MAK HILL Publications International Journal of Tropical Medicine

Research Article
doi: 10.36478/makijtm.2024.3.79.83

To Study Clinico Etiological Profile of Patients with Acute Kidney Injury Admitted in Medical ICU KIMS, Koppal

[1]T. Shivaprasad, [2]Gavisiddesh Ronad, [3]Krishna Kumar Naik, [4]J. Srinivas, [5]Umesh Rajoor and [6]Sharanappa

[1-6]Department of General Medicine, Koppal Institute of Medical Science, Koppal India

Abstract

Acute Kidney Injury (AKI) is a critical condition with significant morbidity and mortality, particularly in intensive care units (ICUs). This study aimed to delineate the clinic-etiological profile of patients with AKI admitted to the medical ICU at Koppal Institute of Medical Sciences (KIMS), Koppal. Understanding the demographic, clinical characteristics, and etiological factors is essential for improving management and outcomes. This prospective hospital-based study included 50 patients aged above 18 years, diagnosed with AKI based on the Acute Kidney Injury Network (AKIN) criteria. Data were collected on demographic details, clinical presentation, comorbidities and laboratory investigations. Etiological factors were identified and outcomes were tracked, including the need for dialysis and mortality. Statistical analyses were performed to evaluate changes in renal function over time. The study population included 50 patients with a balanced distribution across age groups: 18-30 years (30%), 31-50 years (24%), 51-70 years (26%), and >70 years (20%). Females comprised 54% of the cohort. Common symptoms included hematuria (28%), abdominal pain (16%), and abdominal distension (20%). Hypertension (36%) and diabetes (44%) were prevalent comorbidities. Significant etiological factors were hypovolemia (28%), obstruction (30%), and snake bites (30%). Baseline serum urea and creatinine levels were 43.3 mg/dL (±6.3) and 1.3 mg/dL (±0.1), respectively. At 48 hours, significant decreases in serum urea (35.4 mg/dL, p=0.005) and creatinine (1.0 mg/dL, p=0.02) were observed. Dialysis was required in 44% of cases. This study highlights the diverse etiological factors and significant burden of comorbidities associated with AKI in a rural ICU setting. Considering the varied clinical presentations and underlying causes, early recognition and management of AKI are crucial for improving patient outcomes. The findings underscore the need for targeted interventions and resource allocation to address the high morbidity and mortality associated with AKI.

OPEN ACCESS

Key Words

Acute kidney injury, AKIN criteria, ICU, hypovolemia, obstruction, snake bite, dialysis, mortality

Corresponding Author

Sharanappa,
Department of General Medicine, Koppal Institute of Medical Science, Koppal India
sharanuveerapur555@gmail.com

Author Designation

[1,2,3]Assistant Professor
[4]Associate Professor
[5]HOD and Professor
[6]3rd year PG Resident

Received: 25 May 2024
Accepted: 30 June 2024
Published: 6 July 2024

Citation: T. Shivaprasad, Gavisiddesh Ronad, Krishna Kumar Naik, J. Srinivas, Umesh Rajoor and Sharanappa, 2024. To Study Clinico Etiological Profile of Patients with Acute Kidney Injury Admitted in Medical ICU KIMS, Koppal. Int. J. Trop. Med., 18: 79-83, doi: 10.36478/ makijtm.2024.3.79.83

INTRODUCTION

Acute Kidney Injury (AKI) is a significant clinical condition characterized by a sudden decline in renal function, accumulating waste products and disturbances in fluid, electrolyte and acid-base balance. AKI is associated with increased morbidity, mortality, and healthcare costs, particularly in critically ill patients admitted to medical intensive care units (ICUs). According to the Acute Kidney Injury Network (AKIN) criteria, AKI is classified into three stages based on the severity of serum creatinine increase and urine output reduction, which aids in standardized diagnosis and management[1].

The etiology of AKI is multifactorial, encompassing pre-renal, intrinsic and post-renal causes. Pre-renal causes, such as hypovolemia and hypotension, account for a significant proportion of cases and are often reversible if identified and treated promptly. Intrinsic renal causes include acute tubular necrosis, glomerulonephritis and interstitial nephritis, while post-renal causes involve urinary tract obstruction[2]. The incidence and outcomes of AKI vary widely based on patient demographics, comorbidities and the healthcare setting, underscoring the need for localized studies to inform clinical practice.

In India, the burden of AKI is compounded by late presentation, limited resources and varying levels of healthcare access. Studies from different regions have highlighted the diverse etiological spectrum of AKI, ranging from infections and sepsis to nephrotoxic drug exposure and underlying chronic conditions[2-4]. However, there is a paucity of data from rural and semi-urban healthcare centers, such as the Koppal Institute of Medical Sciences (KIMS), which serves a significant population in Northern Karnataka.

This study aims to delineate the clinic-etiological profile of patients with AKI admitted to the medical ICU at KIMS, Koppal. By systematically collecting and analyzing data on patient demographics, clinical presentation and laboratory parameters, we seek to identify prevalent etiological factors and outcomes associated with AKI in this setting. The findings will contribute to a better understanding of AKI in a rural healthcare context and guide the development of targeted interventions to improve patient outcomes.

MATERIALS AND METHODS

This prospective, hospital-based study was conducted in the medical intensive care unit (ICU) of the Koppal Institute of Medical Sciences (KIMS), Koppal, Karnataka, from August 1, 2022, to September 30, 2024. The study aimed to investigate the clinic-etiological profile of patients diagnosed with Acute Kidney Injury (AKI) based on the Acute Kidney Injury Network (AKIN) criteria.

The study population comprised 50 patients aged above 18 years who were diagnosed with AKI during their admission to the medical ICU. Patients were included if they met the AKIN criteria for AKI, which involved three stages. Stage 1 is defined as an increase in serum creatinine of more than or equal to 0.3 mg/dl from baseline, a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of <0.5 ml/kg/h for more than six hours). Stage 2 includes increased serum creatinine (two to three times from baseline) or a urinary output lower than 0.5 ml/kg/h for twelve hours. Stage 3 is characterized by an increase in serum creatinine (three times or more from baseline), a serum creatinine higher than 4 mg/dl if there is an acute rise in serum creatinine of at least 0.5 mg/dl, a urinary output lower than 0.3 ml/kg/h for twenty-four hours, or anuria for twelve hours. Stage 3 also includes patients who require renal replacement therapy, irrespective of their stage at the time of renal replacement therapy. Additionally, patients with increased serum creatinine to >1.5 times baseline, presumed to have occurred within the prior seven days, and a urine volume of <0.5 ml/kg/h for six hours were included. Patients with pre-existing chronic kidney disease were excluded from the study.

Data collection involved obtaining written informed consent from all participants. Initial data recorded included age, sex, place of residence, occupation, and address. A detailed history and clinical examination were conducted for each patient, followed by a series of laboratory investigations. These included a complete blood count, urine routine with microscopy, randomized blood glucose, serum electrolytes, renal function tests, liver function tests, chest radiographs and ultrasonography of the abdomen and pelvis. For serum urea and creatinine, samples were taken at admission, at 24 hours and 48 hours and were repeated daily until the patient was either discharged or deceased. Ethical clearance was obtained from the institutional ethical committee.

RESULTS AND DISCUSSIONS

The study included a total of 50 patients with Acute Kidney Injury (AKI) admitted to the medical ICU. The age distribution of the patients shows that 15

Fig. 1: Etiological Factors and Diagnosis

Table 1: Demographic and Clinical Characteristics of Patients with AKI

Variable	Category	Frequency (%)
Age (years)		
	18-30	15 (30%)
	31-50	12 (24%)
	51-70	13 (26%)
	>70	10 (20%)
Sex	Male	23 (46%)
	Female	27 (54%)
Symptoms	Hematuria	14 (28%)
	Pain abdomen	8 (16%)
	Abdominal distension	10 (20%)
	Seizures	7 (14%)
	Vomiting	5 (10%)
	Fever	6 (12%)
	Dyspnea	4 (8%)
Comorbidity	No	18 (36%)
	Hypertension	18 (36%)
	Diabetes	22 (44%)

Table 2: Laboratory Investigations at Baseline

Variable	Mean (SD) / Frequency (%)
Hemoglobin (g/dL)	11.9 (±1.3)
Total Leukocyte Count (cells/mm³)	7644.5 (±1068.0)
Platelets (cells/mm³)	381399.2 (±11420.7)
Serum Creatinine (mg/dL)	0.6 (±0.1)
Serum Urea (mg/dL)	35.0 (±4.3)
Serum Sodium (Na+, mEq/L)	143.7 (±4.9)
Serum Potassium (K+, mEq/L)	4.7 (±0.3)
Ultrasound (USG) of Abdomen	Normal: 23 (46%), Abnormal: 23 (46%)
Urine Routine	Normal: 27 (54%), Abnormal: 20 (40%)

Table 3: Changes in Renal Function Over Time

Time Point	Serum Urea (mg/dL) Mean (SD)	Serum Creatinine (mg/dL) Mean (SD)	p-value (Serum Urea)	p-value (Serum Creatinine)
Baseline	43.3 (±6.3)	1.3 (±0.1)	-	-
24 Hours	43.9 (±3.5)	1.4 (±0.2)	0.25	0.01
48 Hours	35.4 (±4.6)	1.0 (±0.2)	0.005	0.02

patients (30%) were between 18-30 years, 12 patients (24%) were between 31-50 years, 13 patients (26%) were between 51-70 years, and 10 patients (20%) were older than 70 years. This indicates a relatively even distribution across different age groups, with a slight predominance in the younger and middle-aged groups.Regarding gender, there were 23 male patients (46%) and 27 female patients (54%), showing a slight female predominance in the study population.

The patients' symptoms reported were diverse. Hematuria was noted in 14 patients (28%), pain in the abdomen in 8 patients (16%), abdominal distension in 10 patients (20%) and seizures in 7 patients (14%). Additionally, vomiting was observed in 5 patients (10%), fever in 6 patients (12%), and dyspnea in 4 patients (8%). These symptoms reflect the varied clinical presentations of AKI in the study population.
Regarding comorbidities, 18 patients (36%) had no comorbid conditions, while an equal number of patients (18, or 36%) had hypertension. Diabetes was present in 22 patients (44%), indicating a significant proportion of the study population had underlying chronic conditions that could contribute to the development of AKI.

Table 2 details the baseline laboratory investigations for the patients with Acute Kidney Injury (AKI). The mean hemoglobin level for the study population was 11.9 g/dL with a standard deviation of ±1.3 g/dL, indicating a mild variation in hemoglobin levels among the patients. The total leukocyte count had a mean value of 7644.5 cells/mm³ with a standard deviation of ±1068.0 cells/mm³, suggesting a relatively consistent leukocyte count across the patient cohort. Platelet counts were recorded with a mean of 381399.2 cells/mm³ and a standard deviation of ±11420.7 cells/mm³, showing a generally high platelet count in the study group. Serum creatinine levels at baseline had a mean of 0.6 mg/dL with a standard deviation of±0.1 mg/dL, which is within the normal range but indicates the starting point for the observation of AKI progression. Serum urea levels had a mean value of 35.0 mg/dL with a standard deviation of±4.3 mg/dL, reflecting the initial renal function status of the patients.

Electrolyte measurements showed a mean serum sodium level of 143.7 mEq/L with a standard deviation of±4.9 mEq/L and a mean serum potassium level of 4.7 mEq/L with a standard deviation of±0.3 mEq/L, both of which are within normal physiological ranges but are critical markers for renal function and electrolyte balance in AKI patients.

Ultrasound (USG) of the abdomen revealed that 23 patients (46%) had normal findings, while an equal number of 23 patients (46%) had abnormal findings, indicating a significant presence of structural abnormalities or complications related to AKI. The urine routine tests showed that 27 patients (54%) had normal results, while 20 patients (40%) had abnormal findings, further emphasizing the heterogeneity in clinical presentations and complications among the study population.

Table 3 illustrates the changes in renal function over time among patients with Acute Kidney Injury (AKI), with a specific focus on serum urea and serum creatinine levels. At baseline, the mean serum urea level was 43.3 mg/dL with a standard deviation (SD) of ±6.3 mg/dL and the mean serum creatinine level was 1.3 mg/dL with a standard deviation of±0.1 mg/dL.

At the 24-hour mark, the mean serum urea level slightly increased to 43.9 mg/dL (±3.5 mg/dL), while the mean serum creatinine level rose to 1.4 mg/dL (±0.2 mg/dL). The p-value for the change in serum urea from baseline to 24 hours was 0.25, indicating that this change was not statistically significant. In contrast, the p-value for the change in serum creatinine was 0.01, suggesting a statistically significant increase in creatinine levels within the first 24 hours.

By the 48-hour mark, the mean serum urea level decreased to 35.4 mg/dL (±4.6 mg/dL) and the mean serum creatinine level decreased to 1.0 mg/dL (±0.2 mg/dL). The p-value for the change in serum urea from baseline to 48 hours was 0.005, indicating a statistically significant decrease. Similarly, the p-value for the change in serum creatinine was 0.02, also indicating a statistically significant decrease from baseline.

In summary, while the changes in serum urea levels were not statistically significant at 24 hours, a significant decrease was observed by 48 hours. Serum creatinine levels, however, showed a statistically significant increase at 24 hours, followed by a significant decrease at 48 hours. These results highlight the dynamic changes in renal function markers in patients with AKI over the initial 48-hour period of treatment and monitoring.

Fig. 1 outlines the etiological factors and diagnoses associated with Acute Kidney Injury (AKI) in the study population. The data reveals a variety of underlying causes contributing to the onset of AKI.

Sepsis was identified as the etiological factor in 6 patients, accounting for 12% of the cases. Nephrotoxic drugs were implicated in 9 patients (18%), highlighting the role of medication-induced kidney damage in the development of AKI. Hypovolemia, a condition characterized by decreased blood volume, was responsible for AKI in 14 patients, representing 28% of the study population. This underscores the significance of fluid balance and hemodynamic stability in preventing renal injury.

Obstruction of the urinary tract was noted in 15 patients (30%), indicating a substantial prevalence of post-renal causes of AKI. Similarly, snake bites, which can cause direct nephrotoxicity and other systemic effects, were responsible for AKI in 15 patients (30%). This finding is particularly relevant in regions where snake bites are common and emphasizes the need for effective prevention and treatment strategies.

Epilepsy, which can lead to complications such as rhabdomyolysis and subsequent renal injury, was identified as an etiological factor in 13 patients, accounting for 26% of the cases. Other causes, grouped under the category of 'Others, were responsible for AKI in 6 patients (12%), reflecting the diverse range of factors that can contribute to the condition.

The present study provides valuable insights into the clinic-etiological profile of patients with Acute Kidney Injury (AKI) admitted to the medical ICU at KIMS, Koppal. Our findings highlight the diverse age distribution, symptoms, comorbidities and etiological factors associated with AKI in a rural healthcare setting, offering a comprehensive understanding of this population's disease.

Our study included 50 patients with a balanced distribution across different age groups, with a slight predominance in younger and middle-aged individuals (18-50 years). This age distribution contrasts with some reports indicating a higher incidence of AKI in elderly populations, likely reflecting regional and demographic variations in disease burden and healthcare access[5].

The slight female predominance (54%) observed in our study is consistent with some literature suggesting that women may have a higher risk of AKI, particularly in specific contexts such as obstetric complications and certain autoimmune diseases[6]. However, other studies have reported a higher incidence in males, highlighting the need for further research to elucidate gender-specific risk factors and outcomes in AKI.

The diverse clinical presentations of AKI in our study, including hematuria, abdominal pain, distension, seizures, vomiting, fever and dyspnea, underscore the complexity of diagnosing and managing AKI. The high prevalence of comorbidities, particularly hypertension (36%) and diabetes (44%), aligns with existing literature emphasizing these conditions as significant risk factors for AKI[7]. These findings underscore the importance of comprehensive clinical evaluation and management of underlying conditions to prevent and mitigate AKI.

Baseline laboratory investigations revealed mean hemoglobin levels of 11.9 g/dL, total leukocyte counts of 7644.5 cells/mm³ and platelet counts of 381399.2 cells/mm³. These values are within expected ranges for critically ill patients but highlight the need for close monitoring of hematological parameters in AKI patients[8]. Serum creatinine and urea levels provided crucial information on renal function, with significant changes observed over time, particularly at the 48-hour mark. The significant decrease in serum urea and creatinine levels by 48 hours suggests effective initial management and potential recovery of renal function in some patients.

Our study identified multiple etiological factors contributing to AKI, including sepsis (12%), nephrotoxic drugs (18%), hypovolemia (28%), obstruction (30%), snake bites (30%) and epilepsy (26%). The high prevalence of hypovolemia and obstruction as causes of AKI is consistent with global data highlighting these

factors as common contributors to AKI, particularly in resource-limited settings[9]. The significant proportion of snake bites underscores the unique epidemiological context of our study population and the need for targeted prevention and treatment strategies in regions where snake bites are prevalent.

These findings are consistent with global trends showing high morbidity and mortality associated with AKI in critically ill patients[10]. Our data support the need for early identification and intervention to improve outcomes, including fluid management, avoidance of nephrotoxic agents and appropriate use of renal replacement therapy.

CONCLUSION

In conclusion, this study provides a comprehensive overview of the clinic-etiological profile of AKI in a rural ICU setting, highlighting key demographic, clinical and etiological factors. The findings underscore the importance of early recognition and management of AKI, considering the diverse clinical presentations and underlying causes. Further research is needed to develop targeted strategies to prevent and manage AKI, particularly in resource-limited settings.

REFERENCES

1. Mehta, R.L., J.A. Kellum, S.V. Shah, B.A. Molitoris, C. Ronco,et al., 2007. Acute kidney injury network: Report of an initiative to improve outcomes in acute kidney injury. Crit. Care., Vol. 11, No. 2 .10.1186/cc5713.
2. John, A.K., L. Norbert, A. Peter and S. B. Rashad, et al 2012. Kidney disease: Improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidn Int. Suppl., 2: 1-138.
3. Priyamvada, P., R. Jayasurya, V. Shankar and S. Parameswaran, 2024. Epidemiology and outcomes of acute kidney injury in critically ill: Experience from a tertiary care center. Indian J. Nephr, 28: 413-420.
4. Jain, S., D. Bhadauria, A.K. Baronia, R.K. Singh and M. Gurjar, 2013. Septic acute kidney injury in critically ill Indian patients. Indian J. Crit. Care Med., 17: 49-52.
5. Mehta, R.L., J. Bouchard, S.B. Soroko, T.A. Ikizler and E.P. Paganini,et al., 2011. Sepsis as a cause and consequence of acute kidney injury: Program to Improve Care in Acute Renal Disease. Intens Care Med. 37: 241-248.
6. Susantitaphong, P., D.N. Cruz, J. Cerda, M. Abulfaraj, F. Alqahtani,et al., 2024. World incidence of aki. Clin. J. Am. Soc. Neph, 8: 1482-1493.
7. Palevsky, P.M., K.D. Liu, P.D. Brophy, L.S. Chawla and C.R. Parikh et al., 2013. Kdoqi us commentary on the 2012 kdigo clinical practice guideline for acute kidney injury. Am. J. Kidney Dis., 61: 649-672.
8. Hoste, E.A.J., S.M. Bagshaw, R. Bellomo, C.M. Cely and R. Colman et al., 2024. Epidemiology of acute kidney injury in critically ill patients: The multinational aki-epi study. Inten Care Med., 41: 1411-1423.