

Clinical Profile of Patients with Acute Renal Failure Admitted to Tertiary Care Hospital

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Abstract

Mortality rates for AKI have changed little since the advent of dialysis and have remained at 50%. This curious statistic simply reflects the changing demographics of AKI from community - to hospital - acquired settings. Currently, the mortality rate for hospital - acquired AKI is reported to be as high as 70% and is directly correlated to the severity of the patient's other disease process. A Hospital based Prospective study was conducted in Department of General Medicine for a 2 year period after taking approval from Hospital Ethics and Research Committee. Urine Albumin was present in 31 (29.8%) patients while pus cells and RBC was present in 17 (16.3%) and 10 (9.6%) patients. Mean S. urea level on admission was 98.05 mg, at discharge it was 53.53 mg. The difference at admission, discharge and was statistically significant.

Keywords: Acute Renal Failure; Dialysis; Mean S. Urea Level.

Introduction

Because most cases of community acquired AKI are secondary to volume depletion, as many as 90% of cases are estimated to have a potentially reversible cause. Hospital - acquired AKI often occurs in an ICU setting and is commonly the end result of multiorgan failure [1]. This dichotomy in the etiology of AKI explains the increased mortality rate, dialysis requirements and rates of progression to end-stage renal failure seen in hospital - acquired AKI compared with community-acquired AKI [2].

Mortality rates for AKI have changed little since the advent of dialysis and have remained at 50% [3]. This curious statistic simply reflects the changing demographics of AKI from community - to hospital - acquired settings. Currently, the mortality rate for hospital - acquired AKI is reported to be as high as 70% and is directly correlated to the severity of the patient's other disease process. The mortality rate among patients presenting to the emergency department with pre-renal AKI may be as low as 7%. Despite dialysis and medical advances mortality of AKI requiring dialysis is high [4]. With the advent of dialysis, the most

common causes of death associated with AKI are sepsis, cardiac failure and pulmonary failure.

Survivors of an episode of AKI requiring temporary dialysis, however, are at extremely high risk for progressive CKD, and up to 10% may develop end-stage renal disease. Patients with AKI are more likely to die prematurely after they leave the hospital even if their kidney function has recovered [5].

Because AKI has such a long differential diagnosis, it is mandatory to obtain a directed history along the lines of the pathophysiology of AKI (pre-renal, intrinsic renal, post-renal failure).

Patients commonly present with symptoms related to hypovolemia, including thirst, decreased urine output dizziness and orthostatic hypotension. History of excessive fluid loss via hemorrhage, gastrointestinal losses, sweating or renal sources must be asked. Patients with advanced cardiac failure leading to depressed renal perfusion may present with orthopnea and paroxysmal nocturnal dyspnea. Insensible fluid losses can result in severe hypovolemia in patients with restricted fluid access and should be suspected in the elderly and in comatose or sedated patients.

Patients can be divided into those with glomerular and those with tubular etiologies of AKI. Glomerular disease: nephritic syndrome of hematuria, edema and hypertension is synonymous with a glomerular etiology of AKI. A query about prior throat or skin infections is essential. A history of an earlier episode resembling this symptom complex is often helpful in establishing a different diagnosis [6].

Tubular diseases: ATN should be suspected any patient presenting after a period of hypotension secondary to cardiac arrest, hemorrhage, sepsis, drug overdose or surgery.

A careful search for exposure to nephrotoxins should include a detailed list of all current medications and any recent radiologic examinations (i.e. exposure to radiologic contrast agents). Pigment-induced ARF should be suspected in patients with possible rhabdomyolysis (muscle tenderness, recent coma, seizures, drug abuse, alcohol, excessive exercise, limb ischemia) or hemolysis (recent blood transfusion) [7,8]. Allergic interstitial nephritis should be suspected with recent drug ingestion, fever, rash and arthralgia's.

Post-renal failure usually occurs in older men with prostatic obstruction and symptoms of urgency, frequency and hesitancy. Patients may present with asymptomatic high-grade urinary obstruction because of chronicity of their symptoms. History of prior gynecologic surgery or carcinoma in women often can be helpful in providing clues to the level of obstruction. Flank pain and hematuria should raise a concern about renal calculi or papillary necrosis as the source of urinary obstruction.

Methodology

Study Design

A Hospital based Prospective study was conducted in Department of General Medicine for a 2 year period after taking approval from Hospital Ethics and Research Committee.

Sampling Technique and Sample Size

Universal Sampling Technique was used for selection of study subjects. All the patients coming to medicine department during the study period and fulfilling the inclusion and exclusion criteria were taken for study after taking prior informed consent. The patients included in the study were from both ICU and wards. Final sample

size came to be 104 subjects of Acute Kidney Injury of varied etiology.

Inclusion Criteria

1. Age >18 year.
2. Patients who fulfill the RIFLE criteria for Acute kidney disease (Risk, injury, failure).
3. Minimum of 24 hours of admission.

Exclusion Criteria

1. Patients of established Chronic Kidney Disease and end stage renal disease.
2. Pre-renal factors like volume depletion which is correctable within 48 hours.
3. Discharge against medical advice.
4. Deaths within one day of admission.

Results

A total of 104 patients with AKI were selected for the study after taking inclusion and exclusion criteria on the results were analyzed as follows.

Table 1: Age Distribution

AGE	no of patients
<20	4
21-30	16
31-40	21
41-50	21
51-60	21
>60	21

Over half of the subjects were above 40 years of age with mean age of study subjects was 47.42 years, There were 21 patients with age > 60 yrs. (Table 1)

Table 2: Gender Distribution

Sex	No. of cases	Percent
Female	34	32.7
Male	70	67.3
Total	104	100.0

There were 70(67.3)% males and 34(32.7%) females with M:F ratio of 2.7:1.3 (Table 2)

Table 3: Distribution based on S. Urea Levels

	Minimum	Mean	Std. Deviation
Urea on admin	41.00	98.05	43.99
Urea on discharge	0.00	53.53	29.11

Mean S. urea level on admission was 98.05 mg, at discharge it was 53.53 mg. The difference

at admission, discharge and was statistically significant ($p < 0.01$). (Table 3).

Table 4: Distribution based on S. Creatinine Levels

	Minimum	Mean	Std. Deviation
Creat on admin	1.200	2.629	0.965
Creat on discharge	0.000	1.801	1.297

Mean S. Creatinine level on admission was 2.629 mg, at discharge it was 1.801 mg. The difference at admission, discharge was statistically significant ($p < 0.01$). (Table 4).

Table 5: Distribution based on Input-output Charting

	Minimum	Mean	Std. Deviation
Input on Admin	200.00	1447.69	479.22
Input on Discharge	350.00	1852.50	508.21
Output on Admin	0.00	870.43	584.56
Output on Discharge	0.00	1532.12	649.31

Mean input and output volume of patient significantly improved at discharge from their admission values ($p < 0.01$). (Table 5).

Table 6: Distribution based on Urine Analysis

Urine Analysis	No of cases	%	
Albumin	Nil/ Trace	73	70.2%
	Present	31	29.8%
Pus Cells	No	87	83.7%
	Yes	17	16.3%
RBC	No	94	90.4%
	Yes	10	9.6%

Urine Albumin was present in 31 (29.8%) patients while pus cells and RBC was present in 17 (16.3%) and 10 (9.6%) patients. (Table 6).

Table 7: Distribution based on LFT

LFT	No. of cases	Percent
Abnormal	17	16.3
Normal	87	83.7
Total	104	100.0

Abnormal LFT was observed in 17 (16.3%) patients of Acute Kidney Injury (Table 7).

Table 8: Distribution based on USG Findings

USG Findings	No of cases	Percent
Cystitis	3	2.9
Grade 1 RPC	37	35.6
Grade 2 RPC	10	9.6
Normal kidneys	47	45.2
Obstructive uropathy	2	1.9
Pyelonephritis	4	3.8
Significant post voidal residual urine	1	1.0
Total	104	100.0

On USG, normal kidneys was observed in 47 (45.2%) of the patients while grade I and II RPC was observed in 37 (35.6%) and 10 (9.6%) of the patients. Other findings included Cystitis, Pyelonephritis and obstructive uropathy (Table 8).

Discussion

The study comprised of 104 patients out of admitted. Out of 104 patients, 70 were males and 34 were females.

It is recognized that the epidemiology of AKI in developing No. of cases differs from that of the developed world in many important ways. Whereas in developed regions elderly patients predominate, in developing No. of case, AKI is a disease of the young and children, in whom volume-responsive "prerenal" mechanisms are common. Although overall mortality seems to be lower than in developed No. of cases, this finding is not true across all age groups: In these regions, AKI affects predominantly the young and children and mortality is high [9].

Over half of the subjects were above 40 years of age. There were 21 patients with age > 60 yrs. The mean age in our group was 47.42 years. The mean age reported by Shusterman et al. [10] was 52.4±12, by Shema et al. [11] was 62.3±14.3 and 67.9±7.6 yrs by HSKohli et al. [12]. However, HS Kohli et al. [12] studied only elderly group with age ≥ 60 yrs hence the mean age was higher in their study. The mean age reported by V Jha et al. [13] was 42.9±16.8 yrs as their group had many young patients.

According to J Prakash et al. the mean age of patients getting admitted to the ICU was 44.9 years and there was no statistical significance between survivors and non survivors which was similar to our study. In the study conducted by Sean M. Bagshaw et al in New Zealand, Australia, the mean age of admission was 64.3 years. In present study there were 67.3% males and 32.7% females with M:F ratio of 2.7:1.3.

However in the study conducted by Kohli et al. [12], Jha et al. [13] and various other authors where males outnumbered females.

Similar results were also observed in study by J Prakash et al. [14], incidence of AKI in males was 56.5%. In the study conducted by Marlies Ostermann in Riyadh, 65% were males.

Mean S. urea level on admission was 98.05 mg%, at discharge it was 53.53 mg%. Mean S. creatinine

level on admission was 2.62 mg%, at discharge it was 1.801 mg%. The difference at admission, discharge was statistically significant ($p < 0.01$). Mean input and output volume of patient significantly improved at discharge from their admission values ($p < 0.01$). The results showed continuous improvement in Renal function tests in the patients during the course of treatment.

Bouchard et al. in a similar study observed the mean S. creatinine level on admission as 2.5 mg% and 47% patient's recovered normal renal functions at the time of discharge after treatment. In a study by Ravindra et al. to assess the mortality and recovery of renal functions in ARF, mean S. creatinine level on admission was 4.1 mg% and on discharge 53% patients had S. creat. Levels of < 1.2 mg%. [15]. Nash et al reported complete recovery of renal function in 38.6% of their patients, HS Kohli et al. [12] reported this in 86.3% and Anupama Kaul [16] in 45 % of patients. HSKohli et al. [12] demonstrated that in their group mean peak serum creatinine was 4.2 ± 2.21 (2.0- 10.6 mg/dl).

Urine Albumin was present in 29.8% patients while pus cells and RBC was present in 16.3% and 9.6% patients. Albumin is the preferred urinary protein. Increased urinary excretion of albumin is the earliest manifestation of CKD due to diabetes, other glomerular diseases, and hypertensive nephrosclerosis. Albuminuria may also accompany tubulointerstitial diseases, polycystic kidney disease, and kidney disease in kidney transplant recipients.

On USG, normal kidneys was observed in 47 of the patients (45.2%) while grade I and II RPC was observed in 35.6% and 9.6% patients. Other findings included Cystitis (9.9%), Pyelonephritis (3.8%), and obstructive uropathy (1.9%). ARDS on X-ray was observed in 7.7% while pulmonary oedema in 5.8% patients. Abnormal echo findings were observed in 7.7% patients.

Conclusion

- Mean S. urea level on admission was 98.05 mg%, & at discharge it was 53.53 mg%. The difference at admission & discharge was statistically significant ($p < 0.01$).
- Mean S. Creatinine level on admission was 2.62 mg%, & at discharge was 1.801 mg%. The difference at admission & discharge was statistically significant ($p < 0.01$).
- Urine Albumin was present in 29.8% patients while pus cells and RBC was present in 16.3% and 9.6% patients.

References

1. Khan RZ, Badr KF: Endotoxin and renal function. Perspectives to the understanding of septic acute renal failure and Toxic shock. *Nephrol Dial Transplant* 1999;14:814-18.
2. Shrier Rw, wang. Acute renal failure and sepsis. *N Engl J Med* 2044;351:159-69.
3. Chugh Ks. Snake-bite-induced acute renal failure in India. *Kidney Int.* 1989.pp.891-907.
4. Marcello tonelli. Acute renal failure in intensive care unit: a systemic review of impact of dialytic modality on mortality and renal recovery. *AM J Kidney Dis*, 2002 Nov;40(5):875-85.
5. Wardle EN. Acute renal failure and multiorgan failure. *Nephron* 1994;66:380-85.
6. John W Lohr. A clinical index to predict survival in acute renal failure patients requiring dialysis. *American journal of kidney diseases.* March 1998;254-59.
7. Agrawal Malay et al. acute renal failure. *American family physician* 2000;26:2077-88.
8. Shrier Esson. Diagnosis and treatment of acute tubular necrosis. *Ann of Int Med.* 1993;137(9):744.
9. Cerdá, Jorge, et al. Epidemiology of acute kidney injury. *Clinical journal of the American Society of Nephrology* 2008;3(3):881-86.
10. Shusterman N, Strom BL, Murray TG, et al. Risk factors and outcome of hospital acquired acute renal failure. *Clinical epidemiologic study.* *Am J Med.* 1987 Jul;83(1):65-71.
11. Shema L, Ore L, Geron R, Kristal B. Hospital-acquired acute kidney injury in Israel. *Isr Med Assoc J.* 2009;11(5):269-74.
12. HS Kohli, Madhu C Bhaskaran, Thangamani, Kandavel Thennarasu, Kamal Sud, Vivekanand Jha, Krishan L. Gupta and Vinay Sakhuja Treatment-related acute renal failure in the elderly: a hospital-based prospective study. *NDT* 2000;15:212-17.
13. Jha V, Malhotra HS, Sakuja V, Chugh KS. Spectrum of hospital acquired acute renal failure in the developing countries- Chandigarh study. *Q J Med.* 1992;83(303):497-505.
14. Prakash J, Murthy AS, Vohra R, Rajak M, Mathur SK. Acute renal failure in the intensive care unit. *J Assoc Physicians India* 2006 Oct;54:784-88.
15. Mehta, Ravindra L., et al. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. *Jama* 2002;288(20):2547-53.
16. Davies F, Weldon R. A contribution to the study of "war nephritis". *Lancet* 1917;ii:118-20.