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ORIGINAL ARTICLE

Relationship Between Platelet-to-Lymphocyte Ratio and Presence of Spontaneous Echo Contrast in Patients with Mitral Stenosis

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ABSTRACT

Objective: To evaluate the relationship between platelet-to-lymphocyte ratio (PLR) and spontaneous echo contrast (SEC) in patients diagnosed with mitral stenosis.

Methods: This comparative cross-sectional study was conducted for six months (Nov 2024 to April 2025) at the Cardiology Department of Armed Forces Institute of Cardiology, National Institute of Heart Diseases, Rawalpindi. A consecutive sampling technique was employed to select diagnosed moderate to severe mitral stenosis patients aged ≥18 years. Transthoracic echocardiography (TTE) was performed to identify the presence of SEC. Total platelet count was divided by the lymphocyte count to calculate PLR.

Results: Overall, 100 patients (males: 67, female: 33) having mean age 42.06±7.38 years were the study participants. PLR was significantly elevated in SEC-positive individuals (150.98±44.65 vs. 129.77±50.04, p=0.036). The area under the ROC curve was 0.635, indicating moderate predictive ability. A PLR cutoff 115.23 yielded 80.9% sensitivity and 53.1% specificity, with 95% CI (0.513–0.757). Binary logistic regression confirmed the predictive potential of PLR (β=0.010; 95% CI: 0.01(1.001-1.019) and the significant relationship between PLR and SEC (p<0.05).

Conclusion: PLR is significantly associated with SEC in mitral stenosis patients. Additionally, PLR shows potential as a predictor of SEC.

Keywords: Lymphocyte, Mitral Valve Stenosis, Platelet, Platelet to Lymphocyte Ratio.

Authors' Contribution:
[1,2]Conception; Literature research; manuscript design and drafting; [3,4]Critical analysis and manuscript review; [5,6]Data analysis; Manuscript Editing.

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Introduction

Spontaneous echo contrast (SEC) is an echocardiographic finding marked by swirling, smoke-like echoes within the heart chambers, commonly observed in situations involving stagnant or reduced blood flow.[1,2] Studies have shown that the presence of SEC in the left atrium can be a risk factor for thrombus formation, further increasing the likelihood of thromboembolic events.[3,4] Patients with mitral stenosis are particularly vulnerable as they exhibit an elevated risk of systemic thromboembolism, which is a leading cause of morbidity and mortality.[5] Due to impaired blood flow and stasis, the risk of thrombosis and left atrial SEC development is significantly higher in these patients.[6] Due to the high risk of thrombi, early

diagnosis of thrombosis and recognition of risk factors are of utmost importance for initiating prompt anticoagulant treatment to prevent stroke and other thromboembolic complications. In conditions such as mitral stenosis and atrial fibrillation (AF), the left atrial appendage (LAA) and the left atrium (LA) serve as primary sites for thrombus formation.[7]

In recent years, inflammatory and hematologic biomarkers have gained significant attention as potential predictors of SEC. The pathophysiology of SEC is closely linked to hematologic factors that influence blood echogenicity. Ito and Suwa reported that SEC occurs due to erythrocyte aggregation in the plasma, contributing to swirling, smoke-like echoes within the cardiac chambers.[8] Additionally, some studies have highlighted the role of platelet crit and mean platelet volume in the development of SEC, further emphasizing the importance of platelet-related parameters in this process.[9,10] These findings highlight that hematologic markers, particularly PLR, may provide deep information into the underlying mechanisms of SEC and its association with thromboembolic risk. It reflects both platelet activation, an essential push of thrombus formation, and lymphocyte-mediated immune regulation, making it a valuable marker for assessing the prothrombotic state in mitral stenosis patients.

Despite several studies evaluating hematologic parameters in relation to SEC, the specific role of the PLR in patients with mitral stenosis remains less investigated. While PLR has been recognized as a marker of inflammation and thrombosis, limited research has directly assessed its association with SEC in this patient population. Most existing studies have either focused on broader coagulation profiles or included PLR as part of composite indices rather than evaluating its independent predictive value. Given its accessibility through routine blood tests and its potential to reflect both thrombotic and inflammatory processes, investigating PLR as a standalone marker may offer valuable insights into the pathophysiology of SEC. This study aims to fulfill this gap by examining the relationship between PLR and the presence of SEC in patients with mitral stenosis.

Methodology

This comparative cross-sectional study was conducted for six months (Nov 2024 to April 2025). The study was carried out following the Declaration of Helsinki ethical guidelines and informed consent was ensured from all the study subjects. The sample size was determined to ensure the inclusion of an adequate number of study participants. For this purpose, the WHO sample size calculator was used. The prevalence of mitral stenosis was set at 8.8%,[11] with confidence level and margin of error set at 95% and 5% respectively. Based on these parameters, the final calculated sample size was 123. Consecutive sampling technique was applied for the recruitment of study subjects, which included patients of both genders aged 18 or more and presented with moderate to severe/progressive mitral stenosis. Exclusion criteria were comprised of patients with other moderate or severe valvular heart disease, history of malignancy, left atrium thrombus, on any corticosteroids or non-steroidal anti-inflammatory drug treatment or, history of inflammatory disease, thyroid disease, connective tissue disease, and any other hematological disease. Prior to patient enrollment as study participant, written informed consent was take either from the patients or the legal guardians. Following a careful screening process using the exclusion criteria, mitral stenosis patients diagnosed were included in the study. Experienced cardiologists performed transthoracic echocardiography (TTE) to assess cardiac function and identify the presence of SEC. Based on the echocardiographic findings, patients were categorized as SEC positive and SEC negative. For blood sample collection, venous blood samples were obtained and sent for a complete blood picture test.

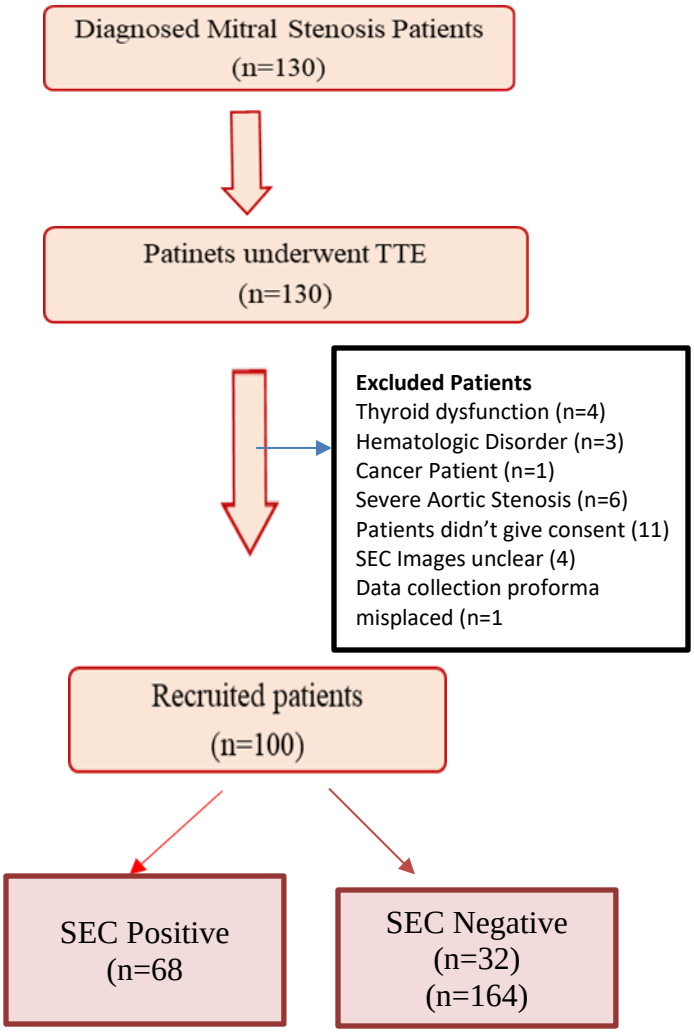

Figure 1: Flow chart for the recruitment of study participants

Platelet as well as lymphocyte count were analyzed to calculate PLR. Qualified cardiologists conducted all echocardiographic examinations. Left ventricular ejection fraction (LVEF) was measured using the parasternal long-axis (PLAX) and apical four-chamber views. SEC was identified by a characteristic swirling, smoke-like echo density in the left atrium.[1]

The area of mitral valve was determined using the planimetry method. Mitral stenosis was defined as a blockage of blood flow into the left ventricle at the level of the mitral valve, caused by structural abnormalities within the mitral valve apparatus. It was categorized as progressive stenosis (Mitral Valve Area >1.5cm^2), severe stenosis (MVA 1-1.5cm^2) and very severe stenosis (MVA <1cm^2). [12]

Data was analyzed on Statistical Package for Social Sciences (SPSS) Version 28.

Frequencies & percentages and mean & standard deviation were calculated for categorical and continuous data respectively. A chi-square test was employed to determine the frequency difference across patients with and without SEC. Student t-test was applied to compare the mean values of numerical data similarly. A receiver operating characteristics curve was drawn for PLR to find its predictive value, sensitivity, and specificity to detect SEC in mitral stenosis patients. Univariate analysis was run to find the independent role of PLR in predicting SEC. Findings were considered significant at p-value ≤0.05.

Ethical review committee of the Armed Forces Institute of Cardiology, National Institute of Heart Diseases, Rawalpindi, under the IERB letter no. 9/2/R&D/2023/286 on 13th September 2023.

Results

Our study included 100 participants to compare SEC-positive (n=68) and SEC-negative (n=32) groups. According to the findings shown in table-I, none of the demographic or clinical variables showed statistically significant differences across groups (p>0.05). The average age was 42.06±7.38 years, with no significant difference between SEC-positive (42.01±7.09) and SEC-negative (42.16±8.10) groups (p>0.05). Males comprised 67% of the participants, with almost similar percent distribution across groups. Among comorbidities, atrial fibrillation and diabetes were more frequent in SEC-positive individuals (44.1% vs. 28.1%; 25% vs. 12.5%; p>0.05 respectively), though not statistically significant. Drug history revealed minor differences in aspirin and warfarin use (20.6% vs. 25%, p=0.812) in both groups.

Table I: Baseline and Clinical Characteristics of Study Participants (n=100)					
Variables		**Total (n=100)**	**SEC(+ve) (n=68)**	**SEC(-ve) (n=32)**	**p-value**
Demographics					
Gender	Male	67(67.0%)	45(66.2%)	22(68.8%)	0.978
	Female	33(33.0%)	23(33.8%)	10(31.3%)	
Age		42.06±7.38	42.01±7.09	42.16±8.10	0.929
Comorbid					
Ischemic Heart Disease		14(14.0%)	9(13.2%)	5(15.6%)	0.243
Hypertension		28(28.0%)	18(26.5%)	10(31.3%)	0.797
Diabetes Mellitus		21(21.0%)	17(25.0%)	4(12.5%)	0.243
Smoker		26(26.0%)	19(27.9%)	7(21.9%)	0.689
Atrial Fibrillation		39(39.0%)	30(44.1%)	9(28.1%)	0.190
Drug History					
History of Aspirin		22(22.0%)	14(20.6%)	8(25.0%)	0.812
History of Warfarin		13(13.0%)	9(13.2%)	4(12.5%)	1.00
Echocardiographic Parameters					
Mitral Regurgitation	Trace	16(16.0%)	11(16.2%)	5(15.6%)	0.363
	Mild	31(31.0%)	20(29.4%)	11(34.4%)	
	Moderate	16(16.0%)	13(19.1%)	3(9.4%)	
	Severe	12(12.0%)	10(14.7%)	2(6.3%)	
Mitral Stenosis	Progressive (>1.5 cm^2)	27(27.0%)	16(23.5%)	11(34.4%)	0.485
	Severe (1-1.5 cm^2)	54(54.0%)	39(57.4%)	15(46.9%)	
	Very Severe (<1cm^2)	19(19.0%)	13(19.1%)	6(18.8%)	
Ejection Fraction (%)		57.75±6.56	58.01±6.47	57.19±6.83	0.559
Mitral valve Area (cm^2)		1.39±0.39	1.38±0.35	1.39±0.48	0.924
LA Size (mm)		40.78±8.33	42.24±7.67	37.69±8.95	0.01
Hematologic Parameters					
Platelet Count (x10^3µL)		316.82±54.6	326.31±53.6	296.66±51.8	0.011
Lymphocyte Count		2412.50±745.7	2339.71±685.9	2567.19±850.2	0.156
PLR		144.19±47.25	150.98±44.65	129.77±50.04	0.036
LA Size=Left Atrial Size; PLR= Platelet to Lymphocyte ratio					

Mitral regurgitation severity did not differ significantly across groups (p>0.05), and mild regurgitation was most common (31%). Mitral stenosis severity was comparable in SEC (+) and SEC (-) patients, with most cases classified as severe (54%). Ejection fraction (%) and mitral valve area were not altered significantly between groups (p>0.05). However, LA size was significantly larger in SEC-positive patients (42.24±7.67 vs. 37.69±8.95, p=0.01), reflecting a strong link with SEC presence.

Regarding hematologic parameters, platelet count (x10^3/µL) was significantly higher in SEC-positive patients (326.31±53.6 vs. 296.66±51.8, p=0.011), reflecting a link between increased platelet levels and SEC formation. Although lymphocyte count was slightly lower (2339.71±685.9 vs. 2567.19±850.2, p=0.156), it was not statistically significant. (Table-I) Additionally, PLR was significantly elevated in SEC-positive individuals (150.98±44.65 vs. 129.77±50.04, p=0.036), depicting a possible inflammatory or

thrombotic role in SEC development. (Figure-2). The receiver operating characteristics curve in Figure-3 demonstrated the predictive role of PLR for SEC in mitral stenosis patients. The AUC (0.635) indicated a moderate predictive ability. A PLR cutoff 115.23 yields 80.9% sensitivity and 53.1% specificity, with 95% CI (0.513–0.757). While PLR shows potential as a predictor, its moderate specificity highlighted the need for additional clinical markers for better SEC detection.

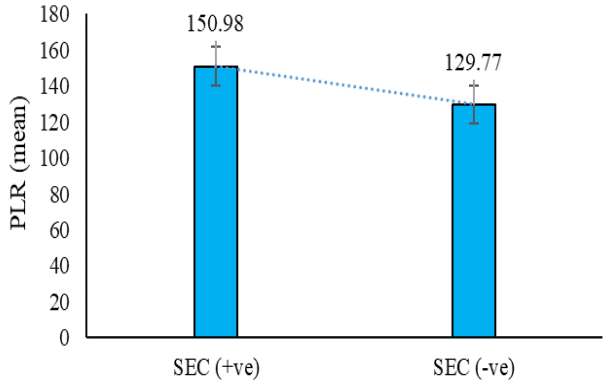

Figure 2: Platelet to lymphocyte ratio across patients with and without SEC (n=100)

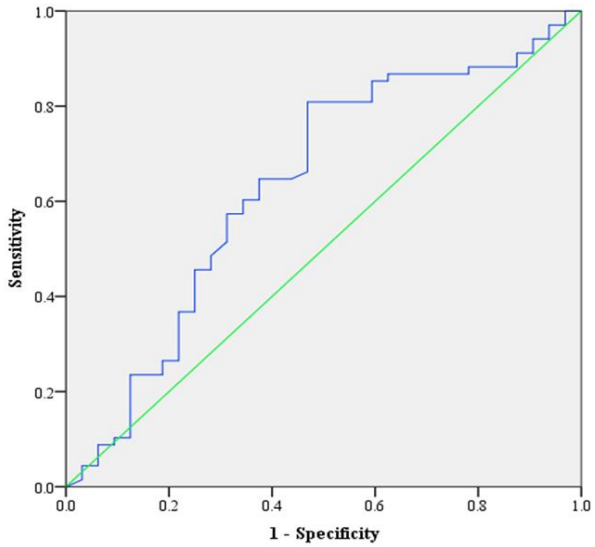

Figure 3: The ROC curve of PRL for predicting SEC in mitral stenosis patients

Findings shown in table-II further confirm the predictive potential of PLR and the significant relationship between PLR and SEC ($p<0.05$). The regression coefficient (β) of 0.010 indicated a positive association. The odds ratio (OR=1.010) implies that for each unit increase in PLR, the likelihood of SEC increases by 1%.

Table II: Independent predicting role of PLR to predict SEC in MS patients (n=100)

Variable	β	p-value	95% CI for EXP(B)	
			Lower	Upper
Platelet-to-lymphocyte ratio	0.010	0.039	1.001	1.019

Discussion

This study demonstrated the significant predictive role of the PLR in the presence of SEC. PLR was markedly elevated in SEC-positive individuals, suggesting a possible inflammatory or thrombotic contribution to SEC development. The area under the ROC curve (0.635) indicated moderate predictive ability, with a PLR cutoff 115.23 yielding 80.9% sensitivity and 53.1% specificity. Additionally, LA size and platelet count were significantly higher in SEC-positive patients. These outcomes are consistent with past studies that have identified PLR as a reliable marker of systemic inflammation and thrombotic risk.

In patients with mitral stenosis, reduced shear stress and low blood flow velocity promote the aggregation of red blood cells by facilitating their interaction with plasma proteins.[6] As a result, SEC may develop in the left atrium, increasing the risk of systemic thromboembolism.[13] Earlier studies have indicated that the presence of spontaneous echo contrast (SEC) in the left atrium is an independent risk factor for thromboembolic complications in patients with stenosis of mitral valve.[2,14,15] Furthermore, the presence of SEC reflects two arms of Virchow's triad, blood stasis and a prothrombotic condition, which are critical factors in thrombus formation.[16,17] This aligns with the current study's

findings, where elevated PLR and increased LA size serve as indicators of an inflammatory and hypercoagulable state. This further validates the role of PLR as a valuable predictive biomarker for SEC.

The findings of the present study can be critically compared with those of Sahin et al.,1 who reported a 40.5% prevalence of SEC. In their study, the mean age of participants was 42.7 ± 11.9 in the SEC-negative group and 45.3 ± 10.5 in the SEC-positive group, with no significant difference between groups. Similarly, comorbid conditions did not differ significantly across the groups. Our study exhibited a higher prevalence of SEC (68.0%), but the mean age across groups was consistent with Sahin's findings: 42.01 ± 7.09 in SEC-positive and 42.16 ± 8.10 in SEC-negative groups ($p > 0.05$). Additionally, comorbidities showed no significant differences across groups, further supported by the findings of Sahin et al. These results highlighted the higher prevalence of SEC in our study sample compared to Sahin et al., which may indicate differences in population characteristics or the severity of underlying cardiovascular conditions. However, the consistency in mean age and comorbidities across SEC-positive and SEC-negative groups exist.

Previous studies have established a strong link between inflammation and the development of SEC, highlighting the role of various inflammatory markers such as the neutrophil-to-lymphocyte ratio (NLR) and PLR in this process.[1,7,18] Kelesgulo et al. also reported the significance of the systemic inflammatory immune index in SEC formation.2 Akpek at al., studied platelet indices as predictors of SEC and their findings demonstrated that SEC-positive patients exhibited a higher platelet count, while lymphocyte counts remained comparable between SEC-positive and SEC-negative groups.[14]

Consistent with these findings, the present study also identified a significantly elevated platelet count in SEC-positive patients. However, unlike previous reports, our study observed a lower lymphocyte count in SEC-positive individuals. This discrepancy may be attributed to differences in patient demographics, underlying pathophysiological mechanisms, or variations in inflammatory responses across study populations. However, the findings reflect the importance of inflammatory and hematological markers in SEC evaluation. Additional studies are needed to investigate the underlying mechanisms driving these relationships and to confirm their predictive value across varied patient groups. When comparing LA diameter across SEC groups, our study revealed a significant mean difference, with a larger LA size observed in the SEC-positive group ($p=0.01$). In contrast, no significant variation was found in the mitral valve area ($p>0.05$). These findings are in line with the study carried out by Macit et al.,[9] who also reported no significant variation in platelet indices, including platelet count, mean platelet volume, plateletcrit, and platelet distribution width (PDW) between SEC-positive and SEC-negative patients. In their study, the mitral valve area was 2.23±0.32 cm^2 and 2.34±0.43 cm^2 in the in the SEC (+ve) and SEC(-ve) group, respectively ($p>0.05$), showing no significant difference. However, similar to our study, LA size was greater in the SEC(+ve) group (53.8±7.8 mm) compared to the SEC(-ve) group (49.9±5.4 mm) with significant mean difference ($p<0.001$). This consistency in study findings highlights the critical role of left atrial enlargement in SEC development, likely due to increased blood stasis and disturbed flow dynamics, which are key contributors to Virchow's triad for thrombus formation. Therefore, LA size can also serve as a valuable echocardiographic marker for identifying patients at higher risk of SEC and subsequent thromboembolic events, even without significant differences in platelet indices or mitral valve area, as evident from a Chinese study.[3]

The current study's findings are further supported by Sahin et al., who reported significantly higher PLR in the SEC-positive group (180.6 ± 108.7 vs. 143.4 ± 77.7; $p < 0.001$). Their ROC curve analysis demonstrated that a PLR ≥123 predicted SEC with 71% sensitivity and 52% specificity in patients with

mitral stenosis. Moreover, PLR was identified as an independent predictor of SEC formation with an odds ratio (OR) of 1.006.[1] According to Tek and Efe, higher PLR levels in patients with a LAA thrombogenic milieu further emphasize the pro-inflammatory and pro-thrombotic role of PLR in the pathogenesis of SEC.[19] These findings, in alignment with our study, suggest that PLR can serve as a simple, inexpensive, and easily accessible inflammatory biomarker for predicting SEC formation and identifying the individuals who have higher risk of thromboembolic events. This could be particularly beneficial for cardiologists in routine clinical practice, especially in resource-limited settings, where advanced imaging modalities like transesophageal echocardiography may not always be feasible.

Thus, the potential use of PLR as a cost-effective and non-invasive screening tool for recruiting patients at higher risk of SEC, particularly in individuals with mitral stenosis. By detecting elevated PLR levels early, clinicians can implement preventive strategies to reduce the risk of thromboembolic events. Furthermore, the lack of significant differences in age and comorbid conditions across groups emphasizes that PLR can serve as a valuable marker regardless of baseline clinical characteristics. This allows for better risk stratification and personalized management of patients predisposed to SEC formation and related thrombotic complications.

Conclusion

Our study demonstrated a significant relationship of PLR with SEC in patients with mitral stenosis. Elevated PLR was observed in SEC-positive individuals, and a PLR cutoff 115.23 showed moderate predictive ability for SEC. Moreover, PLR may serve as a helpful biomarker for classifying patients who are at an increased risk of developing SEC., potentially aiding in risk stratification and management of mitral stenosis.

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