

Original Research Article

Pattern of acute kidney injury and its outcome in a tertiary care centre

Jennie Santhanam¹, Meenakshi Sundari Subramaniyan Natarajan^{1*}, Annam Lakshmanan¹,
Rajeswari K. Balakrishnan²

¹Department of Medicine, SRM Medical College Hospital and Research Centre, SRM IST, Kattankulathur, Tamil Nadu, India

²Department of Medicine, Meenakshi Medical College Hospital and Research Institute, Enathur, Tamil Nadu, India

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*Correspondence:

Dr. Meenakshi Sundari Subramaniyan Natarajan,
E-mail: dr.meenakshisundari@gmail.com

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ABSTRACT

Background: Acute kidney injury is a multiplex disease with severe morbidity and mortality. The trends of acute kidney injury vary according to the regions and the population under study. The aim of this study is to evaluate the trends of acute kidney injury and its outcome in a tertiary care hospital.

Methods: The study was a prospective observational study conducted at a tertiary care hospital in a metropolitan city. A total of 102 patients of acute kidney injury were selected based on the Kidney Disease Improving Global Outcomes guidelines of acute kidney injury. The main trends of acute kidney injury presentation and its outcome were assessed.

Results: Of 102 patients admitted, 42 had a sepsis related diagnosis (42.41%), 17 patients (17.16%) had cardiovascular disease related acute kidney injury and 12 patients (12.12%) had developed acute kidney injury due to drugs and poisons. According to RIFLE (risk of renal failure, injury to kidney, failure and loss of function and end-stage kidney disease) category, 43.96% of patients belonged to the risk category and 30.77% to the injury category. Of 34 patients in failure category, 23 recovered and 11 did not recover. Authors compared the trends of acute kidney injury in patients who recovered and who deteriorated. The mean serum creatinine values were 3.42 mg/dl in patients who didn't recover from acute kidney injury and 2.05 mg/dl in patients who recovered. In patients of the recovered group, the mean urine output value is 783 ml/day; in deterioration group, 445 ml/day.

Conclusions: Most common etiologies of acute kidney injury in this study include sepsis, drugs and poisons, cardiovascular diseases and diarrheal diseases in order of occurrence. High serum creatinine at admission and oliguria were the most common factors that contributed to deterioration in acute kidney injury.

Keywords: Acute kidney injury, Risk of renal failure injury to kidney failure and loss of function and end-stage kidney disease criteria, Sepsis, Serum creatinine

INTRODUCTION

Acute Kidney Injury (AKI) is an abrupt and reversible deterioration of function of kidney, leading to retention of urea and other nitrogenous waste products and with normal or decreased urine output or both and with dysregulation of extracellular fluid volume and electrolytes. AKI is mainly detected by measurement of serum creatinine which is usually used for estimation of

the Glomerular Filtration Rate (GFR). According to Kidney Disease Improving Global Outcomes (KDIGO) guidelines, AKI is defined as following: raise in serum creatinine by ≥ 0.3 mg/dl within 48 hours or increase in serum creatinine to ≥ 1.5 times of baseline, which should have occurred within the prior 7 days or Urine volume < 0.5 ml/kg/h for 6 hours.¹ RIFLE criteria is one of the universally accepted classification system for AKI, which

has increasing grades of severity of AKI down the table as shown in Table 1.

Table 1: RIFLE classification.²

Class	GFR	Urine output
Risk	Serum creatinine: 1.5-fold increase or GFR: 25% decrease	Less than 0.5 ml/kg/hour for 6 hours
Injury	Serum creatinine: 2-fold increase or GFR: 50% decrease	Less than 0.5 ml/kg/hour for 12 hours
Failure	Serum creatinine: 3-fold increase or GFR: 75% decrease or Serum creatinine \geq 4 mg/dl (with acute rise \geq 0.5 mg/dl)	Less than 0.3 ml/kg/hour for 24 hours or anuria for 12 hours
Loss of kidney function	Complete loss of renal function for more than 4 weeks	
End stage renal disease	Complete loss of kidney function for more than 3 months	

In acute care hospital admissions, AKI affects 5 to 7% of patients and up to 30% of admissions in the critical care unit. AKI is associated with a remarkably higher risk of mortality in inpatients, particularly in patients admitted to the critical care unit where the hospital mortality may exceed 50%. The global burden of AKI is estimated at 13.3 million cases per year, with 85% from low- and middle-income countries.^{3,4} The duration and severity of AKI is a risk factor for the development of complications such as a tenfold increase in the risk of CKD (chronic kidney disease) and a threefold risk of end stage renal disease.⁵⁻⁷

The prevalence and causes of AKI differ massively between developed and developing countries, because of the differences in geography, economics, demographics and co morbid disease patterns. Most of the causes for AKI are geographic specific such as envenomation from spiders, snakes, bees; infectious etiologies such as leptospirosis, dengue and malaria etc.

The spectrum of acute kidney injury ranges from mild to severe, at times requiring renal replacement therapy. This study tries to evaluate the etiologies and outcomes of AKI in a developing country with limited resources. AKI is broadly classified into 3 important categories- Prerenal azotemia, intrinsic renal disease and Post renal obstructive diseases. Prerenal azotemia is due to decline in GFR caused by decrease in renal perfusion pressure with no damage to the renal parenchyma.⁸

Some causes of Prerenal azotemia include diarrhoea, vomiting, burns, infections like dengue and leptospirosis, shock, anaphylaxis and renal artery occlusion. Sepsis,

nephrotoxins, ischemia, acute tubular necrosis, acute glomerulonephritis and acute interstitial nephritis are the most common causes of intrinsic AKI.

Post renal causes of AKI include ureteric and bladder stones, cancer cervix, renal cell carcinoma, stroke, multiple sclerosis etc. Signs and symptoms of AKI include oliguria, pedal edema, anasarca, nausea, vomiting, dyspnea, hematuria, hypertension, seizures, confusion and coma.

METHODS

This study is a hospital based prospective observational study.

Inclusion criteria

- The study participants included patients admitted in a tertiary care hospital. Patients above the age of 18, presenting with elevated serum creatinine as per KDIGO guidelines (as given above) and symptoms of acute kidney injury in the medical wards and intensive care units were included in this study.

Exclusion criteria

- Patients who were already diagnosed as chronic kidney disease were excluded.

Convenient sampling method was used to recruit 102 participants. Ethical Clearance was obtained from the ethical committee of the Institute and written and informed consent was sought from the patients. The duration of study is six months from February 2017 to August 2017.

The preliminary workup included a detailed history to know the systemic illnesses, history of volume depletion, fever and use of nephrotoxic drugs that might cause decline in renal perfusion. Meticulous general and systemic examination was done. Urine output was strictly measured daily. Appropriate laboratory evaluation including measurement of serial blood urea and serum creatinine values, complete blood count, fractional excretion of sodium, blood glucose, liver function tests, blood culture and sensitivity, urinalysis, urine culture and sensitivity were done.

Imaging of the kidneys by ultrasonography was performed in all patients, especially in elderly males to rule out obstructive urinary pathology. Patients were assessed for conservative medical management, dialysis requirement and outcome (recovery or deterioration) of the disease. After entering the collected data in excel sheet, it was analyzed. For statistical analysis IBM SPSS version 22 was used. The outcome of the study was analyzed using multivariate logistic regression analysis to predict recovery in acute kidney injury.

RESULTS

Of the 102 patients, there was no statistically significant difference in the outcome (either recovery or deterioration) with regards to age or gender.

From Table 2, authors can see that the recovered group patients exhibited blood urea levels ranging from 75.48

mg/dl (at admission), 83 mg/dl (peak) and 52.11 mg/dl (at discharge) with mean value of 70.20 mg/dl. In the deterioration group, majority exhibited blood urea levels ranging from 96.64 mg/dl (at admission), 109.82 mg/dl (peak) and 91.82 mg/dl (at discharge) with an overall mean value of 99.42 mg/dl. The association between blood urea levels and the outcome groups is not statistically significant since $p > 0.05$.

Table 2: Blood urea levels.

Blood Urea Levels		At Admission	Peak Value	At Discharge	Mean
Recovered Group	N	91	91	91	91
	Mean	75.48	83.00	52.11	70.20
	SD	31.44	35.34	26.28	28.22
Deterioration Group	N	11	11	11	11
	Mean	96.64	109.82	91.82	99.42
	SD	35.10	28.59	43.68	34.12
p value Unpaired t Test		0.0957	0.7572	0.6332	0.6181

According to Figure 1, in the recovered group, the mean serum creatinine values ranged from 2.13 mg/dl (at admission), 2.55 mg/dl (peak) and 1.49 mg/dl (at discharge) with an overall mean value of 2.05 mg/dl. Similarly, in deterioration group, the mean serum creatinine values ranged from 3.25 mg/dl (at admission), 3.94 mg/dl (peak) and 3.05 mg/dl (at discharge) with an overall mean value of 3.42 mg/dl. The decreased mean serum creatinine values in recovered group compared to the deterioration group is significant statistically as the p value is 0.0173.

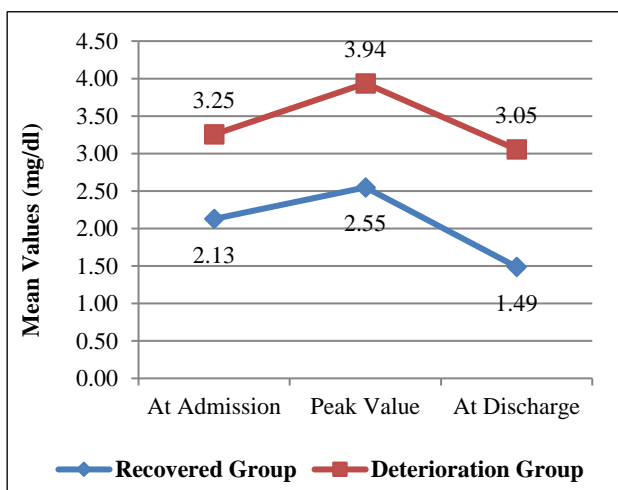


Figure 1: Serum creatinine levels.

Figure 2 shows that in patients in recovered group, the mean of urine output values ranged from 671 ml/day (at admission), 635 ml/day (peak) and 1043 ml/day (at discharge) with an overall mean value of 783 ml/day. In deterioration group, the mean urine output values ranged

from 420 ml/day (at admission), 370 ml/day (peak) and 547 ml/day (at discharge) with an overall mean value of 445 ml/day. The increased mean urine output values in recovered group compared to the deterioration group is statistically significant as the p value is 0.0027.

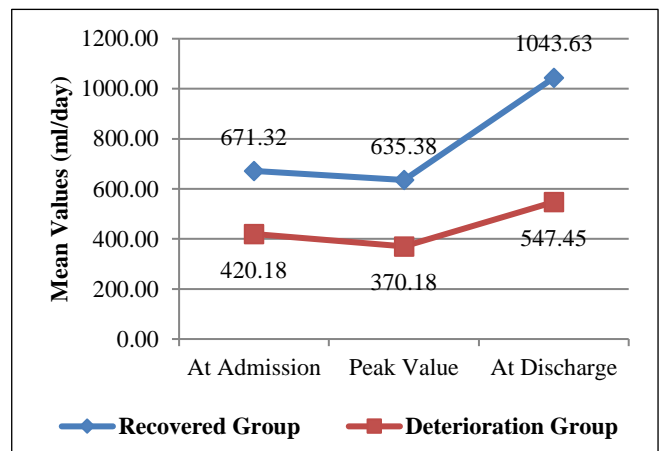


Figure 2: Urine output.

From Figure 3, authors can understand that in patients of recovered group, most of them belonged to the risk category (n=40, 43.96%) and in deterioration group, most of the patients were in the failure category (n=11, 100%). The decreased incidence of failure RIFLE criteria in recovered group compared to the deterioration group is statistically significant as the p value is 0.0388 as per fishers exact test which indicates that the difference is true among outcome groups.

From Figure 4, authors can see that in patients of recovered group, the majority belonged to sepsis etiology

(n=37, 40.66%). Similarly, in deterioration group, majority of the patients belonged to sepsis (n=5, 45.45%).

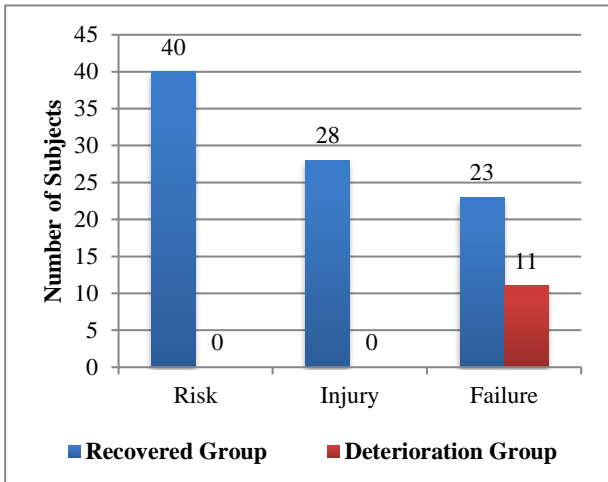


Figure 3: RIFLE criteria.

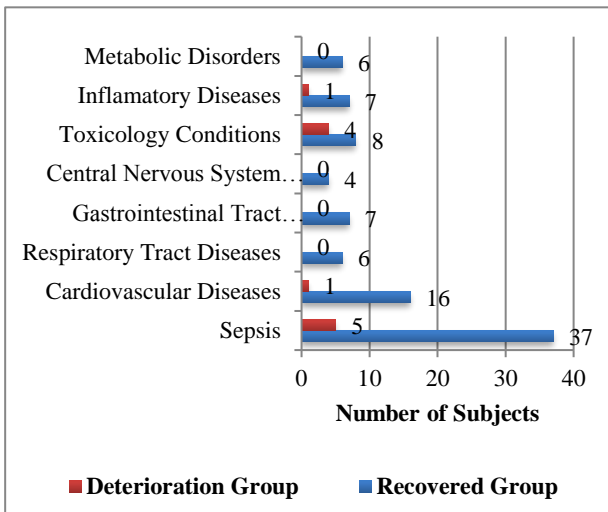


Figure 4: Etiology of AKI.

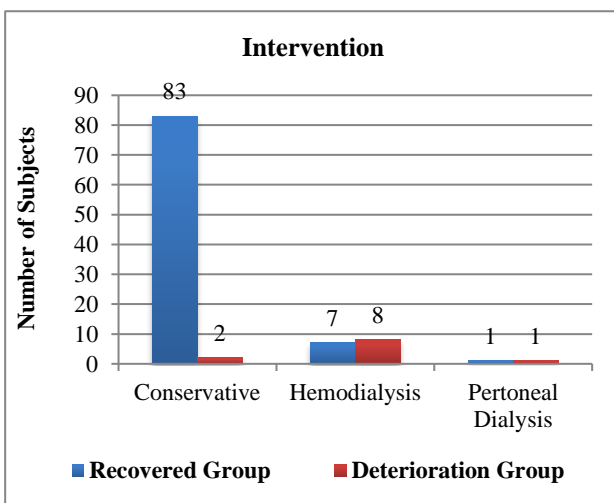


Figure 5: AKI Intervention.

The decreased incidence of toxicology conditions in recovered group compared to the deterioration group is significant as the p value is 0.0030.

From Figure 5, authors can infer that 91% of the patients in the recovered group were conservatively managed and 72% of the patients in the deteriorated group were taken up for hemodialysis.

Table 3 shows that there is 4.19 times higher risk of deterioration in patients with AKI having peak creatinine values > 4.5 mg/dl than in patients with AKI having peak creatinine values < 4.5 mg/dl. It is statistically significant with a p-value of 0.0039. There is 2.44 times higher risk of non-recovery in patients with AKI having urine output < 400 ml/day than in patients with AKI having urine output > 400 ml/day with a p-value of 0.0450.

DISCUSSION

In this study, as authors are comparing people who recovered from AKI and who deteriorated, authors found that there was no statistically significant difference with regards to age and gender of the patients. Majority of the recovered group patients had a mean age of 47.58 years and the deterioration group patients had a mean age of 49.09 years, which was not statistically significant. Majority of the subjects in the recovered group and deteriorated group belonged to the male gender, which was not statistically significant.

The mean serum creatinine levels were less in recovered group compared to the deterioration group by 1.36 mg/dl. This difference of 66% decrease in mean serum creatinine levels in recovered group when compared to the deterioration group is true. Hence authors can infer that higher serum creatinine levels are associated with deterioration in AKI patients.

The mean urine output levels were significantly more in recovered group compared to the deterioration group by 337 ml/day. This shows that lower urine output levels are associated with deterioration in AKI patients.

The incidence of failure category of RIFLE criteria was meaningfully less in recovered group compared to the deterioration group by 75%. In this study authors can conclude that the failure RIFLE criteria were significantly and consistently lower in recovered group compared to the deterioration group. This implies that higher incidence of failure RIFLE criteria is associated with deterioration in AKI patients.

The incidence of toxicological conditions as diagnosis was meaningfully less in recovered group compared to the deterioration group by 28%. This difference of 76% decrease in toxicological conditions as diagnosis in recovered group compared to the deterioration group is true and is not a chance occurrence. In this study authors can safely conclude that the incidence of toxicological

conditions like paraquat, copper sulphate poisoning etc., as diagnosis was consistently lower in recovered group compared to the deterioration group. Hence authors can

infer that higher incidence of toxicological conditions as diagnosis is associated with deterioration and non-recovery outcomes in AKI patients.

Table 3: Multivariate logistic regression model for statistically significant predictor of deterioration in health status.

Independent Variables	Deterioration/Non Recovery		
	Odds Ratio	95% Confidence Interval	p value
Age > 40 years	1.76	0.80-53.35	0.0793
Male	1.5	0.69-15.51	0.1331
Peak Urea Values >100 mg/dl	1.24	0.61-8.78	1.236
Peak Creatinine Values >4.5 mg/dl	4.19	1.12-16.52	0.039
Peak Urine Output Values <400 ml/day	2.44	1.78-3.38	0.045
Peak Potassium Values >5 mmol/L	3.82	0.65-22.62	0.1393

Hou et al., in 1983 published prospective cohort studies of AKI.⁹ The study focused on hospital acquired disease and they excluded patients with AKI which is already established on admission. Study found that the important causes of hospital-acquired AKI were due to decreased renal perfusion (42%), contrast nephropathy (12%), major surgery (8%) and aminoglycoside use (7%). In patients with an increase in serum creatinine of 0.5 to 0.9 mg/dl the in-hospital mortality was 3.8%. In patients with a serum creatinine of ≥ 4.0 mg/dl and in patients those who were not treated with renal replacement therapy the mortality increased progressively to 75%. This study established the significant association between mortality and oliguria in patients with AKI (17% versus 52% without and with oliguria, $P < 0.01$). In this study similarly in deterioration group, the mean serum creatinine values are high compared to recovered group.

Liaño and Pascual, published a prospective study of all types of AKI episodes in 1991 in 13 tertiary care centres in Spain.^{10,11} Of the 748 AKI patients, Acute Tubular Necrosis (ATN) was the most common cause (45%) which was caused by diverse etiologies like sepsis, surgery, renal hypo perfusion and nephrotoxin administration. Other causes were prerenal azotemia (21%), acute on CKD (12.7%) and urinary tract obstruction (10%). In his study, overall 45 % in-hospital mortality rate was obtained and a mortality rate of 65.9% in patients needing dialysis. Shusterman et al, conducted a case-control study in patients with hospital-acquired AKI in 1981.¹² This study found volume depletion, congestive heart failure, septic shock, aminoglycoside use and parenteral contrast infusion as risk factors for AKI. Also, this study found a tenfold increase in odds of death and doubling time of the length of hospital stay among the patients with AKI.

Dr. Tariq Zulfiqar Ali et al, in his study tested the hypothesis of RIFLE classification in AKI.¹³ Sepsis was a

causative factor in 47% of patients. In this study it was 42%. The RIFLE criteria were very useful for predicting recovery of normal kidney function ($P < 0.001$), requirement of renal replacement therapy ($P < 0.001$), in-hospital mortality ($P = 0.035$) and length of hospital stay ($P < 0.001$). His study found that mortality and requirement of RRT was high in patients in the failure category. In this study also many patients in the deteriorated group belonged to the failure category.

Kaul et al, showed in his study that serum creatinine > 4 mg/dl is associated with increased mortality. This study also concludes serum creatinine was significantly higher in the deteriorated group and is associated with poor outcome.¹⁴

Bhattacharya et al, showed in his study that peak serum creatinine and urea > 3 and > 100 mg/dl respectively, hyperkalemia (> 5.5 mEq/L), and severe metabolic acidosis ($\text{pH} < 7.20$) were all significantly associated with increased mortality in patients with AKI. This study didn't have any significant correlation for recovery or deterioration in patients with hyperkalemia.¹⁵

CONCLUSION

Common etiologies of AKI in this study include sepsis, drugs, poisons, cardiovascular disorders and diarrheal diseases. Most significant factors that contributed to deterioration AKI are higher serum creatinine at admission and oliguria. Most of the patients falling in the failure category of RIFLE classification belonged to the non-recovery group and they were more likely to be taken up for hemodialysis than conservative management.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

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