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# Platelet to Albumin Ratio as a Biomarker of Chronic Kidney Disease: A Biochemical and Pathological Perspective

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## ABSTRACT

**Background & objectives:** Chronic kidney disease (CKD) is an escalating global health issue, with a particularly high burden in India due to the widespread prevalence of diabetes, hypertension, and metabolic disorders. In addition to the well-known biomarkers such as serum creatinine, estimated glomerular filtration rate (eGFR), and proteinuria, there is increasing interest in novel, cost-effective markers for early diagnosis, prognosis, and therapeutic management. In this study, we evaluated the platelet-to-albumin ratio (PAR) as a potential biomarker for diagnosing CKD **Methods:** Retrospective data were collected from 120 CKD patients and 90 healthy controls, including clinical, biochemical, and hematological parameters such as serum creatinine, blood urea, uric acid, albumin, CRP, and Complete Blood Count. PAR was calculated and correlated with conventional CKD biomarkers and receiver operating characteristic (ROC) analysis was performed to evaluate its diagnostic value in CKD. **Results:** Our results indicated that PAR was significantly elevated in CKD patients and positively correlated with creatinine and blood urea, while inversely correlated with eGFR. ROC analysis demonstrated that PAR has significant diagnostic accuracy alongside traditional biomarkers. Elevated PAR was also associated with anemia, altered platelet morphology, and hypoproteinemia. Furthermore, PAR effectively reflects inflammation and nutritional status in CKD patients, indicating its strong diagnostic potential. **Interpretation & conclusions:** These findings support the clinical utility of PAR as a reliable, cost-effective biomarker for identifying CKD and assessing its severity, offering improved diagnostic accuracy and aiding comprehensive risk stratification in CKD management.

**Key Words:** Biomarkers, Chronic Kidney Disease, Inflammation, Platelet-to-Albumin Ratio.

## INTRODUCTION

Kidney disease is a significant and growing global health concern, affecting millions of individuals and contributing to substantial morbidity and mortality. Chronic kidney disease (CKD) is characterized by a progressive loss of kidney function over time, potentially leading to end-stage renal disease (ESRD) if left unmanaged. In India, CKD affects approximately 17% of the population, with a higher prevalence in urban areas compared to rural regions [1]. The burden of CKD in India is further exacerbated by the high prevalence of diabetes, hypertension, lifestyle related, and other risk factors that contribute to the development and progression of kidney disease [2, 3]. The growing burden of CKD alongside late-stage presentation in many patients, highlights the urgent need for tools that support early detection and better disease monitoring [4]. While conventional markers, such as serum creatinine, estimated glomerular filtration rate (eGFR), and proteinuria are

widely used, they often detect functional loss only after significant kidney damage has occurred [5]. This has driven a growing interest in the identification of novel, cost-effective, and accessible biomarkers that can improve early diagnosis, prognosis, and personalized management of CKD.

Emerging evidence suggests that systemic inflammation and nutritional status are key contributors to CKD progression [6]. Platelets, primarily known for their role in coagulation, are now recognized as active mediators of inflammation [7]. In CKD, platelet count, and function may be altered due to the uremic environment and the effects of certain medications [8]. Thrombocytopenia, or a low platelet count has been linked to increased bleeding and cardiovascular risk [9]. Conversely, elevated platelet counts may reflect an inflammatory state and contribute to pro-thrombotic conditions [10]. Albumin, a major plasma protein produced by the liver, plays a crucial role in maintaining oncotic pressure and transporting various substances. Low serum albumin levels (hypoalbuminemia) are frequently found in CKD patients and are associated with malnutrition, increased inflammatory burden, and poor clinical outcomes [11]. These interrelated changes in platelet and albumin levels suggest the potential value of composite in dicators that reflect both inflammatory and nutritional status in CKD.

As CKD is driven by chronic inflammation and protein-energy wasting, the platelet-to-albumin ratio (PAR) has emerged as a promising indicator and can be explored as a potential biomarker that reflects both systemic inflammation and nutritional status [12]. Recent studies have demonstrated its predictive value across a range of clinical conditions [13, 14]. In nephrology, PAR has gained attention for its association with diabetic nephropathy and its correlation with complications and disease severity in CKD patients [15, 16]. A recent large-scale study from China further supported its prognostic utility in predicting CKD-related complications and cardiovascular risk, suggesting its relevance in capturing key pathophysiological aspects of the disease [17]. These findings point toward the potential role of PAR in reflecting the chronic inflammatory state that characterizes CKD progression.

Despite these promising findings, evidence supporting the clinical utility of PAR in the Indian CKD population remains limited and underexplored. This gap underscores the need for population-specific validation to assess whether PAR can serve as a practical, cost-effective marker for early detection, risk assessment, and disease monitoring in Indian patients with CKD. In this study, we investigate the variation in PAR among patients with CKD and analyze its correlation with established markers of renal impairment. Additionally, we assess the diagnostic accuracy of PAR in conjunction with traditional biomarkers to evaluate its clinical relevance. Our results suggest that PAR demonstrates a significant correlation with conventional CKD related biomarkers and may serve as a sensitive indicator for CKD diagnosis.

## MATERIAL AND METHODS

A retrospective observational study was conducted at our Tertiary Care Hospital. Medical records of 120 patients diagnosed with CKD and admitted between September 2023, and October 2024 were reviewed. For comparison, a control group of 90 healthy individuals, who underwent routine health examinations at the same institution during the same period, was selected. These individuals had no known kidney disease, inflammatory disorders, or chronic illnesses, and there were no statistically significant differences in age or gender distribution between the two groups.

Demographic clinical and laboratory data, including age, gender, serum creatinine, blood urea, uric acid, serum albumin, C-reactive protein (CRP), and complete blood count (CBC) were collected from electronic medical records. Biochemical tests were performed using an automated analyzer (Vitros 5600, Ortho Clinical Diagnostics, USA), and CBC was analyzed using a hematology analyzer (Sysmex XN-550, Sysmex Corporation, Japan). eGFR was determined using the Chronic Kidney Disease Epidemiology Collaboration equation. The platelet-to-albumin ratio (PAR) was calculated for each subject using the available data. All personal identifiers were removed prior to data analysis, and the requirement for informed consent was waived in accordance with institutional guidelines. The study was conducted following approval from the Institutional Ethics Committee.

### Inclusion & exclusion criteria

The diagnosis of CKD was based on the KDIGO 2024 (Kidney Disease: Improving Global Outcomes) guidelines, which define CKD as abnormalities in kidney structure or function that persist for more than three months and have health implications [18]. In this study, CKD was confirmed in patients with a reduction in eGFR below 60 mL/min/1.73 m² for a minimum of three months. Only patients with complete medical records that included all necessary biochemical and hematological parameters were considered eligible for inclusion. The exclusion criteria included individuals under the age of 18 years, those with eGFR values greater than 60 mL/min/1.73 m², pregnant individuals, patients who had undergone major surgery within the preceding three months, those with cognitive impairments that could interfere with accurate medical documentation, and individuals with documented non-adherence to treatment protocols. Additionally, patients with incomplete data on key variables relevant to the study's objectives were excluded. These criteria were applied to ensure the selection of a well-defined and clinically relevant study population while minimizing confounding factors that could influence systemic inflammation, nutritional status, or renal function.

### Statistical analysis

Statistical analyses were performed using Microsoft Excel, GraphPad Prism, and SPSS software. Continuous variables were expressed as mean ± SD or mean ± SE, as appropriate, and compared using Student's t-test. Pearson's correlation was used to assess associations between PAR and clinical markers. Diagnostic performance was evaluated using receiver operating characteristic (ROC) curve analysis, with area under the curve (AUC) values calculated and optimal cut-off points determined by the Youden index. eGFR was included in the ROC analysis to serve as a performance benchmark, despite being used for CKD classification, to enable comparative evaluation of other biomarkers including PAR. Box plots and scatter plots were used for data visualization. A p-value $< 0.05$ was considered statistically significant.

**RESULTS**

**Participant Characteristics:** A total of 120 patients diagnosed with CKD and 90 healthy individuals were included in this study. The baseline demographic characteristics of both groups are presented in **Table I**. The mean age of the CKD group was 51 ± 12 years, and there were no statistically significant differences in age or gender distribution between the CKD and control groups, indicating appropriate matching (**Figure 1A, 1B**).

**PAR Levels Are Elevated in CKD and Independent of Gender:** PAR was significantly elevated in the CKD group compared to healthy controls (**Figure 1C**). No significant difference in PAR levels was observed between male and female participants within the CKD cohort, indicating that PAR elevation was independent of gender (**Figure 1D**).

**Renal Function Biomarkers Differ Significantly in CKD:** Traditional kidney function biomarkers, including serum creatinine and blood urea, were significantly higher in CKD patients compared to controls, while the eGFR was significantly lower in the CKD group (**Figures 2A, 2B, and 2C**).

**Comparative Analysis of Laboratory Parameters:** To explore the clinical and biochemical differences between CKD patients and healthy controls, a comparative analysis was performed. **Table I** summarizes key laboratory differences, with significant alterations observed in renal function markers, uric acid, PAR, albumin, and inflammatory indicators, reflecting impaired kidney function, systemic inflammation, and nutritional imbalance in CKD.

**PAR Correlates with Renal Dysfunction and Inflammatory Markers:** Correlation analysis revealed that PAR was positively correlated with serum creatinine ($r = 0.424$) and blood urea ($r = 0.317$) and negatively correlated with eGFR ($r = -0.504$), suggesting that higher PAR levels are associated with worsening renal function (**Figures 3A, 3B, and 3C**). Furthermore, as PAR values increased in CKD patients, a significant positive correlation was observed with platelet count ($r = 0.811$), whereas albumin levels were negatively correlated with PAR ($r = –0.614$), (**Figures 3D, 3E**) as shown in **Table II**. These associations indicate that PAR is linked to inflammation and nutritional status, providing complementary prognostic information beyond traditional markers for evaluating and assessing risk in CKD patients.

**Predictive and Diagnostic Performance of PAR in Identifying CKD:** To assess the predictive utility of PAR in CKD diagnosis, receiver operating characteristic (ROC) curve analysis was performed alongside traditional renal biomarkers. The area under the curve (AUC) demonstrated high discriminative ability for urea and eGFR (AUC = 0.992), creatinine (AUC = 0.987), and PAR (AUC = 0.925), highlighting the robust diagnostic performance of all four parameters (**Figures 4A, 4B, and Table III**). The optimal cutoff value for PAR, determined by the Youden index, was $\geq 5.13$, yielding a sensitivity of 81.0% and specificity of 96.7%. These findings underscore the clinical relevance of PAR as a reliable and non-invasive marker with strong sensitivity and specificity for distinguishing CKD patients from healthy individuals. Moreover, PAR-associated ROC analysis also indicated strong predictive potential for identifying underlying inflammation and hypertension, supporting its broader application in the diagnostic and prognostic evaluation of CKD.

**Assessment of Platelet and Albumin Changes in CKD:** To further investigate the factors contributing to elevated PAR in CKD patients compared to controls, individual components of the ratio were examined. Although the mean platelet count in the CKD group was numerically higher than in healthy controls, and the difference was statistically significant, the values remained within the reference range (1.5 to 4.5 Lakh/cu mm) (**Figure 5A**). However, Mean Platelet Volume (MPV) was significantly increased in CKD patients. (**Figure 5B**), and peripheral smear analysis revealed altered platelet morphology in the CKD group compared to healthy individuals (**Figure 5C**), suggesting changes in platelet activation or turnover. In contrast, serum albumin levels were significantly lower in CKD patients than in controls, accompanied by a corresponding decrease in total serum protein levels (**Figures 5D, 5E**). These findings indicate a state of hypoalbuminemia and potential protein-energy malnutrition in the CKD group. Additionally, inflammatory markers were elevated in CKD patients, as evidenced by significantly higher levels of C-reactive protein (CRP) and neutrophil-to-lymphocyte ratio (NLR) compared to healthy controls (**Figures 5F, 5G**). Both parameters are shown on a logarithmic scale to improve visualization of intergroup differences. These results further support the presence of systemic inflammation and compromised nutritional status in CKD patients, both of which are reflected in elevated PAR values.

Figure 1:

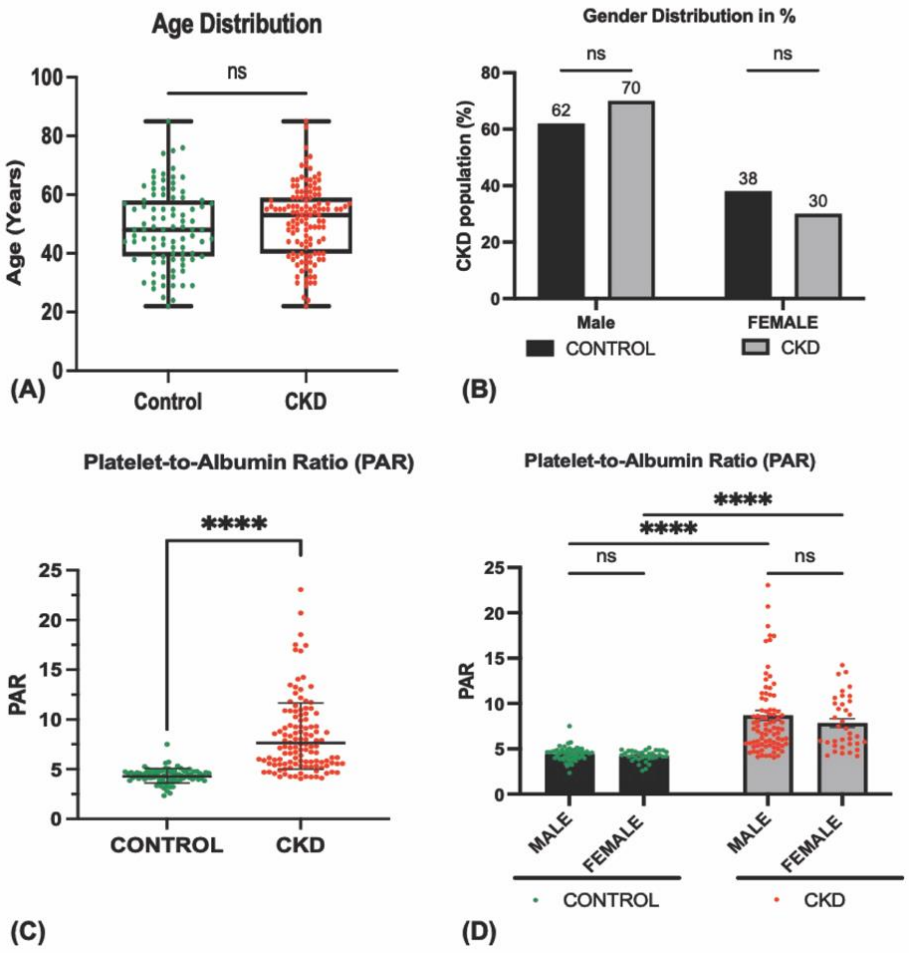


**Figure 1: PAR levels are significantly elevated in CKD patients and independent of gender: (A)** Box plot illustrating age distribution between CKD and control groups, with comparable median ages and no significant difference, confirming appropriate age matching. **(B)** Bar chart showing gender distribution in CKD patients and healthy controls, indicating no statistically significant difference between groups. **(C)** Comparison of platelet-to-albumin ratio (PAR) levels between CKD patients and healthy controls. PAR was significantly elevated in the CKD group, reflecting disease-associated alterations. **(D)** Bar graph showing PAR levels in male and female participants from both CKD and control groups, indicating no significant differences based on gender in either group. Values represent the mean ± SE. P values were determined by Student's t-test. **** P<0.0001, ns = not significant.

Figure 2

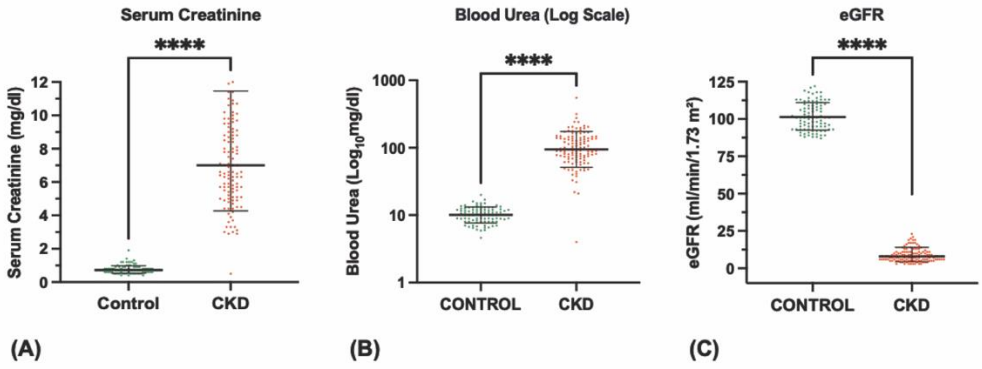


**Figure 2. Renal function biomarkers are significantly altered in CKD patients compared to healthy controls. (A)** Serum creatinine levels are significantly elevated in CKD patients. **(B)** Blood urea levels are markedly higher in CKD patients. **(C)** The estimated glomerular filtration rate (eGFR) is significantly lower in the CKD group. Data are presented as scatter dot plots with mean ± SE. Asterisks indicate statistical significance. Values represent the mean ± SE. P values were determined by Student's t-test. **** P<0.0001.

**Table I:** Baseline demographic characteristics and laboratory parameters between healthy controls and CKD patients.

| Variable | | Control (n=90) | CKD (n=120) | *p*-value |
|---|---|---|---|---|
| Age (years) | | 48.57±13.2 | 51.03±12.3 | ns |
| Gender (%) | Male | 62 | 70 | ns |
| | Female | 38 | 30 | ns |
| Systolic Blood Pressure (mm Hg) | | 114±8.4 | 146±23.6 | $2.46 \times 10^{-31}$ |
| Diastolic Blood Pressure (mm Hg) | | 74.38±8.3 | 86.20±13.3 | $4.55 \times 10^{-14}$ |
| Uric Acid (mg/dl) | | 4.43±0.8 | 7.40±2.6 | $2.18 \times 10^{-21}$ |
| Urea (mg/dl) | | 10.43±2.8 | 111.15±67.9 | $4.69 \times 10^{-53}$ |
| Serum Creatinine (mg/dl) | | 0.76±0.3 | 7.76±3.5 | $1.18 \times 10^{-53}$ |
| Estimated Glomerular filtration rate (ml/min/1.73 $m^2$) | | 101.78±9.3 | 8.82±4.5 | $5.39 \times 10^{-62}$ |
| C-Reactive Protein (mg/L) | | 1.71±0.7 | 69.76±60 | $7.6 \times 10^{-26}$ |
| Albumin(g/dl) | | 4.40±0.4 | 2.93±0.5 | $1.63 \times 10^{-56}$ |
| Total Protein (g/dl) | | 7.31±0.6 | 6.14±0.8 | $3.08 \times 10^{-24}$ |
| Platelet Count (Lakh/ cu mm) | | 1.90±0.3 | 2.42±0.9 | 0.0003 |
| Mean Platelet Volume (fL) | | 7.37±0.3 | 10.07±1.2 | $6.31 \times 10^{-22}$ |
| Red Blood Cells Count (million/ cu mm) | | 4.53±0.83 | 2.92±0.6 | $1.12 \times 10^{-35}$ |
| White Blood Cells Count (cu mm) | | 6203±1787 | 10790±6642 | $6.17 \times 10^{-15}$ |
| Lymphocytes Count (%) | | 29.95±5.14 | 15.28±8.93 | $4.76 \times 10^{-29}$ |
| Neutrophil Count (%) | | 60.85±6.1 | 74.84 ±11.4 | $6.39 \times 10^{-22}$ |
| Neutrophil-to-Lymphocyte Ratio | | 2.1±0.4 | 9.62±7.8 | $1.08 \times 10^{-61}$ |
| Platelet-to-Albumin Ratio | | 4.34±0.7 | 8.44±4.5 | $1.62 \times 10^{-32}$ |

**Figure 3**

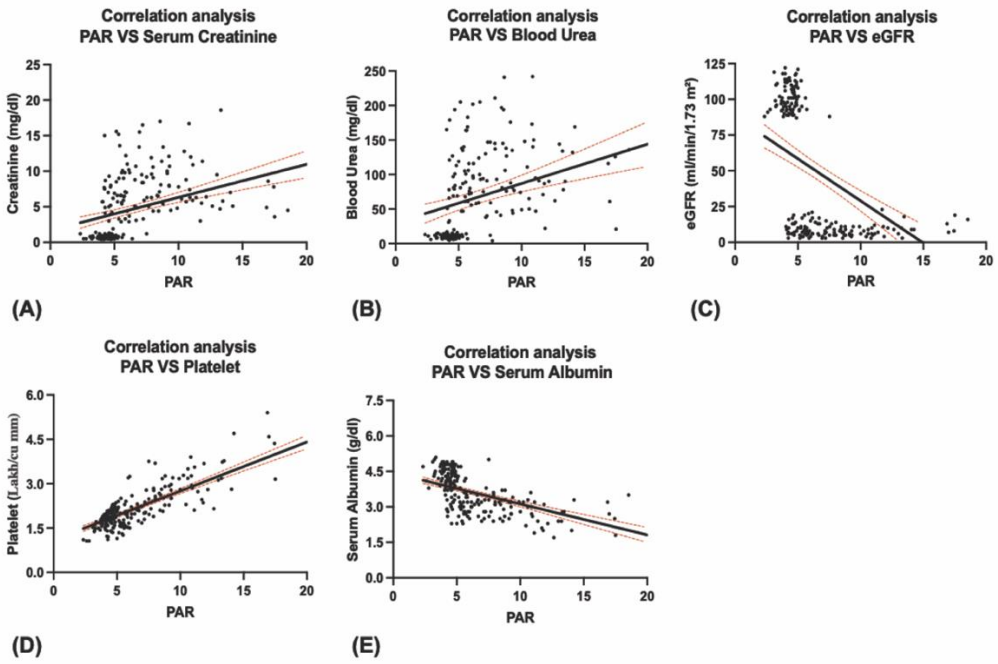


**Figure 3. PAR shows significant correlations with renal function markers in CKD patients. (A)** PAR is positively correlated with serum creatinine (r = 0.424), and **(B)** blood urea (r = 0.317) indicating that higher PAR values are associated with impaired renal function. In contrast, (C) PAR is negatively correlated with the estimated glomerular filtration rate (eGFR) (r = – 0.504) supporting its inverse relationship with kidney function. **(D)** PAR shows a strong positive correlation with platelet count (r = 0.811), and **(E)** a significant negative correlation with serum albumin levels (r = –0.614), reflecting associations with inflammation and nutritional status. Data are shown as scatter plots with trend lines and all correlations were statistically significant. **** P<0.0001.

**Table II:** Correlation of PAR with renal function and nutritional markers in CKD patients.

| Variable (n=210) | Platelet-to-Albumin ratio (PAR) | | |
|---|---|---|---|
| | r | 95% CI | *p*-value |
| **Urea** | **0.317** | **0.19 to 0.43** | **$2.73 \times 10^{-6}$** |
| **Creatinine** | **0.424** | **0.31 to 0.53** | **$1.35 \times 10^{-10}$** |
| **eGFR** | **- 0.504** | **-0.76 to 0.85** | **$5.70 \times 10^{-15}$** |
| **Platelet** | **0.811** | **0.76 to 0.85** | **$1.43 \times 10^{-50}$** |
| **Albumin** | **- 0.614** | **-0.69to -0.52** | **$3.12 \times 10^{-23}$** |

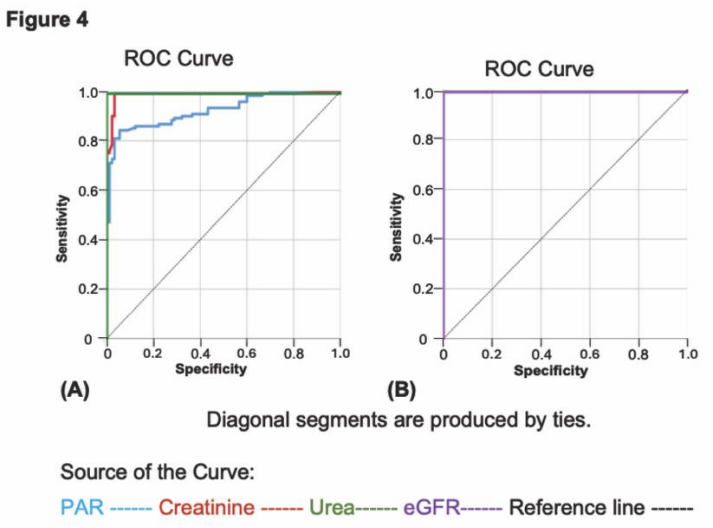


**Figure 4. Receiver operating characteristic (ROC) curve analysis showing the diagnostic performance of PAR and conventional renal biomarkers in CKD. (A)** ROC curves for PAR, serum urea, and creatinine. **(B)** ROC curve for eGFR. The area under the curve (AUC) for PAR, urea, creatinine, and eGFR was 0.925, 0.992, 0.987, and 0.992, respectively, indicating the strong discriminative ability of PAR, comparable to traditional renal biomarkers.

**Table III:** Diagnostic performance of PAR and conventional biomarkers in identifying CKD using ROC curve analysis.

| Measures | CKD Biomarkers | | | |
|---|---|---|---|---|
| | PAR | CREATININE | UREA | eGFR |
| Sensitivity | 0.810 | 0.992 | 0.934 | 0.991 |
| Specificity | 0.967 | 0.967 | 1.000 | 1.000 |
| PPV | 0.970 | 0.976 | 1.000 | 1.000 |
| NPV | 0.791 | 0.989 | 0.909 | 0.989 |
| Accuracy | 0.878 | 0.981 | 0.958 | 0.995 |
| AUC | 0.925 | 0.987 | 0.992 | 0.992 |
| P-value | $4.89 \times 10^{-26}$ | $1.35 \times 10^{-33}$ | $2.79 \times 10^{-34}$ | $2.79 \times 10^{-34}$ |
| Cutoff value | $\geq 5.13$ | $\geq 2.40$ | $\geq 43.00$ | $\leq 60.00$ |
| PAR= Platelet-to-Albumin ratio, PPV= Positive Predictive Value, NPV= Negative Predictive Value, AUC = Area Under the Curve | | | | |

Figure 5:

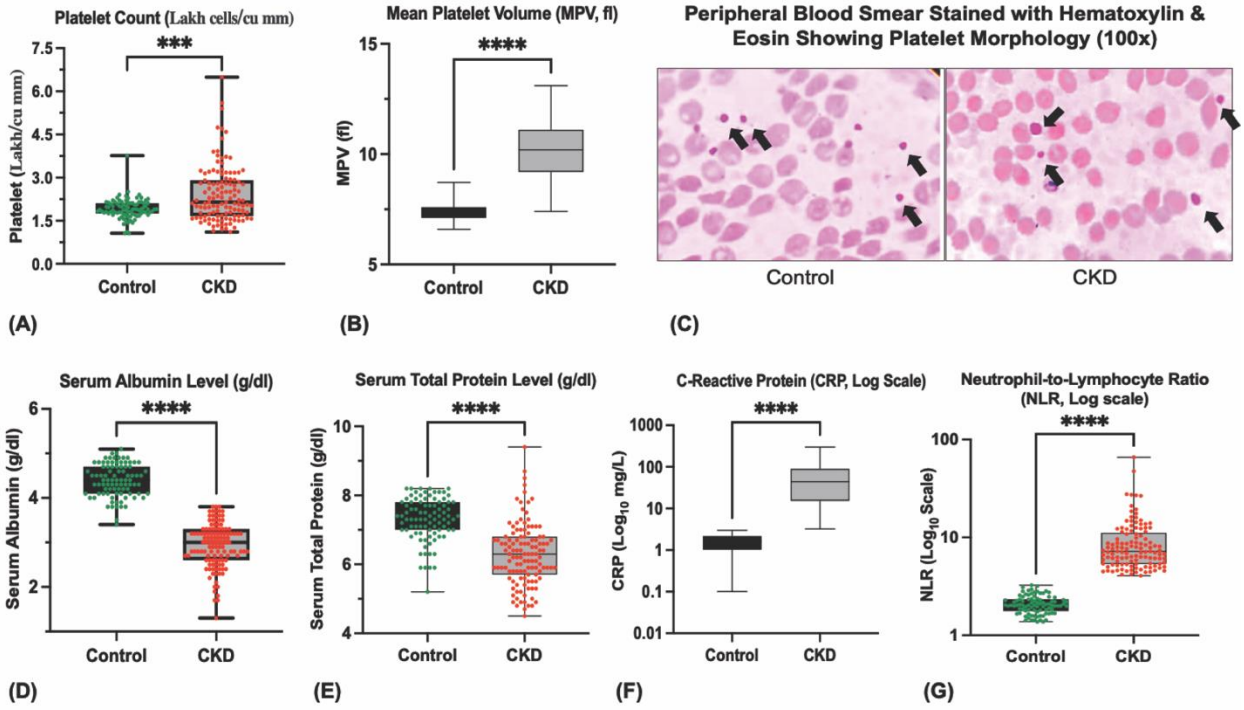


**Figure 5. Alterations in platelet parameters, albumin, and inflammatory markers in CKD patients. (A)** Platelet count was significantly higher in CKD patients compared to healthy controls. **(B)** Mean Platelet Volume (MPV) was markedly elevated in CKD patients, suggesting increased platelet activation (measured in femtoliters, fL). **(C)** Representative hematoxylin and eosin–stained peripheral blood smear images show altered platelet morphology in CKD patients (right panel) compared to healthy individuals (left panel); black arrows indicate platelets. **(D)** Serum albumin and **(E)** total protein levels were significantly lower in the CKD group, reflecting hypoalbuminemia and potential protein-energy malnutrition. **(F)** C-reactive protein (CRP) and **(G)** neutrophil-to-lymphocyte ratio (NLR) were significantly elevated in CKD patients, supporting the presence of systemic inflammation. Both markers are displayed on a logarithmic scale for better visualization of intergroup differences. Data are shown as box plots. Statistical comparisons were performed using two-tailed independent-sample t-tests. Values are presented as mean ± SE. P values were calculated using Student's t-test; ***P < 0.001 and ****P < 0.0001 were statistically significant.

## DISCUSSION

CKD presents a complex interplay of systemic inflammation, nutritional depletion, and metabolic dysregulation that often escapes capture by conventional renal biomarkers [16]. While serum creatinine, blood urea, and eGFR remain the clinical standards for CKD diagnosis, their diagnostic and prognostic precision diminishes, especially in advanced stages [17]. In this study, we propose PAR as an adjunct biomarker that integrates hematologic and biochemical parameters to provide a broader systemic perspective of renal dysfunction.

Our data demonstrate a significant elevation in PAR levels among CKD patients compared to healthy individuals. PAR correlated positively with serum creatinine and blood urea and inversely with eGFR, indicating that it rises along with disease severity. These associations were statistically confirmed by correlation coefficients, indicating that PAR tracks closely with conventional renal indices. In ROC analysis, PAR demonstrated a high diagnostic yield with 81.0% sensitivity and 96.7% specificity underscoring its potential utility as a diagnostic biomarker.

The pathophysiological rationale for PAR as a biomarker lies in the dual relevance of its constituents. Platelets, traditionally associated with hemostasis, are increasingly recognized as active mediators of chronic inflammation and vascular injury [18, 19, and 20]. Our study observed significantly elevated platelet counts and mean platelet volumes (MPV) in CKD patients compared to healthy controls, indicating enhanced platelet turnover and activation. These findings align with earlier reports suggesting that in the uremic environment of CKD, platelet count, and morphology are altered due to systemic inflammation and endothelial dysfunction like conditions [21, 22]. Morphological examination of peripheral smears further revealed distinct abnormalities, including platelet anisocytosis, the presence of giant platelets, and platelet clumping, consistent with altered megakaryopoiesis and heightened platelet reactivity. The structural and quantitative platelet changes in the CKD cohort reinforce the role of platelet dysfunction in renal pathology. Concurrently, serum albumin levels were markedly decreased in CKD patients, reflecting the well-established effects of systemic inflammation, proteinuria, and malnutrition on hepatic protein synthesis and vascular permeability. Previous evidence indicates that hypoalbuminemia in CKD is not merely a consequence of urinary protein loss but also reflects broader disruptions in

nutritional and inflammatory homeostasis, including impaired hepatic protein synthesis [23, 24, and 25]. These opposite changes, higher platelet levels, and lower albumin together reflect both inflammation and poor nutritional status, which PAR brings together in a single measure. The strength of this relationship is underscored by our correlation analy sis, which demonstrated significant associations between PAR and key renal function markers, supporting its utility as a reliable, integrative biomarker for disease assessment.

To explore whether the observed hypoalbuminemia in CKD patients was influenced by systemic inflammation in addition to protein loss, we assessed CRP levels as a representative positive acute-phase reactant. The elevated CRP levels observed in our cohort support the presence of an inflammatory state, indicating that reduced serum albumin may be driven not only by urinary losses but also by inflammation-associated suppression of hepatic protein synthesis [26]. NLR, a derivative of the complete blood count, reflects neutrophil-driven inflammation and relative lymphopenia and has been proposed as a prognostic marker in cardiovascular and renal diseases [27, 28].

These findings highlight the value of PAR as a composite marker reflecting both inflammatory burden and nutritional status, thereby complementing traditional renal function indices. This was further supported by its strong diagnostic performance in ROC analysis. Importantly, PAR's predictive power remained consistent across gender subgroups, supporting its broader applicability. Given that PAR is derived from routine hematological and biochemical tests, it offers a low-cost, scalable option for CKD screening and monitoring, especially in clinical settings with high disease burden and limited access to specialist care.

While the results are promising, some limitations remain to be addressed. This was a retrospective single-center study with a modest sample size. Since most participants had eGFR values below 30 mL/min/1.73 m², the study population primarily represented stage 4 and stage 5 CKD, limiting insights into earlier disease stages. Future studies that evaluate PAR dynamics across all CKD stages may offer more information on its early diagnostic potential. Additionally, longitudinal data assessing PAR changes post-treatment and its association with clinical outcomes such as disease progression or cardiovascular events were not available. Nonetheless, our findings are consistent with prior studies. For example, Tan *et al.* (2022) demonstrated that elevated PAR levels predicted adverse outcomes and histopathological damage in IgA nephropathy [29]. Another study reported increased PAR levels in CKD patients with cardiovascular comorbidities, emphasizing its dual relevance to renal and vascular pathology [30]. Importantly, there remains a dearth of PAR-related studies in the Indian population and our findings thus contribute valuable population-specific data to the existing literature.

Incorporating PAR into clinical practice may enhance current diagnostic frameworks, particularly in advanced CKD stages where traditional markers plateau. Given its simplicity, reproducibility, and systemic relevance, PAR may serve as a valuable adjunct biomarker in the evolving landscape of nephrology. By capturing inflammatory burden and nutritional depletion, PAR adds a novel and clinically meaningful dimension to CKD evaluation.

**Conclusion**

In conclusion, our findings support the utility of PAR as a simple, cost-effective, and clinically informative biomarker in CKD. Its strong correlation with traditional renal parameters and its ability to reflect systemic pathophysiology suggests that PAR can augment diagnostic precision, aid in risk stratification, and potentially enhance prognostication when used alongside established markers. While further multicentric and prospective studies are warranted, this study provides foundational evidence for the integration of PAR into clinical nephrology, especially in populations with high disease burden and limited access to advanced diagnostic resources.

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