

Clinical Presentation and Outcome of Acute Kidney Injury at a Tertiary Care Hospital in Bangladesh: A Retrospective Study

Md. Rezaul Alam^{1*}, Ferdous Jahan¹, Md Rafayet Ullah Siddique², A. K. M Shahidur Rahman³

¹Associate Professor, Department of Nephrology, Bangladesh Medical University (BMU), Dhaka, Bangladesh

²Assistant Professor, Department of Anaesthesia Analgesia & Intensive Care Medicine, Bangladesh Medical University (BMU), Dhaka, Bangladesh

³Medical Officer, Department of Nephrology, Bangladesh Medical University (BMU), Dhaka, Bangladesh

***Author for Correspondence:**

Dr. Md. Rezaul Alam; E-mail: nipponreza@gmail.com

Received: 21 July 2026; Revised: 07 August 2026; Accepted: 30 August 2026; Available Online: 3 September 2026

ABSTRACT

Background: Acute kidney injury (AKI) is a frequent condition and its incidence is increasing. Early identification of precipitating factors and timely management are essential to improve outcomes, particularly in resource-limited healthcare settings. **Objective:** The purpose of this retrospective study was to evaluate the demographic and clinical characteristics, etiological factors, investigation findings, treatment modalities, and short-term in-hospital outcomes of adult patients with acute kidney injury (AKI) admitted to a tertiary-care hospital in Bangladesh. **Methods:** This retrospective observational study was conducted in the Department of Nephrology, Bangladesh Medical University (BMU) Hospital, Dhaka, Bangladesh, over a two-year period from August 2024 to July 2026. A total of 100 patients with AKI were included in this study according to selection criteria. Patients with pre-existing chronic kidney disease or having malignancy/pregnancy were excluded. AKI was defined according to the kidney disease: Improving Global Outcomes (KDIGO) criteria. Demographic characteristics, clinical presentations, underlying diagnoses, treatment modalities, requirement for renal replacement therapy and outcomes at discharge were recorded from hospital records. Outcomes were categorized as recovery with preserved renal function, persistent renal dysfunction at discharge, or in-hospital death. **Results:** The mean age of the patients was 38.52 ± 11.52 years, and 58% were male. Oliguria and oedema were the most frequent clinical presentations, each occurring in 67% of patients, followed by shortness of breath (32%) and diarrhea (24%). Hypovolemia was the leading cause of AKI (24%), followed by NSAID exposure (21%) and rhabdomyolysis (10%). Proteinuria (42%), urinary WBCs (40%), and granular casts (37%) were common urine abnormalities. Serum creatinine >6.8 mg/dL was observed in 70% of patients, blood urea >180 mg/dL in 55%, and pulmonary oedema on chest radiography in 49%. Fluid therapy was administered to 85% of patients, pharmacological treatment to 42% and hemodialysis was required in 12% patients. At discharge, 67% recovered with preserved renal function, 23% had persistent renal dysfunction, and 10% died during hospitalization. **Conclusion:** In this cohort, AKI was predominantly associated with potentially preventable factors, particularly hypovolemia and NSAID exposure. Although two-thirds of patients recovered with preserved renal function, a considerable proportion developed persistent renal dysfunction or died.

Keywords: Acute Kidney Injury (AKI); Hypovolemia; Hemodialysis; NSAID; Renal Dysfunction; Tertiary Care Hospital.

How to cite this article: Alam MR, Jahan F, Siddique MRU, Rahman AKMS. Clinical Presentation and Outcome of Acute Kidney Injury at a Tertiary Care Hospital in Bangladesh: A Retrospective Study. Int J Drug Deliv Technol. 2026;16(86s):676-681. DOI: 10.25258/ijddt.16.86s.72

Conflict of interest: The authors declare that they have no conflict of interest related to this study, its authorship, or publication.

Funding: No external funding was received for this study.

1. INTRODUCTION

Acute kidney injury (AKI) is a clinical syndrome characterized by an abrupt decline in kidney function over hours to days and is often reversible, which is commonly identified by a rise in serum creatinine and/or a reduction in urine output [1]. It can result from a broad range of hemodynamic, infectious, toxic, inflammatory and intrinsic renal insults [1,2]. AKI is associated with increased morbidity, mortality, prolonged hospitalization, greater healthcare utilization and an increased risk of subsequent chronic kidney disease (CKD) [3]. The clinical presentation and etiological spectrum of AKI are heterogeneous. Volume depletion

caused by vomiting, diarrhea, inadequate fluid intake, or blood loss may lead to renal hypoperfusion and potentially reversible kidney injury [4]. Drug-related kidney injury, particularly associated with nephrotoxic medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), represents another important and potentially preventable contributor. Other recognized causes include rhabdomyolysis, sepsis, glomerulonephritis, acute pyelonephritis, obstructive uropathy, contrast exposure, postoperative complications, vasculitis and pregnancy-related disorders [2,5,6]. The relative contribution of these causes varies considerably between populations and

healthcare settings. Studies from developing countries have demonstrated considerable variation in the causes and outcomes of AKI, with infection, volume depletion and other potentially reversible conditions remaining important contributors [7]. AKI may manifest with oliguria or anuria, fluid retention and oedema, dyspnea, electrolyte and acid-base abnormalities, or symptoms related to the underlying precipitating illness [1]. Laboratory evaluation, including assessment of serum creatinine, blood urea, electrolytes, acid-base status, and urine abnormalities, is essential for diagnosis and evaluation of severity. Imaging may also identify complications such as pulmonary oedema or contribute to determining the underlying cause. Early recognition of these abnormalities is particularly important because prompt correction of reversible factors may prevent further deterioration of kidney function. The burden and pattern of AKI may be particularly relevant in low- and middle-income countries, where infectious illnesses, volume depletion, medication-related kidney injury, delayed healthcare presentation and limitations in diagnostic and renal replacement facilities can influence patient outcomes [7,8]. In such settings, identification of locally prevalent and potentially preventable causes is important for developing appropriate clinical strategies. Although AKI has been extensively investigated internationally, data describing its clinical characteristics, etiological profile, management and short-term outcomes in Bangladesh remain limited. Local data are important for recognizing common precipitating factors and potentially preventable exposures. Therefore, this study was undertaken to evaluate the demographic characteristics, clinical presentations, causes, investigation findings, treatment modalities and short-term outcomes of patients with AKI at a tertiary-care hospital in Bangladesh.

2. MATERIALS AND METHODS

2.1 Study design and setting

This was a retrospective observational study conducted in the Department of Nephrology, Bangladesh Medical University (BMU) Hospital, Dhaka, Bangladesh. The study covered a two-year period from August 2024 to July 2026.

2.2 Study population

The study population comprised adult patients who developed AKI and admitted in BMU hospital during the study period. A total of 100 patients with AKI were included in this study according to selection criteria.

Inclusion criteria

Patients were included if they:

1. Were aged ≥ 18 years;
2. Developed AKI and fulfilled the KDIGO criteria for AKI.

Exclusion criteria

Patients were excluded if they had:

1. Pre-existing CKD

2. Having any type of malignancy
3. Pregnancy.

2.3 Definition of acute kidney injury

AKI was defined according to the kidney disease: Improving Global Outcomes (KDIGO) 2012 criteria [1].

2.4 Data collection

Relevant information was retrospectively extracted from hospital records using a pre-designed data collection form. Data included demographic characteristics, sex, clinical presentation, primary clinical diagnosis, relevant comorbidities, laboratory investigations, imaging findings, treatment modalities, dialysis requirement, and clinical outcomes. The source dataset included demographic variables, clinical presentations, clinical diagnoses, treatment modalities, and renal outcomes.

2.5 Treatment

Treatment modalities included fluid management, pharmacological therapy, correction of underlying precipitating conditions and renal replacement therapy when clinically indicated. Hemodialysis was considered when indicated according to the patient's clinical and biochemical status.

2.6 Outcome measures

The principal outcome was the clinical and renal status at hospital discharge. Outcomes were categorized as:

Recovery with preserved renal function: Improvement/stabilization of kidney function without ongoing dialysis.

Persistent renal dysfunction at discharge: Reduced kidney function at discharge.

In-hospital death: Death occurring during the hospital admission.

2.7 Statistical analysis

Data were retrieved, coded and compiled accordingly. Continuous variables were expressed as mean $\pm$ standard deviation (SD), while categorical variables were presented as frequencies and percentages.

2.8 Ethical considerations

Ethics statement: Because this was a retrospective review of hospital records. The Institutional Review Board (IRB) of Bangabandhu Sheikh Mujib Medical University (BSMMU), predecessor of Bangladesh Medical University (BMU) waived the ethical requirement citing ‘no patients were selected for the purpose of research, but rather their past medical record was used after they received regular treatment.’

3. RESULTS

This retrospective study was intended to evaluate the clinical characteristics, causative conditions, treatment modalities and renal outcomes of patients who developed AKI at a tertiary care setting in Bangladesh. Data of 100 patients with AKI who admitted in BMU hospital were evaluated.

3.1 Demographic characteristics

The mean age was 38.52 ± 11.52 years, with an age range of 18-62 years. The largest age group was 41-50 years (28.0%), followed by those aged 31-40 years (26.0%) and >50 years (20.0%). Patients aged ≤ 20 years accounted for 9.0%, while 17.0% were aged 21-30 years. Males predominated, accounting for 58.0% of patients, compared with 42.0% females (Table- 1).

Table 1: Demographic and clinical characteristics of patients with acute kidney injury (N=100)

patients with acute kidney injury (N=100)		
Variables	Number of patients (n)	Percentage (%)
Age group (years)		
≤20	9	9.0
21-30	17	17.0
31-40	26	26.0
41-50	28	28.0
>50	20	20.0
Mean ± SD	38.52 ± 11.52	
Range (Minimum–maximum)	18-62	
Sex		
Male	58	58.0
Female	42	42.0

3.2 Clinical pattern

Among the participants with acute kidney injury, oliguria and oedema were the most frequent clinical presentations, each occurring in 67.0% of patients. Non-oliguric AKI was observed in 23.0%, while anuria was present in 7.0%. Shortness of breath was reported in 32.0% of patients, followed by diarrhea in 24.0%. Hypertension and shock were present in 17.0% and 16.0% of patients, respectively. Drowsiness occurred in 12.0%, while acidotic breath and loin pain were observed in 9.0% and 8.0%, respectively. Hypotension was present in 8.0% of patients, whereas hematuria and convulsion were less frequent, occurring in 5.0% and 6.0%, respectively (Table- 2).

Table 2: Clinical presentations of acute kidney injury among study participants (N=100)

Clinical presentation*	Number of patients (n)	Percentage (%)
Oliguria	67	67.0
Non-oliguric	23	23.0
Anuria	7	7.0
Diarrhea	24	24.0
Shortness of breath	32	32.0
Oedema	67	67.0
Hematuria	5	5.0
Shock	16	16.0
Hypotension	8	8.0
Hypertension	17	17.0
Acidotic breath	9	9.0
Loin pain	8	8.0
Drowsiness	12	12.0
Convulsion	6	6.0

*Multiple response

3.3 Causes of acute kidney injury

Among 100 participants with AKI, hypovolemia was the most common etiological factor (24.0%), followed by NSAID exposure (21.0%) and rhabdomyolysis (10.0%). Septicemia and glomerulonephritis accounted for 8.0% each, while acute pyelonephritis contributed 7.0%. Obstructive uropathy accounted for 6.0%, whereas contrast-induced nephropathy and postoperative AKI each represented 4.0%. Vasculitis and HELLP syndrome accounted for 3.0% each and snake bite was uncommon cause (2.0%) (Table- 3).

Table 3: Causes of acute kidney injury among study participants (N=100)

Cause of AKI	Number (n)	Percentage (%)
Hypovolemia	24	24.0
NSAIDs	21	21.0
Rhabdomyolysis	10	10.0
Septicemia	8	8.0
Glomerulonephritis	8	8.0
Acute pyelonephritis	7	7.0
Obstructive uropathy	6	6.0
Contrast-induced nephropathy	4	4.0
Postoperative	4	4.0
Vasculitis	3	3.0
HELLP syndrome	3	3.0
Snake bite	2	2.0

3.4 Routine urine analysis

Routine urine analysis showed that 46.0% of the 100 participants had apparently normal urine findings. Proteinuria was the most frequent abnormality, detected in 42.0% of patients, followed by white blood cell (WBC) in urine in 40.0%. Granular casts were present in 37.0% of patients, while red blood cell (RBC) in urine and RBC casts were observed in 32.0% and 27.0%, respectively (Table- 4).

Table 4: Findings of routine urine analysis among study participants (N=100)

Routine urine finding	Number of patients (n)	Percentage (%)
Normal	46	46.0
Proteinuria	42	42.0
WBC in urine	40	40.0
RBC in urine	32	32.0
RBC casts	27	27.0
Granular casts	37	37.0

*Multiple response

3.5 Investigation findings

Among the 100 participants with acute kidney injury, elevated serum creatinine (>6.8 mg/dL) was observed in 70.0% of patients, making it the most frequent laboratory abnormality. Blood urea levels greater than 180 mg/dL were present in 55.0% of patients. Pulmonary oedema detected on chest radiography was found in 49.0% of participants. Severe hyperkalemia (serum potassium >7.0 mmol/L) and severe metabolic acidosis (blood pH

<7.2) were comparatively uncommon, occurring in 3.0% and 2.0% of patients, respectively (Table- 5).

Table 5: Investigation findings among patients with acute kidney injury (N=100)

Investigation findings	Number of patients (n)	Percentage (%)
Blood urea >180 mg/dL	55	55.0
Serum creatinine >6.8 mg/dL	70	70.0
Serum potassium >7.0 mmol/L	3	3.0
Blood pH <7.2	2	2.0
Pulmonary oedema on chest X-ray (P/A view)	49	49.0

*Multiple response

3.6 Treatment modalities

Among the patients with AKI, fluid therapy was administered to 85.0%, whereas 15.0% did not receive fluid therapy. Pharmacological treatment was provided to 42.0% of patients, while 58.0% did not receive drug therapy as categorized in the original dataset. Hemodialysis was required in approximately 12.0% of patients (Table- 6).

Table 6: Distribution of study patients according to treatment modalities (N=100)

Treatment modalities	Number of patients (n)	Percentage (%)
Conservative management		
Fluid therapy (Intravenous or oral)		
Yes	85	85.0
No	15	15.0
Pharmacological treatment (Drugs)		
Yes	42	42.0
No	58	58.0
Renal replacement therapy (RRT)		
Hemodialysis	12	12.0

3.7 Outcomes

Approximately two-thirds of the study patients (67.0%) achieved recovery with preserved renal function. Persistent renal dysfunction at discharge was observed in 23.0%, while 10.0% of patients died during hospitalization (Table- 7).

Table 7: Distribution of patients according to outcome following acute kidney injury (N=100)

Outcome	Frequency (n)	Percentage (%)
Recovery with preserved renal function	67	67.0
Persistent renal dysfunction at discharge*	23	23.0
Death	10	10.0

*Persistent renal dysfunction at discharge, not “CKD” as renal impairment was not documented for next 3 months.

4. DISCUSSION

Acute kidney injury (AKI) remains an important clinical problem because it can develop from diverse systemic, hemodynamic, toxic, infectious and intrinsic renal insults

and is associated with both short- and long-term adverse outcomes [9,10]. AKI in low- and middle-income countries frequently affects younger populations and is often associated with potentially preventable conditions such as volume depletion, infections, and nephrotoxic exposures [11]. In the present retrospective study of 100 adult patients with AKI, their mean age was 38.52 ± 11.52 years and males constituted 58% of the study population.

The clinical presentation in this study demonstrated a predominance of oliguria and oedema, each occurring in 67% of patients. Shortness of breath was reported in 32%, while diarrhea occurred in 24%. Shock and hypotension were present in 16% and 8%, respectively. Oliguria is a recognized clinical manifestation and diagnostic component of AKI, although the absence of oliguria does not exclude the diagnosis [12]. The occurrence of oedema in an equal proportion of the study patients may reflect sodium and water retention associated with reduced renal function. These findings also highlight the importance of careful monitoring of urine output and fluid status in patients at risk of AKI.

Hypovolemia was the most frequently identified cause of AKI in this study, accounting for 24% of cases, followed by NSAID exposure in 21% and rhabdomyolysis in 10%. Septicemia and glomerulonephritis each accounted for 8%, while acute pyelonephritis, obstructive uropathy, contrast-induced nephropathy, and postoperative AKI contributed smaller proportions. This etiological pattern is consistent with observations that hypovolemia and infections remain important causes of AKI in low- and middle-income countries [11,13]. NSAID exposure was the second most frequent identified cause. NSAIDs can impair renal autoregulation by inhibiting prostaglandin synthesis, thereby promoting renal vasoconstriction, particularly when renal perfusion is already compromised by volume depletion or other hemodynamic stress [14]. A systematic review and meta-analysis have also demonstrated an increased risk of AKI among users of traditional NSAIDs [15]. Rhabdomyolysis accounted for 10% of cases, representing another clinically important cause of AKI. The presence of glomerulonephritis, vasculitis, and HELLP syndrome among the identified causes also demonstrates that the etiological spectrum in this cohort included intrinsic renal and pregnancy-related disorders in addition to predominantly hemodynamic and toxic causes. According to KDIGO, identifying the underlying cause of AKI is an important component of evaluation because management depends substantially on the precipitating mechanism [1].

Urinalysis demonstrated proteinuria in 42% of patients, urinary WBCs in 40%, granular casts in 37%, urinary RBCs in 32%, and RBC casts in 27%. However, because the present study was retrospective and urine microscopy findings were not correlated with renal biopsy or specific etiological diagnoses, these findings should be

interpreted as supportive rather than definitive evidence of particular pathological lesions. The biochemical findings indicate substantial renal dysfunction among the study participants. Serum creatinine >6.8 mg/dL was observed in 70% of patients, while blood urea >180 mg/dL occurred in 55%. Pulmonary oedema on chest radiography was present in 49%. In contrast, severe hyperkalemia (>7 mmol/L) and severe metabolic acidosis (pH <7.2) were recorded in only 3% and 2%, respectively. Importantly, creatinine concentration alone should not be interpreted as a direct measure of AKI severity without knowledge of baseline kidney function and the temporal pattern of change, as emphasized by KDIGO criteria [1,16].

In this study fluid therapy was the most frequently documented treatment modality followed by pharmacological treatment. Hemodialysis was required in 12% of patients. The predominance of fluid management is consistent with the high frequency of hypovolemia as an identified cause of AKI. However, fluid therapy in AKI must be individualized according to volume status because excessive fluid administration can contribute to pulmonary oedema and other complications. Current AKI guidance emphasizes appropriate assessment and management of volume status and avoidance of both inadequate resuscitation and unnecessary fluid accumulation [1,17].

The overall outcome was relatively favorable in that 67% of patients recovered with preserved renal function at discharge. Nevertheless, 23% had persistent renal dysfunction and 10% died during hospitalization, meaning that approximately one-third of the cohort did not achieve recovery with preserved renal function by discharge. Even after apparent acute recovery, patients may remain at increased risk of subsequent CKD and other adverse outcomes [18]. The KDIGO framework recognizes a continuum between AKI, acute kidney disease, recovery, and later CKD, emphasizing the importance of follow-up after an episode of AKI [1,18]. The 10% in-hospital mortality observed in this study demonstrates the clinical seriousness of AKI despite the relatively high recovery rate. Mortality in AKI varies substantially according to patient population, underlying cause, disease severity, comorbidity, and healthcare setting. AKI in low- and middle-income countries may be influenced by delayed presentation, infectious diseases, limited access to laboratory monitoring, and restricted availability of kidney replacement therapy [11,19].

An important observation from this study is that several of the leading causes of AKI were potentially preventable or modifiable. Hypovolemia and NSAID exposure together accounted for 45% of identified cases. This finding provides a practical opportunity for prevention through early recognition and correction of volume depletion, appropriate use of NSAIDs, avoidance of potentially nephrotoxic medications in high-risk

patients and closer monitoring of renal function during acute illnesses. Medication stewardship and early identification of patients at risk of AKI are particularly relevant because NSAID-associated renal injury is more likely when renal perfusion is compromised [20].

CONCLUSION

AKI in this tertiary-care cohort predominantly affected relatively young adults and commonly presented with oliguria and oedema. Hypovolemia, NSAID exposure, and rhabdomyolysis were the leading causes. Although 67% recovered with preserved renal function, 23% had persistent renal dysfunction and 10% died. These findings emphasize the importance of early recognition of AKI, prompt correction of volume depletion, avoidance of inappropriate NSAID and other nephrotoxic exposure, timely identification of the underlying cause and appropriate supportive and renal replacement therapy when indicated.

Recommendations

Multi-center studies with larger samples are recommended to enhance generalizability. Early recognition, correction of volume depletion, avoidance of nephrotoxins, and appropriate management with follow-up are essential to improve outcomes.

Limitations of the study

Several limitations should be considered when interpreting the findings. First, this was a single-center retrospective study. Second, retrospective data collection depends on the completeness and accuracy of medical records. Third, the study did not provide detailed stratification according to KDIGO AKI stage. Fourth, important prognostic variables such as duration of AKI, baseline and peak serum creatinine, urine output, severity of sepsis, vasopressor requirement, intensive-care admission, mechanical ventilation, and number of affected organ systems were not comprehensively analyzed. Fifth, the study assessed renal status at discharge without long-term follow-up. Sixth, the study was observational and therefore cannot establish causal relationships between the identified clinical diagnoses and outcomes.

Authors' contributions

All authors contributed to the conception and design of the study, data collection and/or interpretation, critical revision of the manuscript and approval of the final version. All authors agree to be accountable for the accuracy and integrity of the work.

Acknowledgements

The authors would like to acknowledge the physicians, nurses, medical officers and staff of Bangladesh Medical University (BMU) Hospital who contributed to the care of the patients included in this study and assisted with the retrieval of relevant clinical records.

REFERENCES

1. Almazmomi MA, Esmat A, Naeem A. Acute kidney injury: definition, management, and promising therapeutic target. *Cureus*. 2023 Dec 28;15(12):e51228.
2. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. *Nephron Clinical Practice*. 2012 Oct 1;120(4):c179-84.
3. Turgut F, Awad AS, Abdel-Rahman EM. Acute kidney injury: medical causes and pathogenesis. *Journal of clinical medicine*. 2023 Jan 3;12(1):375.
4. Abdel-Kader K, Palevsky P. Acute kidney injury in the elderly. *Clinics in geriatric medicine*. 2009 Aug;25(3):331.
5. Hoste EA, Bagshaw SM, Bellomo R, Cely CM, Colman R, Cruz DN, Edipidis K, Forni LG, Gomersall CD, Govil D, Honoré PM. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. *Intensive care medicine*. 2015 Aug;41(8):1411-23.
6. Stewart J, Findlay G, Smith N, Kelly K, Mason M. Acute kidney injury: adding insult to injury. *Natl Confid Enq into Patient Outcomes Death*. 2009:1-22.
7. Kashani K, Macedo E, Burdmann EA, Hooi LS, Khullar D, Bagga A, Chakravarthi R, Mehta R, Group C, Acute Disease Quality Initiative. Acute kidney injury risk assessment: differences and similarities between resource-limited and resource-rich countries. *Kidney international reports*. 2017 Jul 1;2(4):519-29.
8. Mehta RL, Cerdá J, Burdmann EA, Tonelli M, García-García G, Jha V, Susantitaphong P, Rocco M, Vanholder R, Sever MS, Cruz D. International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. *The Lancet*. 2015 Jun 27;385(9987):2616-43.
9. Kellum JA, Lameire N, KDIGO AKI Guideline Work Group. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). *Critical care*. 2013 Feb 4;17(1):204.
10. Ronco C, Bellomo R, Kellum JA. Acute kidney injury. *The Lancet*. 2019 Nov 23;394(10212):1949-64.
11. Cerdá J, Mohan S, Garcia-Garcia G, Jha V, Samavedam S, Gowrishankar S, Bagga A, Chakravarthi R, Mehta R, Group C, Acute Disease Quality Initiative. Acute kidney injury recognition in low-and middle-income countries. *Kidney international reports*. 2017 Jul 1;2(4):530-43.
12. Bianchi NA, Stavart LL, Altarelli M, Kelevina T, Faouzi M, Schneider AG. Association of oliguria with acute kidney injury diagnosis, severity assessment, and mortality among patients with critical illness. *JAMA network open*. 2021 Nov 4;4(11):e2133094.
13. Abebe A, Kebede B, Wobie Y. Clinical profile and short-term outcomes of acute kidney injury in patients admitted to a teaching hospital in Ethiopia: a prospective study. *International Journal of Nephrology and Renovascular Disease*. 2021 Jul 2:201-9.
14. Kunitsu Y, Hira D, Nakagawa S, Tsuda M, Morita SY, Yamamoto Y, Terada T. NSAID-Induced acute kidney injury risk in patients on renin-angiotensin system inhibitors and diuretics: nationwide cohort study. *Journal of Pharmaceutical Health Care and Sciences*. 2025 Aug 18;11(1):77.
15. Ungprasert P, Cheungpasitporn W, Crowson CS, Matteson EL. Individual non-steroidal anti-inflammatory drugs and risk of acute kidney injury: A systematic review and meta-analysis of observational studies. *European journal of internal medicine*. 2015 May 1;26(4):285-91.
16. Pickkers P, Darmon M, Hoste E, Joannidis M, Legrand M, Ostermann M, Prowle JR, Schneider A, Schetz M. Acute kidney injury in the critically ill: an updated review on pathophysiology and management. *Intensive care medicine*. 2021 Aug;47(8):835-50.
17. Tamargo C, Hanouneh M, Cervantes CE. Treatment of acute kidney injury: a review of current approaches and emerging innovations. *Journal of Clinical Medicine*. 2024 Apr 23;13(9):2455.
18. Heung M, Steffick DE, Zivin K, Gillespie BW, Banerjee T, Hsu CY, Powe NR, Pavkov ME, Williams DE, Saran R, Shahinian VB. Acute kidney injury recovery pattern and subsequent risk of CKD: an analysis of veterans health administration data. *American Journal of Kidney Diseases*. 2016 May 1;67(5):742-52.
19. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. Acute kidney injury. *Nature reviews Disease primers*. 2021 Jul 15;7(1):52.
20. Wang H, Liu T, Li Q, Cui R, Fan X, Tong Y, Ma S, Liu C, Zhang J. NSAID treatment before and on the early onset of acute kidney injury had an opposite effect on the outcome of patients with AKI. *Frontiers in Pharmacology*. 2022 May 17;13:843210.