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Incidence and Clinical Outcomes of Acute Kidney Injury among Mechanically Ventilated Patients in a Tertiary Care Hospital: A Prospective Observational Study

Poulami Samanta, Somnath Das*, Anirban Gandhi, Rana Barik, Ayushman Chakraberty, Sumita Yeasmin, S. K. Shamim Raja, Sourav Bhattacharya

Abstract

Introduction: Acute kidney injury (AKI) is a common and clinically important condition in critically ill patients, particularly those requiring invasive mechanical ventilation. Mechanical ventilation can contribute to the development of AKI by increasing intrathoracic pressure, reducing venous return and cardiac output, altering renal perfusion, and increasing renal venous congestion. Early recognition of AKI in mechanically ventilated patients is essential for timely preventive measures and improving patient outcomes. **Methods:** A prospective observational hospital-based study was conducted from April to June 2026 among 108 mechanically ventilated adults. Consecutive sampling was used, with AKI diagnosed and staged according to Kidney Disease: Improving Global Outcomes 2012 criteria. Demographic and laboratory parameters were monitored daily. Outcomes were hospital discharge or death. Continuous and categorical variables were appropriately analyzed using parametric and non-parametric tests. **Results**: Among 108 mechanically ventilated patients, 40.7% developed AKI, with Stage 3 being most common. AKI patients had significantly higher blood urea nitrogen (BUN) and lower urine output. Mortality was higher in AKI patients (45.5% vs. 35.9%), but the difference was not statistically significant. **Conclusion:** Close monitoring for AKI is important in mechanically ventilated patients. Regular monitoring of urine output, serum creatinine and BUN is important for early detection of AKI and appropriate management.

Keywords: Acute kidney injury, ICU outcome, critical care

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Introduction

Acute kidney injury (AKI) is a common and serious condition among critically ill patients admitted to intensive care units (ICUs). It is characterized by an abrupt decline in kidney function, resulting in impaired excretion of metabolic waste products and disturbances in fluid, electrolyte and acid–base balance. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria define AKI based on changes in serum creatinine and urine output, allowing standardized diagnosis and staging of the condition. According to the KDIGO 2012 criteria, AKI is defined by an increase in serum creatinine ≥0.3 mg/dL within 48 h, an increase in serum creatinine to ≥1.5 times the baseline value within 7 days, or urine output <0.5 mL/kg/h for ≥6 h.[1]

The development of AKI is particularly important in patients requiring invasive mechanical ventilation. Positive-pressure ventilation can adversely affect renal function. Increased intrathoracic pressure may reduce venous return, cardiac output, and renal perfusion, while an increase in central and renal venous pressure may contribute to renal venous congestion.[2,3] Mechanical ventilation may also contribute to AKI through hemodynamic and inflammatory mechanisms. It can activate neurohormonal pathways, including the sympathetic nervous system and renin–angiotensin–aldosterone system, resulting in renal vasoconstriction and sodium and water retention. In addition, ventilator-induced lung injury (VILI) and systemic inflammatory responses may contribute to endothelial dysfunction, altered renal microcirculation and tubular injury.[2,3]

The risk of AKI in mechanically ventilated patients may be further increased by coexisting conditions, such as sepsis, septic shock, hypotension, vasopressor use, nephrotoxic drug exposure, pre-existing renal dysfunction and multiorgan failure.[4,5]

Department of Respiratory Medicine, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, West Bengal, India.

Corresponding Author: Dr. Somnath Das, Department of Respiratory Medicine, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, West Bengal, India. E-mail: somnathpulmo@gmail.com

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AKI may lead to fluid overload, electrolyte disturbances, metabolic acidosis, and accumulation of uremic toxins, which can further complicate respiratory and cardiovascular management. Patients with severe AKI may require renal replacement therapy and have a higher risk of adverse clinical outcomes.[1,5] Studies have shown that AKI is associated with increased mortality, and even patients who recover from an episode of AKI may remain at increased risk of subsequent chronic kidney disease (CKD) and persistent renal dysfunction.[1,5]

Early identification of AKI in mechanically ventilated patients is therefore clinically important. Serum creatinine and urine output remain the principal parameters used for diagnosis according to the KDIGO criteria.[1] However, serum creatinine may increase relatively late after renal injury and can be influenced by factors, such as muscle mass, fluid balance and baseline kidney function.[1,6] Continuous monitoring of urine output and serial assessment of renal function are consequently important components of ICU care.

Therefore, studying AKI in mechanically ventilated ICU patients is clinically relevant. Assessment of its incidence, severity, and clinical outcomes may help identify patients at increased risk and facilitate early preventive and therapeutic interventions. The present study is planned to evaluate the incidence of AKI among mechanically ventilated patients admitted to the ICU and to assess their clinical outcomes.

Methods

After getting approval from Ethics Committee and Institutional Research Committee, a hospital-based prospective study was conducted in the ICU of Gouri Devi Institute of Medical Sciences and Hospital, Rajbandh, Durgapur. The total duration of this study was 3 months from April 2026 to June 2026. The primary aim was to determine the incidence and outcome of AKI in mechanically ventilated patients.

In this study, 108 mechanically ventilated patients were taken as the sample. A consecutive sampling method was applied. Patients aged ≥18 years requiring invasive mechanical ventilation for ≥24 h were included after obtaining informed consent. Patients with CKD Stage 4/5, maintenance dialysis, renal transplantation, pre-existing AKI before mechanical ventilation, mechanical ventilation <24 h, pregnancy or incomplete records were excluded from the study. Eligible patients were consecutively enrolled and their demographic data, laboratory parameters, including serum creatinine, blood urea nitrogen (BUN), and urine output were recorded.

Patients with pre-existing AKI before initiation of invasive mechanical ventilation were excluded. Pre-existing AKI was assessed using baseline serum creatinine, urine output, and previously documented serum creatinine values, when available.

The initiation of invasive mechanical ventilation was considered the baseline (time 0), and patients were prospectively followed for development of AKI throughout their ICU stay. The serum creatinine value obtained immediately before or at the initiation of invasive mechanical ventilation was considered the baseline serum creatinine for subsequent AKI assessment.

Patients were monitored daily throughout their ICU stay. AKI diagnosis and staging were done according to KDIGO 2012 criteria (Increase in serum creatinine ≥0.3 mg/dL within 48 h, or increase in serum creatinine to ≥1.5 times baseline within 7 days, or Urine output <0.5 mL/kg/h for ≥6 h, which was based on serum creatinine and urine output. The urine output was recorded in mL/kg/h for KDIGO classification. The outcome was defined as discharge from hospital or death.

The data collected from the study were tabulated in appropriate pro forma. Continuous variables were expressed as mean ± standard deviation for normally distributed data. Categorical variables were expressed as frequencies and percentages. Comparisons of continuous variables between patients with and without AKI were performed using the independent-samples *t*-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to assess the association between AKI and mortality. A two-sided $P < 0.05$ was considered statistically significant.

Results

A total of 108 mechanically ventilated critically ill patients were included in the present study. The mean age of the study population was 54.69 ± 19.37 years, with a median age of 57 years. The study population comprised 63 males (58.3%) and 45 females (41.7%).

Among the 108 mechanically ventilated patients, 44 patients (40.7%) developed AKI, whereas 64 patients (59.3%) had no AKI. Thus, the overall incidence of AKI in the study population was 40.7% (table 1).

The mean age of patients who developed AKI was 55.44 ± 18.46 years, compared with 54.00 ± 20.31 years among patients without AKI.

Among the 44 patients who developed AKI, 6 (13.6%) had Stage 1, 16 (36.4%) had Stage 2, and 22 (50.0%) had Stage 3 AKI, with Stage 3 being the most frequent stage. Among the 44 patients with AKI, 20 patients (45.5%) died, while 24 patients (54.5%) survived. Among the 64 patients without AKI, 23 patients (35.9%) died, while 41 patients (64.1%) survived. Overall, 43 of 108 patients (39.8%) died, while 65 patients (60.2%) survived. The distribution of mortality among the groups is shown in Table 2.

The mortality among patients with AKI was higher than that among patients without AKI. The association between development of AKI and mortality was not statistically significant (Pearson's $\chi^2 = 0.99$, $P = 0.32$). Patients who developed AKI had higher odds of mortality than those without AKI (OR 1.49, 95% CI 0.68–3.25); however, the association was not statistically significant.

There was a marked difference in BUN level between patients with and without AKI. The mean BUN level was 18.90 ± 14.38 mg/dL among patients without AKI compared with 84.46 ± 35.93 mg/dL among patients with AKI. This difference was statistically significant (Mann–Whitney U test, $P < 0.001$).

The mean urine output was 1147.05 ± 490.98 mL/day among patients without AKI and 460.12 ± 451.19 mL/day among patients with AKI. The difference in urine output between the two groups was statistically significant (independent-samples *t*-test, $P < 0.001$).

Stage 3 was the most frequent stage of AKI. Patients who developed AKI had substantially higher BUN levels and lower urine output than those without AKI. Although mortality was higher among patients with AKI, the difference was not statistically significant.

Discussion

Van den Akker *et al*. performed a systematic review and meta-analysis, including 31 studies to evaluate invasive mechanical

Table 1: Incidence of AKI

AKI status	*Number*	*Percentage*
AKI present	44	40.7
AKI absent	64	59.3
Total	108	100

AKI: Acute kidney injury

Table 2: Mortality according to AKI status

Outcome	*Non-AKI n (%)*	*AKI n (%)*	*Total n (%)*
Survived	41 (64.1)	24 (54.5)	65 (60.2)
Death	23 (35.9)	20 (45.5)	43 (39.8)
Total	64 (100)	44 (100)	108 (100)

AKI: Acute kidney injury

ventilation as a risk factor for AKI in 2013. The pooled analysis demonstrated that mechanically ventilated patients had approximately a threefold higher risk of developing AKI (OR 3.16; 95% CI 2.32–4.28). The study concluded that invasive mechanical ventilation is an independent risk factor for AKI, while different tidal volume and positive end-expiratory pressure (PEEP) strategies did not significantly alter the overall risk. So this study corroborates with the present study.[4]

Hoste *et al*., conducted a study in 2015; it was a multinational AKI-EPI study involving 1,802 ICU patients from 97 ICUs. They found that AKI occurred in 57.3% of critically ill patients, many of whom required mechanical ventilation. Increasing AKI severity was associated with increased ICU and hospital mortality, longer ICU stay, and greater need for renal replacement therapy.[5]

Vemuri *et al*., did a retrospective cohort study to evaluate 9,704 ICU patients, of whom 4,484 required invasive mechanical ventilation. Among the mechanically ventilated patients, 2,009 (45%) developed AKI during mechanical ventilation. Mortality among mechanically ventilated patients with AKI was 22.4%, compared with 5% among mechanically ventilated patients without AKI. The study demonstrated a strong association between AKI during invasive mechanical ventilation and adverse ICU outcomes. The incidence of AKI in mechanically ventilated patients corroborates well with this study. However, the mortality outcome is lower than in our study. This may be due to the geographical variation, ICU protocol, patient selection, pre-existing comorbidities, and sepsis.[7]

Mo *et al*. evaluated AKI in 2,325 critically ill patients and reported an incidence of 53.5% in the year 2022. Patients requiring invasive mechanical ventilation had a significantly higher risk of persistent AKI and prolonged ICU stay. The authors highlighted that hypotension, vasopressor use, sepsis, and respiratory failure frequently coexist in ventilated patients and contribute to renal injury. Hence, this study corroborates with the present study.[8]

Lombardi *et al*. did a large multinational retrospective analysis and evaluated the occurrence and risk factors for AKI among patients receiving mechanical ventilation for more than 24 h who had normal renal function at ICU admission. They found that, among the 3,206 mechanically ventilated patients, 700 (21.83%) developed AKI. Most cases occurred early, with 547 cases developing within the first 48 h of ICU admission. The study also demonstrated that the risk-factor profile differed according to the timing of AKI onset. We reported a higher incidence of AKI in mechanically ventilated patients, compared to this study. This difference may be due to the geographical variation, ICU protocol, patient selection, pre-existing comorbidities, and sepsis.[9]

Gao *et al*. did a multicenter retrospective study to evaluate 17,428 mechanically ventilated patients using the MIMIC-IV and eICU-CRD databases. The study investigated whether mean airway pressure was independently associated with AKI. They observed that a higher mean airway pressure was associated with increased risk of AKI, severe AKI, renal replacement therapy and mortality. In the MIMIC-IV cohort, AKI incidence increased from 15.6% in the lowest mean-airway-pressure quartile to 33.0% in the highest quartile.[10]

Kuiper *et al*. reviewed the mechanisms by which mechanical ventilation contributes to AKI. They proposed that positive-pressure ventilation decreases cardiac output and renal perfusion while simultaneously promoting systemic inflammation through VILI. These inflammatory mediators may directly damage renal tubular cells and contribute to AKI in critically ill patients.[3]

Kellum and Lameire, in the KDIGO summary guidelines, emphasized that critically ill patients requiring mechanical ventilation should be closely monitored for AKI using serial serum creatinine measurements and urine output. The authors recommended optimization of intravascular volume, avoidance of nephrotoxic drugs, and maintenance of adequate renal perfusion to prevent ventilator-associated AKI.[6]

Benites *et al*. reviewed ventilation-induced AKI in patients with acute respiratory failure. The authors discussed the role of PEEP, emphasizing that excessive intrathoracic pressure can reduce venous return, cardiac output and renal perfusion. They concluded that lung-protective ventilation strategies should balance adequate oxygenation while minimizing adverse renal effects.[11]

Conclusion

In the present study of 108 mechanically ventilated critically ill patients, AKI occurred in 40.7% of patients. Stage 3 AKI was the most frequently observed (50%) stage. Patients with AKI had significantly higher BUN levels and lower urine output compared with those without AKI. These findings suggest that AKI was frequent among mechanically ventilated patients and was associated with significantly higher BUN levels and lower urine output. Although mortality was numerically higher among patients with AKI, this difference was not statistically significant in the present study. Monitoring of renal function, BUN, and urine output is therefore important in mechanically ventilated patients.

Ethical Approval

Approved.

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