

## Original Research Article

# A study of factors determining outcome of acute kidney injury patients requiring hemodialysis

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### ABSTRACT

**Background:** High mortality rate in acute kidney injury (AKI) has interested many authors to conduct studies about factors predicting its outcome. The need for both dialysis and ICU care defines a group of critically ill patients who may have poor prognosis and consume vast amounts of resources. In this study we determine the variables predicting the outcome of patients with severe acute kidney failure requiring haemodialysis and to ascertain the aetiology of acute kidney injury in this group.

**Methods:** We prospectively analysed 114 patients admitted with severe renal failure requiring renal replacement therapy over a period of one year. The influence of various factors such as demographic variables, pre morbidities, details of admission, clinical presentation and extent of organ dysfunction on the clinical outcome such as mortality and progression to end stage kidney disease were statistically analyzed using SPSS version 12 (SPSS Inc., Chicago, Ill).

**Results:** Univariate and multivariate analysis showed that parameters such as chronic liver disease, preexisting heart disease, mechanical ventilation and vasopressor requirement, oliguria, sepsis, hepatorenal syndrome, cardiogenic shock and admission in ICU were associated with high mortality ( $p < 0.05$ ). Of the 114 patients, 49 died (42.98%), 61 (53.5%) were dialysis independent and 4 patients (3.5%) progressed to end stage renal disease (ESRD).

**Conclusions:** AKI patients requiring hemodialysis were associated with high hospital mortality. Patients who were diagnosed to have acute glomerulonephritis especially rapidly progressing glomerulonephritis as the cause of AKI were more prone to ESRD. Most survivors were dialysis independent at the time of discharge.

**Keywords:** Acute kidney injury, Clinical outcome, Hemodialysis, Mortality

### INTRODUCTION

Acute kidney injury (AKI) is broadly defined as “an abrupt and sustained decrease in kidney function”. Clinical signs include a rapidly decreasing glomerular filtration rate (GFR), resulting in disturbances in electrolyte- and acid-base balance, derangement of extra cellular fluid volume, retention of nitrogenous waste products and often a decreased urine output.<sup>1</sup> Historically, some researchers have argued that patients die with AKI-not of AKI-arguing that it merely denotes an expression

of illness severity. But now strong evidence suggests that AKI has an independent impact on outcome, even after all other variables affecting outcome has been corrected for.<sup>2,3</sup>

Acute kidney injury is a common clinical problem in critically ill patients and is associated with significant morbidity and a high mortality rate. In a general setting, the mortality is up to 40-50 %, whereas, it reaches up to 90% in the ICU setting.<sup>4-7</sup> Only few studies focus on the epidemiology of AKI requiring dialysis within the ICU

(severe acute renal failure of critical illness).<sup>8-10</sup> The need for both dialysis and ICU care defines a specific group of critically ill patients who may particularly have a poor prognosis and who consume vast amounts of resources.<sup>11-13</sup> Information on the overall incidence, details of management, and patient outcome would be useful in assessing current therapeutic approaches, research and resource allocation, and future therapies.

## METHODS

This study was conducted in patients at PSG Institute of Medical Sciences and Research, Coimbatore, India between December 1, 2016 and November 30, 2017. We included all patients admitted with severe AKI during the study period. Severe AKI was defined as any degree of AKI, which, in the opinion of the treating physician, required the commencement of renal replacement therapy.

### *Baseline characteristics*

Demographical parameters such as age, sex, date and details of admission to hospital were obtained. Laboratory investigations on the day of admission, pre-morbid renal function, and serum creatinine at the time of initiation of haemodialysis were also noted. Cause of acute kidney injury was diagnosed after clinical assessment and investigations according to the judgment of the clinician. Information such as use of mechanical ventilation, inotropic/vasopressor drugs, serum creatinine and bilirubin values, Glasgow coma score, and urinary output for the preceding 24 hrs was obtained prior to the start of renal replacement therapy. Information was also obtained on patient outcome, hospital mortality, number of days of renal replacement therapy, progression to End stage kidney disease and duration of hospital stay.

### *Details of management*

Intermittent hemodialysis mode was used in all the patients. A double-lumen catheter-either femoral or jugular-as vascular access was used in all cases. The Polysulfone Diacap (Low Flux 1.2m<sup>2</sup>, KUF 7.9 B Braun Germany) membrane was used in all patients. The approach to anticoagulation was dependant on patient coagulation profile with either heparin free or intermittent heparin as advised by clinician. Renal biopsy was performed on patients in whom the cause of AKI was not clear at the outset, if renal failure is due to glomerular disease or if renal failure was not improving even after 3 months of treatment.

### *Assessment criteria*

Patients were classified as oliguric or non-oliguric based on lowest daily urine output during the azotemic phase. They were also classified according to their admitted specialities as medical, surgical and obstetrics.

## *Sepsis*

In order to assign a possible cause for AKI, following criteria were applied:

A diagnosis of sepsis was made when two or more of the following were present as a result of systemic infection.

- Temperature >38°C or <36°C
- Heart rate >90/mt
- Respiratory rate >20/mt or Pa CO<sub>2</sub> <32mmHg
- WBC >12000/mm<sup>3</sup>, <4000/mm<sup>3</sup> or <20% band form.<sup>14</sup>

## *Organ dysfunction*

We recorded the organs and systems that had failed at the time of initiation of hemodialysis according to the following criteria, which were defined in the PROWESS study.<sup>15</sup>

### *Cardiovascular*

Shock: systolic arterial pressure ≤90mmHg or mean arterial pressure ≤70mmHg, during at least 1 hour despite adequate resuscitation with fluids or adequate intravascular volume; or use of vasopressors (dopamine ≥5µg/Kg/minute; noradrenaline or adrenaline at any dose; dobutamine was not taken into account). Unexplained metabolic acidosis (pH <7.30 or base excess ≤-5 mmol/l) associated with an arterial lactate concentration ≥2mmol/l with no other apparent cause.

### *Respiratory*

Mechanical ventilation; or PaO<sub>2</sub>/FiO<sub>2</sub> <250mmHg if other organ dysfunction was present; or PaO<sub>2</sub>/FIO<sub>2</sub> <200mmHg if only pulmonary dysfunction was present.

### *Nervous system*

Encephalopathy with GCS <13 without sedation unexplained by other causes.

### *Liver*

Bilirubin >3mg/dl or increase in prothrombin time related to a hepatic cause.

### *Hematological*

Platelets <80,000/ml<sup>3</sup> or decrease of 50% in the 3 previous days.

### *Stopping of hemodialysis*

It is considered if serum creatinine <3, normal serum electrolytes normal urine output.

*Measured outcomes*

The outcome was measured based on mortality and progression of AKI to end stage kidney disease.

*Statistical analysis*

Relationship between demographics, premorbidities and clinical parameters with outcome measures were studied. The influence of various factors such as age of patient, premorbidities like cardiac failure, liver failure, hypertension and diabetes, serum creatinine at the time of admission and at the time of initiation of dialysis, presence or absence of oliguria, presence of organ dysfunction, major causes of acute renal failure and duration of dialysis on the outcome of acute renal failure patients were analysed.

Data were expressed as number of patients (%) for categorical data or mean ± standard deviation (SD) for numerical data unless specified. Fisher’s exact test was used for categorical data and Mann-Whitney U test and student t test for continuous data in univariate analysis with survivors and non survivors as dependent variable. A p value of less than 0.05 was considered as significant. Multivariate analysis was performed using survivors and non survivors as dependent variable. Multiple logistic was performed for organ dysfunction. Data analysis was performed by SPSS version 12 (SPSS Inc., Chicago, Ill).

**RESULTS**

About 114 patients with AKI were analyzed for a period of one year. The mean age of these patients was 52.95 + 15.75 (range 18-89 years). The total number of males were 72 (63.2%) and 42 were females (36.8%). Patients were grouped based on their admitted specialities, cause of AKI and urine output (oliguric or non-oliguric). The mean creatinine was 5.36±2.78mg/dl. The mean duration of hospital stay was 12.09±8.23 days with a range of 2-50 days. The mean duration of hemodialysis was 12.81±18.2 days.

*Outcome*

About 49 died (42.98%), 61 (53.5%) recovered to have normal renal function or mild renal dysfunction not requiring hemodialysis during follow up and 4 patients (3.5%) progressed to end state renal disease (ESRD). In non ICU (ward) patients, the mortality is 6 (12.2 %), whereas, it is 43 (87.8%) in the ICU setting (Table 1).

*Univariate analysis of factors predicting outcome*

Univariate analysis of factors affecting AKI outcome showed that history of chronic liver disease, AKI secondary to cardiogenic shock, hepatorenal syndrome, use of vasopressors and mechanical ventilation and low urine output were significantly associated with mortality (Table 2).

**Table 1: Baseline characteristics of the study population.**

<b>Total no. of patients</b>	<b>114</b>
Mean Age (yrs)	52.95±15.75
Males:Females	72:42
<b>Details of admission</b>	
<b>Speciality</b>	
Medical	96 (84.2%)
Surgical	16 (14%)
Obstetrics	2 (1.8%)
ICU:non ICU	84:30
<b>Premorbidities</b>	
Cardiovascular disease	16 (14%)
Chronic liver disease	11 (9.6%)
Diabetes mellitus	34 (29.8%)
Systemic hypertension	21 (18.4%)
Pre-existing chronic kidney disease	10 (8.8%)
<b>Cause of AKI</b>	
Sepsis	46 (40.4%)
Hypovolemic shock	11 (9.6%)
Cardiogenic shock	10 (8.8%)
Hepatorenal syndrome	11 (9.6%)
Acute glomerulonephritis	7 (6.1%)
Drug induced kidney injury	4 (3.5%)
Acute Pancreatitis	4 (3.5%)
Malaria	4 (3.5%)
Snake Bite	2 (1.8%)
Obstructive Renal failure	6 (5.3%)
Following major surgery	4 (3.5%)
Post CABG	2 (1.8%)
Rhabdomyolysis	2 (1.8%)
Pregnancy Related	1 (0.9%)
<b>Presentation</b>	
Oliguric: Non oliguric	97:17
<b>Details of management</b>	
Mean serum creatinine	5.36±2.78
Duration of dialysis (days)	12.81±18.2
Mean duration of hospital stay (days)	12.09±8.23
<b>Outcome</b>	
Non survivors	49 (42.98%)
Recovered or mild renal dysfunction	61 (53.5%)
ESRD	4 (3.5%)

Univariate analysis of variables indicating organ dysfunction and their effect on survival showed that such as low GCS score (less than 8), serum creatinine, bilirubin, lactate, platelet count at the time of initiation of haemodialysis were associated with mortality (Table 3).

*Multivariate analysis of factors predicting outcome*

Multivariate analysis showed parameters directly related to AKI and was found to be significantly associated with mortality were chronic liver disease, pre-existing heart disease, requirement of mechanical ventilation, oliguria, sepsis, cardiogenic shock, and admission in ICU (Table 4).

**Table 2: Univariate analysis of factors affecting outcome of AKI.**

Disease	All patients		survivor			non survivor			p value		
	Present	Absent	Present	%	Absent	%	Present	%		Absent	%
<b>Premorbidities and mortality</b>											
Diabetes	34	80	25	73.5	40	50.0	9	26.5	40	50.0	0.016
Hypertension	21	93	14	66.7	51	54.8	7	33.3	42	45.2	0.230
Heart	16	98	6	37.5	59	60.2	10	62.5	39	39.8	0.077
Liver	11	103	2	18.2	63	61.2	9	81.8	40	38.8	0.008
Pre-existing chronic renal disease	10	104	9	90.0	56	53.8	1	10.0	48	46.2	0.026
<b>Cause of AKI and mortality</b>											
Sepsis	46	68	26	56.5	39	57.4	20	43.5	29	42.6	0.541
Hypovolemic Shock	11	103	9	81.8	56	54.4	2	18.2	47	45.6	0.073
Heart failure	10	104	2	20.0	63	60.6	8	80.0	41	39.4	0.016
Hepatorenal syndrome	11	103	2	18.2	63	61.2	9	81.8	40	38.8	0.008
Acute glomerulonephritis	7	107	6	85.7	59	55.1	1	14.3	48	44.9	0.115
Acute pancreatitis	4	110	2	50.0	63	57.3	2	50.0	47	42.7	0.578
Malaria	4	110	3	75.0	62	56.4	1	25.0	48	43.6	0.422
Snake bite	2	112	0	0.0	65	58.0	2	100.0	47	42.0	0.183
Drug induced Renal failure	7	107	1	100.0	64	56.6	0	0.0	49	43.4	0.570
Obstructive Renal failure	6	108	6	100.0	59	54.6	0	0.0	49	45.4	0.031
Following Major surgery	4	110	0	0.0	65	59.1	4	100.0	45	40.9	0.032
Post CABG	2	112	2	100.0	63	56.2	0	0.0	49	43.8	0.323
Rhabdomyolysis	2	112	2	100.0	63	56.2	0	0.0	49	43.8	0.323
Pregnancy related	1	113	1	100.0	64	56.6	0	0.0	49	43.4	0.570
<b>Urine output and mortality</b>											
Urine output	97	17	48	49.5	17	100.0	49	50.5	0	0.0	0.000
<b>Supportive measures and mortality</b>											
Vasoactive drugs	59	55	18	30.5	47	85.5	41	69.5	8	14.5	0.000
Mechanical Ventilation	44	70	10	7	55	78.6	34	77.3	15	21.4	0.000

**Table 3: Univariate analysis of variables indicating organ dysfunction predicting outcome in the study population using Mann Whitney U tests.**

	Survivor	Non survivor	Total	P value
Hb (mean ±SD)	11.13 ± 2.23	11.43 ± 2.83	11.26±2.51	0.826
Total leukocyte count (mean ±SD)	15658.46±6398.65	20602.04±12730.81	17783.33±9903.36	0.012
Platelet count (mean ±SD)	216843.75±131184.37	161142.85±105188.32	193631.5789±123116.1236	0.020
Sr. Sodium (mean ± SD)	133.49±7.14	131.84±8.24	132.7807±7.65	0.203
Sr. Potassium (mean ± SD)	4.64±1.26	5.04±1.42	4.81±1.34	0.119
Arterial PH (mean ± SD)	7.29±.078	7.20±.132	7.25±0.11	0.000
Arterial lactate (mean ± SD)	2.27±1.94	6.91±4.96	4.26±4.23	0.000
S. Creatinine (mean ±SD)	5.62±2.72	5.01±2.84	5.36±2.78	0.045
Sr. Bilirubin (mean ± SD)	1.73±2.30	5.95±7.79	3.55±5.75	0.000
GCS (mean ±SD)	14.35±1.59	10.33±2.99	12.62±3.04	0.000

**Table 4: Multivariate analysis of variables predicting outcome in the study population.**

Factor	B	Significant
<b>Age</b>	-0.020	0.170
<b>Sex</b>	-0.418	0.278
<b>Cormorbidities</b>		
Diabetes	-0.656	0.095
Hypertension	0.142	0.750
Heart disease	2.131	0.026
Liver disease	-5.601	0.000
Pre-existing chronic renal disease	0.073	0.885
<b>Cause of AKI</b>		
Sepsis	3.035	0.000
Hypovolemic shock	1.875	0.026
Cardiogenic shock	7.054	0.000
Hepatorenal Syndrome	2.368	0.154
Acute glomerulonephritis	0.250	0.784
Drug induced renal failure	2.876	0.033
Acute pancreatitis	2.638	0.073
Malaria	0.004	0.997
Snake bite	13.073	0.990
Obstructive renal failure	1.385	0.179
Following major surgery	14.861	0.963
Post CABG	4.186	0.020
Rhabdomyolysis	5.044	0.001
Pregnancy related	7.223	0.000
Ward	1.098	0.010
Mechanical ventilation	-3.559	0.000
Vasoactive drugs	-0.079	0.869
Urine output	0.950	0.028

**Organ dysfunction**

Stepwise multiple regression analysis was done to check the impact of the organ dysfunction present at the time of initiation of hemodialysis. As the variables were added, the impact factor increased from 0.04% to 48.1% (Table 5).

**Survivors’ characteristics and outcome**

Of those patients who survived, 61 (53.5%) recovered to have normal renal functions or mild renal dysfunction not requiring hemodialysis on follow up and 4 (3.5%) progressed to ESRD. All patients with medical causes of AKI progressed to ESRD unlike patients with surgical or obstetric causes. Patients who were diagnosed to have acute glomerulonephritis as the cause of AKI were more prone to ESRD. The mean age of the patients who developed ESRD did not differ from those who recovered renal functions completely (44.5±8.18 years vs. 52.62±15.7 yrs p>0.05). The duration of dialysis, as expected was significantly different between the two groups. Peak serum creatinine was 5.59±2.79mg/dl in patients who recovered and 5.97±1.34mg/dl in patients who developed ESRD (Table 6).

**DISCUSSION**

The mean age of our patients was 52.95±15.75years. Though in tropical countries young patients are more prone to develop AKI studies done by Liano et al and Feest et al showed similar age group people, like our study.<sup>16-19</sup>

**Table 5: Multiple regression analysis to analyse the impact of organ dysfunction on survival.**

Variables Entered	R	R Square	Adjusted R square
Platelet count	0.225	0.051	0.042
Mechanical ventilation, platelet count	0.568	0.322	0.310
Systolic BP<90mmHg, platelet count, mechanical ventilation	0.637	0.406	0.389
Bilirubin, mechanical ventilation, platelet count, systolic BP<90mmHg	0.677	0.458	0.438
GCS, Platelet count, bilirubin, systolic BP <90mmHg, mechanical ventilation	0.710	0.504	0.481
Creatinine, GCS, platelet count, bilirubin, systolic BP <90mmHg, mechanical ventilation	0.714	0.509	0.481

**Cause of AKI**

**Medical**

Septicemia was the most common cause of severe AKI in our study which is similar to other studies.<sup>20-23</sup> This information highlights the fact that any successful way of decreasing the incidence of severe AKI is likely to be based on the development of more effective therapies for

the prevention or rapid treatment of sepsis. Diarrhoeal diseases and snake bite showed a significant decline (9.6%) when compared to previous studies.<sup>23, 24</sup>

**Surgical**

The incidence of surgical AKI in our centre was 16 (14%) patients and obstructive Uropathy secondary to stones was the most frequent aetiology which is similar to

study done by Sakhuja et al.<sup>25</sup> Trauma and operative complications contribute to only 2-5% of cases of community-acquired AKI in the tropics when compared

to developed countries where road traffic accidents, drugs, complicated cardiac, vascular and abdominal surgeries are the leading causes of surgical AKI.<sup>13,23,25</sup>

**Table 6: Analysis of survivors' characteristics and outcome using Mann Whitney u test and Student t-test.**

	Recovered	ESRD	p value
<b>Age (Mean ± SD)</b>	52.62±15.70	44.50±8.18	0.257
<b>Sex (n %)</b>			
Male	35(57.4)	3(75.0)	0.496
Female	26(42.6)	1(25.0)	
<b>Pre morbidities (n %), Diabetes (n %)</b>			
Yes	23(37.7)	2(50.0)	0.631
No	38(62.3)	2(50.0)	
<b>Hypertension (n %)</b>			
Yes	11(18.0)	3(75.0)	0.007
No	50(82.0)	1(25.0)	
<b>Heart disease (n %)</b>			
Yes	6(9.8)	0(0.0)	0.518
No	55(90.2)	4(100.0)	
<b>Liver disease (n %)</b>			
Yes	2 (3.3)	0 (0.0)	0.718
No	59(96.7)	4(100.0)	
<b>Pre existing chronic renal disease (n %)</b>			
Yes	9(14.8)	0(0.0)	0.416
No	52(85.2)	4(100.0)	
<b>Speciality (n %)</b>			
Medicine	48(78.7)	4(100.0)	0.338
Surgery	11(18.0)	0(0.0)	
Obstetrics	2(3.3)	0(0.0)	
<b>AKI cause (n %)</b>			
Sepsis	26(42.6)	0(0.0)	0.095
Hypovolemic shock	9(14.8)	0(0.0)	0.416
Cardiogenic shock	2(3.3)	0(0.0)	0.718
Hepatorenal Syndrome	2(3.3)	0(0.0)	0.718
Acute glomerulonephritis	2(3.3)	4(100.0)	0.000
Drug induced renal failure	4(6.6)	0(0.0)	0.800
Pancreatitis	2(3.3)	0(0.0)	0.718
Malaria	3(4.9)	0(0.0)	0.656
Snake bite	0(0.0)	0(0.0)	-
Obstructive renal failure	6(9.8)	0(0.0)	0.518
Following major surgery	0(0.0)	0(0.0)	-
Post CABG	2(3.3)	0(0.0)	0.718
Rhabdomyolysis	2(3.3)	0(0.0)	0.718
Pregnancy related	1(1.6)	0(0.0)	0.800
<b>Mechanical Ventilation (n %)</b>			
Yes	10(16.4)	0(0.0)	0.387
No	51(83.6)	4(100.0)	
<b>Vasoactive drugs (n%)</b>			
Yes	18(29.5)	0(0.0)	0.207
No	43(70.5)	4(100.0)	
<b>Urine output (n %)</b>			
Yes	44(72.1)	4(100.0)	0.226
No	17(27.9)	0(0.0)	
<b>Hospital duration in days (Mean ± SD)</b>	14.06 ± 7.38	11.0 ± 4.08	0.450
<b>Ward (n %)</b>			
ICU	37(60.7)	4(100.0)	0.118
No ICU	24(39.3)	0(0.0)	

### *Obstetrics*

In some of the developing countries like India, the incidence of obstetric AKI has shown a decline from 22% (of all AKI) in 1960s to 8% in 1990s.<sup>26</sup> On the other hand, in Ethiopia, septic abortion is the underlying cause of AKI in 52% of all patients<sup>27</sup> and in Argentina gynecologic and obstetric complications still account for 32% of cases of AKI.<sup>27</sup> The incidence of severe AKI in our study was low which was similar to other studies.<sup>28</sup>

### *Outcome*

Mortality varies according to the setting in which AKI developed. In non ICU patients, the mortality is up to 40-50 %, whereas, it reaches up to 90% in the ICU setting.<sup>13,28</sup> In our study mortality of patients admitted in ICU was high (87.8%) than patients admitted in non ICU (ward) (12.2%)

### *Demographic variables*

Increasing age has been identified as an adverse prognostic factor in many studies.<sup>29,30</sup> Despite some previous studies which observed a trend towards an increasing number of AKI cases among male patients and male gender to be an effective factor on AKI mortality in our study no significant difference was found between sex and AKI outcome.<sup>29,30</sup>

### *Cause of AKI and outcome*

Among the causes of AKI, sepsis was associated with poor outcome in previous studies.<sup>28,30</sup> Sepsis contributes to mortality by its associated cardiopulmonary failure and has poor prognosis. In our study outcome of sepsis causing renal failure was not significant in univariate analysis however, it was significant in multivariate analysis. AKI due to cardiogenic shock secondary to acute myocardial infarction, acute decompensated cardiac failure and hepatorenal syndrome were associated with poor outcome.<sup>31,32</sup>

A better survival was seen in patients with glomerular disease leading to AKI. Presence of diabetes mellitus and pre-existing kidney disease was not significantly associated with increased mortality. Presence of pancreatitis adversely affected the outcome by mortality and required longer duration of RRT as observed similarly by Rasmussen et al.<sup>33</sup> All of our patients admitted with snake bite and treated with RRT expired unlike other studies where most patients ended with severe renal failure.<sup>34</sup> Patients with myoglobinuric renal failure and obstructive uropathy tended to have a better prognosis, but the small numbers of patients in such categories made it impossible to assign statistical significance to this observation.

### *Clinical presentation and outcome*

Presence of hypotension and oliguria at the time of admission were associated with poor outcome.

### *Organ dysfunction and outcome*

In agreement with other studies, cardiovascular dysfunction at the time of initiation of haemodialysis was associated with a high mortality rate.<sup>31,33,34</sup> Presence of hepatic failure at the time of initiation of haemodialysis was associated with increased mortality in a few series.<sup>35</sup> In our study presence of liver failure at the time of initiation of haemodialysis was associated with significant mortality.

Patients with CNS dysfunction at the time of initiation of haemodialysis have been observed to have a poor outcome in some studies.<sup>33,36</sup> Univariate analysis revealed that patients who had CNS dysfunction were more prone to death. Presence of respiratory dysfunction and requirement of mechanical ventilation have been consistently associated with high mortality. Our data suggest that the presence of metabolic acidosis increased the risk of mortality. Patients with acute glomerulonephritis especially rapidly progressive glomerulonephritis more often progressed to ESRD. Bonomini et al, observed ESRD in 16.2% of the survivors.<sup>37</sup> In our study, the incidence of ESRD among patients who recovered was 6.1%.

This was an observational study making it difficult to establish the cause and effect relationship between the various factors and outcomes. As this is a single center experience, results cannot be generalized. Also, we had not classified severe AKI secondary to hospital acquired or community acquired causes.

### **CONCLUSION**

Our study showed that AKI patients requiring hemodialysis were associated with high hospital mortality. Univariate and multivariate analysis showed that parameters such as chronic liver disease, pre-existing heart disease, mechanical ventilation and vasopressor requirement, oliguria, sepsis, hepatorenal syndrome, cardiogenic shock, admission in ICU were associated with high mortality. Patients who were diagnosed to have acute glomerulonephritis especially rapidly progressing glomerulonephritis as the cause of AKI were more prone to ESRD. Most survivors were dialysis independent at the time of discharge.

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