cases of DM patients whereas albumin increased almost in every case of AKI suggesting strongly correlated to AKI.

In the present study, It was observed that most of the patients in AKI stage 3 had unlined sepsis, acute gastroenteritis and DM2, while in AKI stage 2 apart from sepsis patients had malaria and cardiac as common causes. AKI stage 1 almost every cause had equally contribution.

In the present study, it was observed that maximum patients of sepsis, cardiac, acute gastroenteritis required hemodialysis. Patients with co-morbidities HTN and DM2 had increased requirement for hemodialysis. It was observed that most of the patients in AKI stage 3 had required hemodialysis.

In the present study, maximum mortality was seen due to sepsis, cardiac and AGE causes.

RESULTS AND DISCUSSION

About 48% of patients were in the 4th and 5th decades with the mean age of 49.78±13.63 years (Table 1).

Table 1: Age wise distribution

	No. of patients (n = 100)	
Age (years)	No.	Percentage
18-30	11	11
31-40	15	15
41-50	22	22
51-60	26	26
61-70	19	19
>70	7	7
Total	100	100
Mean age	----------49.78±13.63 ----------	

Table 2: Sex wise distribution

	No. of patients (n = 100)	
Sex	No.	Percentage
Male	58	58
Female	42	42
Total	100	100
M:F ratio	----------1.38:1 ----------	

Table 3: Co morbidities wise distribution

	No. of patients (n = 100)	
Co-morbidities	No.	Percentage
DM	34	34
HTN	21	21
CAD	18	18
COPD	12	12
CLD	8	8
CVA	3	3

Table 4: Clinical symptoms of AKI clinical symptoms

	No. of patients (n = 100)	
	No.	Percentage
Oligouric outcome	68	68
Fever	63	63
Vomiting	56	56
Breathlessness	43	43
Altered sensorium	32	32
Jaundice	26	26
Edema	25	25

Table 5: Etiological profile of AKI

	No. of patients (n = 100)	
Etiological profile	No.	Percentage
Sepsis	35	35
Acute gastroenteritis	11	11
Malaria, dangue	10	10
Cardiac diagnosis	10	10
Respiratory diagnosis	9	9
Hepatic cause	8	8
Drug induced	5	5
Metabolic/poisoning diagnoses	4	4
Primary neurologic diagnoses	3	3
Snake bite	3	3
Renal stone disease	2	2

The male patients (58%) were predominantly higher then female (42%). The sex ratio is 1. 38:1 (Table 2).

DM was present in (34%) patients followed by HTN (21%) and IHD (18%) patients, respectively (Table 3).

Oliguria (68%) was the most common clinical symptoms of AKI then Fever (63%) and vomiting (56%) (Table 4).

Sepsis (35%) was the most common cause of AKI followed by acute gastroenteritis (11%), respectively (Table 5).

The most common changes of AKI was seen in serum urea and serum creatinine by using biochemical parameters (Table 6).

Table 6: Biochemical parameters of AKI

Biochemical parameters	Mean±SD
Hemoglobin	9.74±1.63
TC (mm^3)	7020.82±2059.31
APC (lakhs)	3.08±0.77
RBS (mg dL^{-1})	106.13±5.25
Serum creatinine (mg dL^{-1})	4.48±2.74
Serum urea (mg dL^{-1})	96.48±47.61
Serum sodium (meq L^{-1})	135.81±4.90
Serum potassium (meq L^{-1})	4.16±0.84
Total protein (g dL^{-1})	6.25±0.72
Serum albumin (g dL^{-1})	3.00±0.54

Table 7: Urine R/M examination

	No. of patients (n = 100)	
Stage	No.	Percentage
Cast	12	12
Albumin	93	93
Pus	21	21
Sugar	34	34

Table 8: AKI stage and its distribution

	No. of patients (n = 100)	
Stage	No.	Percentage
AKI 1	18	18
AKI 2	30	30
AKI 3	52	52
Total	100	100

Table 9: Treatment wise distribution

	No. of patients (n = 100)	
RRT	No.	Percentage
Conservative	63	63
Hemodialysis	27	27
Total	100	100

Table 10: Outcome wise distribution

	No. of patients (n = 100)	
Outcome	No.	Percentage
Recovery	79	79
Mortality	21	21
Total	100	100

AKIN criteria such as AKIN 1, AKIN 2 and AKIN 3 stages were found in 18, 30 and 52% patients, respectively (Table 7).

About (27%) patients required hemodialysis support while remaining (69%) patients were treated conservatively (Table 8).

About (79%) of patients had recovery. Moreover, the overall in hospital mortality (Table 9).

CONCLUSION

Timely diagnosis and management of this disease condition confer a favourable prognosis to the patient. The progression of Oliguric AKI, Higher AKIN stage and sepsis lead to increased hospital mortality. Patients with co-morbid condition like HTN and DM2 were more prone to AKI. Patients with etiology of sepsis had higher risk of progression of AKI stage 3, oliguria and mortality.

REFERENCES

1. Almeida, A.F. and J.P. Kothari, 2012. Acute Kidney Injury: API Text Book of Medicine. 19 Edn., Jaypee, Pages: 1291.

2. Waikar, S.S. and V. Joseph, 2012. Bonventre-Acute Kidney Injury. In: Harrison's Principles of Internal Medicine, Lango, D.L., D.L Kasper. J. Loscalzo, J.L. Jame, (Ed.)., Mcgrawhill, New York.
3. Utas, C., C. Yalçindag, H. Taskapan, M. Güven, O. Oymak and M. Yücesoy, 2000. Acute renal failure in Central Anatolia. Nephrol. Dial. Transplant., 15: 152-155.
4. Muthusethupathi, M.A. and S. Shivakumar, 1987. Acute renal failure in south India: Our experience with 187 patients. J. Assoc. Physicians. India., 35: 504-507.
5. Prakash, J., K. Tripathi, V. Malhotra, O. Kumar AND P.K. Srivastava, 1995. Acute renal failure in Eastern India. Nephrol. Dial. Transplant 10: 2009-2012.
6. Bellomo, R., C. Ronco, J.A. Kellum, R.L Mehta, P. Palevsky and ADQI, 2004. Acute renal failure-definition, outcome measures, animal models, fluid therapy and information technology needs: The second international consensus conference of the acute dialysis quality initiative (ADQI) group. Critical Care, Vol. 8. 10.1186/cc2872
7. Kellum, J.A., C. Ronco, R. Mehta and R. Bellomo, 2005. Consensus development in acute renal failure: The acute dialysis quality initiative. Curr. Opin. Crit. Care., 11: 527-532.
8. Leblanc, M., J.A. Kellum, R.T.N. Gibney, W. Lieberthal, J. Tumlin and R. Mehta, 2005. Risk factors for acute renal failure: Inherent and modifiable risks. Curr. Opin. Crit. Care., 11: 533-536.
9. Mehta, R.L., J.A. Kellum, S.V. Shah, B.A. Molitoris, C. Ronco, D.G. Warnock and A. Levin, 2007. Acute kidney injury network: Report of an initiative to improve outcomes in acute kidney injury. Crit. Care., Vol. 11. 10.1186/cc5713