

Clinical profile and prognosis of patients with acute kidney injury from a tertiary care hospital in south India

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Abstract

Introduction: Acute kidney injury (AKI) is associated with significant mortality and prognosis depends on spectrum of etiologies and management. There are regional differences in etiology and outcomes of AKI. This study was undertaken to determine clinical characteristics, etiologies of AKI, modality of treatment and prognosis of AKI in our cohort of population.

Methodology: This is a retrospective observational study conducted over a period of 6 months from a tertiary care hospital. All patients above 15 years admitted with AKI are included in the study. Clinical parameters, risk factors, cause of AKI (as per KDIGO criteria) and treatment received were studied. Patients with underlying chronic kidney disease (CKD), obstructive causes, drugs and toxin mediated AKI were excluded from the study.

Results: A total of 50 patients with AKI were studied in this period. The mean age of patient was 41 years with slightly predominant male patients. The common etiologies of AKI included: infections (48%), snake bite (18%), hepatorenal syndrome (18%), cardiorenal causes (10%), and rhabdomyolysis in 6%. Oliguria was most common symptom at presentation (46%) followed by fever and breathlessness. Dialysis was required in 22 patients. Dialysis was equally effective in infective and non-infective causes of AKI. Mean duration of hospital stay was 7.8±2.5 days. Two patients died during study period due to multiorgan failure.

Conclusion: Infections are common cause for AKI in this study. Renal replacement therapy is equally effective in both infective and non-infective causes of AKI with favourable prognosis.

Keywords: acute kidney injury; infections; hemodialysis; mortality

Introduction

Acute kidney injury (AKI) is one of the leading causes of morbidity and mortality among patients in developing countries, which may be secondary to multiple etiologies. Apart from infections, AKI may occur due to noninfectious causes such as contrast induced nephropathy, rhabdomyolysis, drug and toxin induced AKI, hepatorenal syndrome (HRS), ischemic nephropathy, and glomerulonephritis [1, 2]. AKI occurs in 5–7% of acute hospital admissions and up to 30% of Intensive care unit (ICU) admissions [1-3]. AKI may need renal replacement therapy and a single episode of AKI increases the risk of chronic kidney disease (CKD) by 10 fold and end stage renal disease (ESRD) by 3 fold [2-4].

AKI has been defined according to KDIGO [5] as increase in serum creatinine by 0.3 mg/dl within 48 hours; or

increase in creatinine to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days, or urine volume 0.5 ml/kg/h for 6 hours. AKI is associated with complications like fluid overload, hyperkalemia and life threatening complications like

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cardiac arrhythmia, myocardial infarction, pulmonary embolism, gastrointestinal ulcers, seizures, coma, haemolysis, bleeding tendencies and severe infections [6].

Since AKI is associated with higher mortality, timely identification and intervention can mitigate the poor outcomes associated with it. The prognosis of AKI varies depending on etiological spectrum, presence of comorbidities, severity of disease and timely management including renal replacement therapy (RRT). This study was undertaken to determine the etiological spectrum for AKI, its clinical characteristics and to assess prognosis of these patients in our hospital.

Materials and methods

The study was conducted after the approval of institutional ethical committee. This is a retrospective observational study conducted for a period of 6 months from January 2018 to June 2018 at Bangalore Medical College and Research Hospital, Bangalore, south India. All patients with AKI diagnosed by KDIGO criteria [5] irrespective of the etiology and causes were included. Detailed demographic details, clinical history, comorbidities, treatment required and discharge/ death creatinine were noted. Underlying CKD, toxin mediated AKI and obstructive causes of AKI were excluded from the study since the outcomes varies according to treatment and dialysability of the toxin. All the patients were considered for optimum management protocol including need for peritoneal or hemodialysis. Duration of dialysis and duration of hospital stay and recovery were also noted. Patients were monitored closely during hospital stay and development of complications/ need for dialysis, duration of requirement of dialysis, recovery/ mortality were also noted.

Statistics

The data were analyzed descriptively using means, standard deviations and percentages. Tests of significance were performed using independent student 't' tests and chi square analyses as appropriate for the variables used in the comparison. The level of significance was set at 0.05. All analysis were performed with the statistical package for the social sciences for windows version 16; SPSS, Chicago.

Results

A total of 50 patients who developed AKI satisfying the inclusion criteria were included. Mean age was 41.4 ± 14.6 years (18-75 years). Males constituted 52% (n=26) of the cases, and females 48% of cases (n=24). 20% (n=10) patients were diabetics, 4% (n=3) patients had underlying hypertension, 10% (n=5)

patients had history of coronary artery disease. Presenting symptoms of patients were oliguria in 46% (n=23) patients, breathlessness in 32% (n=16) of the patients, fever was seen in 42% (n=21), and fluid overload in 26% (n=13) patients. Clinical characteristics of study patients are shown in Table 1.

Table 1: Clinical and hematological parameters in patients with acute kidney injury.

Parameters	Number of cases (n=50)
Age (in years)	41.4±14.6
Gender (Male:Female)	26:24
Symptoms	
Oliguria	23 (46%)
Breathlessness	16 (32%)
Fever	21 (42%)
Generalised anasarca	13 (26%)
Hemoglobin (g/dl)	11.3±1.7
WBC count (cells/cu.mm)	10100±3136
Platelet count (Lakh cells/cu.mm)	2.3±1.1
Urea (mg/dl)	68±21.3
Creatinine (mg/dl)	3.1±1.9
Potassium (mEq/l)	4.12±0.87
Duration of hemodialysis (in days)	1.08±1.8 (0-6)
Duration of hospital stay (in days)	7.8±2.5(3-15)

Underlying infections was the most common cause of AKI in our study accounting for 48% (n=24) of the total cases. Other causes of AKI included snake bite in 18% (n=9) patients, rhabdomyolysis in 6% (n=03), hepato renal syndrome (HRS) in 18% (n=09) and cardio renal syndrome (CRS) in 10% (n=05) patients. Among the infectious causes urinary tract infection was seen in 14% (n=07), pneumonia in 4% (n=02), acute gastroenteritis in 08% (n=04), cellulitis in 4% (n=02) and tropical fevers in 18% (n=09) of the patients as shown in Table 2.

Among hematological parameters, mean haemoglobin was 11.3 ± 1.7 g/dl, with mean total leucocyte count of 10100 ± 3136 cells/cumm. Mean platelet count was 2.3 ± 1.1 lakh cells/cumm. Mean serum urea was 68 ± 21.3 mg/dl, mean serum creatinine was 3.1 ± 1.9 mg/dl and mean serum potassium was 4.2 ± 0.89 mg/dl. 28% (n=14) of the patients required renal replacement therapy in the form of haemodialysis (HD) and 16% (n=08) patients underwent peritoneal dialysis. Mean duration

of HD was 1.08 ± 1.8 days. Mean duration of hospital stay was 7.8 ± 2.5 days (Table 3).

Table 2: Etiological spectrum for mortality in our cohort of acute kidney injury patients.

<i>Etiology of AKI</i>	<i>Number of cases (n=50)</i>
Snake bite	9
Rhabdomyolysis	3
Hepatorenal syndrome	9
Cardiorenal syndrome	5
Infective causes:	
Urinary tract infection- 7	
Pneumonia-2	
Gastroenteritis-4	24
Cellulitis- 2	
Tropical infections-9	

Table 3: Comparison of infective and non-infective factors for acute kidney injury patients.

	<i>Infective causes N=24</i>	<i>Non-infective cause N=26</i>	<i>P value</i>
Age	40.7 ± 16.1	42.1 ± 13.4	0.73
Hemoglobin (g/dl)	11.1 ± 1.6	11.5 ± 1.7	0.49
WBC count (cells/cu.mm)	10292 ± 3694	9924 ± 2580	0.68
Platelet count (Lakh cells/cu.mm)	2.1 ± 0.9	2.6 ± 1.2	0.13
Creatinine (mg/dl)	2.8 ± 1.5	3.5 ± 2.2	0.204
Duration of haemodialysis (in days)	0.7 ± 1.6	1.3 ± 2.07	0.306
Duration of hospital stay (in days)	8.1 ± 2.6	7.6 ± 2.5	0.75

Out of 44%(n=22) of the total AKI cases which required haemodialysis or peritoneal dialysis 91% (n=20) of the cases recovered completely with discharge serum creatinine of 1.1 ± 0.6 mg/dl. 4%(n=02) of the cases died during the course in the hospital secondary to severe sepsis and multi organ failure. Among the remaining 56%(n=28) of the cases who were managed conservatively, 100%(n=28) of patients recovered with discharge serum creatinine of 0.8 ± 0.6 mg/dl.

Discussion

In the present study, etiological profile and prognosis of patients admitted with AKI were studied. The mean age of the study population was 41.4 ± 14.6 years. This is comparable to other studies from India where the range is between 40-50 years [7-9]. A relatively younger population involvement has been shown in one of study of intensive care settings [10]. Males constituted 52% of the total cases, similar to a study by Patel et al. [9] where males constituted to about 64% of the total cases. 20%(n=10) patients were diabetics, 4%(n=3) patients had underlying hypertension, in contrast to a study done by Patel et al. [9], where diabetics constituted 10% of cases and hypertension found in 25% of the cases.

Most common presentation of AKI was oliguria in 46%, followed by fever and breathlessness. This is similar to other studies [10, 11]. Mahajan et al had showed that oliguria was present in upto 71% and had significant co-relation to mortality [12].

Infections were the most common cause of AKI in our study seen in 48% of our study, and tropical fever and urinary tract infection was the most common cause among infections. Singhal et al who found malaria was the predominant cause of AKI seen in 46% of patients followed by snakebite (20%) [11]. Studies from India have reported varying range of 30 to 86% of AKI from infections and sepsis. The increased incidence of infections, socio-economic factors, prevalence of uncontrolled sugars and underlying diabetic nephropathy, poor reach for tertiary care and inadequate antibiotic usage have all been contributing to increased incidence of sepsis related AKI in developing countries. Sepsis also denotes a multisystem involvement and poor prognosis/mortality in these patients [12, 13]. Apart from tropical infections, pneumonia and volume depletion secondary to gastroenteritis are also common in this part of country. This is comparable to other studies from developing nations [14, 15].

In the present study, 44% of the cases required renal replacement therapy in the form of either haemodialysis or peritoneal dialysis. These rates are relatively higher as compared to other studies where RRT were utilized in upto 20-30% of patients [7, 9]. However mortality rates were significantly lower compared to these studies. The mortality rate in AKI varies from as low as 7% [16] to as high as 80-90% [17, 19]. 4% of our study cases expired during course of illness which was lower than a study by Kumar et al [20]. In another study [21] from South India, mortality in AKI was 41%.

None of our patients required readmission during the study period whereas in other studies

[22] found 11% of patients with AKI were readmitted to the hospital. Similarly in a study by Sawhany et al [23] readmission rates at 30 day was 18% and 40% at 5 year.

When we compared the demographic and laboratory features between infective and noninfective causes of AKI as shown in Table 3 none of the parameters were statistically significant. This signifies the importance of early renal replacement therapy in these patients and timely initiation of antibiotics which would reflect in similar prognosis between infective Vs non infective etiologies of AKI.

Conclusion

Infections were the primary cause for AKI in our cohort of population. Younger populations are more affected, with fever and oliguria being predominant symptoms. Renal replacement therapy is equally effective in both infective and non-infective causes of AKI. Mortality rates are similar between infective and non-infective etiologies of AKI. Early identification of at risk patients for AKI and timely intervention with supportive therapy or renal replacement therapy can reduce the mortality and long term complications.

Limitations

It is a single center study and relatively lower cohort of patients. Study period was short. No follow up of recovered patients was done. However it gives indirect evidence on prevalence of AKI in the community and also effectiveness of renal replacement therapy. It also guides on prevailing mortality rates secondary to various etiologies of AKI. Follow up of recovered patients could have thrown light on the long term complication associated.

Conflicts of interest

Authors declare no conflicts of interest.

References

- [1] Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol.* 2005; 16(11):3365–3370.
- [2] Chawla LS, Amdur RL, Amodeo S, Kimmel PL, Palant CE. The severity of acute kidney injury predicts progression to chronic kidney disease. *Kidney Int.* 2011; 79(12):1361–1369.
- [3] Coca SG, Singanamala S, Parikh CR. Chronic kidney disease after acute kidney injury: A systematic review and meta-analysis. *Kidney Int.* 2012; 81(5):442–448.
- [4] Ishani A, Nelson D, Clothier B, Schult T, Nugent S, et al. The magnitude of acute serum creatinine increase after cardiac surgery and the risk of chronic kidney disease, progression of kidney disease, and death. *Arch Intern Med.* 2011; 171(3):226–233.
- [5] Piccinni P, Cruz DN, Gramaticopolo S, Garzotto F, Dal Santo M, et al. Prospective multicenter study on epidemiology of acute kidney injury in the ICU: a critical care nephrology Italian collaborative effort (NEFROINT). *Minerva Anestesiol.* 2011; 77(11):1072–1083.
- [6] Brady HR, Brenner BM. *Acute renal failure the kidney vol. I.* 5th edition. Saunders WB. Philadelphia:2000.
- [7] Khan MY, Deepak P, Kumar AP, Kumar TVK. Study of etiology, clinical profile and outcome of acute kidney injury (AKI) in medical intensive care unit. *Int J Contemp Med Res.* 2017; 4(11):2225–2228.
- [8] Bernieh B, Levy DW, Chaudhuri MD. Pattern of acute renal failure. *Transplantations proceeding.* 2003; 36:1780–1783.
- [9] Patel UR, Pasari AS, Balwani MR, Bhawane A, Tolani PR, et al. Clinical profile of acute kidney injury in a tertiary care center in the Tropical Region. *J Integr Nephrol Androl.* 2018; 5(4):130–133.
- [10] Liano F, Abero K. Acute renal failure. *Kidney International.* 2006; 50:820–824.
- [11] Singhal AS, Salkar AR, Chaudhary A, Fuscly SM. Clinical profile of acute renal failure. *JAPI.* 2002; 50:71–73.
- [12] Mahajan S, Tiwari S, Bharani R, Bhowmik D, Ravi S, et al. Spectrum of acute renal failure and factors predicting its outcome in an intensive care unit in India. *Renal Failure.* 2006; 28(2):119–124.
- [13] Pillai VSN, Varghese CJ, Pais CC, Rai VG, Chakrapani M. Clinical profile and outcomes of acute kidney injury patients in an intensive care unit in India. *International Journal of Clinical Trials.* 2020; 7(4):245–249.
- [14] Korula S, Balakrishnan S, Sundar S, Paul V, Balagopal A. Acute kidney injury-incidence, prognostic factors, and outcome of patients in an Intensive Care Unit in a tertiary center: A prospective observational study. *Ind J Crit Care Med.* 2016; 20(6):332–336.
- [15] Singh TB, Rathore SS, Choudhury TA, Shukla VK, Singh DK, et al. Hospital-acquired acute kidney injury in medical, surgical, and intensive care unit: A comparative study. *Ind J Nephrol.* 2013; 23(1):24–29.
- [16] Eswarappa M, Gireesh MS, Ravi V, Kumar D, Dev G. Spectrum of acute kidney injury in critically ill patients: A single center study from South India. *Ind J Nephrol.* 2014; 24(5):280–285.
- [17] Sural S, Sharma RK, Singhal MK, Kher V, Gupta A, et al. Acute renal failure in an intensive care unit in India--prognostic factors and outcome. *J Nephrol.* 1999; 12(6):390–394.
- [18] Prakash J, Murthy AS, Vohra R, Rajak M, Mathur SK. Acute renal failure in the intensive care unit. *J Assoc Physicians India.* 2006; 54:784–788.
- [19] Maxvold NJ, Bunchman TE. Renal failure and renal replacement therapy. *Crit Care Clin.* 2003; 19(3):563–575.
- [20] Kumar S, Raina S, Vikrant S, Patial RK. Spectrum of acute kidney injury in the Himalayan region. *Indian J Nephrol.* 2012; 22(5):363–366.
- [21] Mathew MK, Radha TR. Etiological factors and clinical profile of acute kidney injury in medical intensive care unit. *J Curr Med Res Opinion.* 2019; 2(11):350–366.
- [22] Abebe A, Kebede B, Wobie Y. Clinical profile and short-term outcomes of acute kidney injury in patients admitted to a teaching hospital in Ethiopia: A prospective study. *Int J Nephrol Renovasc Dis.* 2021; 14:201–209.
- [23] Sawhney S, Marks A, Fluck N, McLernon DJ, Prescott GJ, et al. Acute kidney injury as an independent risk factor for unplanned 90-day hospital readmissions. *BMC Nephrol.* 2017; 18(1):9.