# **Original Research Article**

Title: To determine prospectively the variables predicting the outcome of

patients with severe acute kidney injury requiring haemodialysis

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# **Abstract**

**Background & Methods:** The aim of the study is to determine prospectively the variables predicting the outcome of patients with severe acute kidney injury requiring haemodialysis. The Polysulfone Diacap (Low Flux 1.2m2, KUF 7.9 Polymed) 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.

**Results:** Sepsis contributed to AKI in 39 patients (39%), Hypovolemic shock in 10 patients (10%), Hepatorenal syndrome in 10 patients (10%) Cardiogenic shock in 09 patients (09%), acute glomerulonephritis in 7 (07%), Obstructive renal failure in 05 patients (05%), drug related AKI, malaria, acute pancreatitis, following major surgery.

Conclusion: Among the causes of AKI, sepsis contributed predominantly (39%), followed by hypovolemic shock hepatorenal syndrome in 10% each. Results obtained on analysis of the role of co-existing diseases and organ system failures are as follows. The following factors were associated with hospital mortality: history of chronic liver disease causes of AKI like cardiogenic shock, hepatorenal syndrome, use of vasopressors and mechanical ventilation, low urine output, serum creatinine, bilirubin, lactate, platelet count at the time of initiation of haemodialysis.

Keywords: severe, acute, renal & haemodialysis.

Study Design: Observational Study.

# Introduction

AKI is a devastating illness that is associated with a high risk of mortality. Despite several decades of clinical experience with acute kidney injury, the advent of newer antibiotics, vastly improved dialytic techniques, an impressive growth in the availability of vasoactive drugs and aggressive nutritional support, the mortality rate of AKI has shown only a modest improvement over the last few decades[1]. Bisenbach et al in an analysis of 710 cases over a period of 15 years, observed a reduction in the overall mortality from 70 % in 1975-79 to 48% in 1985-89[2]. However, Forts et al noted a mortality of 46 % which did not change significantly over a period of 12 years.

The unchanged mortality in acute kidney injury has been attributed to many reasons. First there has been a change in the population susceptible to AKI12 Previously, the patients were predominantly young quite often from the armed forces or women of fertile age, whereas now it is more frequent among older patients[3]. Secondly, there have been new development in medical techniques and aggressive interventions which can maintain people alive, who would previously have died before having time to present with AKI[4]. The appearance of multiple organ system failure has contributed to a situation in which life can be prolonged and maintained indefinitely by artificial means in a patient who will inexorably die after lengthy suffering. The most precise way of establishing the vital prognosis of an entity is by determination of its mortality rate[5]. Acute kidney injury continues to have a high mortality and it varies according to the setting in which AKI developed. In a general setting, the mortality is upto the tune of 40-50 %, whereas, it reaches up to 90% in the ICU setting[6]. The fact that mortality in AKI is very high has interested many authors to conduct studies of factors predicting the outcome in AKI.

Numerous studies have been conducted both prospectively and retrospectively, and various prognostic factors have been proposed to predict the outcome in AKI patients. Some of the studies have been performed in specific situations like AKI following aortic aneurysm surgery, 14 post traumatic AKI 15 and AKI in the ICU setting[7].

# **Material and Methods**

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.

### **Exclusion criteria:**

Patients who had received RRT for indications other than AKI like, prevention of contrast nephropathy and drug poisoning; Known end-stage renal disease (ESRD) patients who had been receiving chronic renal dialysis before admission; Patients having been started on RRT for AKI in other units before admission. End stage renal Disease was defined as GFR <15 ml/min/1.73 m2 body surface area.

Information was also obtained on patient outcome, hospital mortality, number of days of renal replacement therapy, patients who developed ESRD, and duration of hospital stay renal Intermittent HD Was the RRT modes used in all the patients A double – lumen catheter was used for vascular access in all cases. The femoral position was chosen in 80 (70.2%) patients, and the jugular in 34 patients (29.8%).

### Result

Table No. 1: Demographic Profile

S. No.	Gender	No.	Percentage	P Value
1	Male	69	69	0.317
2	Female	31	31	
	ICU:NON ICU	No.	Percentage	
1	ICU	77	77	0.042
2	NON ICU	23	23	
		Mean	SD	
1	Mean Duration of Hospital stay( days)	12.46	2.63	-
2	Duration of Dialysis (days)	12.71	5.75	-

Mean Age (years)  $51.9 \pm 6.6$ 

**Table No. 2: Cause of AKI** 

S. No.	Gender	No.	Percentage
1	Sepsis	39	39
2	Hypovolemic shock	10	10
3	Cardiogenic shock	09	09
4	Hepatorenal	10	10
	syndrome		
5	Acute	07	07
	glomerulonephritis		
6	Drug induced renal	03	03
	failure		
7	Acute Pancreatitis	03	03
8	Malaria	03	03
9	Snake Bite	02	02
10	Obstructive Renal	05	05
	failure		
11	Following major	04	04
	surgery		
12	Post CABG	02	02
13	Rhabdomyolysis	02	02
14	Pregnancy Related	01	01

Sepsis contributed to AKI in 39 patients (39%), Hypovolemic shock in 10 patients (10%), Hepatorenal syndrome in 10 patients (10%) Cardiogenic shock in 09 patients (09%), acute glomerulonephritis in 7 (07%), Obstructive renal failure in 05 patients (05%), drug related AKI, malaria, acute pancreatitis, following major surgery.

100

S. No. **GCS Score Non Survivor P Value** Survivor No.=100  $N_0=100$ 1 Less than or 03 33 equal to 8 9-12 0.042 2 12 49 3 More than or 85 18 equal to 13

Table No. 3: Relationship between GCS & Survivor or Non survivor

Table No. 4: Mean Stats

100

S. No.		Mean±SD	P Value	
1	Hb	11.26±2.51	26±2.51	
2	TLC	17563.33±9903.36		
3	Platelet Count	193485.5789 ±		
		123116.1236		
4	S.Sodium	$131.7807 \pm 7.65$	<0.0001	
5	S.Potassium	4.61±1.34		
6	Arterial PH	7.52±0.11		
7	Arterial Lactate	4.29±4.23		
8	S. Creatinine	5.64±2.78		
9	S. Bilirubin	3.79±5.75		

Mean peak serum creatinine at  $5.64 \pm 2.78$  mg/dl. Mean serum bilirubin at  $3.79 \pm 5.75$  mg/dl. Mean arterial lactate at  $4.29 \pm 4.23$  mg/d.

#### **Discussion**

4

Total

AKI in the western societies is now largely a consequence of road traffic accidents, cardiovascular surgery, drugs, multi-organ failure and renal transplant rejection. This sharp decline in the incidence of community acquired AKI in the developed countries contrasts with hospitals in tropical countries which continue to cater to AKI associated with diarrheal diseases, obstetrical accidents, toxins and infections specific and unique to their respective

regions, The patterns of AKI encountered in the tropics have, however, shown changes similar to those in the west, though at a much slower pace[8]. Amongst the medical causes of AKI, etiological factors leading to AKI in tropical countries are very different from those seen in the developed world. Diarrheal diseases, intravascular hemolysis due to G6PD deficiency, copper sulfate poisoning, snake bites and insect stings together constitute over 40% of all causes of AKI in India and these causes are rarely encountered in the west.

Improvements in obstetrical care have led to a virtual disappearance of AKI related to pregnancy in the advanced countries. Even 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. On the other hand, in Ethiopia, septic abortion is the underlying cause of AKI in 52% of all patients 72 and in Argentina and Nigeria, gynecologic and obstetric complications still account for 32% and 25% of cases of AKI respectively[9]. This high incidence is due to the prevalence of unsafe home deliveries and abortions conducted by untrained personnel. The obstetric patients with severe AKI in the our study was 2 (1.8%); and they are increasingly rare elsewhere as well. However this may not be the true incidence in india as ours is a single centre and private set up where a limited number of patients would have been seen.

Acute kidney injury continues to have a high mortality .Despite several decades of clinical experience with acute kidney injury, the advent of newer antibiotics, vastly improved dialytic techniques, and an impressive growth in the availability of vasoactive drugs and aggressive nutritional support, the mortality rate of AKI has shown only a modest improvement over the last few decades[10].

Mortality varies according to the setting in which AKI developed. In NON ICU patients, the mortality is upto the tune of 40-50 %, whereas, it reaches upto 90% in the ICU setting. In our study mortality in patients admitted in ICU was (87.8%) than patients admitted in non ICU (ward) (12.2%).

# Conclusion

Among the causes of AKI, sepsis contributed predominantly (39%), followed by hypovolemic shock hepatorenal syndrome in 10% each. Results obtained on analysis of the role of co-existing diseases and organ system failures are as follows. The following factors were associated with hospital mortality: history of chronic liver disease causes of AKI like cardiogenic shock, hepatorenal syndrome, use of vasopressors and mechanical ventilation,

low urine output, serum creatinine, bilirubin, lactate, platelet count at the time of initiation of haemodialysis.

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