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Original Research Article

Incidence of conversion of AKI to CKD: A retrospective study in a tertiary care hospital

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Abstract:

Background: Acute Kidney Injury (AKI) is characterized by an abrupt decrease in kidney function, posing a substantial clinical challenge worldwide. AKI often results in adverse outcomes including progression to chronic kidney disease (CKD), which increases long-term morbidity, healthcare costs, and mortality. Understanding factors influencing this progression is critical to improving patient care and preventing chronic renal impairment.

Aim: This study aimed to determine the incidence of progression from AKI to CKD among hospitalized patients and identify key risk factors, emphasizing the role of appropriate and timely antibiotic use in AKI caused by infections.

Methods: A retrospective cohort study was performed at a tertiary care hospital, enrolling 200 patients diagnosed with AKI. Data on demographics, AKI severity, etiologies, and outcomes were collected. CKD conversion was defined as sustained reduction of GFR below 60 mL/min/1.73 m^2 beyond 3 months post-AKI. Statistical analyses included Kaplan-Meier survival curves and Cox regression to evaluate progression risks.

Results: Out of 200 patients, 40 (20%) progressed to CKD. CKD incidence correlated with AKI severity: 8.2% in Stage 1, 20.6% in Stage 2, and 52.9% in Stage 3 ($p < 0.001$). Intrinsic renal AKI showed the highest progression (39.5%). Timely interventions, particularly appropriate antibiotic administration in infection-related AKI, significantly reduced progression to 13.2%.

Conclusion: AKI severity and cause strongly influence CKD development. Early, cause-specific treatment—especially judicious antibiotic use—is vital to prevent long-term renal damage and improve prognosis.

Keywords: Acute Kidney Injury, Chronic Kidney Disease, Antibiotic Therapy, Renal Outcomes, AKI Progression

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Introduction

Acute Kidney Injury (AKI) is a rapidly developing condition marked by an abrupt decline in kidney function, leading to retention of nitrogenous waste, electrolyte imbalances, and volume disturbances [1]. It occurs frequently in hospitalized patients, with reported incidences ranging from 5% to 30% depending on clinical setting and

patient population. Despite advances in supportive care, AKI remains associated with high mortality and morbidity [2].

Historically, AKI was regarded as a reversible syndrome; however, accumulating evidence reveals that AKI often predisposes patients to the development or acceleration of chronic kidney disease (CKD) [3]. This AKI-to-CKD continuum represents a paradigm shift in nephrology, emphasizing the importance of identifying patients at risk and intervening early to prevent irreversible renal damage. The pathophysiology underlying AKI progression to CKD involves sustained tubular injury, inflammation, vascular rarefaction, and fibrosis that result in permanent nephron loss. Severity of AKI is strongly associated with poor renal recovery, with Stage 3 AKI patients having the highest risk of progression [4,5].

Etiologically, intrinsic renal causes such as sepsis-induced acute tubular necrosis and drug-induced interstitial nephritis are associated with worse outcomes due to direct parenchymal damage. Timely and appropriate management of AKI is paramount. In particular, infections leading to sepsis-associated AKI require prompt and targeted antibiotic therapy to halt ongoing inflammation and prevent further kidney injury [6,7].

Although comorbidities such as diabetes and hypertension are known risk factors, this study focuses on the role of AKI severity, cause, and early therapeutic interventions—especially appropriate antibiotic use—in reducing CKD progression.

Materials and Methods

Study Design and Setting

This retrospective cohort study was conducted at a tertiary care teaching hospital from November 18, 2022, to April 22, 2025. The aim was to assess AKI progression to CKD and analyze clinical outcomes based on etiology and management.

Participants

We included 200 patients diagnosed with AKI during hospitalization, selected via systematic review of medical records. AKI diagnosis and staging were based on KDIGO criteria, considering serum creatinine and urine output changes.

Inclusion Criteria

- Patients aged ≥18 years with AKI due to:
    - **Pre-renal causes:** Volume depletion (vomiting, diarrhea), hemorrhage, burns, acute pancreatitis.
    - **Intrinsic renal causes:** Sepsis, drug-induced nephritis, acute glomerulonephritis, thrombotic microangiopathy.
    - **Post-renal causes:** Urinary tract obstruction due to benign prostatic hyperplasia (BPH), stones, or tumors.

Exclusion Criteria

- Pre-existing CKD or end-stage renal disease.
- Incomplete medical records.
- Polycystic kidney disease and chronic tubulointerstitial nephritis.

Treatment Approach

Management was tailored according to AKI etiology:

- **Volume depletion:** Prompt intravenous fluid resuscitation to restore renal perfusion.
- **Sepsis-associated AKI:** Early administration of appropriate, culture-guided antibiotics was emphasized to control infection and minimize inflammatory injury.
- **Drug-induced AKI:** Offending medications were promptly discontinued.

- **Obstructive uropathy:** Early surgical or catheter-based decompression was performed.
- **Severe AKI:** Dialysis initiated when indicated (refractory electrolyte imbalance, fluid overload, uremic symptoms).

Supportive care included nutritional optimization (target ~30 kcal/kg/day) and close monitoring of renal function.

Data Collection

Patient demographics, clinical details, laboratory values, AKI staging, and outcomes were extracted by trained personnel from electronic health records. Follow-up data were collected to identify progression to CKD, defined as estimated GFR <60 mL/min/1.73 m² persisting for >3 months post-AKI.

Statistical Analysis

Data were analyzed using SPSS v23.0. Descriptive statistics summarized baseline characteristics. Kaplan-Meier survival curves compared CKD-free survival by AKI stage. Cox proportional hazards regression identified independent predictors of CKD progression. A p-value <0.05 was considered statistically significant.

Results

Patient Characteristics and CKD Incidence

Of 200 patients with AKI, 40 (20%) progressed to CKD over a median follow-up of 24 months. Patients who progressed were older (mean 64.5 vs. 56.8 years) and had more severe AKI.

AKI Stage	Patients (n)	CKD Cases (n)	CKD Rate (%)
Stage 1	98	8	8.2
Stage 2	68	14	20.6
Stage 3	34	18	52.9

Stage 3 AKI patients had a significantly higher CKD progression risk compared to Stage 1 ($p < 0.001$).

Etiology and CKD Progression

Intrinsic renal causes had the highest progression rate:

Etiology	Patients (n)	CKD Cases (n)	Progression Rate (%)
Pre-renal	94	18	19.1
Intrinsic renal	76	30	39.5
Post-renal	30	8	26.7

Intrinsic AKI causes, such as sepsis and drug-induced nephritis, showed nearly double the risk compared to pre-renal causes.

Impact of Timely Intervention and Antibiotic Use

Among 144 patients receiving timely, etiology-specific interventions, including early and appropriate antibiotics for infection-related AKI, CKD progression was reduced to 13.2%. This reduction highlights the critical role of targeted therapy in altering disease course.

Survival Analysis

Kaplan-Meier curves demonstrated significantly reduced CKD-free survival in Stage 3 AKI (68.2% at 12 months) compared to Stage 1 (94.5%).

Risk Factors for CKD Progression

Multivariable Cox regression identified:

Variable	Hazard Ratio (HR)	95% CI	p-value

Age (per 10 years)	1.28	1.05–1.57	0.015
Baseline eGFR	1.35 (per 10 ↓)	1.12–1.62	0.001
AKI Stage 3	4.78	2.20–10.37	<0.001

Severe AKI stage and lower baseline renal function were strong independent predictors of CKD conversion.

Discussion

The findings of this retrospective study reveal that approximately one in five patients who experienced acute kidney injury (AKI) progressed to chronic kidney disease (CKD) within a median follow-up period of two years. This rate aligns well with previously reported figures and highlights the substantial long-term impact AKI can have on kidney health.

Importantly, the risk of progression to CKD was strongly correlated with the severity of the initial AKI episode. Patients classified as Stage 3 AKI faced the greatest risk, with more than half progressing to CKD. This clearly emphasizes that the extent of kidney damage at the time of injury plays a pivotal role in determining renal recovery and long-term outcomes. Severe AKI likely causes irreversible nephron loss through mechanisms such as extensive tubular injury and interstitial fibrosis, limiting the kidney's ability to fully regenerate [8,9].

The underlying cause of AKI also influenced the likelihood of progression. Intrinsic renal causes, including sepsis-associated acute tubular necrosis and drug-induced nephritis, were associated with the highest risk of developing CKD. These findings are consistent with existing knowledge that intrinsic renal injuries cause direct damage to kidney tissue, setting in motion maladaptive repair processes and fibrosis that contribute to chronic renal impairment. Conversely, pre-renal causes such as volume depletion, which are often reversible with timely intervention, showed comparatively lower rates of progression to CKD [10].

A particularly noteworthy aspect of this study was the critical importance of timely and appropriate therapeutic interventions, especially the administration of targeted antibiotics in cases of infection-related AKI. Infection is a common and serious precipitating factor for AKI, and delays or inadequacies in antibiotic treatment can exacerbate systemic inflammation and worsen renal injury. Early and appropriate use of antibiotics helps control the underlying infection, reduces the release of inflammatory mediators, and thereby mitigates ongoing kidney damage [11,12].

The study demonstrated that patients receiving such prompt interventions, including appropriate antibiotic therapy, had a significantly lower rate of progression to CKD, underscoring the vital role that antimicrobial stewardship plays in managing AKI effectively. These clinical observations are supported by current research that elaborates on the pathophysiological continuum from AKI to CKD. Studies have shown that sustained injury and maladaptive repair mechanisms, involving endothelial damage and chronic inflammation, are central drivers of fibrotic kidney remodeling and irreversible functional decline [13,14]. The concept of Acute Kidney Disease (AKD) as an intermediate stage between AKI and CKD has emerged, highlighting a therapeutic window during which interventions can prevent permanent kidney damage [15,16].

Additionally, the study found that lower baseline renal function and advanced age were independent risk factors for the advancement of CKD. Reduced renal reserve and regenerative capacity are probably reflected in these characteristics, making these individuals more susceptible to long-term harm after AKI. This conclusion emphasises the necessity of active therapy and enhanced surveillance in older adults and those with mild pre-existing renal impairment. In order to

concentrate on the effects of AKI severity and treatment, the study excluded individuals with diabetes and hypertension; nevertheless, this may restrict its generalizability to larger patient populations where these comorbidities are prevalent. However, it shows that even without them, the severity and cause of AKI, along with prompt treatment, significantly impact long-term results.

Clinically, these findings underscore the importance of early identification of AKI severity and etiology, followed by rapid, cause-specific management. In particular, the judicious and prompt use of antibiotics in infection-related AKI is critical to reducing inflammation-driven kidney injury and preventing progression to CKD. Similarly, preventing or correcting volume depletion and relieving urinary obstruction remain cornerstone strategies to minimize pre-renal and post-renal injury, respectively.

Future research is required to validate these results and improve antibiotic methods that successfully strike a balance between nephrotoxicity reduction and infection control. Finding biomarkers that indicate the progression of AKI to CKD may also help with risk assessment and treatment planning. Lastly, the creation of multidisciplinary care guidelines that emphasise long-term monitoring and AKI prevention may enhance patient outcomes and lessen the prevalence of CKD worldwide. This study emphasises that the likelihood of developing chronic kidney disease is mostly determined by the severity and aetiology of AKI, as well as the timing and suitability of management, especially antibiotic therapy in infectious patients. Future studies aiming at preventing chronic renal impairment after acute injury should be guided by these insights, which should also influence clinical practice.

Conclusion

AKI severity and etiology strongly predict CKD progression, with intrinsic renal causes posing the highest risk. Early, appropriate interventions, especially judicious antibiotic use in infection-driven AKI, substantially reduce long-term renal damage. Clinicians should prioritize early diagnosis, targeted therapy, and follow-up to mitigate CKD burden post-AKI.

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