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ORIGINAL ARTICLE

C-reactive protein as a predictor of acute kidney injury in ICU patients: A prospective observational cohort study.

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ABSTRACT… Objective: To evaluate baseline C-reactive protein (CRP) as a predictor of acute kidney injury (AKI) in critically ill ICU patients. **Study Design:** Prospective Observational Cohort study. **Setting:** ICU of Shifa International Hospital Islamabad. **Period:** 1st November 2025 to 28th February 2026. **Methods:** A total of 194 patients aged 16-92 years were enrolled using consecutive sampling. Baseline CRP was measured within 12 hours of ICU admission. Mann–Whitney U test compared CRP levels between AKI and non-AKI groups. ROC curve analysis determined predictive accuracy. Binary logistic regression identified independent predictors of AKI. A p-value ≤0.05 was considered statistically significant. **Results:** AKI occurred in 66 patients (34.0%). Baseline CRP was significantly higher in patients who developed AKI (mean rank 116.52 vs. 88.45, U = 3002.0, p = .020). ROC analysis demonstrated acceptable discriminatory ability (AUC = 0.645). On multivariate logistic regression, age (OR = 1.034, p = .036), male gender (OR = 4.024, p = .012), APACHE II score (OR = 1.099, p = .027), and baseline CRP (OR = 1.006, p = .031) were independent predictors of AKI. AKI was significantly associated with ICU mortality (p = .003) and mechanical ventilation (p = .030), but not with length of ICU stay (p = .069). **Conclusion:** Elevated baseline CRP is independently associated with development of AKI in critically ill ICU patients. However, its discriminatory ability is modest (AUC 0.645), suggesting that CRP should be interpreted in conjunction with established severity scores such as APACHE II rather than used as a standalone predictor.

Key words: Acute Kidney Injury, APACHE II, C-reactive Protein, Intensive Care Unit, Predictor, ROC Curve.

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INTRODUCTION

Acute kidney injury (AKI) is a sudden renal dysfunction leading to significant morbidity and mortality.[1] It occurs in around 30% of ICU patients and 7% in normal hospitalized patients.[2] most common underlying pathologies in these patients are ischemic or reperfusion injuries to kidney parenchyma, hemodynamic instability and toxicity.[3]

C-reactive protein (CRP), an acute-phase reactant that increases during systemic inflammation, has been proposed as a potential predictor of AKI development and progression. It also exacerbate AKI by inducing fibrosis and damage to renal tubules and parenchyma.[4,5]

In ICU patients CRP is raised often in systemic sepsis and which also indirectly lead to AKI.[6] Not only progressive rise of CRP lead to such conditions but also baseline CRP at time of admisssion has also shown to have strong prediction regarding AKI occurrence in ICU stay.[7]

Value of CRP is directly proportional to extent of damage of kidneys. more CRP levels, more patients has shown to be requiring renal replacement therapy and increase length of stay in these patients.[8,9]

A study by Nigerian ICU study (n=204), AKI developed in 55.9% and RRT was required in 21.6%. Patients needing RRT had lower albumin and higher APACHE II/SOFA scores, with markedly higher mortality. CRP/albumin ratio (CAR) was modestly elevated but showed poor discrimination (AUC 0.643 for AKI, 0.575 for RRT). CAR correlated moderately with illness severity (SOFA r=0.442, APACHE II r=0.383) but was not an independent predictor.[10]

As Acute kidney injury (AKI) is prevalent and lethal complication for ICU cases with increased healthcare cost; Even if critical care has gotten better, it's

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still hard to find AKI patients early on. Serum creatinine levels and urine production frequently exhibit delays, perhaps failing to adequately reflect early renal injury, so hindering prompt intervention. pathophysiology of AKI in ICU is contingent upon systemic inflammation, particularly in cases of sepsis, trauma, and multi-organ dysfunction. liver makes C-reactive protein (CRP), which is an acute-phase reactant that is cheap, easy to get, and typically tested in people who are very sick. High levels of CRP mean that there is inflammation, which could harm kidneys. Early identification of individuals at risk may enable timely interventions and reduce morbidity. Establishing this association could improve prognostication and guide clinical decision-making in ICU setting.

METHODS

This was a prospective observational cohort study conducted in Intensive Care Unit of Shifa International Hospital Islamabad. Study duration was four months (1st November 2025 to 28th February 2026) after synopsis approval (wide IRB no. 576-25 Shifa international hospital). Sample size was calculated using WHO Calculator with expected AKI incidence of 55.6%[10], with 95% confidence level and 7% margin of error, and a sample size of 194 patients was calculated. Non-probability consecutive sampling technique was used.

Patients aged ≥16 to 92 years, admitted to ICU requiring monitoring for at least 48 hours, and with CRP measurement available within 12 hours of ICU admission were included. Patients with known chronic kidney disease (eGFR <60 mL/min/1.73 m^2 for >3 months), AKI present at ICU admission, autoimmune diseases, malignancy under active treatment, or severe liver dysfunction that may alter CRP levels, and pregnancy were excluded.

Acute Kidney Injury (AKI) was defined according to KDIGO 2012 criteria as: increase in serum creatinine ≥0.3 mg/dL within 48 hours, or increase to ≥1.5 times baseline within 7 days, or urine output <0.5 mL/kg/hour for ≥6 hours. Assessment was done by consultant intensivist. Development of AKI was defined as presence or absence of AKI during ICU stay. Renal Replacement Therapy (RRT) was defined as requirement of intermittent hemodialysis or continuous renal replacement therapy (CRRT), as decided by nephrology/ICU team based on condition of patient. ICU mortality included death occurring during stay in ICU. Length of ICU stay was recorded as number of days patient stayed in ICU after admission.

Approval was obtained from ethical review board prior to commencement of study. Informed consents were obtained from guardians of patients. Data (age, gender, comorbidities (DM, HTN, COPD, IHD), BMI, smoking, primary diagnosis andrelevant details were documented. On ICU day one, APACHE II and SOFA were done and documented. Within 12 hours of ICU admission, 5 mL venous blood sample from any access point was taken and sent to laboratory of hospital to measure CRP and baseline renal function such as creatinine. Patients followed in ICU stay to look for AKI, RRT, ICU stay, and ICU mortality. All data were recorded on a predesigned questionnaire.

After collection of data, analysis was done on SPSS version 26. Mean± standard deviations or median (interquartile) were calculated for age, baseline CRP levels (mg/L), creatinine (mg/L), APACHE II score, and ICU stay duration in days. Normality was checked by Shapiro Wilk test. Gender, AKI status (yes/no), RRT requirement (yes/no), and mortality (yes/no) were presented as frequencies and percentages. Comparison of baseline CRP levels with AKI status was done using independent t-test or Mann–Whitney U test depending on normality of CRP levels. Predictive accuracy of CRP for AKI, and later ROC curve was plotted. To identify independent predictors of AKI, multivariate logistic regression was performed using CRP, age, sex, comorbidities, and severity scores (APACHE II and SOFA score) as covariates. A p-value ≤0.05 was considered statistically significant.

RESULTS

Total of 194 critically ill ICU patients were enrolled in this study with complete data available for all variables (0% missing). cohort comprised 82 males (42.3%) and 112 females (57.7%), with a median age of 63.0 years (IQR: 42.5–72.0; range 16–92 years). majority of patients were non-smokers (81.4%, n=158), while 36 patients (18.6%) were

active smokers. median APACHE II score at ICU admission was 11.0 (IQR: 7.0–16.0).

Baseline CRP levels demonstrated a wide and right-skewed distribution with a median of 44.2 mg/L (IQR: 12.8–94.0; range 0.48–490.0 mg/L). median ICU length of stay was 4.0 days (IQR: 3.0–6.0). AKI developed in 66 /128 cases (34%). Mechanical ventilation was required in 40 patients (20.6%), and renal replacement therapy (RRT) in 6 patients (3.1%), with 2 patients in each category refusing intervention. ICU mortality was recorded in 14 patients (7.2%), with an overall ICU survival rate of 92.8%. Complete baseline characteristics are presented in Table-I.

Given non-normal distribution of CRP, Mann-Whitney U test was used to compare baseline CRP levels between patients who developed AKI (n=66) and those who did not (n=128). Patients in AKI group had a significantly higher mean rank for baseline CRP (mean rank = 116.52) compared to those without AKI (mean rank = 88.45), with a statistically significant difference (U = 3002.0, Z = −2.326, p = .020). This indicates that higher baseline CRP levels are significantly associated with AKI development during ICU stay. ROC curve analysis was performed to evaluate predictive accuracy of baseline CRP for AKI. area under curve (AUC) was 0.645, indicating acceptable but modest discriminatory ability. An AUC of 0.645 reflects that in 64.5% of randomly selected AKI-non-AKI patient pairs, AKI patient had a higher baseline CRP. While group difference was statistically significant, AUC suggests that baseline CRP has limited accuracy as a standalone diagnostic predictor of AKI in this population. Results are presented in Figure-1. Binary logistic regression using Enter method was performed to identify independent predictors of AKI, with Age, Gender, Smoking status, APACHE II score, and Baseline CRP entered simultaneously as covariates. Full regression results are presented in Table-III. AKI was associated with increased risk of mechanical ventilation, ICU mortality but wasn't associated with increase length of ICU stay as tabulated in Table-IV.

TABLE-I

Baseline demographic and clinical characteristics (n=194)

Variable	Total Cohort (n = 194) Median (IQR) / n (%)
Age (years)	63.0 (42.5 – 72.0)
Gender	
Male	82 (42.3%)
Female	112 (57.7%)
Smoking Status	
Yes	36 (18.6%)
No	158 (81.4%)
APACHE II Score	11.0 (7.0 – 16.0)
Baseline CRP (mg/L)	44.2 (12.8 – 94.0)
Length of ICU Stay (days)	4.0 (3.0 – 6.0)
Acute Kidney Injury (AKI)	
Yes	66 (34.0%)
No	128 (66.0%)
Need for RRT	
Yes	6 (3.1%)
No	186 (95.9%)
Refused	2 (1.0%)
Need for Mechanical Ventilation	
Yes	40 (20.6%)
No	152 (78.4%)
Refused	2 (1.0%)
ICU Mortality	
Yes	14 (7.2%)
No	180 (92.8%)

Continuous variables presented as Median (IQR); categorical variables as n (%). IQR =L Interquartile Range; CRP = C-Reactive Protein; APACHE II = Acute Physiology and Chronic Health Evaluation II; AKI = Acute Kidney Willysy; RRT = Renal Replacement Therapy; MV = Mechanical Ventilation. Refused = intervention declined by patient or family.

TABLE-II

Comparison of baseline CRP between AKI groups and ROC curve analysis (n = 194)

	AKI - Yes (n = 66)	AKI - No (n = 128)	Test Statistic	P-Value
Baseline CRP (mg/L) Mean Rank	116.52	88.45	U = 3002.0 Z = −2.326	.020*

* Statistically significant at p ≤ 0.05. Mann-Whitney U test used due to non-normal distribution of CRP.

TABLE-III

Binary logistic regression — independent predictors of AKI (n = 194)

Variable	B	S.E.	Wald	df	P-Value	OR Exp(B)
Age (years)	0.033	0.016	4.404	1	.036*	1.034
Gender (Male vs. Female)	1.392	0.556	6.262	1	.012*	4.024
Smoking (No vs. Yes)	0.625	0.679	0.846	1	.358	1.868
APACHE II Score	0.094	0.043	4.914	1	.027*	1.099
Baseline CRP (mg/L)	0.006	0.003	4.643	1	.031*	1.006
Constant	−5.508	1.517	13.182	1	< .001	0.004

* Statistically significant at p ≤ 0.05. Method: Enter (forced entry). OR = Odds Ratio = Exp(B). Reference categories: Gender — Female; Smoking — Yes. Model $\chi^2(5) = 55.516$, p < .001. Nagelkerke $R^2 = 0.344$. Hosmer-Lemeshow p = .592 (good fit). Overall classification accuracy = 75.3%; Sensitivity = 57.6%; Specificity = 84.4%.

TABLE-IV

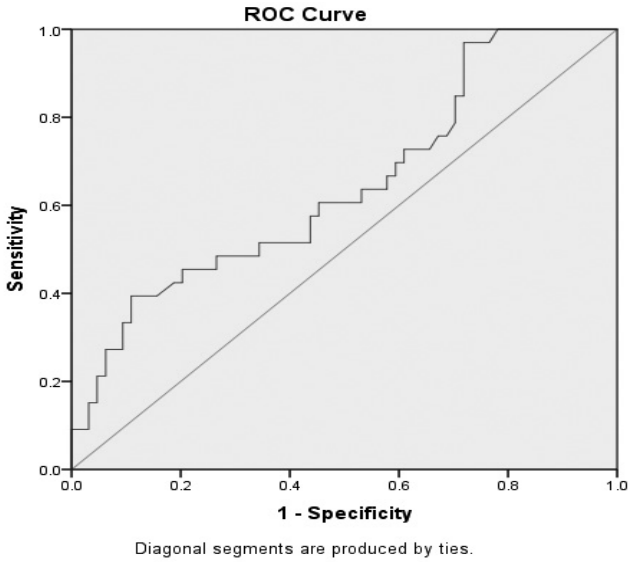
Association of clinical outcomes with AKI status (n = 194)

Outcome Variable	AKI — No (n = 128)	AKI — Yes (n = 66)	Total (n = 194)	Test Statistic	P-Value
Mechanical Ventilation				Pearson $\chi^2 = 5.086$ df = 2	.079
No	108 (84.4%)	44 (66.7%)	152 (78.4%)		
Yes	20 (15.6%)	20 (30.3%)	40 (20.6%)		
Refused	0 (0.0%)	2 (3.0%)	2 (1.0%)		
ICU Mortality				Pearson $\chi^2 = 8.982$ df = 1	.003*
No	126 (98.4%)	54 (81.8%)	180 (92.8%)	Fisher's Exact Test	.006*
Yes	2 (1.6%)	12 (8.2%)	14 (7.2%)		
Length of ICU Stay (days) Median (Mean Rank)	45.34 (mean rank)	56.11 (mean rank)	—	Mann-Whitney U = 821.5 Z = −1.819	.069

* Statistically significant at p ≤ 0.05. Values presented as n (%) for categorical variables and mean rank for continuous variable.

FIGURE-1

ROC of Baseline CRP for AKI



DISCUSSION

AKI occurred in 34.0% of patients, which is comparable to incidence reported in critically ill populations. Our findings are comparable with finding of FINNAKI Study which reported incidence of 39% AKI.[11] In contrast a study by Mo S et al. similarly reported 53.5 % frequency of AKI among ICU patients, which is higher than our findings. Similarly, incidence assessed by KDIGO in ICU patients was 57%.[12,13]

Baseline CRP was significant in AKI cases. This indicates association of systemic inflammation and renal injury. Similar findings were reported by Colak OY et al, Qian J et al., Abdullah MD et al., and Jung CY et al., who demonstrated that elevated CRP levels were associated with an increased risk of AKI in critically ill patients.[10,14,15,16]

ROC analysis in present study demonstrated modest predictive ability of CRP for AKI (AUC = 0.645). Comparable findings were described by Abdullah MD et al., and Liu B et al., who reported moderate predictive performance of CRP for AKI.[14,17]

Multivariable analysis identified age, male gender, APACHE II score, and baseline CRP as independent predictors of AKI. Similar associations between increasing age and AKI risk were reported by Banda J et al.

We found that patients who had AKI had more mortality and need of mechanical ventilation compare to Non AKI patients. These findings are supported by multiple studies which reported increase incidence of mortality and need for mechanical ventilation in AKI patients.[10,14,15,17,18]

ROC AUC of 0.645 indicates modest standalone predictive accuracy of CRP for AKI, and no optimal cut-off value was identified. Additionally, CRP is a non-specific inflammatory marker that may be influenced by underlying comorbidities and active infections, potentially confounding observed association. Finally, post-ICU renal outcomes were not assessed.

Results of current study correspond with expanding evidence highlighting significance of systemic inflammation in etiology of acute kidney injury. CRP, an acute phase reactant, indicates presence of an inflammatory burden that leads to endothelial dysfunction, microvascular injury, and renal tubular damage. In critically ill patients, especially those with sepsis or multi-organ dysfunction, elevated CRP levels may signify a pro-inflammatory environment that predisposes to renal hypoperfusion and ischemic injury. This biological plausibility enhances observed correlation between elevated baseline CRP levels and subsequent onset of AKI. Nevertheless, comparatively low odds ratio for CRP (OR = 1.006) indicates that, although statistically significant, its clinical impact per unit increase is minimal, underscoring notion that AKI is multifactorial and cannot be elucidated by a singular biomarker alone.[14,17,19]

Another important finding from this study is that CRP (AUC = 0.645) doesn't work well as a standalone predictive tool. This finding aligns with existing literature indicating that inflammatory markers, while correlated with AKI, exhibit restricted sensitivity and specificity when utilized in isolation. On other hand, composite scoring systems like APACHE II and SOFA take into account more than one physiological variable and give a better picture of how serious a patient's condition is. Consequently, incorporating CRP into multimarker models or amalgamating it with established scoring systems may enhance predictive accuracy. New studies also show that combining CRP with other biomarkers like procalcitonin, neutrophil gelatinase-associated lipocalin (NGAL), or cystatin C may help find AKI earlier, especially in ICU patients who are at high risk.[16,20,21]

This study's findings demonstrate significant correlation between high C-reactive protein (CRP) levels and start of acute kidney injury (AKI) in ICU patients, underscoring importance of systemic inflammation as critical element in renal impairment during severe illness. These results are consistent with research by Siew ED et al.[23] which demonstrated that inflammatory biomarkers, including CRP, are independently associated with an increased risk of AKI in hospitalized patients. Grams ME and colleagues[24] demonstrated that elevated levels of inflammatory markers correlate with both incidence and severity of acute kidney injury (AKI), hence reinforcing association between inflammation and renal dysfunction. This study extends previous findings to a prospective ICU cohort, emphasizing CRP as a valuable surrogate marker for early inflammatory burden preceding substantial renal failure.

CRP is a less specific but more generally available and affordable option than biomarkers like NGAL and cystatin C. This is especially significant in places where there aren't many resources. Haase M et al.'s research has shown that novel biomarkers are better at finding acute kidney injury (AKI) early on; nevertheless, they can't be used all time because they are too expensive and hard to get. Conversely, findings similar to those provided by Zhang Z suggest that CRP retains moderate predictive value when integrated with clinical indications. This data

supports this position, indicating that while CRP may not replace novel biomarkers, it can serve as a valuable adjunct for early risk stratification. This means that CRP is a useful tool in ICU, especially when more advanced testing aren't possible.

LIMITATIONS

There are some methodological and clinical limitation with this study that need to be recognized. Because this is a single-center study, it may not be able to be used in other ICU settings. sample size, while sufficient, may not completely represent heterogeneity among various critical illness subgroups. CRP was assessed solely at baseline, and sequential measurements were not conducted, which might have yielded enhanced understanding of dynamic inflammatory alterations and their association with AKI progression. Residual confounding remains a possibility, especially from unmeasured variables like fluid balance, nephrotoxic drug exposure, and comprehensive sepsis severity indices. lack of long-term renal outcomes further constrains comprehension of prognostic relevance of CRP beyond duration of ICU admission.

RECOMMENDATIONS

Future research should emphasize multicenter designs with expanded sample sizes to enhance generalizability and external validation. It is important to look into serial monitoring of CRP and use of other new renal biomarkers to make better predictive models for AKI. It is also necessary to determine clinically significant CRP cut-off values that facilitate early risk stratification. Incorporating CRP into composite scoring systems or machine learning-based predictive models may augment its clinical utility. Moreover, interventional studies are advocated to assess whether early identification of high-risk patients via inflammatory markers can facilitate targeted therapies and enhance renal and overall ICU outcomes.

CONCLUSION

Elevated baseline CRP is independently associated with development of AKI in critically ill ICU patients. However, its discriminatory ability is modest (AUC 0.645), suggesting that CRP should be interpreted in conjunction with established severity scores such as APACHE II rather than used as a standalone predictor.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Muhammad Ahmad Ali:** Data collection, analysis, writing.
2	**Aftab Akhtar:** Data collection, writing.
3	**Aayesha Qadeer:** Literature review, data entry.
4	**Sheher Bano:** Data analysis, review of manuscript.
5	**Sidra Bibi:** Critical revisions.
6	**Sidra Nazir:** Discussion writing.