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Original Research Article

CORRELATION BETWEEN C-REACTIVEPROTEIN ALBUMIN RATIO AND ANKLEBRACHIAL INDEX IN CKD PATIENTS:ACROSS-SECTIONAL STUDY

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ABSTRACT

Background: Chronic Kidney Disease (CKD) is linked to high cardiovascular morbidity, with peripheral arterial disease (PAD) being common. The C-reactive protein/albumin ratio (CAR) integrates inflammatory and nutritional status, while the ankle-brachial index (ABI) is a non-invasive PAD marker.
Materials and Methods: This cross-sectional hospital-based study included 150 CKD patients at AGMC & GBP Hospital (Jan 2023–Jun 2024). Serum CRP (latex agglutination), albumin (BCG method), and creatinine (enzymatic method) were measured. CAR was calculated and ABI assessed. Data were analysed using SPSS; $p<0.05$ was significant.
Results: PAD (ABI<0.9) was present in 57.33% of patients. CRP and CAR both showed strong negative correlations with ABI ($r = –0.90$ and $–0.92$, $p<0.001$). Higher CAR was associated with greater arterial disease severity.
Conclusion: CAR is strongly and inversely correlated with ABI in CKD, suggesting its utility as a simple, cost-effective tool for cardiovascular risk assessment.
Keywords: Chronic kidney disease, C-reactive protein/albumin ratio, ankle-brachial index, inflammation, peripheral arterial disease.

INTRODUCTION

Chronic Kidney Disease (CKD) is a major non-communicable condition and ranks among the leading causes of death globally. It disproportionately affects older adults, women, racial and ethnic minorities, and individuals with diabetes mellitus or hypertension.[1] CKD significantly elevates the risk of cardiovascular complications and progression to end-stage kidney disease (ESKD).[2] In India, kidney disease stands as a leading cause of mortality.[3] Research from Europe, Australia, and Asia further highlights the widespread nature of CKD. Since 2006, World Kidney Day has been observed annually on the second Thursday of March. This global health initiative aims to raise awareness and emphasize to the public, policymakers, healthcare providers, patients, and their families that CKD is not only common and burdensome but also manageable and treatable.[4]

CKD leads not only to kidney failure but also to a range of serious complications, including a heightened risk of cardiovascular disease (CVD), acute kidney injury, anaemia, mineral and bone disorders—leading to fractures—and cognitive decline.[5] Notably, individuals with CKD are far more likely to die from cardiovascular complications than to progress to end-stage kidney disease (ESKD).[6] Because the early stages of CKD are typically silent, routine screening is essential for early detection. Identifying CKD in seemingly healthy individuals allows for timely intervention to prevent disease progression, minimize further kidney damage, and address associated cardiovascular risks. Emerging anecdotal and clinical evidence indicates that CKD can be diagnosed through simple laboratory tests.[7] Moreover, certain treatments have shown promise in slowing disease progression, preventing complications, and reducing cardiovascular risk.

To make a meaningful global impact, these medical advances must be translated into accessible and practical strategies for public health implementation. Among diagnostic tools, estimated Glomerular Filtration Rate (eGFR) remains the cornerstone for CKD detection. However, eGFR is not routinely

measured in individuals without symptoms,[8] and the equations currently used—originally developed for Western populations—may not be entirely applicable to the Indian demographic.[9] Consequently, there is an ongoing effort to identify alternative routine biomarkers for early detection and disease monitoring.[9] One such overlooked marker is serum albumin. Studies have demonstrated that even a slight reduction in serum albumin levels is strongly correlated with increased risks of cardiovascular disease, heart failure, and mortality—particularly in vulnerable groups such as the elderly and individuals living with HIV.[10]

Albumin is the most abundant plasma protein from mid-gestation through the lifespan. As a negative acute-phase protein (APP), its levels decrease in response to both acute and chronic inflammation, making hypoalbuminemia a common marker of inflammatory states.[11] While relatively few studies have specifically examined the relationship between low serum albumin and kidney function decline, existing evidence suggests a significant association. For instance, findings from the Cardiovascular Health Study revealed that lower serum albumin levels were independently linked to a greater risk of declining kidney function, whereas seven other inflammatory markers showed no such association.[12] Some studies have further reported that hypoalbuminemia correlates with an accelerated decline in glomerular filtration rate (GFR).[13]

C-reactive protein (CRP) is an acute-phase reactant found in the serum of individuals experiencing acute illness, notable for its ability to bind the C-polysaccharide component of the Streptococcus pneumoniae cell wall. As an early and highly responsive biomarker of inflammation, CRP shows significant elevations during acute inflammatory states.[11] Elevated CRP levels have also been strongly linked to an increased risk of cardiovascular disease (CVD) in older adults, with particularly robust associations observed in cases of myocardial infarction (MI).[14] Immunohistochemical studies using both polyclonal and monoclonal anti-human CRP antibodies have demonstrated the presence of CRP in necrotic regions of human atherosclerotic plaques, while it is notably absent in non-necrotic arterial tissue. The degree of CRP immunoreactivity has been found to correlate positively with intimal thickening and inversely with lumen diameter, suggesting that inflammatory mechanisms may underlie arterial remodelling and narrowing.[15] This is particularly relevant in individuals with advanced renal insufficiency, where atherosclerotic cardiovascular disease represents the leading cause of morbidity and mortality. These patients often exhibit profound metabolic disturbances, including uraemia, dyslipidaemia, anaemia, and acid-base imbalances.[16] CRP may serve as a marker for identifying patients with end-stage renal failure (ESRF) who are at higher risk of hospitalization.[17] Inflammatory responses observed during haemodialysis could be triggered by factors such as the use of bioincompatible dialysis membranes, residual chemicals in the tubing and solutions, or the presence of comorbid conditions, including both occult and overt infections.[18]

Several studies have shown that CRP is correlated with low serum albumin levels, which serves as both a negative inflammatory marker and an indicator of visceral protein nutrition.[19] This relationship has been observed in both haemodialysis and peritoneal dialysis patients.[20] Utilizing a ratio of CRP to albumin could create a composite marker that integrates the information provided by both biomarkers, offering an index that correlates with disease severity. A higher CRP/albumin ratio (CAR) would indicate a more pronounced inflammatory state. By combining these two markers into a single parameter, the CRP/albumin ratio could prove useful for monitoring disease activity, tracking progression, and guiding treatment decisions. One study demonstrated that an elevated CAR is associated with increased mortality in Intensive Care Unit (ICU) patients. [21,22] Another study done in China concluded that a higher CAR was an independent risk factor for CKD. The ability of the CAR to predict CKD was better than that of CRP or albumin alone. The CAR provides an important reference index for predicting the risk of CKD.[23]

Many countries maintain registries for patients undergoing dialysis or transplantation; however, these programs often overlook individuals with advanced chronic kidney disease (CKD) who die before reaching kidney failure, or those who, despite the onset of kidney failure, do not receive dialysis or a transplant.[24] Ideally, a surveillance system targeting the early stages of CKD would allow countries to better monitor the size and care needs of this high-risk, high-cost population. Such a system could potentially help reduce both the progression to end-stage kidney failure and the associated costs of dialysis and transplantation.[25]

MATERIALS AND METHODS

This hospital-based observational study was conducted in the Department of Biochemistry in collaboration with the Department of Medicine at AGMC and GBP Hospital, spanning the period from January 2023 to June 2024. The study followed a cross-sectional design and included a total of 150 patients diagnosed with Chronic Kidney Disease (CKD).

Participants were selected based on specific inclusion and exclusion criteria. Inclusion criteria comprised patients diagnosed with CKD as per the National Kidney Foundation definition, individuals who provided written informed consent to participate, and those aged 18 years and above. Exclusion criteria involved patients unwilling to participate, as well as those with nephrotic syndrome, gastrointestinal diseases, severe organ failure, those undergoing steroid therapy, or suffering from active infections or

acute vasculitis. Additionally, patients with a history of substance abuse were also excluded from the study.

Serum albumin levels were estimated using the Bromo Cresol Green (BCG) method with the XL-640 Fully Automated Autoanalyzer. C-Reactive Protein (CRP) was measured using the Latex Slide Agglutination Test. The CRP titre was calculated using the formula: CRP (µg/ml) = 7 × D, where D represents the highest serum dilution showing visible agglutination, and 7 µg/ml is the sensitivity of the test.

Serum creatinine was estimated using the enzymatic method on the XL-640 Fully Automated Autoanalyzer. The estimated Glomerular Filtration Rate (e-GFR) was calculated using the CKD-EPI 2021 updated formula:

The CKD-EPI 2021 equation is: eGFR = 142 x min(standardized Scr/K,1) $^{\alpha}$ * max(standardized Scr/K,1) $^{-1.200}$ * 0.9938 $^{\text{age in years}}$ * 1.012 [if female].

Data entry was performed using Microsoft Excel, and statistical analysis was carried out using SPSS software on a Windows-based PC. Categorical data were represented through text, tables, and charts. A p-value of less than 0.05 was considered statistically significant.

Rationalization & Objectives: This study investigated the correlation between the CRP/Albumin Ratio (CAR) and Ankle Brachial Index (ABI) in chronic kidney disease (CKD) patients, given the significant cardiovascular risk in this population. Both CRP, an inflammatory marker, and low albumin levels are associated with poor prognosis and increased mortality in CKD patients, while ABI is a key indicator of peripheral arterial disease (PAD), a common comorbidity in CKD. By combining the information from CAR and ABI, this study seeks to explore whether CAR can serve as a comprehensive marker of inflammation and vascular health, providing an additional tool for assessing cardiovascular risk and guiding treatment in CKD patients.

This study was done to evaluate the levels of serum C-reactive protein (CRP) and serum albumin in Chronic Kidney Disease (CKD) patients at Agartala Government Medical College and Govind Ballabh Pant Hospital. It had two key focuses: first, to investigate the relationship between serum CRP, serum albumin, and peripheral artery disease in CKD patients; and second, to assess the correlation between the CRP/Albumin Ratio and Ankle Brachial Index (ABI) within this population.

RESULTS

The table classifies CKD participants based on their Ankle Brachial Index (ABI). Among the 150 participants, 4.67% (7 patients) have a normal ABI, indicating no detectable arterial disease. 38% (57 patients) have an acceptable ABI, suggesting mild arterial disease or stiffness. 28% (42 patients) show some arterial disease, with observable abnormalities. A group, 29.33% (44 patients), have moderate arterial disease, indicating significant arterial blockages or severe issues.

Table 1: Prevalence of CKD patients in each ABI class

ABI Class	Range	Number of CKD pt.	Percentage (%)
Calcification	>1.4	0	0 %
Normal	1.0-1.4	07	4.67%
Acceptable	0.9-1.0	57	38.0%
Some Arterial Disease	0.8-0.9	42	28.0%
Moderate Arterial Disease	0.5-0.8	44	29.33%
Severe Arterial Disease	<0.5	0	0%

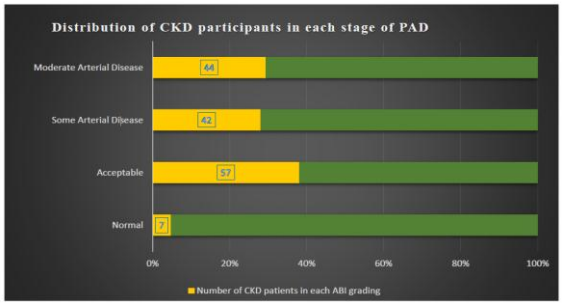

Figure 1: Distribution of CKD participants in each stage

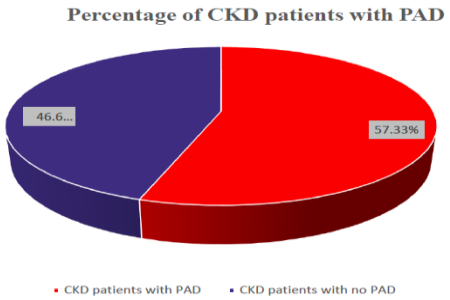

Figure 2: Percentage of CKD patients with PAD

The bar diagram visualizes the distribution of ABI classifications in the CKD study population, with categories on the vertical axis and participant percentages represented by yellow bars on the x-axis. It provides a clear overview of the prevalence of varying levels of arterial disease among the participants.

The pie chart shows that 57.33% of the 150 CKD participants have Peripheral Artery Disease (PAD), as indicated by their Ankle Brachial Index (ABI) values. PAD, a common complication in CKD, is linked to factors like inflammation, atherosclerosis, and vascular calcification, leading to reduced blood flow to the limbs.

Table 2: Distribution of Serum CRP ranges in each class of ABI

ABI Class	Serum CRP (mg/dl)						Total	
	(1.0-3.0) mg/dl		(3.1-10) mg/dl		>10 mg/dl			
	No.	%	No.	%	No.	%	No.	%
Normal	7	4.67%	0	0%	0	0%	7	4.67%
Acceptable	57	38%	0	0%	0	0%	57	38%
Some Arterial Disease	10	6.67%	32	21.33%	0	0%	42	28%
Moderate Arterial Disease	0	0%	6	4%	38	25.33%	44	29.33%
Total	74	49.34%	38	25.33%	38	25.33%	150	100%

In CKD patients, higher CRP levels are linked to greater arterial disease severity. With CRP levels of 1-3 mg/dl, most patients have acceptable ABI, indicating moderate arterial health, while some show early signs of arterial disease. At CRP levels of 3.1-10 mg/dl, more patients develop early or moderate arterial disease. CRP levels >10 mg/dl strongly correlate with significant arterial impairment, marking the most severe cases of inflammation and arterial damage.

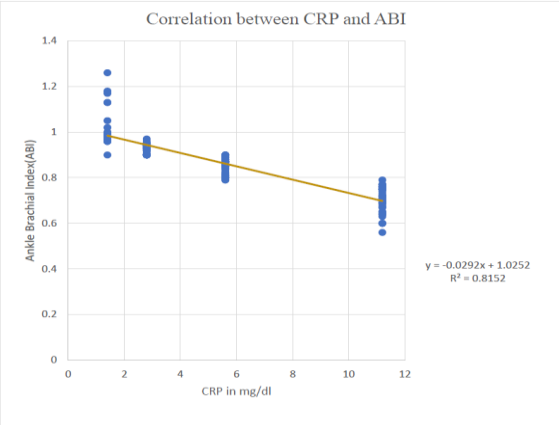

Figure 3: Correlation between CRP and ABI

The graph shows a negative correlation between C-reactive protein (CRP) and Ankle-Brachial Index (ABI), with the golden trendline indicating that higher CRP levels are associated with lower ABI values. The equation for the trendline is $y=-0.0292x+1.0252$, where each unit increase in CRP decreases ABI by about 0.0292 units. The R^2 value of 0.8152 suggests a strong correlation, explaining 81.52% of the variability in ABI.

Table 3: Correlation between CRP & Ankle Brachial Index

	Mean ± S.D.	P value (Anova)	r (correlation coefficient)
CRP	5.3 ± 3.76	<0.001	-0.9
ABI	0.87 ± 0.12		

The table shows a strong, statistically significant negative correlation between C-reactive protein (CRP) and Ankle-Brachial Index (ABI), with a p-value less than 0.001 and a correlation coefficient of r = -0.9. This indicates that as CRP levels increase, ABI values decrease significantly.

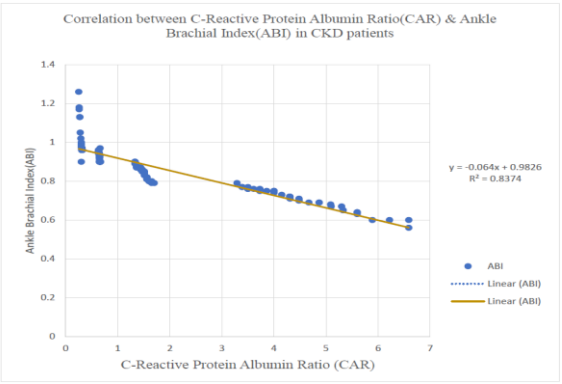

Figure 4: Correlation between C-Reactive Protein Albumin Ratio (CAR) & Ankle Brachial Index (ABI) in CKD patients

The graph shows a negative correlation between Serum C-reactive Protein Albumin Ratio (CAR) and Ankle-Brachial Index (ABI) in CKD patients. A golden trendline, with the equation $y=-0.064x+0.9826$ indicates that for each unit increase in CAR, ABI decreases by about 0.064 units. The R^2 value of 0.8374 suggests that 83.74% of the variability in ABI can be explained by CAR, highlighting a strong negative linear relationship.

Table 4: Correlation between CAR and ABI

	Mean ± S.D.	P value (Anova)	r (correlation coefficient)
CAR	1.76 ± 1.74	<0.001	-0.92
ABI	0.87 ± 0.12		

The correlation coefficient between CAR and ABI is -0.92, indicating a strong negative correlation, meaning as CAR increases, ABI decreases. The p-value is less than 0.001, confirming the correlation is highly significant. The mean CAR is 1.76 with a standard deviation of 1.74, showing considerable variability. The mean ABI is 0.87 with a lower standard deviation of 0.12, indicating less variability.

DISCUSSION

The findings of our study demonstrate a strong and statistically significant negative correlation between the C-reactive protein/albumin ratio (CAR) and Ankle Brachial Index (ABI) among patients with chronic kidney disease (CKD). This suggests that higher levels of systemic inflammation, as indicated by elevated CAR, are associated with poorer peripheral vascular health, reflected in lower ABI values.

Inflammation is a well-established contributor to the pathogenesis and progression of both CKD and atherosclerotic cardiovascular disease. Our results

reinforce this understanding, revealing that CRP levels alone showed a strong negative correlation with ABI ($r = -0.90$, $p < 0.001$), and the strength of correlation increased slightly when CRP was assessed in relation to albumin (CAR), with an even stronger correlation coefficient ($r = -0.92$, $p < 0.001$). This highlights that CAR may be a more sensitive marker than CRP or albumin alone for assessing vascular impairment in CKD patients.

The inverse relationship between CAR and ABI aligns with earlier studies suggesting that CAR reflects both inflammatory burden and nutritional status. Low serum albumin, often considered a marker of malnutrition or chronic inflammation, when combined with high CRP, indicates a state of heightened inflammation and poor prognosis. The strong correlation between CAR and ABI found in our study suggests that CAR can potentially serve as a dual-purpose marker—capturing both the inflammatory and vascular risk in CKD populations.

Moreover, the distribution of ABI classes among our study participants provides insight into the burden of subclinical and overt peripheral arterial disease (PAD) in CKD. Over 57% of patients had ABI values indicative of PAD, with 29.33% showing moderate arterial disease. These findings are in line with previous research indicating that PAD is common in individuals with CKD and is frequently underdiagnosed due to its asymptomatic nature in early stages.

Another important observation is the progressive decline in ABI with increasing CRP levels, which suggests a dose-response relationship between systemic inflammation and arterial stiffness or obstruction. This relationship supports previous studies that have implicated CRP as not only a marker but a possible mediator of vascular pathology, including endothelial dysfunction and arterial remodelling.

The relatively high mean CAR (1.76 ± 1.74) and low mean ABI (0.87 ± 0.12) in our study population reflect a high-risk profile typical of Indian CKD patients, who often present with multiple comorbidities and advanced disease at diagnosis. These findings underscore the need for early cardiovascular risk stratification and comprehensive inflammation management in CKD care.

Despite the significant findings, the cross-sectional nature of this study limits the ability to infer causality. Longitudinal studies are warranted to explore whether changes in CAR predict future declines in ABI or cardiovascular events. Additionally, while we controlled for several confounding factors through our exclusion criteria, residual confounders such as unmeasured comorbidities, lifestyle factors, or medication use could have influenced the results.

CONCLUSION

This cross-sectional study highlights a strong and statistically significant negative correlation between the C-reactive protein/albumin ratio (CAR) and Ankle Brachial Index (ABI) in patients with chronic kidney disease (CKD). The findings suggest that elevated CAR—a marker combining systemic inflammation and nutritional status—is closely associated with reduced ABI, an established indicator of peripheral arterial disease (PAD). This relationship underscores the interplay between chronic inflammation and vascular dysfunction in CKD.

Given its simplicity, cost-effectiveness, and clinical relevance, CAR could serve as a valuable tool for early identification of CKD patients at high risk of cardiovascular complications. Integrating CAR assessment alongside traditional cardiovascular risk markers may enhance screening, risk stratification, and targeted interventions in this vulnerable population. Further longitudinal studies are recommended to evaluate the prognostic value of CAR in predicting cardiovascular events and disease progression in CKD.

Limitations

While this study provides valuable insights into the relationship between the CRP/Albumin Ratio (CAR) and Ankle Brachial Index (ABI) in CKD patients, several limitations must be acknowledged:

1. Cross-sectional design: The study captures a single point in time, limiting the ability to establish causality or track the progression of inflammation or vascular disease over time.
2. Single-centre setting: Conducted at a single tertiary care hospital, the findings may not be generalizable to broader populations or different geographic or socioeconomic groups.
3. Limited sample size: Although 150 patients were included, a larger sample could offer greater statistical power and subgroup analysis, such as differences across CKD stages.
4. Exclusion of confounding conditions: While this reduced heterogeneity, it may limit real-world applicability, as many CKD patients present with multiple comorbidities.
5. Non-quantitative CRP measurement method: The use of the latex agglutination method, while accessible, lacks the precision of high-sensitivity CRP (hs-CRP) assays, potentially affecting the accuracy of CAR values.
6. Lack of long-term outcome data: The study does not assess whether elevated CAR predicts cardiovascular events, hospitalization, or mortality, which would provide deeper clinical relevance.

Recommendations

Based on the findings and limitations of this study, the following recommendations are proposed:

1. Routine use of CAR in CKD assessments: Incorporating CAR as part of routine laboratory evaluation in CKD patients could aid in early

detection of cardiovascular risk, especially in resource-limited settings.

2. Larger multicentred studies: Future research should involve diverse populations across multiple centres to improve generalizability and explore ethnic or regional variations in CAR and ABI relationships.
3. Prospective longitudinal studies: Tracking CAR and ABI over time would help determine whether CAR can predict disease progression, cardiovascular events, or mortality in CKD patients.
4. Refinement of biomarker measurement: Using high-sensitivity CRP (hs-CRP) assays could enhance the accuracy and reliability of CAR calculations, especially in early or low-grade inflammation.
5. Integration into clinical decision-making: CAR may serve as a guide for therapeutic decision-making, particularly in prioritizing anti-inflammatory or cardiovascular interventions in high-risk CKD patients.
6. Explore other inflammatory markers: Future studies could compare CAR with other composite indices (e.g., neutrophil-to-lymphocyte ratio, IL-6 levels) to evaluate their relative predictive values.

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