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Original Research Article

A Tertiary Hospital Based Study of the Clinical Profile, Outcome, and Prognostic Factors of Acute Kidney Injury

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Abstract:

Background: Acute kidney damage (AKI) is a significant factor in hospital mortality, particularly in patients who are critically unwell. Different clinical presentations are seen. To determine prospective areas for intervention, a thorough understanding of AKI is necessary. The course of renal disease can be stopped by early detection and treatment of AKI in a possibly reversible stage. These study objectives were to evaluate the clinical profile of acute kidney injury, including its etiologies, and to identify its prognostic factors and results.

Methods: This observational study took place in a hospital. The study population was evaluated once the relevant inclusion and exclusion criteria had been applied. The clinical profile of AKI was evaluated, including the etiology, symptoms, indicators, and blood tests. AKI's stage and result were identified. In an effort to better monitor patients with AKI, factors that influence AKI outcome were sought for. Using the chi square test and Kruskal Wallis tests, associations were identified.

Results: The study population clinical profile was discovered to be comparable to prior research. The most frequent type of renal failure was intrinsic, and sepsis was the most frequent cause. A significant correlation between the result and eGFR, hospital days, and KDIGO stage of AKI was found. Poor outcome was linked to the need for hemodialysis, ventilator support, and inotropes. The 24 hour urine output, blood urea, and serum creatinine were the best predictive indicators.

Conclusion: Any patient who is brought to the hospital should be kept on an input-output chart and have their urine output regularly watched for any changes. Hypotension and sepsis need to be treated right away. In the case of AKI, conservative treatment is the recommended course of action because, as with all diseases, prevention is always preferable than cure.

Keywords: Acute kidney injury, 24 hour urine output, blood urea, serum creatinine, hemodialysis, sepsis.

Introduction

Acute kidney injury (AKI), formerly known as acute renal failure, is characterized by an abrupt decline in renal function that causes the retention of waste products including nitrogenous waste that the kidneys would normally eliminate. AKI suggests that kidney injury is a continual process with a wide variety of manifestations, from moderate to severe.

Uncertainties in the epidemiology and clinical management of AKI have been reduced by the latest KDIGO definition and classification based on objective indicators such as urine output and creatinine levels.[1]

AKI is common in hospitalized patients, especially those who are severely sick, and both its incidence and fatality rates1-31% for the former and 28-82% for the latter vary greatly globally.[2] It is now a significant risk factor for mortality in intensive care units and is no longer only a passive observer reflecting co-existing illnesses.

Prerenal (induced by decreased renal perfusion, primarily due to volume depletion), intrinsic renal (caused by injury to the kidney itself), and postrenal (caused by blockage to urine flow) are the three broad categories of causes of AKI.

Either the community or the hospital may have acquired it. While medicines, distal obstructions, and medications are the main causes of fluid loss in the former, the major causes of fluid loss in the latter include sepsis, operations, heart or liver failure, contrast administration, and drugs. Envenomation, leptospirosis, and malaria are a few region-specific etiologies that are seen in this area

of the country. The reasons vary greatly from country to country. [3]

To reduce mortality, early identification and aggressive care of AKI are essential. Due to late presentation to tertiary care facilities and a lack of recording of medical care, the true epidemiological picture of AKI in a nation like India is still not fully recognized, and these are the main causes for starting this study. This research may aid in the early and reversible identification of AKI, halting its progression to total renal failure and even death. In order to prevent renal damage, this study also provides a bird's-eye view of the key prognostic indicators that should be monitored if a patient is in early renal failure.

Materials and Methods

The study was a hospital based non-interventional observational descriptive study conducted in Between September 2021 and February 2022 at Department of Medicine, Atal Bihari Vajpayee Institute of Medical Sciences and Ram Manohar Lohia Hospital, New Delhi.

All patients over the age of fifteen who arrived at our hospital during the study period with a sudden rise in serum creatinine of at least 0.3 mg/dl from baseline within 48 hours, a rise in serum creatinine of at least 50% over baseline within one week, or oliguria (a decrease in urine output of less than 0.5 ml/kg/hour for more than 6 hours) were included in the study. People with known renal illness, established diabetes or hypertensive nephropathy, connective tissue disorders such systemic lupus erythematosis, and refusal to consent to participate in the study were all excluded from participation.

90 of the 128 patients who were interviewed during the study period after applying the aforementioned inclusion and exclusion criteria were included in the final analysis. Twenty patients were unwilling to participate in investigations, while 18 patients refused to grant consent. For this investigation, a significant sample size of 75 was estimated and statistically validated.

The complete set of gathered data was imported into Microsoft Excel 2020 and put through the proper statistical analysis software. Chi-square and Kruskal Wallis tests with p values <0.05 were considered to indicate a significant connection.

Results

The study total sample size was 90. Only 16 people in the research population, with a mean age of 58.39 years and a standard deviation of 18 years, were under the age of 40. Out of 90 people, 53 men (58.9%) were present. A total of thirty patients from medicine wards (33.3%) and forty patients from the medical intensive care unit (44.4%) made up the study population. 8 and 12 patients from the surgical ICU and wards, respectively, were included. Fever was the most prevalent symptom, occurring in 44 (48.8%) of the sample group, with a mean duration of 4 days. Symptoms of the study population over time are shown in table 1 below.

Table 1: Symptoms of AKI with duration

Symptom	Frequency (percentage)	Duration in days(mean ± SD)
Fever	44 (48.8%)	4.36 ± 3.56
Oliguria	33 (36.6%)	2.61 ± 1.99
Vomiting	22 (24.4%)	3.95 ± 9.93
Edema	22 (24.4%)	5.27 ± 6.54
Breathlessness	21 (23.3%)	3.43 ± 2.95
Altered sensorium	20 (22.2%)	1.60 ± 2.08
Abdominal pain	15 (16.6%)	4.40 ± 2.55
Dysuria	5 (5.5%)	9.33 ± 4.61

43% of the study sample exhibited pitting pedal edema and 43% had pallor. Mean systolic blood pressure was 135.89± 37.98 millimeters of mercury, and mean diastolic blood pressure was 83.11± 18.338 millimeters of mercury. The average respiratory rate was 21.49±4.71 beats per minute and the average pulse rate was 91.86± 17.227 beats per minute. Another important symptom was the presence of bilateral basal crepitations, which were found in 26.6% of cases. Table 2 below lists the median results of first-day blood tests.

Table 2: First day blood investigations

Investigation	Mean	Standard deviation
Hemoglobin (grams/dl)	11.279	2.04
WBC count (cells/cumm)	14310.56	8134.82
Platelet count (cells/cumm)	240762.22	113026.28
ESR (mm after 1 hour)	43.61	24.14
Blood urea (mg/dl)	70.94	46.95
Serum creatinine (mg/dl)	3.079	2.9479
Blood sugar (mg/dl)	128.78	63.23
eGFR	34.7147	22.6133

Total bilirubin (mg/dl)	1.218	0.972
Direct bilirubin (mg/dl)	0.329	0.67
SGPT (IU)	80.64	95.82
SGOT (IU)	68.11	42.097
ALP (IU)	96.7	82.342
Albumin (mg/dl)	3.12	0.68
Globulin (mg/dl)	3.08	0.62
Sodium (mEq/L)	129.38	15.54
Potassium (mEq/L)	4.098	0.92
Calcium (mg/dl)	8.043	0.8946
Phosphorous (mg/dl)	4.541	1.801

90 patients were treated, and 28 patients (31.1%) had 1+ albuminuria, 14 had 2+, and 7 had 3+. Only 13 individuals (14.4%) had routinely negative albumin tests. There was hematuria in 20 patients (22.2%). The only other acid base condition identified was metabolic acidosis, which was found in 57 patients, or 63.3% of the research group. 75.5% of the study group had hypocalcemia, which was followed by hyponatremia in 66.67%, hypokalemia in 26.67%, hypophosphatemia in 22.2%, and hyperkalemia in 13.33% of the population.

70% of the study group had intrinsic renal failure, followed by 6.7% of postrenal failure and 23.3% of pre-renal failure. Applying clinical data and computing sodium fractional excretion, the same result was discovered. Stage 3 AKI affected 44 of the 90 patients (48.9%), stage 2 affected 28, and stage 1 affected 18 of the 90 patients (20%). In this study, 92.2% of AKI cases were community-acquired, and only 8 patients developed AKI in hospitals, of whom 3 had used vancomycin, 2 had taken aminoglycosides, and 3 had experienced substantial diarrhea while receiving hospital care. Sepsis was the most frequent cause of AKI in this study, accounting for 43 patients (47.7%), as shown by a relative rise in total leukocyte count in the blood tests mentioned above. Among individuals with infections, 8.8% had cellulitis, skin and soft tissue infections, 14.4% had respiratory tract infections, and 21.1% had urinary tract infections. AKI was induced by acute glomerulonephritis in 7.8% of cases and accelerated hypertension in 5.6%.

AKI results can be graded as expired, partially recovered, or fully recovered. 90 people were examined; 16 (17.8%) died from their illnesses, while 35 (38.9%) recovered totally and 39 (43.3%) only partially. Only fourteen patients required hemodialysis throughout their hospital stay, and 84% of patients received conservative care. A total of 79 individuals (87.8%) received antibiotics. 14 (15.6%) patients required inotrope support, and 13 (14.4%) required ventilator support. Five of the fourteen patients who received hemodialysis died, eight required long-term maintenance hemodialysis, just one recovered fully.

The accompanying table (table 3) displays the patient follow-up, mean blood urea, mean serum creatinine, and mean 24-hour urine output for the first five days.

As the major focus of this investigation, it was attempted to determine the relationship between various characteristics and the outcome of AKI in order to identify potential prognostic indicators. Age, sex, premorbidities like diabetes, hypertension, and addictions did not affect how AKI turned out. Similar high blood counts, abnormal liver function tests, and serum electrolyte levels had no effect on the result. A few variables and the outcome were found to be significantly correlated; these variables are combined in table 4 below.

Table 3: Follow up of the study population

Urine output	**Mean (ml/day)**	**Standard deviation**
Day 1	1067.96	611.75
Day 2	1222.137	701.50
Day 3	1300.462	665.69
Day 4	1211.191	585.40
Day 5	1241.522	705.99
Blood Urea	**Mean (mg/dl)**	**Standard Deviation**
Day 1	71.75	50.21
Day 2	71.03	51.12
Day 3	68.21	48.71
Day 4	71.94	53.45
Day 5	70.64	55.98
Serum Creatinine	**Mean (mg/dl)**	**Standard Deviation**

Day 1	3.097	2.94
Day 2	3.092	2.43
Day 3	2.849	2.04
Day 4	2.923	1.86
Day 5	2.74	1.77

Table 4: Association of various factors to outcome of AKI

Factor	**Outcome**			**Kruskal Wallis test(P value)**
	Expired (mean ± SD)	**Partial recovery (mean ± SD)**	**Complete recovery (mean ± SD)**	
Mean hospital stay in days	7.94 ± 3.24	10.69 ± 6.35	6.86 ± 3.566	0.01*
eGFR	34 ± 20.78	29.09 ± 26.68	41.30 ± 16.44	0.002*
Stage of kidney disease	**Expired (number of patients)**	**Partial recovery (number of patients)**	**Complete recovery (number of patients)**	**Chi square calculated/p value**
Stage 1	0 (0%)	3 (16.66%)	15 (83.3%)	42.730*/0.0001*
Stage 2	2 (7.1%)	10 (35.7%)	16 (57.7%)	
Stage 3	14 (31.8%)	26 (66.67%)	4 (9%)	

*Statistically significant data

The length of the hospital stay, eGFR, and stage of acute renal damage were all significantly correlated with the outcome. Out of 21 prerenal AKI patients, 12 fully recovered, 4 partially recovered, and 5 passed away. Out of 63 patients with intrinsic renal disease, 8 passed away, 32 only recovered partially, and 23 fully. Three of the six patients with post-renal AKI died, while three others only partially recovered. Sepsis was the most frequent reason for patient admission, and of those patients, 9 died, 8 just partially recovered, and 11 totally recovered. Out of the 76 patients who received conservative care, 44.7% made a full recovery, 40.7% made a partial recovery, and 14.4% passed away from their illnesses. May be the group of patients taken for hemodialysis were more sick than the conservatively managed group; outcome of the former was also bad. Out of 14 patients taken for hemodialysis only one had complete recovery, 8 had partial recovery and 5 succumbed to illness.

Regarding the monitoring of AKI patients, the Kruskal Wallis test was used to determine the statistical significance of the associations between daily blood urea and serum creatinine concentrations and the course of the disease. The results are displayed in table number 5 below. Therefore, a poor prognostic indicator for AKI is a progressive decrease in urine production and a progressive rise in blood urea and serum creatinine.

Table 5: Association of 24 hour urine output, blood urea and serum creatinine to outcome of AKI

Variable	**Expired (mean ± SD)**	**Partially recovered (mean ± SD)**	**Fully recovered (mean ± SD)**	**Kruskal Wallis Test (P value)**
Urine Output Day 1 (ml)	964.188 ± 473.73	982.967 ± 621.63	1210±643.87	0.262
Urine Output Day 2 (ml)	1020.063±541.9	1142.213±720.06	1403.571±720.21	0.163
Urine Output Day 3 (ml)	935±483.11	1310.092±735.58	1456.8±603.46	0.014*
Urine Output Day 4 (ml)	839.286±426.37	1186.766±702.64	1492.857±298.61	0.004*
Urine Output Day 5 (ml)	872.222±487.07	1041.170±686.4	1852.417±484.19	0.002*
Blood Urea Day 1 (mg/dl)	82.725±62.69	75.51±55.96	62.54±34.18	0.667
Blood Urea Day 2 (mg/dl)	105.38±81.72	73.46±44.47	52.63±27.79	0.019*
Blood Urea day 3 (mg/dl)	112.56±73.36	66.97±40.06	49.31±27.34	0.001*
Blood Urea day 4 (mg/dl)	139.77±79.17	61.00±24.27	45.05±18.70	0.001*
Blood Urea day 5 (mg/dl)	152.14±96.90	62.74±22.04	38.25±9.17	0.001*
Serum creatinine day 1 (mg/dl)	2.37±1.26	4.126±3.64	2.283±2.22	0.02*
Serum creatinine day 2 (mg/dl)	3.35±1.60	4.144±2.97	1.80±1.20	0.01*
Serum creatinine day 3 (mg/dl)	3.963±1.68	3.646±2.28	1.45±0.69	0.01*
Serum creatinine day 4 (mg/dl)	4.43±1.65	3.438±1.69	1.29±0.58	0.01*
Serum creatinine day 5 (mg/dl)	4.81±1.27	3.056±1.53	0.97±0.09	0.01*

Discussion

Ninety AKI patients who were hospitalized participated in this study. The average age was 58, which was comparable to research by Turney et al. and research by Prakash et al. in India. [4,5] Despite their being no statistically significant correlation, the mean age of expired patients was much higher than that of recovered patients. In

contrast, a comparable Spanish study found that getting older had an impact on the outcome of AKI. [6] This study's detection of a greater incidence of AKI in men is consistent with numerous other investigations. [7] Unknown genetic factors could have a role in the emergence of AKI. In this study, patients from the departments of medicine and surgery were included, and 62 of the 90 participants came from intensive care units. Increased co-morbidities including hypotension and infections may be to blame for this. In this investigation, it was discovered that fever was the most typical presenting symptom. Oliguria is predicted to be the most prevalent sign of AKI in the majority of research conducted globally. [8] Sepsis is the most frequent cause seen here, which may explain this discrepancy. Anemia affected 43% of the study's participants. Anemia was a factor in 53 out of 56 individuals with AKI in a Canadian study, which earlier brought attention to this issue. [9] Since renal failure was preceded by anemia in about one-third of cases, anemia was multifactorial. Anemia may be a sign of chronic kidney disease or other long-term illnesses. Volume overload was the main factor in the findings of the systemic assessment. Acute pulmonary edema may cause bilateral basal crepitations, and peritoneal fluid buildup may cause abdominal distension. A substantial correlation between the incidence, progression, or outcome of AKI and co-morbid conditions such diabetes or hypertension could not be found.

Apart from anemia, neutrophilic leukocytosis was the most frequent finding on a routine hemogram. Leukocytosis and luekocytosis are also linked to higher mortality rates, albeit no analogous results were found here. [10] In this study, the majority of the patients had very little proteinuria. In this investigation, the urine microscopy proved useful in diagnosing AKI. However, there was no connection between the results of the microscopy and the AKI. Bagshaw and colleagues had previously taken blood and urine samples from 83 severely ill sepsis patients, 52% of whom had AKI. They developed a urine microscopy score based on the measurement of renal tubular epithelial cells and granular casts in sediments and shown that, despite both AKIs being equally severe, septic AKI was linked with more urine microscopy signs of kidney injury. [11] The likelihood of increasing AKI was likewise predicted by a higher urine microscopy score. The most prevalent electrolyte abnormality linked to AKI in this investigation was hypocalcemia, which affected 75.5% of the sample group. Hyponatremia, hypophosphatemia, hypokalemia, and hyperkalemia came next. Electrolyte abnormalities and AKI result were not shown to be significantly correlated. The metabolic abnormalities of hypocalcemia, hyper phosphatemia, and a rise in immunoreactive parathyroid hormone are particularly frequent, and calcium and parathyroid abnormalities continue into the diuretic stage of AKI. [12] According to some research, hyponatremia and hypocalcemia were the most prevalent electrolyte abnormalities after hyperkalemia. Hypoalbuminemia was the sole anomaly seen in the liver function tests. Administration of human albumin solution has shown some potential in AKI prevention, and hypoalbuminemia may unintentionally contribute to the development of AKI. [13]

Based on the greatest AKI stage that each patient had achieved throughout their hospital stay, the patients were categorized into stages 1, 2, and 3. This study unmistakably demonstrated that the result is strongly impacted by the disease's progression. The short- and long-term effects of AKI have been demonstrated to be correlated with the stage of the disease. The same Kidney Disease Improving Global Outcomes (KDIGO) criteria employed here were discovered to be an effective predictor of 30-day death in patients who had increased post-operative serum creatinine and underwent heart operations in a Brazilian investigation. [14] The studies that are available show that even temporary changes in renal function increase mortality. The majority of the study population—70%—had postrenal failure, 23% had prerenal failure, and the remaining were in intrinsic renal failure. Sepsis was the most frequent etiology for the same, accounting for 92.2% of the study population's causes as community acquired conditions. There are notable differences between common causes and risk factors of AKI in developed and poor nations. The most frequent cause of acute tubular necrosis in the tropics continues to be community acquired infections, which are less common than trauma, workplace accidents, medications, cardiogenic reasons, and rejection of renal transplants in industrialized nations. The primary causes of AKI in India were sepsis, hypotension, and aminoglycosides.[15] These are the several situations where we should be watchful of the patient's renal condition.

In terms of the management, of the 76 patients who were conservatively managed, 11 (14.4%) passed away from the illness, 31 had a partial recovery, and 34 made a full recovery. The mainstay of treatment for AKI was intravenous fluids, however this was done carefully while closely monitoring urine output to prevent pulmonary edema. Another crucial component of treatment, particularly for sepsis patients, was the use of antibiotics. A few patients in the study population required a ventilator and inotrope assistance. Eight out of thirteen patients who needed a ventilator and seven out of fourteen patients who needed an inotrope passed away as a result of their illnesses, so these two requirements may be interpreted as having a

negative prognostic indicator. Every time an antibiotic is administered, the dose must be calculated depending on the patient's creatinine clearance.

Diuretics are a typical drug used in the conservative therapy of AKI. However, employing diuretics on patients who have AKI was compared to beating a horse that was about to expire. The same was proved in trials that showed diuretics were unsuccessful in avoiding AKI or improving its results. [16] Despite decades of research, there is now just supportive care available as a specialized therapy for AKI. Patients started on hemodialysis had a worse result than those under conservative care, which may be related to the higher severity of the disease and the elevated stage of AKI. Only one of the 14 patients who were started on dialysis fully recovered; eight had to be kept on hemodialysis, and five patients passed away. In the SHARF (Stuivenberg Hospital Acute Renal Failure) research, fatality rates for AKI patients who had received hemodialysis were 58% and 43%, respectively. As a result, when starting renal replacement therapy for AKI, a cautious approach is necessary.

Without considering the monitoring and prognostication elements of AKI, the discussion will fall short. In this investigation, it was discovered that daily urine output, blood urea, and serum creatinine were excellent indicators of AKI outcome. Oliguric AKI was discovered to have a much worse prognosis than non-oliguric AKI.[17] In this investigation, poor survival outcomes were demonstrated by deteriorating blood urea and creatinine values despite optimal conservative therapy. According to our study, these are the three most significant predictors of the outcome of AKI, and we strongly advise monitoring them in critically sick patients in order to detect AKI very early and prevent it from getting worse. In this investigation, only in-hospital outcomes were taken into account for determining the final result. Hospital deaths made up 17.8% of the study population, whereas partial and full recoveries made up 43.3% and 38.9%, respectively. Patients who reacted to treatment but did not return to baseline creatinine levels over the research period made up the bulk of the partial recovery group. AKI patients' reported hospital mortality rates ranged from 13.3% to 49.1%. [18] A prospective study of AKI in ICU patients in India discovered a death rate of 52% over time. [19] According to studies, the majority of AKI deaths happen within 60 days, so a follow-up period of 60 to 90 days would be sufficient for a trustworthy examination of the mortality rate. [20]

Conclusion

The clinical illness known as acute kidney injury has a variety of etiologies, pathophysiologies, and prognostic variables. The key clinical characteristics are oliguria and fever. The most typical cause of AKI is sepsis. Intrinsic renal failure was the most typical kind of AKI. Premorbid conditions including diabetes and hypertension had little impact on the outcome of AKI. The following variables had an impact on the outcome: eGFR, KDIGO stage of AKI, hospital stay time, requirement for inotrope support, ventilator use, and hemodialysis. Antibiotics and intravenous fluid are the cornerstones of the conservative care strategy, which has been proven to be the most effective way to treat AKI. By measuring the patient's 24-hour urine output, blood urea, and serum creatinine on a daily basis, AKI patients can be monitored.

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