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Pentraxin 3 And Cardiovascular Risk in Patients with Systemic Lupus Erythematosus: Correlation with Left Ventricular Strain and Disease Activity

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ABSTRACT

Background: Systemic lupus Erythematosus (SLE) increases cardiovascular risk due to both traditional risk factors and disease-specific mechanisms like inflammation and autoimmunity. Pentraxin3 (PTX3) has emerged as a key biomarker, with elevated levels correlating with disease activity and subclinical cardiovascular dysfunction. PTX3 may assist identify people with increased cardiovascular risk and monitor the effectiveness of treatments aimed at reducing inflammation and preventing heart damage.

Objective: This study aimed to investigate the relationship between disease activity, pentraxin levels, and Global Longitudinal Strain (GLS), hypothesizing that higher disease activity is associated with elevated pentraxin levels and a decrease in GLS.

Patients and methods: This case-control study included 50 SLE patients and 50 age- and sex-matched healthy controls to compare these factors.

Results: The study found significant differences between SLE patients and control subjects in terms of clinical symptoms, laboratory parameters, and elevated PTX3 levels, which correlated with disease activity. Cardiac parameters were mostly normal, though reduced GLS suggested potential myocardial dysfunction. PTX3 showed high sensitivity and specificity as a biomarker for differentiating SLE patients from healthy controls and was strongly correlated with SLE disease activity, supporting its potential role in monitoring the disease.

Conclusion: In patients with SLE, a strong correlation between PTX3 levels and LV systolic function as measured by LV GLS, with the disease activity, potentially represents new tools to improve risk stratification in these patients. And reinforces its potential as an effective screening tool for early detection of subclinical LV dysfunction in patients with SLE, though further research is needed to fully establish its clinical utility.

Keywords: SLE, Pentraxin3 (PTX3), Cardiovascular Risk, Global Longitudinal Strain (GLS).

INTRODUCTION

SLE is a chronic autoimmune disorder marked by systemic inflammation that affects multiple organs, including the cardiovascular system. Patients with SLE are at increased risk for cardiovascular events, such as atherosclerosis, myocardial infarction, and heart failure, even in the absence of traditional risk factors like hypertension or hyperlipidemia. Cardiovascular disease (CVD) etiology in SLE is complicated, encompassing both classic cardiovascular risk factors and disease-specific processes, such as inflammation and autoimmune [(1)].

Pentraxin3, an acute-phase protein generated locally in inflammatory areas by a variety of cells, including smooth muscle cells, endothelial cells, and macrophages, is one important biomarker that has surfaced in recent years. Pentraxin3 is essential for immunological modulation, pathogen binding, and complement system activation, in contrast to C-reactive protein (CRP), which is mostly generated by the liver [(2)]. According to the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), Pentraxin3 levels increase in SLE during active illness episodes and are associated with increased disease activity. In these individuals, elevated Pentraxin3 levels are linked to vascular damage, endothelial dysfunction, and systemic inflammation, all of which raise the risk of cardiovascular disease [(3)].

The cardiovascular risk in SLE is influenced by both conventional factors (e.g., hyperlipidemia, smoking, and obesity) and lupus-specific factors, including chronic inflammation, autoantibodies, and corticosteroid use [(4)]. Chronic inflammation, in particular, plays a central role in endothelial dysfunction, atherosclerosis, and vascular damage. This inflammation also affects the heart, with left ventricular (LV) strain serving as an early indicator of myocardial stress and dysfunction—often before clinical heart failure becomes evident [(5)].

PTX3 has shown promise as a biomarker for both disease activity and cardiovascular risk in SLE patients [(6)]. Elevated PTX3 levels are correlated with increased LV strain and subclinical LV dysfunction, suggesting that PTX3 may reflect myocardial inflammation even before noticeable changes in heart function occur. As SLE-related inflammation drives endothelial dysfunction and vascular damage, PTX3 could serve as an early marker for cardiac involvement, providing an opportunity for early intervention to prevent further damage [(7)].

The correlation between PTX3 and cardiovascular risk underscores the need for closer cardiovascular monitoring in SLE patients. Increased PTX3 levels may be useful in identifying people who are more susceptible to future cardiovascular events since they are linked to both disease activity and subclinical myocardial dysfunction, especially during

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the asymptomatic phase [8]. Furthermore, PTX3 could offer insights into the effectiveness of anti-inflammatory treatments, helping clinicians evaluate their impact on disease control and potential reductions in cardiovascular risk [9]. Therefore, this study aimed to investigate the relationship between disease activity, pentraxin levels, and LV function assessed by GLS.

PATIENTS AND METHODS

This case-control study comprised 100 individuals in total, 50 of whom were SLE patients and 50 of whom were healthy controls who were matched for age and sex. All SLE patients were enrolled from the Outpatient Clinic of the Rheumatology, Rehabilitation and Physical Medicine Department. Control subjects were recruited from individuals who did not meet the criteria for SLE or any other inflammatory disease and have no history of cardiovascular or infectious diseases.

Inclusion criteria: For SLE patients included individuals over 18 years of age who met the American College of Rheumatology (ACR) criteria and the 2012 Systemic Lupus Erythematosus International Collaborating Clinics (SLICC) classification criteria for SLE [10].

Exclusion criteria: Individuals over 50 years of age, those diagnosed with antiphospholipid syndrome (APS), pregnant or postpartum women, patients with malignancy or ischemic heart disease (IHD), and those with active infections or recent trauma. Additionally, patients with a history of cerebrovascular accidents, multi-infarct dementia, cerebral venous thrombosis, cardiomyopathy, hypertension, diabetes mellitus (DM), other autoimmune diseases, or transverse myelopathy were excluded. The control group contained individuals with no history of inflammatory or autoimmune diseases, CVD, or infectious diseases.

Demographic information, including age, sex, disease duration, and medical history, were gathered for all participants. Clinical evaluation involved the measurement of BP and HR, as well as a comprehensive examination of joints and skin. Disease activity in SLE patients was assessed using the SLEDAI to determine the level of disease activity in each participant.

Blood and urine samples were collected from both SLE patients and healthy controls for a range of laboratory investigations. These include tests for ESR, CRP, CBC, ANA, dsDNA, C3, and C4 levels, as well as kidney function tests, including serum creatinine, urea, and uric acid. Urinalysis and 24-hour urine protein collection were also performed. Additionally, plasma PTX3 concentrations were measured, with plasma samples stored at −75 °C until analysis using a sandwich ELISA kit (Hycult, USA) on an automated platform (DSX, Technogenetics, USA). To quantify PTX3, a sandwich ELISA was used, which involves two specific antibodies: One to capture PTX3 on a microplate and another to detect it, conjugated to an enzyme for colorimetric analysis.

Echocardiography

Conventional echocardiographic Doppler study, as well as tissue Doppler imaging and 2D-speckle tracking imaging were performed using Vivid 9, General Electric Healthcare (GE Vingmed, Norway) equipped with harmonic M5S variable frequency (1.7–4 MHz) phased-array transducer according to recommendations of the American Society of Echocardiography [11].

2D-speckle tracking echocardiography

2D strain analysis was performed offline using the Echopac software (General Electric version 1.8.1.X-Vingmed). Global LV longitudinal strain (GLS) was obtained from averaging peak values of 18 LV segments and compared between the 2 groups.

Ethical approval: Menoufia Faculty of Medicine's Ethics Committee has given its approval for this study (IRB-No.: 1/2025 PMRR22). All participants signed their consents after receiving all the information. The Helsinki Declaration was followed throughout the whole study.

Statistical analysis

The recorded data were examined using SPSS version 23.0. Both the SLE and control groups' baseline characteristics were obtained using descriptive statistics. Frequencies and relative percentages were used to illustrate the qualitative data. The mean ± SD was used to express quantitative data. With the use of suitable statistical techniques, such as Pearson or Spearman correlation analysis, the relationship between PTX3 levels, disease activity (SLEDAI), and LV strain was evaluated. Independent t-tests or Mann-Whitney U-tests, if applicable, were used to compare the differences between the two groups (SLE vs. controls). To find variables that are independently linked to PTX3 levels and LV strain, multivariate regression analysis may be employed. P values ≤ 0.05 were regarded as statistically significant.

RESULTS

Table (1) compared the baseline characteristics of 50 SLE patients (cases) and 50 control subjects across various parameters. In terms of demographics, the mean age of the SLE patients (35.8 ± 9.5 years) and control group (35.3 ± 10.2 years) was similar, with no significant difference (p-value = 0.801). The SLE group also had higher proportion of females (94%) compared to the control group (84%), although this difference was not statistically significant (p-value = 0.178). Notably, the SLE group showed significant clinical manifestations, with 96% experienced arthritis, 66% had malar rash, and 72% reported oral ulcers. Other common features include steroids therapy (100%), antimalarial therapy (60%), and immunosuppressant therapy (44%), with 70% testing positive for Anti-Ds-DNA, a hallmark of lupus.

Regarding laboratory parameters, the SLE group had significantly lower hemoglobin levels (10.7 ± 1.96 g/dL) compared to the control group (11.88 ± 1.29 g/dL, p-value < 0.05), and significantly higher erythrocyte sedimentation rate (46.34 ± 10.94 mm/h) and CRP (12.26 ± 2.87 mg/L), indicating ongoing inflammation. 24-hour urinary protein was significantly higher in the SLE group (85.51 ± 21.13 mg vs. 20.13 ± 5.02 mg, p-value < 0.05). Complement proteins C3 and C4 were significantly lower in the SLE group, suggesting complement consumption (p-values < 0.05). Lastly, no significant differences were found between the groups for lipid profile parameters, including total cholesterol, HDL, LDL, and triglycerides.

Table (1): the baseline characteristics of the SLE patients and the control subjects

Parameter	Case (n = 50)	Control (n = 50)	p-value
Age (years)	35.8 ± 9.5	35.3 ± 10.2	0.801
Sex (Female %)	94.0%	84.0%	0.178
Clinical manifestations		0	NA
Arthritis	96.0%		
Malar rash	66.0%		
Oral ulcers	72.0%		
Steroids therapy	100.0%		
Antimalarial therapy	60.0%		
Immunosuppressant therapy	44.0%		
Presence of Anti-Ds-DNA	70.0%		
Degree of SLEDAI score (High)	30.0%		
Value of SLEDAI score			
6.00	32.0%		
8.00	16.0%		
10.00	22.0%		
12.00	6.0%		
14.00	20.0%		
16.00	4.0%		
Hemoglobin (g/dL)	10.7 ± 1.96	11.88 ± 1.29	< 0.05
WBC (x10^3/µL)	5.71 ± 1.41	6.63 ± 1.64	0.178
Platelets (PLT) (x10^3/µL)	243.3 ± 59.88	213.66 ± 35.71	0.070
Erythrocyte Sedimentation Rate (ESR) (mm/h)	46.34 ± 10.94	7.73 ± 1.42	< 0.05
CRP (mg/L)	12.26 ± 2.87	3.95 ± 0.89	< 0.05
SGOT (AST) (U/L)	27.56 ± 6.69	13.48 ± 2.64	< 0.05
SGPT (ALT) (U/L)	28.62 ± 7.11	28.88 ± 4.85	0.79
Serum Urea (mg/dL)	33.54 ± 5.11	26.96 ± 6.54	0.070
Serum Creatinine (mg/dL)	0.97 ± 0.23	0.66 ± 0.14	0.070
24-hour Urinary Protein (mg)	85.51 ± 21.13	20.13 ± 5.03	< 0.05
Total Cholesterol (mg/dL)	202.74 ± 26.35	200.76 ± 23.65	0.72
HDL Cholesterol (mg/dL)	52.33 ± 10.46	51.94 ± 9.69	0.79
LDL Cholesterol (mg/dL)	133.59 ± 19.43	134.12 ± 19.05	0.91
Triglycerides (mg/dL)	159.54 ± 35.53	154.88 ± 34.91	0.72
C3 (mg/L)	82.5 ± 20.10	102.5 ± 24.91	< 0.05
C4 (mg/L)	13.1 ± 3.10	31.0 ± 7.60	< 0.05
Anti-dsDNA (IU/mL)	62.58± 15.37	0	NA

Figure (1): The case group exhibited a higher mean pentraxin-3 level (6.6580) compared to the control group (2.3180), the difference in pentraxin-3 levels between the two groups was highly significant, with a p-value of 0.000.

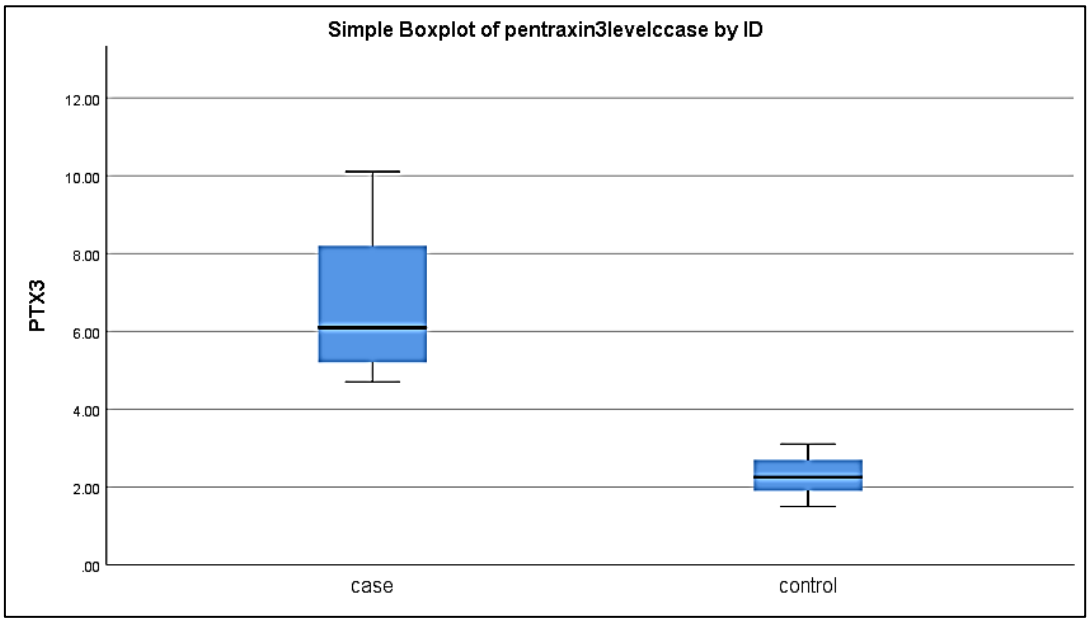

Figure (1): The PTX3 plasma levels in SLE patients and control subjects.

Table (2) provides an overview of key cardiac parameters. The **left ventricular ejection fraction (LVEF) 66.34%** indicated normal systolic function. **GLS** of -14.13 suggests reduced myocardial strain, which indicates impaired LV systolic function.

Table (2): Various cardiac measurements for patient with SLE

Variable	Mean	Standard Deviation
Global Longitudinal Strain (GLS)	-14.1282	3.51822
Fractional Shortening (fs)	36.7400	3.33081
Left Ventricular Ejection Fraction (LVEF)	66.3400	4.17773

The optimal cut-off point for pentraxin3 was found to be 3.05, showing excellent performance with a sensitivity of 96% and specificity of 94%, along with an impressive AUC of 0.988. This threshold effectively differentiates between the two groups, accurately identifying most positive cases while minimizing false positives, making it a highly reliable measure for distinguishing between the groups **(Figure 2).**

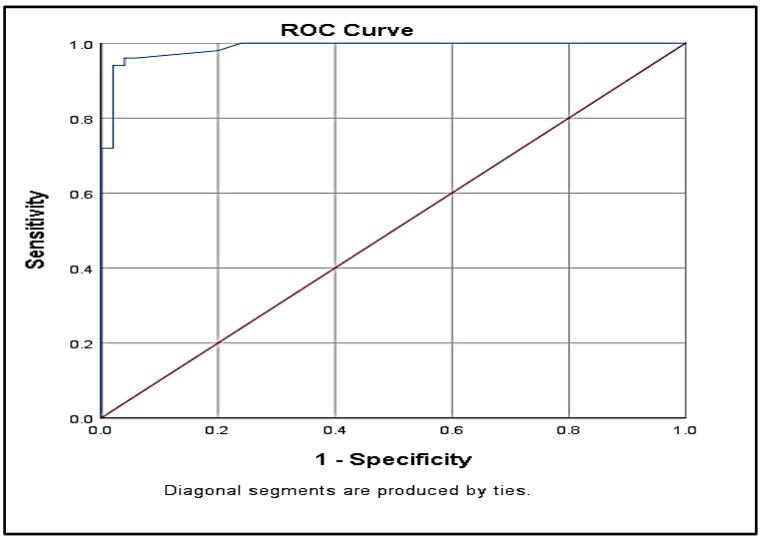

Figure (2): ROC curve analysis to determine pentraxin3 cut-off point between lupus patients and healthy controls.

The AUC of 0.516 indicated poor discrimination between the groups, close to random chance. At a cut-off of 5.65, sensitivity was 71.1%, but specificity was low at 41.7%, leading to a high false positive rate. While there was a balance between sensitivity and specificity, the test's overall performance was suboptimal due to the low AUC and high false positives

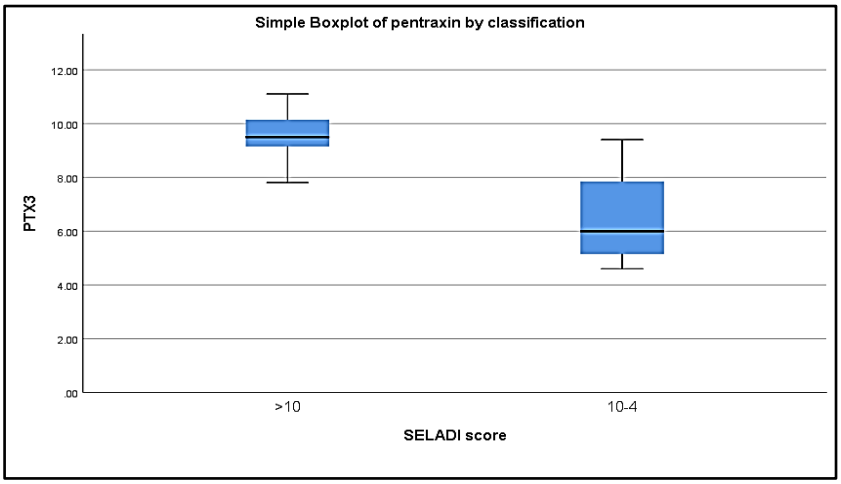

Figure (3): Boxplot curve of pentraxin3 plasma levels and SLEDAI patients.

The Pearson correlation between SLEDAI and PTX3 plasma levels was 0.922, showing a strong positive relationship. This suggested that higher PTX3 levels are linked to increased disease activity in SLE. The correlation was statistically significant ($p < 0.01$). PTX3 could be a potential biomarker for disease activity, but more research is needed to explore its role (Table 3 and figures 4 & 5).

Table (3): Correlations between pentraxin3 Plasma Levels and SLEDAI

Variable	Pearson Correlation	Sig. (2-tailed)	N
SLEDAI	1	0.000	50
Pentraxin	0.922**	0.000	50

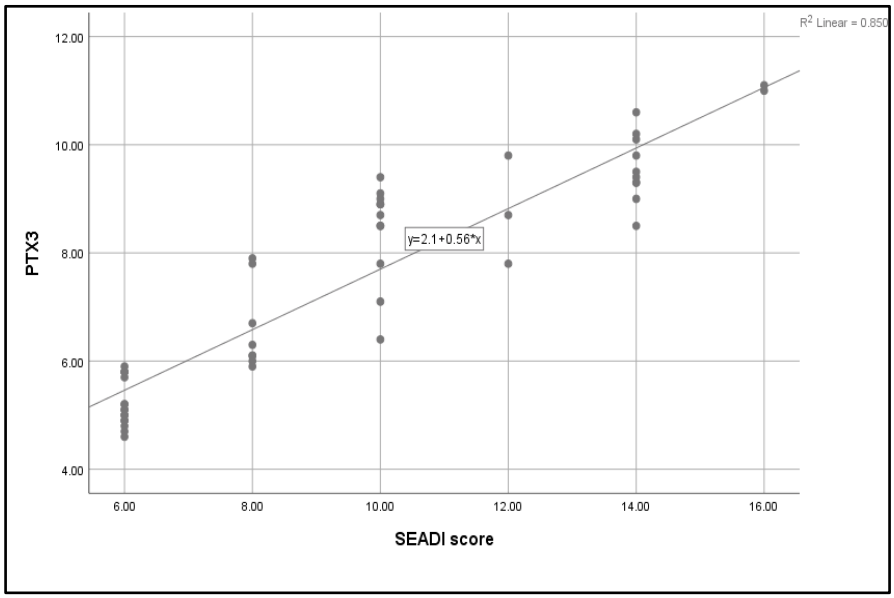

Figure (4): Scatterplot curve showing positive correlation between pentraxin-3 plasma levels and SLEDAI score

Table (4): Correlation of global LV longitudinal strain (GLS) with Disease Activity score (SLEDAI Score) and pentraxin-3 plasma levels.

Variable	Correlation with GLS (r)	p-value
Pentraxin 3 (ng/mL)	-0.666	< 0.001
SLEDAI Score	0.693	< 0.000

This table indicates a strong positive correlation between GLS and the SLEDAI score ($r = 0.693$, $p = 0.000$), suggesting that increased disease activity in SLE is associated with a statistically significant reduction in GLS reflecting impairment of myocardial function in those patients. In addition, the association between serum pentraxin-3 levels and average LVGLS was statistically significant as confirmed by correlation studies ($r = -0.666$, $p < 0.001$) implying that pentraxin-3 levels are significantly related to LV myocardial strain in SLE patients.

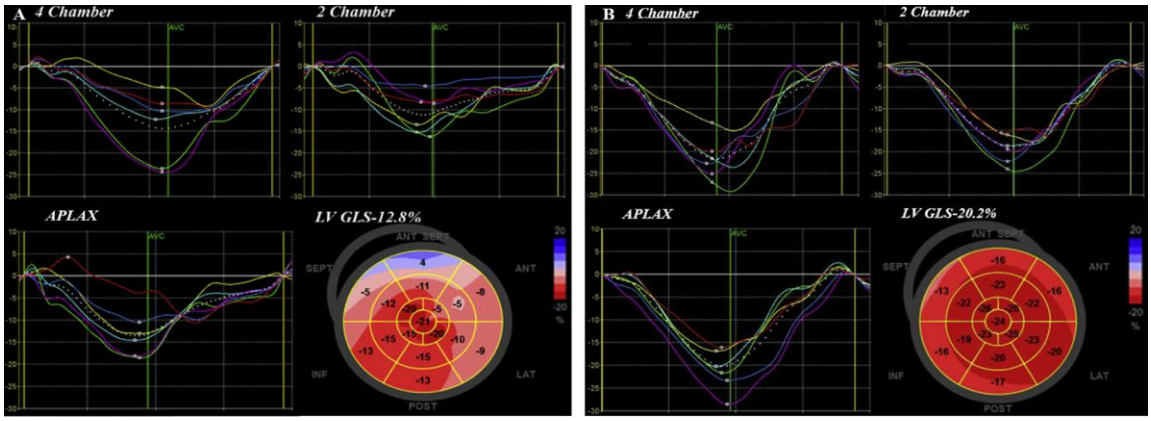

Figure (4): Example of assessment of LV GLS by speckle-tracking strain echocardiography in patients with SLE (A; LV GLS = -12.8%) compared with the control group (B; LV GLS = - 20.2%) displayed with color-coded bull's-eye plots for longitudinal strain. Curves of longitudinal strain per segment and averaged among the segments (dotted line for the three-chamber, four-chamber, and two-chamber apical views) are also displayed. APLAX, Apical long-axis.

DISCUSSION

SLE, a chronic autoimmune illness, is linked to an elevated cardiovascular risk. Pentraxins-3, an acute-phase protein, is a biomarker that is getting more attention in recent years. Global LV strain (GLS) stands out as an indicator for assessment of myocardial performance and early detection of subclinical LV dysfunction in apparently healthy subjects. Serum levels of PTX3 have been demonstrated to increase during periods of active disease and correlate with endothelial activity and vascular injury in SLE patients[12]. This study aimed to explore the correlation between PTX3 levels, left ventricular GLS, and disease activity in SLE. By examining these relationships, we seek to promote early detection of subtle cardiovascular complications and monitoring of disease progression in SLE patients.

The comparison of baseline characteristics between 50 SLE patients and 50 controls showed similar age and gender distributions, but the SLE group had more pronounced clinical manifestations, including arthritis, malar rash, and oral ulcers. Laboratory results revealed lower hemoglobin, elevated ESR, CRP, and 24-hour urinary protein, indicating inflammation and potential renal impairment. Complement proteins C3 and C4 were lower, suggesting active autoimmune activity. There were no significant differences in lipid profiles, suggesting that the increased cardiovascular risk in SLE patients may not be explained by conventional cardiovascular risk factors. Similarly, **Assandri *et al.*'s**[13] study revealed no appreciable variations in BP, BMI, or lipid profiles between SLE patients and healthy controls. Certain clinical manifestations such as renal involvement, arthritis, and anemia were observed in the SLE group,

Our study demonstrated a significant difference in pentraxin-3 (PTX3) levels between the SLE case group (6.6580 ± 1.664281) and the control group (2.3180 ± 0.46891), with a highly significant p-value of 0.000. These findings suggest that PTX3 could serve as a reliable biomarker for inflammation and disease activity in SLE, offering a potential role for distinguishing SLE patients from healthy controls. Various studies report different PTX3 levels in healthy individuals due to the use of different sample matrices (plasma vs. serum). **Hollan *et al.*** [14] found serum PTX3 in healthy subjects to be 1.21 ± 0.59 ng/mL, while **Yamasaki *et al.*** [15] found that an average plasma concentration of 2.00 ng/mL. **Shimada *et al.*** [16] found a similar plasma concentration of 2.2 ± 1.1 ng/mL. In SLE patients, **Hollan *et al.*** [14] demonstrated that a lower serum Pentraxin-3 level of 0.38 ± 0.50 ng/mL, with some patients not having active disease. **Assandri *et al.*** [13] found a plasma value of 2.3 ± 1.1 ng/mL, which is consistent with other studies. While, **Wu *et al.*** [17] revealed that SLE patients had significantly higher PTX3 levels than controls.

The reduced left ventricular GLS in the SLE patient group suggested potential early signs of LV dysfunction, which aligns with findings from several studies in the literature. GLS, a sensitive marker of myocardial function, has been shown to detect subclinical cardiac impairment before changes in traditional measures such as ventricular dimensions or ejection fraction become apparent [18]. In conditions like SLE, where cardiovascular involvement may be subtle and asymptomatic, GLS provides valuable insight into early myocardial dysfunction, which may be missed using standard assessments[19]. Additionally, research has shown that GLS is a sensitive method for measuring LV function in patients with autoimmune disorders [20]. Reduced GLS has been linked to an increased risk of cardiovascular events, making it an important tool for early detection and intervention. Therefore, while traditional cardiac measures like ejection fraction and ventricular dimensions remain critical, integrating GLS could improve early identification of myocardial impairment, particularly in populations at risk for subclinical heart disease, such as those with SLE.

In our study, the optimal cut-off point for pentraxin-3 plasma level (PTX3) at 3.05 showed excellent diagnostic accuracy, with 96% sensitivity, 94% specificity, and an AUC of 0.988. This indicated that PTX3 is highly effective in distinguishing SLE

patients from healthy controls, making it a reliable biomarker for clinical use. **Assandri *et al.* [13]** assessed Pentraxin-3 levels in SLE patients at various phases of SLEDAI and found an operational cut-off value of 2.8 ng/mL that had great specificity (80%) and high sensitivity (100%).

The strong positive correlation between SLEDAI and Pentraxin-3 levels highlights Pentraxin-3 as a potential biomarker for assessing disease activity in SLE patients. The statistical significance of this relationship supports the use of PTX3 for monitoring disease progression. One of the key insights of the **Wu *et al.* [17]** study was the positive correlation between Pentraxin-3 levels and disease activity in SLE. The study found that individuals with more active illness had greater levels of PTX-3, implying that PTX-3 might be useful for assessing disease activity in clinical settings.

While, the study demonstrated increased PTX-3 levels in SLE patients, PTX-3 was not strictly specific to SLE. Higher PTX-3 levels have been observed in other autoimmune and inflammatory diseases, which may restrict its application as a diagnostic marker for SLE by itself. However, PTX-3 may provide additional complementary information if it is combined with other biomarkers or clinical data in respect of diagnosing a case of or monitoring of SLE.

CONCLUSION

This study revealed significant differences between SLE patients and control subjects. Notably, PTX3 plasma levels were significantly higher in SLE patients and strongly correlated with disease activity, indicating its potential as a biomarker for monitoring SLE and attract more attention towards its prognostic, as well as, diagnostic value.to monitor acute cardiac events. SLE, Moreover reduced LV global myocardial strain (GLS) was obvious among SLE patients and showed a positive correlation with disease activity demonstrating a promising diagnostic tool for assessing the magnitude and severity of myocardium injury in such patients.

In addition association between both PTX3 plasma levels and GLS with disease activity in SLE patients support the concept that those patients warrant a comprehensive cardiovascular assessment and additional research is needed to confirm its clinical utility.

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