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Original Article

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A Prospective Observational Study of Predictors and Clinical Outcomes of Acute Kidney Injury in Hospitalized Adults with Community-Acquired Pneumonia

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ABSTRACT

Background: Acute kidney injury (AKI) is a frequent complication of community-acquired pneumonia (CAP) and worsens outcomes. Prospective data on its predictors from Indian cohorts are limited.

Methods: This prospective observational study at the Department of Internal Medicine, Malabar Medical College, Kerala (May 2025–June 2026) enrolled 110 consecutive adults hospitalized with CAP. We defined and staged AKI by KDIGO criteria. The primary outcome was in-hospital AKI; secondary outcomes were AKI stage, ICU admission, mechanical ventilation, length of stay, in-hospital mortality and renal replacement therapy (RRT). Predictors were assessed by univariable and multivariable logistic regression.

Results: AKI developed in 28 patients (25.5%): KDIGO stage 1 in 17, stage 2 in 7 and stage 3 in 4. Patients with AKI were older (68.2 vs 58.7 years) and more often had pre-existing CKD (32.1% vs 8.5%), hypotension, hypoxemia and CURB-65 ≥3. The incidence of AKI rose from 11.1% to 24.4% and 42.4% across CURB-65 categories 0–1, 2 and ≥3 (trend P = 0.003). AKI was associated with ICU admission (53.6% vs 19.5%), mechanical ventilation (32.1% vs 11.0%), longer median stay (10 vs 7 days) and higher in-hospital mortality (21.4% vs 6.1%); three patients required RRT. Independent predictors were pre-existing CKD (adjusted OR 3.8, 95% CI 1.3–11.2), systolic BP <90 mmHg (3.5, 1.2–10.1), CURB-65 ≥3 (3.1, 1.1–8.8) and higher admission creatinine (2.7, 1.1–6.8).

Conclusion: One in four adults hospitalized with CAP developed AKI, which was associated with markedly worse outcomes. Baseline CKD, hypotension and severe pneumonia identify high-risk patients at admission who merit intensified renal monitoring and preventive care.

Key-words: Community-acquired pneumonia; Acute kidney injury; KDIGO; CURB-65; Risk factors; ICU admission; Mortality

INTRODUCTION

Community-acquired pneumonia (CAP) remains one of the commonest reasons for hospital admission among adults and a leading infectious cause of death worldwide [1,2].

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Its clinical spectrum ranges from mild illness managed in the community to severe disease complicated by sepsis, respiratory failure and multiorgan dysfunction. The development of extrapulmonary organ failure substantially influences prognosis [1,2].

Early assessment of disease severity is essential for determining the appropriate level of care. The Pneumonia Severity Index (PSI) combines demographic, clinical and laboratory variables to identify low-risk patients [3]. The CURB-65 score, based on confusion, elevated blood urea, respiratory rate ≥30/min, low blood pressure and age ≥65 years, is a simpler bedside tool [4]. However, these scores were primarily developed to estimate mortality risk and guide decisions regarding the

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site of care rather than predict specific organ complications.

Acute kidney injury (AKI) is an important extrapulmonary complication of CAP. It is characterized by an abrupt decline in kidney function, resulting in impaired fluid, electrolyte and acid–base regulation. AKI is associated with prolonged hospitalization, increased mortality and an elevated risk of subsequent chronic kidney disease (CKD) [5,6]. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria provide a standardized definition based on changes in serum creatinine and urine output and classify AKI into three stages according to severity [5]. Studies in hospitalized and critically ill populations have demonstrated that AKI is common and associated with adverse clinical outcomes [7–9].

Evidence from pneumonia cohorts also indicates that AKI may occur even in patients with non-severe pneumonia and is associated with an increased inflammatory response and reduced survival [10]. The International Society of Nephrology has highlighted the substantial burden of AKI and the scarcity of epidemiological data from low- and middle-income countries, emphasizing the need for locally generated evidence [11].

The development of AKI in pneumonia is multifactorial and involves interacting haemodynamic, inflammatory and treatment-related mechanisms [12–15]. Systemic infection can trigger inflammatory responses, endothelial dysfunction and disturbances in renal microcirculation. Sepsis-associated AKI may occur even in the absence of sustained systemic hypotension, owing to microcirculatory and cellular abnormalities [13,14]. Hypotension, vasodilatation and dehydration may reduce renal perfusion, while hypoxemia can compromise oxygen delivery to renal tissues. Nephrotoxic antimicrobials, contrast agents and other medications may further aggravate renal injury, particularly in patients with reduced renal reserve [15]. Advanced age and pre-existing CKD, along with other comorbidities, may increase susceptibility to AKI [12,15].

Despite the available evidence, the reported incidence and predictors of AKI in pneumonia vary according to patient characteristics, diagnostic definitions and monitoring intensity. Prospective data from Indian hospitals using standardized KDIGO criteria remain limited [5,8,11]. Identifying readily available admission predictors may help clinicians recognize high-risk patients early and institute appropriate renal monitoring, optimize haemodynamic status and avoid preventable nephrotoxic exposure [5,12].

Therefore, the present prospective study was conducted among adults hospitalized with CAP to determine the incidence and severity of AKI, evaluate its association with in-hospital clinical outcomes, and identify independent admission predictors that may support early risk stratification and preventive measures.

MATERIALS AND METHODS

Study design, setting and period- This was a prospective observational study conducted in the Department of Internal Medicine, Malabar Medical College, Kozhikode, Kerala, India, over one year from May 2025 to June 2026. The study was reported in accordance with the STROBE recommendations for observational studies.

Inclusion Criteria- Consecutive adults (≥18 years) admitted to the Department of Internal Medicine with CAP were screened. CAP was defined as an acute lower respiratory tract infection with a new infiltrate on chest radiography or computed tomography, occurring in a patient who was not hospitalized or resident in a healthcare facility at onset, together with compatible features such as fever, cough, sputum production, dyspnoea, tachypnoea or pleuritic chest pain. Patients were enrolled after written informed consent until 110 participants had been recruited.

Exclusion Criteria- Patients were excluded if they had hospital-acquired or ventilator-associated pneumonia, end-stage kidney disease or maintenance dialysis, kidney transplantation, post-renal obstruction, recent major surgery or major trauma likely to cause AKI independently, or pregnancy, or if clinical or laboratory data were insufficient to assess renal outcomes. Patients with CKD that was not end-stage were retained, because baseline renal dysfunction was a candidate predictor.

Data collection- A structured proforma recorded demographics (age, sex, residence, smoking, alcohol use), comorbidities (diabetes mellitus, hypertension, CKD, coronary artery disease, heart failure, chronic obstructive pulmonary disease, cerebrovascular disease), and admission clinical parameters (temperature, heart rate, respiratory rate, blood pressure, oxygen saturation, mental status, respiratory distress, dehydration and oxygen requirement).

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Laboratory investigations comprised complete blood count, urea, creatinine, electrolytes, glucose, liver function tests, albumin, C-reactive protein (CRP), procalcitonin where available, arterial blood gas and serum lactate in clinically appropriate patients. Microbiological tests (blood and sputum culture, respiratory viral testing) were performed according to clinical indication and institutional practice. Serial serum creatinine values and urine output were recorded throughout hospitalization. Baseline renal function was taken as the most recent serum creatinine obtained 7–365 days before admission. When no prior value existed, the admission creatinine was interpreted in the light of the clinical history and subsequent trend. CKD was identified from previous records and history.

Assessment of pneumonia severity- Pneumonia severity at admission was graded by the CURB-65 score (confusion, urea elevation, respiratory rate ≥30/min, systolic blood pressure <90 mmHg or diastolic ≤60 mmHg, and age ≥65 years; 0–5 points) [4]. The Pneumonia Severity Index was calculated where adequate data were available [3].

Definition and staging of AKI- AKI was defined and staged according to KDIGO criteria [5] (Table 1). The maximum stage reached during hospitalization and the interval from admission to AKI were recorded. Patients were classified as AKI (Group 2) or no AKI (Group 1).

Table 1: KDIGO definition and staging of acute kidney injury

Stage	Serum creatinine criterion	Urine output criterion
Stage 1	1.5–1.9 × baseline, or increase ≥0.3 mg/dL	<0.5 mL/kg/h for 6–12 h
Stage 2	2.0–2.9 × baseline	<0.5 mL/kg/h for ≥12 h
Stage 3	≥3.0 × baseline, or creatinine ≥4.0 mg/dL, or initiation of RRT	<0.3 mL/kg/h for ≥24 h, or anuria ≥12 h

AKI is diagnosed by a rise in creatinine ≥0.3 mg/dL within 48 h, or to ≥1.5 × baseline within 7 days, or urine output <0.5 mL/kg/h for 6 h. RRT, renal replacement therapy.

Outcomes- The primary outcome was development of AKI during hospitalization. Secondary outcomes were maximum KDIGO stage, ICU admission, mechanical ventilation, length of hospital stay, in-hospital mortality and requirement for RRT. Variables evaluated as potential predictors were age, sex, comorbidities, admission vital signs and oxygenation, altered mental status, dehydration, CURB-65 and PSI, sepsis, and admission urea, creatinine, haemoglobin, leukocyte count, platelet count, CRP, albumin, lactate and electrolytes. Treatment-related exposures (nephrotoxic drugs, vasopressors, mechanical ventilation) were recorded but not used as admission predictors.

Statistical analysis- Data were analysed using IBM SPSS Statistics. Normality was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± SD or median (IQR), as appropriate, and categorical variables as frequencies and percentages. Between-group comparisons were performed using Student's t-test or Mann–Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables.

For presentation, unadjusted odds ratios with Wald 95% CIs were derived from the 2 × 2 tables of the reported counts, exact (Fisher) p-values were calculated for categorical outcomes, and a Cochran–Armitage test was used for the trend in AKI incidence across ordered CURB-65 categories.

Ethical considerations- The study was approved by the Institutional Ethics Committee of Malabar Medical College. Written informed consent was obtained from all participants. Data were recorded against study identification numbers, confidentiality was maintained, and management followed the treating physician's judgment and institutional protocols without interference from the study.

RESULTS

A total of 110 adults hospitalized with CAP were enrolled and followed to discharge or death. Their mean age was approximately 61 years and 71 (64.5%) were men. AKI developed in 28 patients (25.5%), whereas 82 (74.5%) did not develop AKI (Fig. 1A). Among those with AKI, the maximum KDIGO stage was 1 in 17 (60.7%), 2 in 7 (25.0%) and 3 in 4 (14.3%) patients (Fig. 1B).

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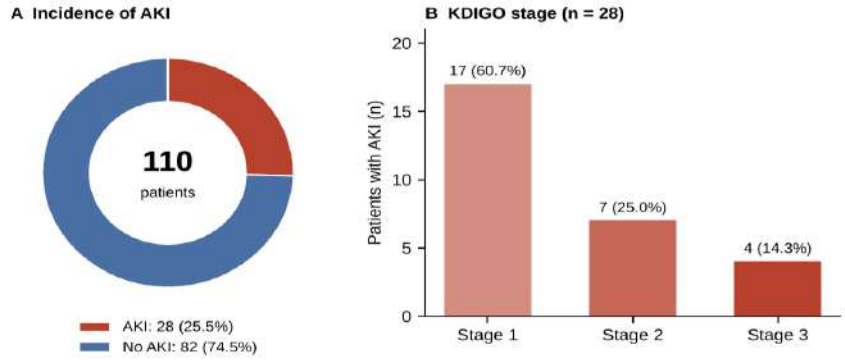


Fig. 1: Incidence and severity of acute kidney injury (AKI). *(A) Proportion of 110 patients with CAP who developed AKI. (B) Distribution of maximum KDIGO stage among the 28 patients with AKI.*

Patients who developed AKI were significantly older (68.2 ± 12.1 vs 58.7 ± 14.3 years; p = 0.001), whereas sex distribution was similar (Table 2). Pre-existing CKD was the comorbidity most strongly associated with AKI (32.1% vs 8.5%; p<0.001). Diabetes mellitus, hypertension, coronary artery disease and heart failure were numerically more frequent in the AKI group but did not reach statistical significance.

Table 2: Admission characteristics of patients according to AKI status

Variable	AKI (n = 28)	No AKI (n = 82)	p-value
Demographics			
Age, years (mean ± SD)	68.2 ± 12.1	58.7 ± 14.3	0.001
Male sex	19 (67.9)	52 (63.4)	0.67
Comorbidities			
Pre-existing CKD	9 (32.1)	7 (8.5)	<0.001
Diabetes mellitus	16 (57.1)	31 (37.8)	0.08
Hypertension	20 (71.4)	43 (52.4)	0.08
Coronary artery disease	8 (28.6)	12 (14.6)	0.09
Heart failure	5 (17.9)	5 (6.1)	0.07
COPD	7 (25.0)	14 (17.1)	0.35
Clinical parameters			
SBP <90 mmHg	9 (32.1)	7 (8.5)	0.002
Heart rate ≥100/min	15 (53.6)	22 (26.8)	0.009
Respiratory rate ≥30/min	16 (57.1)	21 (25.6)	0.002
SpO_2 <90%	12 (42.9)	14 (17.1)	0.005
Altered sensorium	7 (25.0)	8 (9.8)	0.04
Supplemental oxygen	22 (78.6)	40 (48.8)	0.006
CURB-65 score			
0–1	4 (14.3)	32 (39.0)	—
2	10 (35.7)	31 (37.8)	—
≥3	14 (50.0)	19 (23.2)	—
Laboratory parameters (mean ± SD)			
Haemoglobin, g/dL	11.1 ± 2.0	11.8 ± 1.9	0.09
Leukocyte count, /µL	13,200 ± 5,100	11,600 ± 4,400	0.12
Urea, mg/dL	61.4 ± 29.2	38.7 ± 18.6	<0.001
Creatinine, mg/dL	1.48 ± 0.63	0.94 ± 0.24	<0.001
CRP, mg/L	126 ± 72	82 ± 59	0.002
Albumin, g/dL	3.1 ± 0.5	3.4 ± 0.5	0.006
Lactate, mmol/L	2.8 ± 1.5	1.8 ± 0.9	<0.001

Values are n (%) unless stated. CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; SBP, systolic blood pressure; SpO_2, oxygen saturation; CRP, C-reactive protein.

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Patients who subsequently developed AKI presented with greater physiological derangement. Systolic blood pressure <90 mmHg (32.1% vs 8.5%; p=0.002), heart rate ≥100/min (53.6% vs 26.8%; p=0.009), respiratory rate ≥30/min (57.1% vs 25.6%; p=0.002), SpO_2 <90% (42.9% vs 17.1%; p=0.005), altered sensorium (25% vs 9.8%; p=0.04) and need for supplemental oxygen (78.6% vs 48.8%; p=0.006) were all more frequent in the AKI group (Table 2).

On admission, patients with AKI had higher urea (61.4 vs 38.7 mg/dL), creatinine (1.48 vs 0.94 mg/dL), CRP (126 vs 82 mg/L) and lactate (2.8 vs 1.8 mmol/L), and lower albumin (3.1 vs 3.4 g/dL) than patients without AKI (all p ≤0.006; Table 2). Haemoglobin and leukocyte count did not differ significantly. Half of the patients with AKI had CURB-65 ≥3, compared with 23.2% of those without AKI (Fig. 2A). Conversely, the incidence of AKI increased stepwise with severity: 11.1% (4/36) for CURB-65 0–1, 24.4% (10/41) for score 2 and 42.4% (14/33) for score ≥3 (Cochran–Armitage trend=0.003; Fig. 2B).

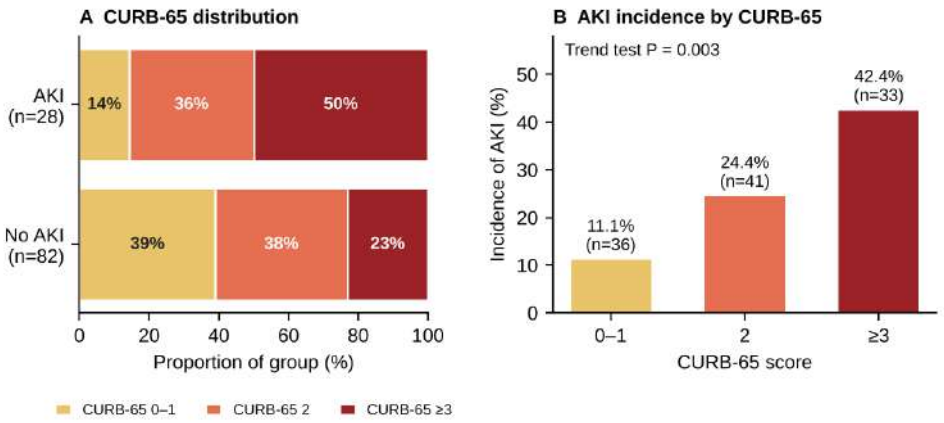


Fig. 2: Pneumonia severity (CURB-65) and AKI. *(A) Distribution of CURB-65 categories within the AKI and no-AKI groups. (B) Incidence of AKI within each CURB-65 category; the number of patients in each category is shown. P value from the Cochran–Armitage trend test.*

AKI was associated with significantly worse in-hospital outcomes (Table 3). ICU admission was required in 53.6% of patients with AKI versus 19.5% of those without (p = 0.001), and mechanical ventilation in 32.1% versus 11.0% (p = 0.016). Median hospital stay was 10 days in the AKI group and 7 days in the no-AKI group (p <0.05). In-hospital mortality was 21.4% (6/28) with AKI versus 6.1% (5/82) without (p = 0.030). Three patients with AKI (10.7%) required renal replacement therapy; all had severe AKI and were managed in the ICU.

Table 3: Clinical outcomes according to AKI status

Outcome	AKI (n = 28)	No AKI (n = 82)	p-value
ICU admission	15 (53.6)	16 (19.5)	0.001
Mechanical ventilation	9 (32.1)	9 (11.0)	0.016
Median hospital stay, days	10	7	<0.05
In-hospital mortality	6 (21.4)	5 (6.1)	0.030
Renal replacement therapy	3 (10.7)	-	-

Values are n (%) unless stated. p-values for categorical outcomes by Fisher's exact test.

On univariable analysis, pre-existing CKD (OR 5.1, 95% CI 1.7–15.4), systolic BP <90 mmHg (5.1, 1.7–15.4), respiratory rate ≥30/min (3.9, 1.6–9.5), supplemental oxygen (3.9, 1.4–10.5), SpO_2 <90% (3.6, 1.4–9.4), CURB-65 ≥3 (3.3, 1.3–8.2) and heart rate ≥100/min (3.1, 1.3–7.7) were significantly associated with AKI (Table 4). Altered sensorium showed a similar magnitude of association (OR 3.1) with a lower confidence limit of 1.0. Associations for diabetes mellitus, hypertension, coronary artery disease, heart failure and COPD were positive in direction but imprecise.

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Table 4: Unadjusted odds ratios for AKI

Variable	AKI, n/28	No AKI, n/82	Unadjusted OR (95% CI)
Pre-existing CKD	9/28	7/82	5.1 (1.7–15.4)
Diabetes mellitus	16/28	31/82	2.2 (0.9–5.2)
Hypertension	20/28	43/82	2.3 (0.9–5.7)
Coronary artery disease	8/28	12/82	2.3 (0.8–6.5)
Heart failure	5/28	5/82	3.3 (0.9–12.6)
COPD	7/28	14/82	1.6 (0.6–4.5)
SBP <90 mmHg	9/28	7/82	5.1 (1.7–15.4)
Heart rate ≥100/min	15/28	22/82	3.1 (1.3–7.7)
Resp. rate ≥30/min	16/28	21/82	3.9 (1.6–9.5)
SpO_2 <90%	12/28	14/82	3.6 (1.4–9.4)
Altered sensorium	7/28	8/82	3.1 (1.0–9.5)
Supplemental O_2	22/28	40/82	3.9 (1.4–10.5)
CURB-65 ≥3	14/28	19/82	3.3 (1.3–8.2)

Odds ratios and Wald 95% confidence intervals were calculated from the reported 2 × 2 counts. OR, odds ratio.

Presence of a single admission risk factor identified a group at roughly two- to three-fold higher absolute risk (Fig. 3). The incidence of AKI was 56.3% (9/16) in patients with pre-existing CKD versus 20.2% (19/94) without, and 56.3% (9/16) in those with systolic BP <90 mmHg versus 20.2% (19/94) without. Corresponding figures were 46.2% versus 19.0% for SpO_2 <90%, 43.2% versus 16.4% for respiratory rate ≥30/min, 42.4% versus 18.2% for CURB-65 ≥3 and 46.7% versus 22.1% for altered sensorium.

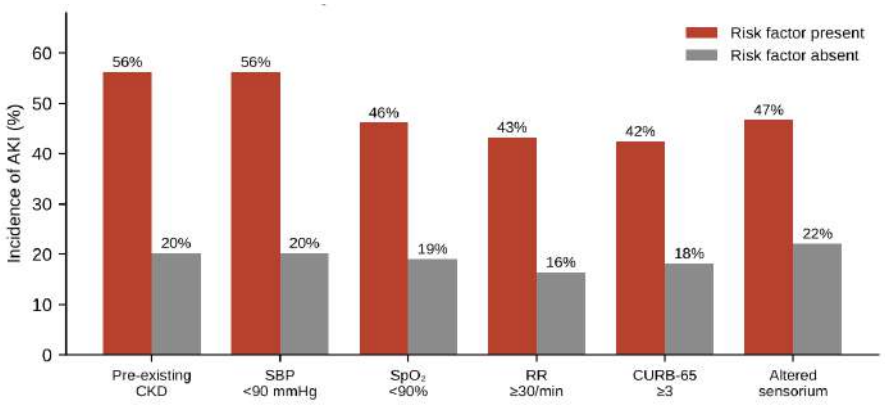


Fig. 3: Absolute risk of AKI according to the presence or absence of admission risk factors.
Percentages are the incidence of AKI among patients with and without the factor.

In the multivariable logistic regression model, four variables remained independently associated with AKI: pre-existing CKD (aOR 3.8, 95% CI 1.3–11.2; p = 0.015), systolic BP <90 mmHg (aOR 3.5, 1.2–10.1; p = 0.020), CURB-65 ≥3 (aOR 3.1, 1.1–8.8; p = 0.034) and elevated admission creatinine (aOR 2.7, 1.1–6.8; p = 0.041) (Table 5).

Table 5. Multivariable logistic regression of independent predictors of AKI

Predictor	Adjusted OR	95% CI	p-value
Pre-existing CKD	3.8	1.3–11.2	0.015
SBP <90 mmHg	3.5	1.2–10.1	0.020
CURB-65 ≥3	3.1	1.1–8.8	0.034
Elevated admission creatinine	2.7	1.1–6.8	0.041

OR, odds ratio; CI, confidence interval; SBP, systolic blood pressure.

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DISCUSSION

In this prospective cohort of 110 adults hospitalized with CAP in Kerala, one in four developed AKI. Although most episodes were KDIGO stage 1, AKI was accompanied by a roughly threefold higher frequency of ICU admission, mechanical ventilation and in-hospital death, a three-day longer median stay and, in a small number of patients, a need for renal replacement therapy. Pre-existing CKD, hypotension, severe pneumonia (CURB-65 ≥3) and higher admission creatinine independently predicted AKI. All four are available at the bedside or in the first set of laboratory results, which gives the findings practical value for triage.

An incidence of 25.5% is high but plausible for an inpatient population in whom AKI was sought systematically with KDIGO criteria. Published incidence figures in pneumonia and sepsis vary widely because of differences in case-mix, in baseline kidney function, in the definition applied and in how often creatinine and urine output are measured [7,8]. Hospitalized CAP patients are, moreover, a selected, more severely ill group than those treated in the community, and many in our cohort were elderly with multiple comorbidities and a substantial prevalence of hypoxemia and haemodynamic instability. The predominance of stage 1 AKI (60.7%) is consistent with the usual pyramid of AKI severity, but the 14.3% progressing to stage 3 form a clinically important subgroup that accounted for all dialysis requirement [6,7].

Patients with AKI were on average nearly ten years older. Ageing reduces renal reserve through nephrosclerosis, impaired autoregulation and a greater burden of hypertension, diabetes and vascular disease, and older patients are more prone to dehydration and drug-related injury during acute illness [12,15]. Pre-existing CKD was the strongest single predictor, with an unadjusted OR of 5.1 and an adjusted OR of 3.8, and more than half of patients with CKD developed AKI. A kidney with fewer functioning nephrons has less capacity to tolerate the haemodynamic and inflammatory stress of pneumonia, and CKD and AKI reinforce one another in a bidirectional relationship [5,11]. Identifying CKD from records or admission creatinine should therefore be part of the initial assessment of every patient with CAP.

Systolic hypotension was independently associated with AKI, with a nearly four-fold difference in prevalence between groups (32.1% vs 8.5%). Renal blood flow and glomerular filtration depend on adequate perfusion pressure, and persistent hypotension produces ischaemic tubular injury [13,15]. Yet hypotension does not fully explain AKI in sepsis: injury may occur despite apparently preserved systemic haemodynamics, through microcirculatory dysfunction and inflammatory and metabolic mechanisms [13,14]. Our data are consistent with this broader view. Patients with AKI also had higher heart rate, respiratory rate, oxygen requirement, lactate and CRP and lower albumin, a constellation that points to greater systemic inflammatory burden and tissue hypoperfusion. The association with hypoxemia is biologically plausible, since the renal tubule has high oxygen demand, but cannot be separated here from the severity of the pneumonia itself. The inflammatory component is in line with observations in pneumonia cohorts in which AKI was accompanied by a heightened immune response and reduced survival [10]. Lower albumin may reflect inflammation, capillary leak and poor nutritional reserve rather than a direct renal effect.

The stepwise rise in AKI incidence across CURB-65 categories (11.1%, 24.4% and 42.4%) illustrates that a score created to predict mortality and site of care also stratifies renal risk [4]. This is expected, since confusion, tachypnoea, hypotension and raised urea reflect the same organ dysfunction pathways that injure the kidney. A caveat is that CURB-65 includes urea, which shares variance with admission creatinine; the two should not be interpreted as wholly independent. CURB-65 ≥3 can nevertheless be used as a simple flag: roughly four in ten such patients in our series developed AKI.

ICU admission and mechanical ventilation were more frequent with AKI, which probably reflects both shared severity and the added complexity that kidney injury imposes on fluid, electrolyte and acid–base management. Ventilation itself can alter intrathoracic pressure and venous return and may require vasoactive drugs, adding further renal stress [12,15]. The longer stay is likely bidirectional: AKI prolongs hospitalization through monitoring, medication adjustment and treatment of complications, while longer hospitalization increases exposure to nephrotoxic and haemodynamic insults [19,20]. Mortality was 21.4% in the AKI group and 6.1% in the comparison group. This is consistent with the established association between AKI and death in hospitalized and critically ill patients [6,7,9]. Because our comparison is unadjusted and the number of deaths small, we cannot determine whether AKI is itself causal

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or mainly a marker of the severity of illness; most likely it is both [12]. Only three patients required renal replacement therapy, which shows that most AKI in CAP is manageable without dialysis, but also that dialysis capacity must be available for the few with stage 3 injury [11].

The findings suggest a simple admission checklist for every adult with CAP (Table 6). Patients with CKD, hypotension, CURB-65 ≥3 or an elevated admission creatinine can be flagged as high-risk and managed with a bundle based on KDIGO principles: serial creatinine and strict urine output monitoring, early restoration of volume and perfusion pressure, avoidance or dose adjustment of nephrotoxic drugs and contrast, and early review in patients whose renal function worsens [5,12]. Prospective testing is required to establish whether such a strategy lowers the incidence or severity of AKI.

Table 6: Suggested admission risk checklist derived from the study findings

Admission variable	Observed AKI incidence if present	Suggested response
Pre-existing CKD	56.3% (aOR 3.8)	Baseline eGFR/creatinine; avoid nephrotoxins; daily creatinine
SBP <90 mmHg	56.3% (aOR 3.5)	Early volume and haemodynamic optimization; urine output chart
CURB-65 ≥3	42.4% (aOR 3.1)	High-dependency monitoring; serial creatinine
Admission creatinine elevated	Higher at admission (1.48 vs 0.94 mg/dL; aOR 2.7)	Repeat within 24 h; review drug doses

Incidence values are crude proportions from this cohort. The suggested responses are derived from KDIGO general principles and are not outcomes of this study. eGFR, estimated glomerular filtration rate.

CONCLUSIONS

AKI complicated about one quarter of hospital admissions for CAP in this prospective Kerala cohort and was associated with higher rates of ICU admission and mechanical ventilation, longer hospital stay and higher in-hospital mortality. Pre-existing CKD, systolic hypotension, severe pneumonia (CURB-65 ≥3) and higher admission creatinine were independent predictors, and AKI incidence rose steadily with pneumonia severity. These readily available variables can help clinicians identify patients at admission who warrant intensified renal monitoring and early preventive measures. Larger multicentre studies are needed to validate these predictors and to test whether risk-guided interventions improve outcomes.

CONTRIBUTION OF AUTHORS

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