Original Research Article

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

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ABSTRACT

Background: AKI, or acute kidney injury, plays a major role in hospital mortality, especially in patients with severe illness. There are several clinical manifestations observed. A comprehensive understanding of AKI is required in order to identify potential areas for intervention. When AKI is discovered early and treated while it may still be reversible, the progression of renal damage can be prevented. **Methods:** From March 2023 to February 2024, this observational study was carried out in the Department of Medicine, DMCH, Laheriasarai, Bihar. After the pertinent inclusion and exclusion criteria had been met, the study population was assessed. An assessment was conducted on the clinical profile of AKI, encompassing the aetiology, symptoms, signs, and blood tests. The stage and outcome of AKI were determined. Finding the elements that affect the outcome of AKI was one way to try and monitor patients with AKI more effectively. Associations were found using the Kruskal Wallis and chi square tests. **Results:** The clinical profile of the study population was found to be similar to previous investigations. Sepsis was the most common cause of renal failure, which was intrinsic in nature. The outcome and the eGFR, hospital days, and KDIGO stage of AKI were found to be significantly correlated. The use of inotropes, ventilator support, and hemodialysis was associated with poor outcomes. The most accurate prognostic markers were serum creatinine, blood urea, and urine output over a 24-hour period. **Conclusions:** Upon admission to the hospital, every patient ought to be monitored closely for any alterations in their urine production and charted accordingly. Sepsis and hypotension require immediate medical attention. As is the case with other diseases, conservative treatment is advised in the case of AKI since prevention is always better than cure. **Keywords:** Acute kidney injury, 24 hour urine output, blood urea, serum creatinine, hemodialysis, sepsis

INTRODUCTION

An abrupt reduction in renal function leading to the retention of waste products, especially nitrogenous waste that the kidneys would ordinarily remove, is known as acute kidney injury (AKI), formerly known as acute renal failure. According to AKI, renal damage is a continuous process that can take many different forms, ranging from mild to severe. The most recent KDIGO criteria and classification, which is based on objective measures like urine output and creatinine levels, has helped to lessen uncertainties in the epidemiology and clinical management of AKI.[1]

AKI occurs often in hospitalized patients, particularly in those who are very ill. Its incidence and death rates vary widely throughout the world, ranging from 1-31% for the former to 28-82% for the latter.[2] It is no longer merely a passive observer reflecting co-existing illnesses; rather, it is becoming a significant risk factor for mortality in intensive care units.

The three main categories of causes of acute kidney injury (AKI) are postrenal (caused by blockage to urine flow), intrinsic renal (caused by injury to the kidney itself), and prerenal (produced by decreased renal perfusion, typically owing to volume depletion).

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It might have been obtained by the hospital or the community. The primary causes of fluid loss in the former group are medications, distal obstructions, and pharmaceuticals; in the latter group, sepsis, surgeries, heart or liver failure, contrast delivery, and drugs are the main causes of fluid loss. Some of the region-specific etiologies that are observed in this part of the country are envenomation, leptospirosis, and malaria. The causes differ significantly between nations.[3]

Early detection and intensive care for AKI are crucial for lowering mortality. The actual epidemiological picture of AKI in a country like India is still not fully recognized due to late presentation to tertiary care institutions and a lack of recording of medical care. These are the key reasons why this study is being conducted. This study could help detect AKI early and treat it effectively, preventing complete renal failure and even death. This study also offers an overview of the important prognostic markers that need to be watched if a patient is experiencing early renal failure in order to avoid kidney damage. The aetiology, prognostic factors, prognosis, and clinical features of acute renal injury were all assessed in this investigation.

MATERIAL AND METHODS

From March 2023 to February 2024, a non-interventional observational descriptive study was conducted in the department of Medicine at Darbhanga Medical College and Hospital, Laheriasarai, Bihar. The study included all patients over the age of fifteen who visited DMCH during the study period and had oliguria (a decrease in urine output of less than 0.5 ml/kg/hour for more than 6 hours) or an acute rise in serum creatinine of at least 0.3 mg/dl from baseline within 48 hours of admission, at least 50% greater than baseline within one week of admission. People with known renal illness, established diabetes or hypertensive nephropathy, connective tissue disorders such systemic lupus erythematosus, and refusal to consent to participate in the study were all disqualified 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.

After receiving informed consent, patients were told about the study. They were assessed through in-person interviews using a statistically verified proforma that placed an emphasis on a thorough medical history, clinical examinations, and pertinent investigations. Investigations and interviews were conducted on the same day without causing any discomfort to the research population.

According to KDIGO recommendations, AKI staging was carried out after the examination. Prerenal, intrinsic renal, postrenal, community- and hospital-acquired, and other etiologies were found as the probable causes. Following a thorough diagnosis, the patient was often offered two treatment choices dependent on the severity of the disease: conservative and hemodialysis. Anuria, refractory rapidly deteriorating renal failure, hyperkalemia, refractory metabolic acidosis, uremic pericarditis, and uremic encephalopathy were the criteria for hemodialysis. Every day, the 24-hour urine output, blood urea, and serum creatinine readings of every patient were monitored. Mortality, a partial recovery, and a full recovery of renal functions were the in-hospital outcomes of AKI taken into consideration. The outcome and the variables influencing it were carefully examined, and an effort was made to identify some prognostic indications for the outcome in AKI. The full set of gathered data was imported into Microsoft Excel 2010 and put via the proper statistical analysis software. Chi-square and Kruskal Wallis tests with p value <0.05 were considered to indicate a significant connection.

RESULTS

The study total sample size was 90. Only 16 people in the study 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 (40.8%) 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

Pallor and pitting pedal edoema were seen in 43% of the study population. The average blood pressure was 135.89± 37.98 mmHg for the systolic component and 83.11±18.338 mmHg for the diastolic component. The mean respiratory rate was 21.49 beats per minute and the mean pulse rate was 91.86 beats per minute. Another important symptom was the 26.6% of cases in which bilateral basal crepitations were observed. Table 2 below lists the average 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/cmm)	14310.56	8134.82
Platelet count (cells/cmm)	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%, hyperphosphatemia 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 diarrhoea 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.

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 (ml/day)	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

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 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%)	

The length of the hospital stay, eGFR, and stage of acute renal damage were all significantly correlated with the outcome. Out of 21 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 74 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. Perhaps the patients who had hemodialysis were sicker than the individuals who received conservative management; the former's results were likewise poor. Only one of the 14 patients who received hemodialysis recovered completely, 8 only partially, and 5 ultimately passed away.

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 leukocytosis 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. 83 severely ill sepsis patients, of whom 52% had AKI, had blood and urine samples previously collected by Bagshaw and colleagues. 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, hyperphosphatemia, hypokalemia, and hyperkalemia came next. Electrolyte abnormalities and AKI result were not shown to be significantly correlated. The metabolic abnormalities of hypocalcemia, hyperphosphatemia, 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 based on the patient's creatinine clearance. Diuretics are a typical medication used in the conservative management 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] Similarly worsening blood urea and creatinine value despite optimum conservative management showed poor survival outcomes in this study. 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 mortality 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, Patho physiologies, 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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