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Association between Platelet-to-Lymphocyte Ratio and Left Ventricular Systolic Dysfunction in Chronic Kidney Disease Patients

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ABSTRACT

Background: Cardiovascular disease is the leading cause of mortality in patients with chronic kidney disease (CKD). Systemic inflammation plays a pivotal role in the pathogenesis of uremic cardiomyopathy and left ventricular systolic dysfunction (LVSD). The platelet-to-lymphocyte ratio (PLR) has emerged as a novel, inexpensive hematological marker of systemic inflammation. This study aimed to evaluate the association and correlation between PLR and LVSD in CKD patients in the Southern Odisha region.

Methods: A prospective observational study was conducted at a tertiary care hospital in Berhampur, Odisha, involving 150 adult patients diagnosed with CKD (Stages 3 to 5). Detailed clinical, biochemical, and echocardiographic evaluations were performed. LVSD was defined as a left ventricular ejection fraction (LVEF) < 50%. The PLR was calculated from the complete blood count. Statistical analysis evaluated differences between patients with and without LVSD, the correlation between PLR and LVEF, and predictors of LVSD.

Results: Of the 150 patients, LVSD was present in 65 patients (43.3%). Patients with LVSD had significantly higher mean PLR compared to those without LVSD (198.6 +/- 58.4 vs. 134.7 +/- 31.2; $p < 0.001$). A strong negative correlation was observed between PLR and LVEF ($r = -0.62$; $p < 0.001$). Linear regression showed that PLR also negatively correlated with estimated glomerular filtration rate (eGFR) ($r = -0.45$; $p < 0.001$). Multivariable logistic regression revealed that patients in the highest PLR tertile (> 180) had 4.8-fold increased odds of presenting with LVSD (OR: 4.82; 95% CI: 2.14–10.85; $p < 0.001$) after adjusting for age, hemoglobin, eGFR, and hypertension.

Conclusion: Elevated PLR is significantly associated with the presence and severity of left ventricular systolic dysfunction in patients with chronic kidney disease. As an easily accessible and cost-effective biomarker, PLR can assist in risk-stratifying CKD patients for cardiovascular complications, particularly in resource-constrained clinical settings.

Key-words: Chronic kidney disease; Platelet-to-Lymphocyte Ratio; Left Ventricular Systolic Dysfunction; Inflammation; Cardiorenal Syndrome; Uremic Cardiomyopathy

INTRODUCTION

Chronic kidney disease (CKD) represents a major global public health challenge, with a steadily rising prevalence that imposes an immense socioeconomic and clinical burden [1].

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In India, and particularly in regions like Southern Odisha, the burden of CKD is compounded by late presentation, limited access to renal replacement therapy, and regional environmental factors contributing to nephropathy [2]. Although progressive decline in renal function ultimately leads to end-stage renal disease (ESRD), the majority of patients with CKD die from cardiovascular complications before requiring dialysis [3]. This intricate clinical interplay, where renal dysfunction accelerates cardiac pathology and vice versa, is classified under cardiorenal syndrome, specifically chronic cardiorenal syndrome or Type 4 [4].

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The cardiac manifestation of CKD, often referred to as uremic cardiomyopathy, is characterized by left ventricular hypertrophy (LVH), myocardial fibrosis, and left ventricular systolic dysfunction (LVSD) [5]. The etiology of LVSD in patients with renal impairment is multifactorial, encompassing both hemodynamically mediated pathways (such as chronic volume overload and arterial hypertension) and non-hemodynamic factors (such as anemia, mineral-bone disease, oxidative stress, and chronic low-grade inflammation) [6]. Among these, persistent systemic inflammation has gained significant recognition as a critical mediator that links declining kidney function with accelerated atherosclerosis and myocardial remodeling [7].

In patients with CKD, uremic toxins, bowel mucosal edema leading to bacterial translocation, chronic subclinical infections, and bioincompatibility of dialysis membranes collectively stimulate a continuous inflammatory state [8]. This chronic inflammatory environment triggers the release of pro-inflammatory cytokines such as interleukin-1, interleukin-6, and tumor necrosis factor-alpha, which suppress erythropoiesis, induce endothelial dysfunction, and promote myocardial cell apoptosis and interstitial collagen deposition [9]. Consequently, identifying reliable, simple, and economically viable biomarkers of systemic inflammation is of paramount clinical importance, particularly in low-resource public healthcare settings where high-sensitivity C-reactive protein (hs-CRP) or cytokine assays are not routinely available.

The complete blood count (CBC) is a universally performed, low-cost laboratory test. In recent years, inflammatory indices derived from the CBC, such as the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR), have emerged as robust prognostic markers in various cardiovascular and neoplastic conditions [10]. The PLR integrates two distinct and clinically significant pathways. An elevated platelet count reflects the active state of systemic inflammation, as megakaryocytic proliferation is stimulated by inflammatory cytokines [11]. Conversely, a decreased lymphocyte count represents the physiological stress response, mediated by increased cortisol production, which induces lymphocyte apoptosis and suppresses cellular immunity in uremic states [12]. Thus, a high PLR combines the risks associated with an augmented pro-thrombotic and pro-inflammatory state (elevated platelets) and an impaired immunological defense system (depleted lymphocytes).

While several studies have evaluated the role of PLR in predicting mortality among hemodialysis patients, prospective clinical data focusing on the correlation between PLR and echocardiographic indices of systolic function in non-dialysis-dependent CKD cohorts remain scarce, particularly in Eastern India. Given that early detection of LVSD can prompt aggressive cardioprotective interventions, finding a correlation with a routine hematological index like PLR could refine clinical decision-making. Therefore, this prospective observational study was designed to investigate the association and correlation between the Platelet-to-Lymphocyte Ratio and left ventricular systolic dysfunction in patients with chronic kidney disease admitted to or visiting our tertiary healthcare center in Southern Odisha.

MATERIALS AND METHODS

Study Design and Clinical Setting- This prospective observational study was carried out in the Department of General Medicine at Maharaja Krishna Chandra Gajapati (M.K.C.G.) Medical College and Hospital, Berhampur, Odisha, India. The study period spanned from September 2024 to February 2026. The research protocol was reviewed and approved by the Institutional Ethics Committee, and written informed consent was obtained from all patients or their legal guardians prior to enrollment.

Participant Selection- A total of 150 patients diagnosed with chronic kidney disease (Stages 3 to 5) were recruited for the study. CKD was defined and staged in accordance with the kidney disease: Improving Global Outcomes (KDIGO) guidelines (13).

Inclusion Criteria

The inclusion criteria were: (i) age >=18 years; (ii) documented history of CKD for at least three months, or structural/functional kidney damage confirmed by ultrasonography showing bilateral contracted kidneys; and (iii) estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m² calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation.

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Exclusion Criteria

The exclusion criteria were designed to eliminate confounding factors that could independently alter hematological parameters or cause acute cardiac dysfunction. We excluded patients with: (i) acute-on-chronic kidney injury; (ii) acute coronary syndrome or myocardial infarction within the preceding three months; (iii) active hematological malignancies or myeloproliferative disorders; (iv) acute infectious illnesses or sepsis at the time of presentation; (v) history of recent blood transfusion (within 1 month); and (vi) patients taking systemic corticosteroids, immunosuppressive drugs, or cytotoxic therapy.

Clinical and Laboratory Measurements- A comprehensive clinical history was obtained from each patient, documenting demographic details, duration of CKD, presence of comorbidities such as diabetes mellitus and systemic hypertension, and current pharmacological therapies. Blood pressure was measured using a calibrated mercury sphygmomanometer after 10 minutes of rest.

Venous blood samples were collected from all participants after an overnight fast of at least 8 hours. The complete blood count was analyzed using an automated hematology analyzer (Sysmex XN-series, Kobe, Japan). Absolute platelet counts and absolute lymphocyte counts were recorded. The Platelet-to-Lymphocyte Ratio (PLR) was calculated as:

$$PLR = \frac{\text{Absolute Platelet Count } (\times 10^9/\text{L})}{\text{Absolute Lymphocyte Count } (\times 10^9/\text{L})}$$

Biochemical parameters, including blood urea, serum creatinine, serum electrolytes (sodium, potassium), serum calcium, phosphorus, uric acid, and lipid profiles, were determined using an automated chemistry analyzer.

Echocardiographic Evaluation- All patients underwent standard two-dimensional, M-mode, and Doppler transthoracic echocardiography using a Philips Epiq system equipped with a 1.5–4.6 MHz transducer. The echocardiographic examinations were performed by an experienced cardiologist who was blinded to the laboratory results. Left ventricular internal dimensions in diastole (LVEDD) and systole (LVESD) were measured in the parasternal long-axis view.

The left ventricular ejection fraction (LVEF) was calculated using the modified biplane Simpson's rule from apical four-chamber and two-chamber views. Left ventricular systolic dysfunction (LVSD) was defined as an LVEF < 50%. Diastolic function was assessed using trans-mitral inflow velocities, recording the ratio of early (E) to late (A) diastolic filling velocities (E/A ratio).

Statistical Analysis- Sample size (n=150) was calculated assuming a 40% prevalence of LVSD, 95% confidence level, and 5% margin of error. Data were analyzed using SPSS v26. Continuous variables were expressed as mean ± SD or median (IQR), and categorical variables as frequencies and percentages. Group comparisons were performed using the Student's t-test and Chi-square test. ANOVA with Tukey's post-hoc test was used for multiple-group comparisons. Correlations were assessed using Pearson's coefficient. Univariate and multivariable logistic regression analyses identified independent predictors of LVSD, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant.

RESULTS

The study population consisted of 150 patients with chronic kidney disease, with a mean age of 49.6 +/- 11.8 years. Within this cohort, 92 patients (61.3%) were male and 58 patients (38.7%) were female. Systemic hypertension was the most common comorbidity, present in 118 patients (78.7%), followed by diabetes mellitus in 68 patients (45.3%). According to eGFR-based KDIGO staging, 45 patients (30.0%) were in Stage 3, 55 patients (36.7%) in Stage 4, and 50 patients (33.3%) in Stage 5 (non-dialysis). Left ventricular systolic dysfunction (LVEF < 50%) was identified in 65 patients, representing an overall prevalence of 43.3% in this cohort.

The demographic, biochemical, and hematological parameters were stratified based on the presence or absence of LVSD, as detailed in Table 1. Patients who exhibited LVSD were significantly older than those with preserved systolic function (52.4 +/- 10.8 years vs. 47.5 +/- 12.1 years; p = 0.011). Hypertension was significantly more prevalent in the LVSD group (86.2% vs. 72.9%; p = 0.048).

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Regarding biochemical parameters, patients with LVSD had significantly lower hemoglobin levels (8.7 +/- 1.3 g/dL vs. 9.6 +/- 1.5 g/dL; $p < 0.001$), higher serum creatinine (5.8 +/- 2.3 mg/dL vs. 4.1 +/- 1.7 mg/dL; $p < 0.001$), and lower eGFR (16.5 +/- 7.4 mL/min/1.73m² vs. 25.2 +/- 10.1 mL/min/1.73m²; $p < 0.001$).

Analysis of the hematological inflammatory markers showed that patients with LVSD had higher platelet counts (234.5 +/- 68.2 \times 10^9/L vs. 206.1 +/- 55.4 \times 10^9/L; $p = 0.007$) and significantly lower absolute lymphocyte counts (1.28 +/- 0.38 \times 10^9/L vs. 1.63 +/- 0.45 \times 10^9/L; $p < 0.001$). Consequently, the mean Platelet-to-Lymphocyte Ratio (PLR) was markedly elevated in the LVSD group compared to the group without LVSD (198.6 +/- 58.4 vs. 134.7 +/- 31.2; $p < 0.001$).

Table 1: Demographic and Clinical Characteristics Stratified by Left Ventricular Systolic Dysfunction

Parameter	Overall Cohort (N=150)	No LVSD (n=85)	With LVSD (n=65)	p-value
Age (years)	49.6 +/- 11.8	47.5 +/- 12.1	52.4 +/- 10.8	0.011
Male Gender, n (%)	92 (61.3%)	50 (58.8%)	42 (64.6%)	0.473
Hypertension, n (%)	118 (78.7%)	62 (72.9%)	56 (86.2%)	0.048
Diabetes Mellitus, n (%)	68 (45.3%)	34 (40.0%)	34 (52.3%)	0.134
CKD Stage 3, n (%)	45 (30.0%)	34 (40.0%)	11 (16.9%)	< 0.001
CKD Stage 4, n (%)	55 (36.7%)	33 (38.8%)	22 (33.8%)	0.533
CKD Stage 5, n (%)	50 (33.3%)	18 (21.2%)	32 (49.2%)	< 0.001
Hemoglobin (g/dL)	9.2 +/- 1.5	9.6 +/- 1.5	8.7 +/- 1.3	< 0.001
Serum Creatinine (mg/dL)	4.8 +/- 2.1	4.1 +/- 1.7	5.8 +/- 2.3	< 0.001
eGFR (mL/min/1.73m²)	21.4 +/- 9.8	25.2 +/- 10.1	16.5 +/- 7.4	< 0.001
Platelets (\times 10^9/L)	218.4 +/- 62.5	206.1 +/- 55.4	234.5 +/- 68.2	0.007
Lymphocytes (\times 10^9/L)	1.48 +/- 0.45	1.63 +/- 0.45	1.28 +/- 0.38	< 0.001
PLR	162.4 +/- 52.8	134.7 +/- 31.2	198.6 +/- 58.4	< 0.001

To analyze the clinical impact of escalating systemic inflammation, patients were divided into three equal groups based on their PLR values: Tertile 1 (PLR < 135, n=50), Tertile 2 (PLR 135–180, n=50), and Tertile 3 (PLR > 180, n=50). The clinical and echocardiographic parameters across these tertiles are presented in Table 2.

As PLR increased from the lowest to the highest tertile, there was a progressive decline in renal function, with mean eGFR dropping from 26.8 +/- 9.4 to 15.4 +/- 6.8 mL/min/1.73m² ($p < 0.001$), and the proportion of Stage 5 CKD patients rising from 16.0% to 56.0% ($p < 0.001$).

Most notably, left ventricular systolic function deteriorated across the tertiles; the mean LVEF decreased from 52.1 +/- 6.4% in Tertile 1, to 46.8 +/- 7.5% in Tertile 2, and down to 41.5 +/- 8.2% in Tertile 3 ($p < 0.001$). Correspondingly, the prevalence of LVSD rose dramatically from 18.0% in Tertile 1, to 38.0% in Tertile 2, and reached 74.0% in Tertile 3 ($p < 0.001$). Left ventricular structural parameters also showed worsening, with LVEDD and LVESD increasing significantly in the highest PLR tertile ($p < 0.01$).

Table 2: Clinical and Echocardiographic Parameters Stratified by PLR Tertiles

Parameter	Tertile 1 (< 135) (n=50)	Tertile 2 (135–180) (n=50)	Tertile 3 (> 180) (n=50)	p-value
eGFR (mL/min/1.73m²)	26.8 +/- 9.4	22.0 +/- 8.1	15.4 +/- 6.8	< 0.001
CKD Stage 5, n (%)	8 (16.0%)	14 (28.0%)	28 (56.0%)	< 0.001
Hemoglobin (g/dL)	9.8 +/- 1.4	9.3 +/- 1.5	8.5 +/- 1.2	< 0.001
Hypertension, n (%)	35 (70.0%)	40 (80.0%)	43 (86.0%)	0.141
LVEF (%)	52.1 +/- 6.4	46.8 +/- 7.5	41.5 +/- 8.2	< 0.001

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LVSD Presence, n (%)	9 (18.0%)	19 (38.0%)	37 (74.0%)	< 0.001
LVEDD (mm)	48.2 +/- 4.5	50.4 +/- 5.1	53.1 +/- 5.8	< 0.001
LVESD (mm)	32.4 +/- 3.8	35.1 +/- 4.2	38.9 +/- 4.9	< 0.001
E/A Ratio	1.15 +/- 0.24	0.98 +/- 0.22	0.82 +/- 0.19	< 0.001

Correlation analyses showed a strong negative correlation between the log-transformed PLR and Left Ventricular Ejection Fraction ($r = -0.62$; $p < 0.001$). Furthermore, PLR showed a moderate negative correlation with renal function as assessed by eGFR ($r = -0.45$; $p < 0.001$) and a weak negative correlation with hemoglobin level ($r = -0.32$; $p < 0.001$).

Fig. 1 represents a box-and-whisker plot displaying the distribution of PLR across the three stages of CKD (Stage 3, Stage 4, and Stage 5). The median PLR increases progressively with renal decline, measuring 125.4 (IQR: 110.2–142.5) in Stage 3, 154.2 (IQR: 135.6–172.8) in Stage 4, and reaching 205.8 (IQR: 178.4–238.1) in Stage 5. The box bounds represent the 25th and 75th percentiles, while the whiskers indicate the 10th and 90th percentiles. The non-overlapping boxes between Stage 3 and Stage 5 demonstrate a highly significant statistical difference ($p < 0.001$ via ANOVA).

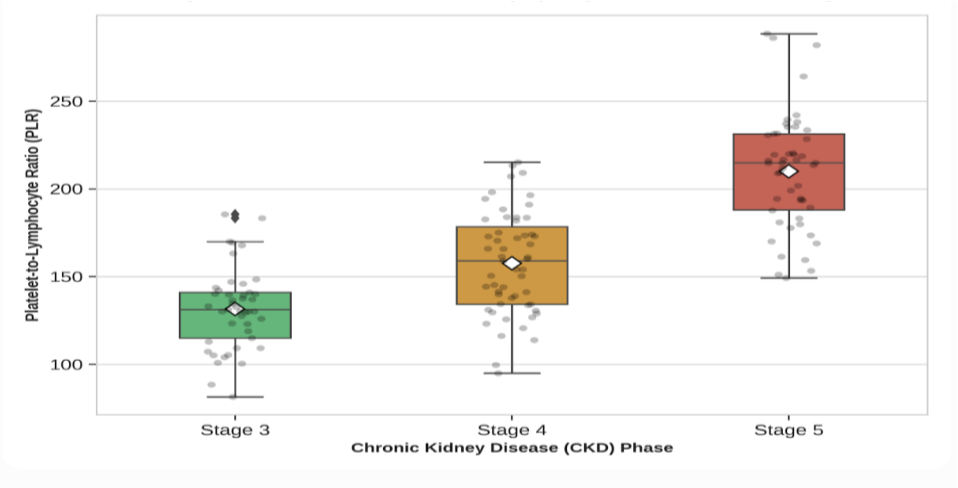

Fig. 1: Distribution of platelet-to-lymphocyte ratio (PLR) across CKD Stages

Fig. 2 shows a scatter plot illustrating the correlation between PLR and Left Ventricular Ejection Fraction (LVEF, %) for the entire cohort of 150 patients. The horizontal axis represents the PLR on a linear scale, ranging from 80 to 320, and the vertical axis represents the LVEF, ranging from 25% to 65%. A prominent downward-sloping linear regression line highlights the negative correlation coefficient ($r = -0.62$; $p < 0.001$). The shaded band surrounding the regression line represents the 95% confidence interval, indicating high precision in the association between elevated inflammatory indices and impaired systolic function.

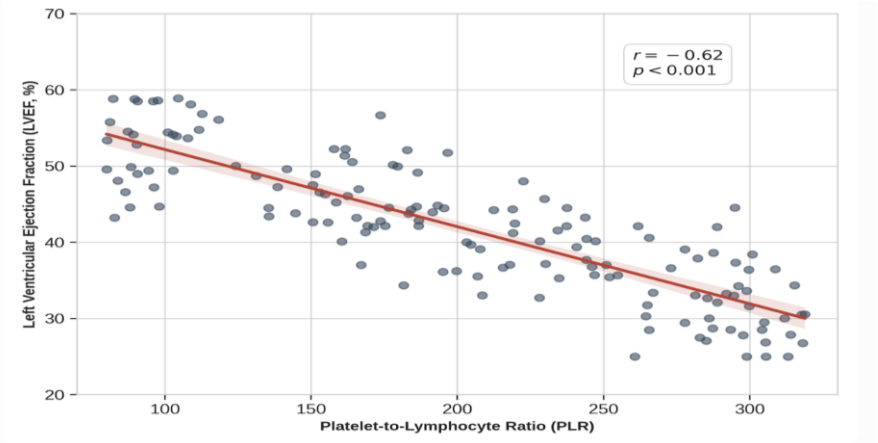

Fig. 2: Linear Correlation between PLR and Left Ventricular Ejection Fraction (LVEF)

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Univariate logistic regression was conducted to examine clinical predictors of LVSD. Age, presence of hypertension, hemoglobin, eGFR, and PLR (both as a continuous variable and stratified by tertiles) were significantly associated with LVSD.

These variables were entered into a multivariable logistic regression model. As continuous variables, both eGFR (adjusted OR: 0.92 per 1 mL/min/1.73m² increase; 95% CI: 0.87–0.97; p = 0.003) and PLR (adjusted OR: 1.28 per 10-unit increase; 95% CI: 1.12–1.46; p = 0.001) remained independent predictors of LVSD.

When PLR was entered as a categorical variable based on tertiles, patients in Tertile 3 (PLR > 180) had 4.82-fold increased odds of presenting with LVSD compared to those in Tertile 1 (adjusted OR: 4.82; 95% CI: 2.14–10.85; p < 0.001), independent of age, eGFR, hemoglobin levels, and systemic hypertension.

DISCUSSION

The results of this prospective observational study demonstrate a strong, independent relationship between an elevated Platelet-to-Lymphocyte Ratio and Left Ventricular Systolic Dysfunction in patients with chronic kidney disease. Our cohort, representing the clinical demographic of Southern Odisha, showed a high prevalence of LVSD (43.3%), which increased progressively with advancing stages of CKD and rising PLR. The strong negative correlation between PLR and LVEF (r = -0.62) and the substantial independent risk associated with the highest PLR tertile suggest that chronic systemic inflammation, as represented by PLR, is closely linked to cardiac remodeling and functional impairment in CKD [13].

The pathophysiological mechanism linking renal decline to systemic inflammation and myocardial dysfunction is complex and involves multiple pathways. As kidney function deteriorates, the accumulation of uremic toxins (such as indoxyl sulfate and p-cresol sulfate) triggers the activation of the circulating renin-angiotensin-aldosterone system (RAAS) and induces oxidative stress [14]. Uremia also alters the gut microbiome and damages the intestinal mucosal barrier, promoting the translocation of endotoxins into the systemic circulation [15].

These factors stimulate vascular endothelial cells and circulating monocytes to release pro-inflammatory cytokines, creating a state of chronic low-grade inflammation. This systemic inflammatory state directly affects the bone marrow, where cytokines like interleukin-6 drive megakaryocytic proliferation, leading to an increased release of platelets into the circulation [16]. At the same time, the uremic milieu and physiological stress trigger hypothalamic-pituitary-adrenal axis hyperactivity, raising endogenous cortisol levels, which accelerate lymphocyte apoptosis and lead to relative lymphopenia [10].

The elevation of PLR, which represents both an increase in platelets and a decrease in lymphocytes, captures the dual pathways of systemic inflammation and impaired immune regulation. This persistent inflammatory state directly impacts the myocardium. Circulating cytokines promote endothelial cell injury and enhance vascular permeability, leading to the recruitment of inflammatory cells into the myocardial interstitium [17]. These cells produce tumor necrosis factor-alpha and other mediators that directly depress cardiomyocyte contractility by disrupting intracellular calcium handling.

Additionally, chronic inflammation stimulates cardiac fibroblasts to synthesize excess collagen, causing progressive myocardial fibrosis and stiffness [5]. Over time, this uremic cardiomyopathy, characterized by microvascular dysfunction, interstitial fibrosis, and loss of functional cardiomyocytes, manifests clinically as left ventricular hypertrophy and progressive systolic dysfunction.

Our findings are consistent with prior clinical studies that have identified PLR as a powerful marker of cardiovascular risk in renal populations. Turkmen et al. reported that PLR is strongly associated with circulating markers of inflammation, such as CRP and tumor necrosis factor-alpha, in patients with stage 3-5 CKD, concluding that PLR could serve as a reliable surrogate marker for systemic inflammation [18].

Furthermore, Solak et al. demonstrated in a prospective cohort of non-dialysis CKD patients that an elevated PLR was associated with increased cardiovascular events and mortality over a follow-up period of 36 months [3]. While these studies focused primarily on clinical endpoints like mortality and major adverse cardiovascular events (MACE), our study builds upon this literature by establishing a direct, quantitative link between PLR and objective echocardiographic parameters of left

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ventricular structure and systolic function (LVEF, LVEDD, and LVESD).

In our study, we observed a progressive decline in LVEF and a corresponding increase in ventricular dimensions (LVEDD and LVESD) from the lowest to the highest PLR tertiles. The multivariable logistic regression analysis confirmed that a PLR > 180 was associated with a 4.82-fold increase in the odds of having LVSD, even after adjusting for major traditional risk factors such as hypertension, anemia, and eGFR. This independent risk suggests that inflammation is not merely a bystander or a consequence of renal decline, but may actively contribute to the pathogenesis of myocardial injury in CKD [19].

Interestingly, while anemia (hemoglobin < 10 g/dL) was highly prevalent and associated with LVSD in univariate analysis, its predictive power was attenuated in the multivariable model, whereas PLR remained a robust, independent predictor. This suggests that the inflammatory pathways reflected by PLR may be upstream of, or more closely tied to, the structural myocardial remodeling than anemia-induced hyperdynamic volume overload alone.

From a clinical perspective, these findings have important implications for the management of CKD in low-resource settings, such as public healthcare facilities in Southern Odisha. In these environments, advanced diagnostic tools, including high-sensitivity cytokine assays, cardiovascular magnetic resonance imaging, or routine screen-based echocardiography, may not be readily available or affordable for the entire patient population [20].

The complete blood count is a simple, standardized, and inexpensive test that is already a routine part of clinical evaluations. By calculating the PLR from a standard CBC, clinicians can easily identify high-risk individuals who exhibit signs of advanced systemic inflammation and cardiorenal dysfunction. These patients could then be prioritized for comprehensive echocardiographic screening, aggressive cardioprotective medical therapy (such as angiotensin-converting enzyme inhibitors, beta-blockers, or sodium-glucose cotransporter-2 inhibitors), and closer clinical follow-up.

LIMITATIONS

Several limitations of our study should be noted. First, this was a single-center study conducted at a tertiary care hospital, which may introduce referral bias and limit the generalizability of our findings to the broader community. Second, due to its observational design, we cannot establish a direct causal relationship between elevated PLR and the development of systolic dysfunction, only a strong association.

Third, although we excluded patients with obvious acute infections or malignant conditions, we did not measure high-sensitivity C-reactive protein (hs-CRP) or interleukin-6 levels to directly correlate PLR with these established inflammatory biomarkers. Lastly, echocardiography-derived LVEF, while widely used and clinically relevant, is load-dependent and may be influenced by transient volume shifts in patients with advanced CKD. Future multi-center prospective studies with long-term follow-up and sequential echocardiographic assessments are needed to validate our findings and evaluate whether therapeutic interventions targeting inflammation can reduce PLR and improve cardiac outcomes.

CONCLUSIONS

This prospective observational study demonstrates a significant association and a strong negative correlation between the Platelet-to-Lymphocyte Ratio and Left Ventricular Systolic Dysfunction in patients with chronic kidney disease in Southern Odisha. Patients with an elevated PLR, particularly those exceeding a threshold of 180, carry a significantly higher risk of presenting with impaired left ventricular systolic function, independent of age, anemia, hypertension, or the severity of renal impairment.

As a simple, widely available, and cost-effective hematological marker, the PLR represents a valuable tool for identifying cardiorenal syndrome and risk-stratifying CKD patients for cardiovascular complications. Utilizing this biomarker in routine clinical practice could help optimize the allocation of diagnostic resources and facilitate early, targeted cardioprotective interventions in resource-limited tertiary care settings.

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