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Original Research Article

Neutrophil to lymphocyte ratio and platelet to lymphocyte ratio as a marker of disease activity in patients with systemic lupus erythematosus

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a female predominant multiple system involving autoimmune inflammatory disorder. Considering the affordability and wide availability of the recently studied marker of inflammation neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR), this study was done to investigate the relationship between NLR and PLR with the disease activity in patients with SLE.
Methods: In this hospital based cross sectional study done in Assam medical college and hospital, Dibrugarh done over one year period, newly diagnosed SLE patients above the age of 13 years were included. Patients with underlying disorders or on medications that may affect cell counts, drug induced lupus and recent history of blood transfusion were excluded. The disease activity was calculated using SLE disease activity index 2000 (SLEDAI-2K) and the patients were divided in two groups, group-A (SLEDAI-2K score $\leq$9) and group-B (SLEDAI-2K score >9) and correlation was sought with NLR and PLR.
Results: In this study 49 patients were included, with a mean age of 26.33 years and a female predominance (98%). NLR was found to be higher in group-B (11.37 vs 8.05, $p<0.001$) and NLR positively correlated with SLEDAI-2K score ($p<0.001$), ESR ($p<0.001$) and CRP ($p=0.212$). PLR had a positive correlation with SLEDAI-2K score, CRP, and ESR, although it was the correlation with ESR that was statistically significant ($p=0.029$).
Conclusions: NLR and PLR was found to have a positive correlation with SLEDAI-2K scores, CRP and ESR and therefore can serve as an inexpensive marker of disease activity in patients with SLE.

Keywords: SLE, NLR, PLR, SLEDAI-2K

INTRODUCTION

Systemic lupus erythematosus (SLE) is the prototypic autoimmune disease characterized by the production of auto-antibodies against the components of the cell nucleus, in association with diverse clinical manifestation encompassing almost all organ systems. The nature of the disease is complex as it has variable presentations, course, and prognosis, that is characterized by remissions and flares. Extreme diverseness of the disease has led some investigators to propose that SLE represents a syndrome rather than a single disease.[1]

SLE effects women of childbearing age, with a male-to-female ratio of about 9:1 commonly reported.[2]

SLE is a multifactorial disease with evidence of genetic susceptibility, environmental effects, and disturbances in both innate and adaptive immunity manifest by disturbances in apoptotic cell clearance, cytokines, B-cell immunity, and T-cell signaling. Although it has been more than six decades that we know about the presence of

autoantibodies in SLE, efforts are still being made to understand the pathogenetic, diagnostic and prognostic meaning of such autoantibodies. However, the mere presence of an autoantibody is not equivalent to disease and absence of an autoantibody does not exclude a disease.[3]

Polyclonal B cell activation is responsible for autoimmunity which involves release of multiple cytokines and immunoglobulin, which can serve as a marker of inflammation.[4] Any systemic inflammation is accompanied by a change in the number and composition of circulating blood cells such as normocytic normochromic anemia and thrombocytopenia, SLE is no exception to this.[5] Neutrophils and platelets are also involved in the production of these cytokines which in turn causes the activation of more neutrophils and platelets.

NLR and PLR is the proportion of absolute neutrophil count and platelet count to absolute lymphocyte count respectively. NLR is more stable than other WBC subsets as it is less affected by physiological, pathological, and physical factors unlike the traditional markers of inflammation like ESR and CRP.[2]

The role of NLR as a marker of inflammation has been demonstrated in psoriasis and Rheumatoid arthritis, whereas NLR and PLR have been studied as a prognostic index in malignancies.[3]

Recent studies have shown that both NLR and PLR is also increased in SLE patient.[2-4]

The aim of this study is to investigate the relationship of NLR and PLR with inflammatory response and disease activity in SLE patients.

METHODS

The present study, "NLR and PLR as a marker of disease activity in patients with SLE", was carried out in the department of medicine, Assam medical college and hospital, Dibrugarh, with the aim to study NLR and PLR as a marker of disease activity in SLE. This was a cross-sectional hospital based observational study carried out in the above-mentioned department for a period of one year from 1st July 2021 till 30th June 2022. The study population comprised of all the newly diagnosed cases of SLE attending the various out-patient departments of our institute. The sample size was calculated considering a 95% confidence interval with a power of 90% and a correlation of NLR with SLEDAI-2K to be 0.471, a sample size for the present study rounded off to 50.[6]

Inclusion criteria

All SLE patients attending rheumatology OPD and other outpatient departments or in various wards of the department of medicine at Assam medical college and hospital, who fulfilled the EULAR/ACR 2019 SLE criteria, were taken up for the study and patients having age ≥13 years were included.

Exclusion criteria

Diagnosed case of malignancy, evidence of acute or chronic infections, chronic diseases like CLD (chronic liver disease), CKD (chronic kidney disease), CAD (coronary artery disease), etc. Other autoimmune disorders like rheumatoid arthritis, Sjogren syndrome, etc. History of ingestion of drugs that affect blood cell counts like steroid, chloramphenicol, etc. History of blood transfusion in the past 4 months. Patients who were uncooperative or did not give consent and drug-induced lupus were excluded.

Ethical clearance

Due permission from the institutional ethical committee (H) of Assam medical college and hospital was taken before the commencement of the study (ID-AMC/EC/PG/5460 on 7th June 2021).

Data extraction

The disease severity was assessed using the SLEDAI-2K scoring system. Based on this scoring, patients were divided into two groups; group A with a SLEDAI-2K score of ≤9 (with mild disease activity) and group B with a SLEDAI-2K score of >9 (with moderate to severe disease activity).

Data analysis

The statistical analysis was performed using statistical package for social sciences (SPSS for Windows, version 20.0. Chicago, SPSS Inc.) and Microsoft excel 2016. For continuous measurements, results are presented as mean ± standard deviation and comparison was done using the student t test. For discrete data, it was expressed as numbers (%) and was analyzed using the chi square test and Fischer's exact test (where the cell counts were <5 or 0). To measure the associations among continuous variables Pearson's correlation coefficient (r) was used. For all analyses, the statistical significance was fixed at a 5% level ($p<0.05$).

Routine investigations done as a part of the study included complete blood count, renal function test, liver function test, antinuclear antibody panel, C-reactive protein, erythrocyte sedimentation rate, complement factors (C3 and C4), 24 h urinary protein and other selected investigations to rule out any other underlying condition.

RESULTS

Fifty SLE patients who fulfilled the classification criteria were included in this study. The following tables and figures illustrate the important features and results of the study.

The maximum number of patients were seen in the age group of 20-29 years (51%), followed by the age group 30-39 (22%) (Figure 1). The minimum age was 13 years and the maximum age was 60 years. The average age of the patients was 26.33±8.989 SD. In our study 49 (98%) were female patients while only 1 (2%) were male, with an approximate female to male ratio of 9:1.

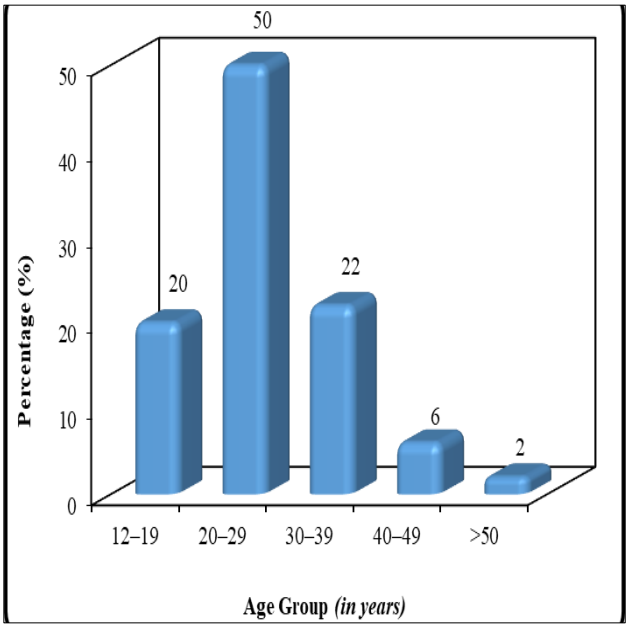

Figure 1: Age distribution.

The 58% of the patients had moderate to severe disease activity (SLEDAI-2K score >9) and 42% had mild disease activity (SLEDAI-2K score ≤9) (Figure 2).

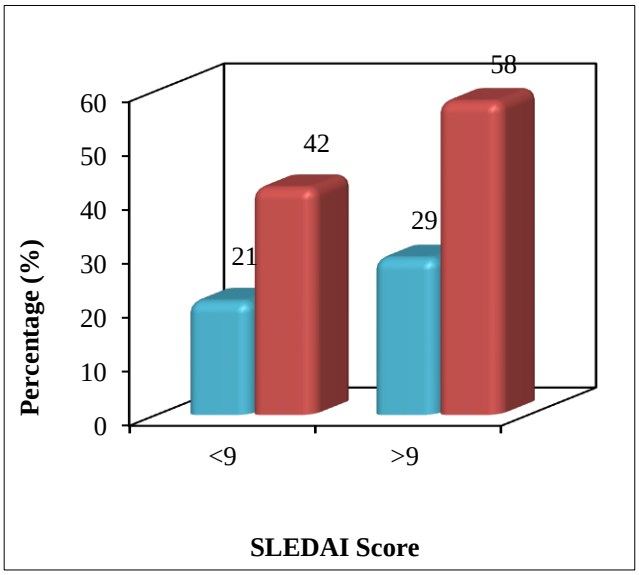

Figure 1: Distribution of disease activity in SLE patients

Table 1 shows the mean values of various demographic, clinical, and laboratory parameters, the mean age of the patients in our study was 26.33±8.98. Disease activity was measured using SLEDAI-2K scoring system, the mean value was 10.08±1.902. Inflammatory markers, ESR and CRP had a mean value of 64.17±42.97 mm AEFH and 3.86±2.47 mg/dl respectively.

The mean TLC was 7546.79±3037.03 per cumm, the mean neutrophil count was 5384.9±2296.4 per cumm, the mean lymphocyte count was 1667.4±774.3 per cumm, and mean platelet count was 1.739×10^5 per cumm. The mean values of key markers that are being studied in this study, the NLR and PLR were 3.45±1.16 and 173.95±61.15 respectively.

Table 1: Demographic, clinical characteristics and laboratory results of patients.

Parameters	Mean	SD (±SD)
Age (in years)	26.33	8.99
SLEDAI score	10.08	1.90
TLC (/cu mm)	7546.79	3037.03
Platelet-count ($\times10^4$/cumm)	173.95	61.16
NLR	3.45	1.16
PLR	120.12	74.78
C3 (mg/dl)	37.14	22.08
C4 (mg/dl)	8.34	6.58
CRP (g/L)	3.87	2.47
ESR mm AEFH	64.17	42.97

Table 2 compares various parameters between SLE patients with mild disease from the patients with moderate to severe disease. In this study, patients who were in group A (SLEDAI-2K score <9) had a mean NLR of 2.505 and a mean PLR of 87.113. The mean NLR and PLR in group B (SLEDAI-2K >9) were 4.05 and 143.34 respectively. NLR and PLR are found to be higher in group B patients and the difference was significant (NLR<0.001, PLR=0.01). TLC, neutrophil count, lymphocyte count, ESR, CRP, C3, and C4 have also been compared in Table 3.2. TLC, neutrophil count, ESR, and CRP were higher in group B whereas lymphocyte count, C3, and C4 higher in group A, but the difference was statistically significant only for lymphocyte count (p<0.001) and ESR (p=0.032).

NLR had a statistically significant positive correlation with the SLEDAI-2K score and ESR (p<0.001). NLR also had a positive correlation with CRP but it was not statistically significant (Figure 3 A-C). PLR had a positive correlation with SLEDAI-2K score, CRP, and ESR, although it was only the correlation with ESR that was statistically significant (p=0.029) (Figure 3 D-E). SLEDAI-2K Score had a positive correlation with both ESR (r=0.737) and CRP (r=0.256), but the correlation with only ESR was statistically significant (p<0.001). SLEDAI-2K score had a negative correlation with complement factors, both C3 and C4 (Figure 4 A-D).

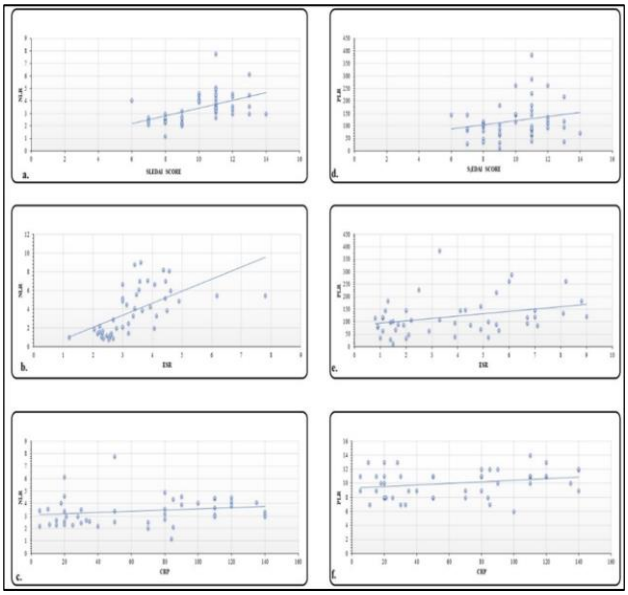

Figure 3 (A-F): Correlation of NLR and PLR with SLEDAI score, ESR and CRP respectively.

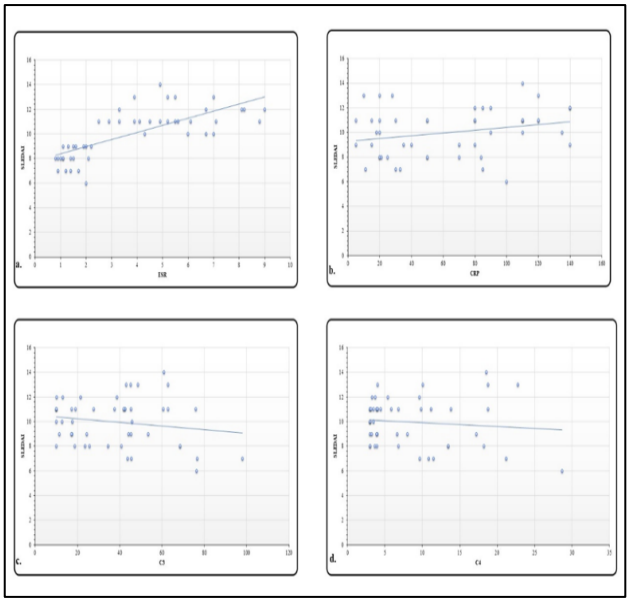

Figure 4 (A-D): Correlation of SLEDAI-2K score with ESR, CRP, C3 and C4 respectively.

Table 2: Laboratory results according to SLEDAI-2K score.

Parameters	Group A (SLEDAI 2K score ≤9)		Group B (SLEDAI 2K score >9)		P value
	Mean	±SD	Mean	±SD	
WBC (per cumm)	8545.13	3597.45	6955.19	2540.96	0.097
ESR mm AEFH	53.16	36.30	74.86	45.30	0.032
Neutrophil (per cumm)	5721.10	2.75	5.14	1.92	0.407
Lymphocyte (per cumm)	2152.60	0.83	1.33	0.52	<0.001
Platelet (×10^4/cumm)	166444.00	58.10	179.36	63.88	0.501
NLR	2.51	0.56	4.05	1.04	<0.001
PLR	87.11	44.36	143.35	83.42	0.010
C3 (mg/dl)	40.82	24.73	34.56	20.10	0.350
C4 (mg/dl)	9.47	7.36	7.55	5.99	0.335
CRP (mg/l)	1.51	0.44	5.56	1.79	0.67

Table 4: Similar studies comparing NLR between low and high disease activity in SLE patients.

Study	Patients with active SLE			Patients with inactive SLE		
	Cases	Mean	±SD	Cases	Mean	±SD
Wu et al[10]	64	3.25	0.70	52	2.34	0.41
Soliman et al[17]	60	3.88	1.33	60	2.21	1.50
Qin et al[18]	58	6.32	1.28	42	4.06	1.33
Yu et al[19]	64	3.25	0.70	52	2.34	0.41
Present study (2021-22)	29	4.05	1.04	21	2.51	0.56

Table 4: Correlation of NLR with SLEDAI in various studies.

Study	Year	R value	P value
Qin et al[18]	2015	0.471	<0.001
Wu et al[16]	2016	0.312	<0.001
Kim et al[23]	2017	0.282	0.002
Soliman et al[17]	2018	0.525	<0.001
Yu et al[19]	2018	0.211	0.002
Present study	2021-22	0.503	<0.001

Table 5: Correlation of PLR with SLEDAI in various studies.

Study	Year	R value	P value
Qin et al[18]	2015	0.440	<0.001
Wu et al[16]	2016	0.559	0.001
Kim et al[23]	2017	0.117	0.204
Soliman et al[17]	2018	0.512	<0.001
Xie et al[21]	2018	-0.159	0.105
Present study	2021-22	0.220	0.142

DISCUSSION

SLE is a female predominant multisystem involving autoimmune inflammatory disorder that occurs as a result of immune-dysregulation in the presence of certain predisposing factors that are both genetic and environmental. The circulating blood cells, especially the leukocytes actively participate in mediating the inflammation and this is associated with changes in their composition in blood.

NLR and PLR is a relatively new marker of inflammation and sepsis that has been studied in acute pancreatitis, malignancies, rheumatological disorders, and also in COVID-19. Recently these two markers have been investigated in multiple studies, which has demonstrated fruitful results. Considering the affordability and wide availability of this marker, this study was done to investigate the relationship between NLR with the inflammatory response and disease activity in patients with SLE.

Age distribution

In our study maximum patients belonged to the age group of 20-29 years (50%, n=25), followed by the age group of 30-39 (22%, n=11). The minimum age was 13 years whereas the maximum age was 60 years, with a mean age of 26.33 years. In multiple studies done across the nation, similar results were found. In a study done by Saigal et al the mean age of SLE patients was 28 years and the maximum number of patients belonged to the age group of 20-29 years.[7] In a similar study done in the central part of India, the mean age was 26.18 years whereas in a study done in the southern part of the country the mean age was 25.45 years.[8,9] In a study done in China by Wu et al the mean age was 26 years, similar to our results.[10] However, in a study done in the United Kingdom by Nightingale et al the mean age of diagnosis was 49.4 years and the maximum number of patients belonged to the age group of 40-49 years.[11]

Gender distribution

Out of the 50 newly diagnosed patients, 2% (n=1) were male and the rest were female 98% (n=49). In a study done by Jagadish et al 96.7% were females whereas in a study done by Santharam et al, 86% were females.[9,11] In a study done in northeast India, females constituted 96.4% of the study population.[13] In a study done in Sweden with over 1200 participants, 86.46% of patients were female, thus making it evident that geographical location influences gender distribution in SLE.[14]

Disease activity in SLE patients

To assess the relation of disease activity with NLR and PLR, patients were categorized into 2 groups according to their SLEDAI-2K scores; group A with a score of $\leq$9 and group B with a score of >9. A SLEDAI-2K score of greater than equal to 10 is consistent with high disease activity status.[15] In our study, 42% of the newly diagnosed SLE patients belonging to group A had a score of $\leq$9, and 58% of patients, belonging to group B had a score >9. A score of less than equal to 9 is associated with mild disease activity whereas a score of more than 9 is associated with moderate to severe disease activity. The average SLEDAI-2K score in our study was 10.08±1.902. In a similar study done by Wu et al in newly diagnosed SLE patients, 55.17% of patients had a SLEDAI-2K score of more than 9.[16]

Comparing NLR of SLE patients with mild disease activity with moderate to severe disease activity

In this study, SLEDAI-2K scoring was done for all the patients, and patients were divided into two groups, one with mild disease activity (Group A) and one with moderate to severe disease activity (Group B). Various parameters were compared between the two groups, with special reference to NLR, PLR, c3, c4, CRP, and ESR. The mean NLR was higher in the group with higher disease activity, which was statistically significant (Group A 2.505, group B 4.054, p<0.001). Similar findings were seen in multiple studies done internationally, most of them were done in China.

Comparing PLR of SLE patients with mild disease activity with moderate to severe disease activity

In our study mean PLR was also higher in patients with moderate to severe disease activity (Group A 87.112±44.35, group B 143.34±83.42, p=0.01). There are a few studies with similar results where PLR was more in SLE patients with higher disease activity. Wu et al in their study found the mean PLR was higher in patients with higher disease activity, (130.54 vs 171.79, p=0.040).[16] Qin et al also found that PLR positively correlated with SLEDAI score.[18] Interestingly, Yolbas et al did not find any significant difference in PLR between patients with active and inactive disease, and on the other hand, Xie et al found that the PLR was higher in patients with SLEDAI-2K score of $\leq$9, than a score of >9, although it was not statistically significant (179.68 vs 168.96, p=0.589).[20,21]

The difference in findings in the latter two studies could be because the inclusion of cases was not restricted to only newly diagnosed patients of SLE and patients might have been already on medications that could have affected the cell counts.

Correlation of NLR with SLEDAI-2k score, CRP and ESR

In our study, we found a positive correlation of NLR with SLEDAI-2K score (r=0.503, p<0.001), CRP (r=0.186, p=0.212), and ESR (r=0.593, p<0.01), however, the correlation was not statistically significant with CRP. A positive correlation of NLR with SLEDAI-2K score has been established in multiple studies, that was evaluated in a meta-analysis by Ma et al mentioned below in table.[22]

A positive correlation of NLR with CRP and ESR has also been established in various studies. Soliman et al found a statistically significant correlation of NLR with CRP and ESR, {(NLR and CRP, r=0.363, p=0.021), (NLR and ESR, r=0.383, p=0.015)}.[17] Qin et al found similar results, {(NLR and CRP, r=0.509, p<0.01), (NLR and ESR, r=0.610, p<0.01)}.[18]

Correlation of PLR with SLEDAI-2k score, CRP and ESR

In our study, we found a positive correlation of PLR with SLEDAI-2K score (r=0.220, p=0.142) and ESR (r=0.322, p=0.029) and a negative correlation with CRP (r=-0.014, p=0.928), although it was not statistically significant. Ma et al also performed a meta-analysis where correlation of PLR with SLEDAI score was investigated, it has been mentioned below.[22]

CONCLUSION

In this observational study we found that NLR and PLR were higher in patients with moderate to severe disease activity. NLR had a significant positive correlation with the SLEDAI-2K score and ESR, whereas PLR had a significant positive correlation with only ESR. SLEDAI-2K score positively correlated with both CRP and ESR and had an inverse correlation with complement components, C3 and C4. Thus, both NLR and PLR can be used as a cheap and easily available markers for both disease activity and inflammation in SLE patients.

However, like any other study, our study has its limitations that need to be mentioned. The sample size of this study was relatively small but the findings of this study emphasize the need for further study in a larger population. The cross-sectional design of the study limited the ability to deduce a causal relationship between the NLR and PLR and SLEDAI-2K scores. Also, NLR and PLR alone may not be enough to differentiate an underlying infection from a flare. The analyses that were done were based on a single measurement of whole blood count and therefore it may not reflect the relation over time.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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