Sapporo Medical Journal
Volume 54, Issue 05, May, 2020

Blood Indices in Rheumatoid Arthritis and Systemic Lupus Erythematosus Patients and Their Relation to Disease Activity

Samar H Goma[1], Nihal Ahmed Fathi[1], Maha Gamal Seddek[1], SafaaA Mahran[1]

[1] Physical Medicine, Rheumatology and Rehabilitation Department

Abstract

Abstract— Rheumatoid arthritis (RA) and systemic lupus erythematosus are considered chronic inflammatory autoimmune diseases with unknown etiology and systemic involvement.Both PLR and NLR are associated with the inflammatory status. Based on this, wehypothesized that both NLR and PLR may also be correlated with the disease activity of RA and SLE.

Patients and Methods— Fifty RA patients, 50 SLE patients and 50 healthy controls were enrolled in this study. The disease activity in RA patients was assessed by DAS 28 and the disease activity in SLE patients was assessed by SLEDAI score.

Results— In RA group, NLR mean±SD of 2.6±1.3,1.7±1.03 **,**1.9±1.8in low, moderate and high activity respectively ,and PLR mean±SD of 231.4±161.8, 163.01±86.8, 187.5±73.9in low, moderate and high activity respectively ,and there is no statistical significant correlation **(p-value > 0.05)** between disease activity (DAS score) and (NLR & PLR). In SLE group, NLR mean±SD2.42±1.49, 3.42±3.14 and 3.06±1.31in low, moderate and high activity respectively, and PLR mean±SD was 201.90±107.02, 253.29±255.47and 241.57±134.19in low, moderate and high activity respectively. Median – IQR measured for PLR and was (198.2 – 125.8) due to odd numbers in patients with moderate SLEDAI. There was no statistical significant correlation **(p-value > 0.05)** between disease activity (SLEDAI) and (NLR & PLR).

Conclusion— This study confirmed that Rheumatoid arthritis &Systemic Lupus Erythematosus cause rising in blood indices in the form of Neutrophil/Lymphocyte ratio and Platelet/Lymphocyte ratio but no correlation between them and disease activity.

Keywords— Rheumatoid Arthritis (RA), Systemic Lupus Erythematosus (SLE), Neutrophil Lymphocyte Ratio (NLR), Platelet Lymphocyte Ratio (PLR)

Introduction

Rheumatoid arthritis (RA) and systemic lupus erythematosus are considered chronic inflammatory autoimmune diseases with unknown etiology and systemic involvement (1)(2)(3)

A common characteristic of all autoimmune disease is the chronic inflammatory process, which is triggered by autoantigens and maintained by both environmental and genetic factors.(4)

Inflammatory status was assessed using many different markers of inflammation, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), interferon (IFN) and interleukin-6(IL-6). For a large number of malignancy studies, the neutrophil to lymphocyte ratio (NLR) and the platelet to lymphocyte ratio (PLR) were recently investigated as new prognostic indicators. (5)(6).

Usually there is alterations in blood cells quantity and composition in case of systemic inflammation. That is why the components of blood can be used for the assessment of inflammation in systemic diseases (7).
Previous studies have shown that NLR and PLR are associated with morbidity and mortality in many chronic diseases including hypertension,heart failure, infective endocarditis, acute coronary syndromes and type 2 diabetes (8)(9)(10)(11)(12)

Neutrophils are primed in active RA patients. Neutrophils can aggravate disease by secretion of B-lymphocyte stimulator (BLyS), tumor necrosis factor, IL-17, proteases, prostaglandins, and reactive oxygen intermediates causing activation of other cells (13)(14)(15).

Neutrophil to lymphocyte ratio (NLR) is the proportion of absolute neutrophil count to lymphocyte count, which is derived from routine complete blood count (CBC) test.(16)(17)

NLR has been a marker of subclinical inflammation in recent years, and has been used in many diseases in c onjunction with other inflammatory markers to assess
inflammation (18)

Extensive evidence shows that PLTs, including monocytes, neutrophils and T cells, can bind to leukocytes. Still, surprisingly, the amount of aggregates of circulating leukocytes-
PLTs increases after activation of PLTs. (19)(20)

In patients with autoimmune inflammatory diseases such as systemic lupus
erythematosus (SLE), rheumatoid arthritis (RA), and multiple scelerosis, the number of circulating activated PLTs is increased.(21)(22)

We aimed to investigate NLR and PLR in RA and SLE patients and, their correlation with disease activity.

Patients and methods

This cross-sectional study was conducted at Physical Medicine,Rheumatology and Rehabilitation Department and outpatient clinic at Assiut University Hospitals, in the period between January 2019 and April 2019. Fifty patients were diagnosed as rheumatoid arthritis according to 2010 ACR revised criteria(23), 50 patients were diagnosed as systemic lupus erythematosus according to 2015 ACR/SLICC revised criteria (24) and 50 healthy age and sex matched subjects who served as controls. Patients aged > 18 years for both RA and SLE were included in the study.

Exclusion criteria for RA&SLE

1) Subjects with hematologic disorders other than anemia, concomitant infectious or inflammatory diseases such as ulcerative colitis, liver, or kidney disease
2) coronary heart disease
3) other immunological diseases other than RA&SLE including overlap syndrome.

Complete history was recorded and thorough general and musculoskeletal examination was performed. Various basal characteristics and parameters such as age, sex, disease duration, age of onset, duration of morning stiffness, presence of extra-articular manifestations and their current treatment were recorded. The disease activity in RA patients was assessed by the 28 joint count Disease Activity Score (DAS 28) using the number of swollen and tender joints, erythrocyte sedimentation rate (ESR) and patient's global status, and pain evaluated by the visual analogue scale (VAS) range from 0 to 100 mm. The patients were allocated to 3 groups according to the Disease Activity Score for 28 joint counts (DAS28): DAS28 < 2.6 indicated remission, DAS28 > 2.6 < 5.1 low/moderate activity and DAS28 > 5.1 high disease activity. We assessed the disease activity for SLE patients according to the SLE Disease Activity Index 2000.The SLEDAI is a global index that evaluates disease activity over the previous 10 days and includes 24 items collecting specific manifestations in 9 organ systems: neurological, musculoskeletal, renal, mucocutaneous, general, heart, respiratory, vascular, and hematological. The maximum score is 105. Activity categories have been defined on the basis of SLEDAI scores: no activity (SLEDAI= 0), mild activity (SLEDAI= 1-5), moderate activity (SLEDAI =6-10), high activity (SLEDAI =11-19), very high activity (SLEDAI ≥20).(25)

Full laboratory investigations had been done for RA and SLE patients

***CBC** (neutrophils, platelets, lymphocytes) assessment of NLR by dividing the absolute neutrophil count on the absolute lymphocyte count and assessment of PLR by the platelet count dividing on the absolute lymphocyte count .

***ESR**
***Immunological tests** (RF, anti-CCP) for RA patients, and (ANA, anti-dsDNA, C3, C4) for SLE patients.

Ethical Considerations

The research was done according to Assiut medical colleague ethics committee for research, keeping patient confidentiality, safety, and verbal and written consent was taken from patients

Statistical analysis:

Data were analyzed using Statistical Program for Social Science (SPSS) version 15.0. Quantitative data were expressed as mean± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The following tests were done:

Chi-square test: was used when comparing between non-parametric data.
A one-way analysis of variance (ANOVA): when comparing between more than two means.
Independent-samples t-test of significance: was used when comparing between two means.

Probability (P-value)

- P-value <0.05 was considered significant.
- P-value <0.001 was considered as highly significant.
- P-value > 0.05 was considered insignificant.

Results

Our results showed that the age of patients with RA ranged between 20 and 74 years with mean was (49 ± 12.9) years. Out of them, 47 (94%) patients were females and 3(6%) patients were males. The disease duration ranged from (4 -360) months with mean was (115.8± 83.7).

The age of patients with SLE ranged between 20 and 62 years with mean of (39.2 ± 12.5) years. Out of them, 47 (94%) patients were females and 3(6%) patients were males. The disease duration ranged from (3 – 204) months with mean±SD was (63.6 ± 42.4). The age of control ranged between (34 – 59yrs)with mean±SD of 42.8 ± 7.09, 3of them were males (6%) and 47 females (94%).

No statistical difference was detected between control group and either RA nor SLE groups as regarding age and sex.

Our results showed that in RA group, ESR ranged between 10-140 with mean±SD (43.2±26.6), and (68%) had positive RF. Complete blood count done showed white blood cells ranged 2.7 – 15.8, with neutrophils 0.9 – 11.5 and lymphocytes0.8 – 3.98, Hemoglobin levels ranged between 8.2 – 14.5, platelets ranged between169 – 641. Neutrophils/lymphocytes ratio(NLR) calculated ranged between 0.5 – 8.3with mean±SD1.8 ± 1.4 and platelets /lymphocytes(PLR) ratio also calculated ranged between 72.2 – 567.25, and mean±SD(175.9 ± 87.2).

While in SLE group, ESR range was 15-130 with mean±SD (49.76±30.48), ANA was Positive in 41 patients (82%) , and as regard anti-ds DNA38 patients were positive (76%) .Complete blood count was done showed white blood cells ranged between 2.2 – 15.9, with neutrophils 0.95–9.4 and lymphocytes0.2 –5.3, Hemoglobin levels ranged between7.8 – 14.5, platelets ranged between 104.6 – 442. Neutrophils/lymphocytes ratio(NLR) calculated ranged between0.4 – 16.1with mean±SD of 2.9 ± 2.4 and platelets /lymphocytes(PLR) ratio also calculated ranged between 50.2 – 1378 with mean±SD of 232.1 ± 193.9 as shown in **table (1).**

In RA patients our results showed no statistical significant difference (p-value > 0.05) & negative correlation (r = - 0.11) between ESR and NLR. No statistical significant difference (p-value > 0.05) & positive correlation (r = 0.088) between ESR and PLR as shown in **figure 1&2.**

As regarding SLE patients we found no statistical significant difference (p-value > 0.05) & positive correlation (r = 0.152) between ESR and NLR. No statisticalsignificant difference (p-value < 0.05)&positive correlation (r = 332) between ESR and PLR as shown in **figure 3&4**.

As regardingDAS28 score in RA group, there were 3(6%) patients with low disease activity,29(58%) with moderate disease activity,and 18(36%) patients with high disease activity with mean±SD (2.9±0.14, 4.6±0.43, 6.1±0.57) respectively.

At the same time, SLEDAI of SLE patients showed that19(38%) patients had mild activity, 24(48%) patients had moderate activity and 7(14%) patients had high activity with mean±SD of 5.2±1.3, 9.5±1.6, 16.9±3.02 respectively.

Our results showed that **,** in RA group NLR mean±SD of 2.6±1.3,1.7±1.03 **,**1.9±1.8in low, moderate and high activity respectively ,and PLR mean±SD of 231.4±161.8, 163.01±86.8, 187.5±73.9in low, moderate and high activity respectively ,and there was no statistical significant correlation **(p-value > 0.05)** between disease activity (DAS score) and (NLR & PLR) in RA group. Shown in **table (2)**

In SLE group, NLR mean±SD2.42±1.49, 3.42±3.14 and 3.06±1.31in low, moderate and high activity respectively, and PLR mean±SD was 201.90±107.02,253.29±255.47and 241.57±134.19in low, moderate and high activity respectively. Median – IQR measured for PLR and was(198.2 – 125.8)due to odd numbers in patients with moderate SLEDAI. There is no statistical significant correlation **(p-value > 0.05)** between disease activity (SLEDAI) and (NLR & PLR) in SLE group**table (3).**

Discussion

Rheumatoid arthritis (RA) is a chronic, symmetrical, inflammatory autoimmune disease that initially affects small joints, progressing to larger joints, the skin, eyes, heart, kidneys, and lungs. Often, the bone and cartilage of joints are destroyed, and tendons and ligaments weaken.(26)

SLE is a complex autoimmune disease with a possible involvement of multiple organs including skin and mucosa, kidneys, central and peripheral nervous system, cardiovascular system, bone and peripheral blood and many others. (27)

In RA patients, our results showed that Neutrophils/Lymphocytes ratio range was (0.5- 8.3) &Platelet/Lymphocytes ratio range was(72.2 – 567.25). High disease activity was found in (36%) of the patients, while moderate disease activity was found in (58%) & low disease activity patients was found in (6%). In the SLE patients, our results showed that Neutrophils/Lymphocytes ratio range was (0.4– 16.1) & Platelet/Lymphocytes ratio range was (50.2 – 1378). High disease activity was found in (14%) of patients, those with moderate disease activity (48%), and low disease activity patients were (38%).

Inflammatory processes in rheumatic inflammatory diseases include inflammatory cells and molecules that cause changes in the number, shapes, and sizes of cells in the bone marrow and peripheral blood. The effect of inflammatory diseases on hematological indices is controversial.(28)

The cause of an increased NLR level is increased cytokines and the inflammatory process.(29)
A number of studies have suggested that joint destruction and disease activity in inflammatory arthritis directly correlate with the recruitment of neutrophils in the synovium(30). **Uslu et al.(2015)** demonstrated that NLR and PLR were two new inflammatory markers which could be used to assess disease activity in patients with RA.(31)
In RA patients,we found that NLR&PLR are increased in RA, but they did not correlate with DAS28.
These results agree with the study of **Mikhael et al.(2013)** who showed that NLR did not correlate with RA disease activity .They also noted that NLR didn't differ significantly between highly and moderately

active RA patients.So,they concluded that NLR could not be used as a marker of disease activity but could be used as a marker of on going inflammation. (32)

Imtiaz et al.(2012) found that there was no significant relationship between NLR and arthritis of rheumatoid arthritis patients.(33)

Similarly **Bhatnager et al.(2017)** found that PLR did not significantly correlate with DAS-28 score.(34)
On the other hand, **Fawzy et al.(2017)** showed that NLR significantly correlated with disease activity indices in recent onset RA patients thus reflecting systemic inflammation with its advantages of beingavailable, easy and cost accessible being as reliable as the DAS-28 hence it could be used as a marker of disease activity.(35)
Fu et al.(2015) found thatIn RA patients both NLR and PLR were enhanced and positively correlated with CRP and DAS28. Additionally, NLR was positively correlated to ESR, but not to PLR. Knowing that ESR, CRP, and DAS28 are useful indices for estimating RA activity, their findings suggest that both NLR and PLR could be potential indices for evaluating disease activity in RA.(36)

During inflammation, the increase in NLR may be due to either a drop in lymphocyte count or an increase in neutrophil count, or both. NLR represents two immune system compartments: the neutrophil representing the innate system, and the adaptive system representing the lymphocyte.(37) Other studies found significant correlation of PLR and NLR with disease activity .(31)(37)(38)

In the SLE group, Our results showed that NLR&PLR are increased , but also they did not correlate with SLEDAI score .Similarly ,**Oehadianet al.(2013)** reported that NLR did not correlate with disease activity and clinical subsets.(39)

Xie et al.(2018) demonstrated that PLR was significantly higher in patients with SLE, but a correlation between PLR and SLEDAI was not found.(40)

Conversely, **Qin et al.(2016) &Li et al (2015)** reported that NLR was associated with disease activity and the presence of nephritis and PLR also correlated with SLEDAI.(41)(42). Likewise , **kim et al.(2017)** reported that both PLR and NLR reflect activity of SLE and NLR was more valuable than PLR for predicting nephritis.

Yolbas et al . (2016)reported that PLR was significantly higher in the SLE and it may be an important biomarker for the diagnosis of SLE.(43) Also**, Wu et al (2016)** reported that PLR and NLR were significantly ↑ in patients with SLE compared with healthy controls. Both PLR and NLR were positively correlated with SLEDAI-2K ($r=0.298$, $P<0.001$ and $r=0.312$, $P<0.001$, respectively) (44).

This study has some limitations as small sample size, the cross-sectional nature of this study so, longitudinal study with follow up of the patients is recommended. Also, study specific organ affection in both diseases in correlation to blood parameters (example, arthritis in RA,nephritis in SLE) are preferred.

Authors declare no conflict of interest.

Conclusion

This study confirmed that Rheumatoid arthritis &Systemic Lupus Erythematosus cause rising in blood indices in the form of Neutrophil/Lymphocyte ratio and Platelet/Lymphocyte ratio but no correlation between them and disease activity.

References

[1] Assayag D, Lee JS. Rheumatoid arthritis associated interstitial lung disease: a review. Med (Buenos Aires). 2014;74(2).
[2] Willemze A, Toes RE, Huizinga TW, Trouw LA. New biomarkers in rheumatoid arthritis. Neth J Med. 2012;70(9):392–9.
[3] Anisur R, David A. Systemic lupus erythematosus. N Engl J Med. 2008;358(9):929–39.
[4] Goodnow CC. Multistep pathogenesis of autoimmune disease. Cell. 2007;130(1):25–35.
[5] Templeton AJ, Ace O, McNamara MG, Al-Mubarak M, Vera-Badillo FE, Hermanns T, et al. Prognostic role of platelet to lymphocyte ratio in solid tumors: a systematic review and meta-analysis. Cancer Epidemiol Prev Biomarkers. 2014;23(7):1204–12.
[6] Templeton AJ, McNamara MG, Šeruga B, Vera-Badillo FE, Aneja P, Ocaña A, et al. Prognostic role of neutrophil-to-lymphocyte ratio in solid tumors: a systematic review and meta-analysis. JNCI J Natl Cancer Inst. 2014;106(6).
[7] Kisacik B, Tufan A, Kalyoncu U, Karadag O, Akdogan A, Ozturk MA, et al. Mean platelet volume (MPV) as an inflammatory marker in ankylosing spondylitis and rheumatoid arthritis. Jt Bone Spine. 2008;75(3):291–4.
[8] Tamhane UU, Aneja S, Montgomery D, Rogers E-K, Eagle KA, Gurm HS. Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. Am J Cardiol. 2008;102(6):653–7.
[9] Guasti L, Dentali F, Castiglioni L, Maroni L, Marino F, Squizzato A, et al. Neutrophils and clinical outcomes in patients with acute coronary syndromes and/or cardiac revascularisation. Thromb Haemost. 2011;106(10):591–9.
[10] Uthamalingam S, Patvardhan EA, Subramanian S, Ahmed W, Martin W, Daley M, et al. Utility of the neutrophil to lymphocyte ratio in predicting long-term outcomes in acute decompensated heart failure. Am J Cardiol. 2011;107(3):433–8.
[11] Afsar B. The relationship between neutrophil lymphocyte ratio with urinary protein albumin excretion in newly diagnosed patients with type 2 diabetes. Am J Med Sci. 2014;347(3):217–20.
[12] Sunbul M, Gerin F, Durmus E, Kivrak T, Sari I, Tigen K, et al. Neutrophil to lymphocyte and platelet to lymphocyte ratio in patients with dipper versus non-dipper hypertension. Clin Exp Hypertens. 2014;36(4):217–21.
[13] Kouri V-P, Olkkonen J, Ainola M, Li T-F, Björkman L, Konttinen YT, et al. Neutrophils produce interleukin-17B in rheumatoid synovial tissue. Rheumatology. 2014;53(1):39–47.
[14] Cascao R, Rosario HS, Souto-Carneiro MM, Fonseca JE. Neutrophils in rheumatoid arthritis: more than simple final effectors. Autoimmun Rev. 2010;9(8):531–5.
[15] McInnes IB, Schett G. The pathogenesis of rheumatoid arthritis. N Engl J Med. 2011;365(23):2205–19.
[16] Torun S, Tunc BD, Suvak B, Yildiz H, Tas A, Sayilir A, et al. Assessment of neutrophil-lymphocyte ratio in ulcerative colitis: a promising marker in predicting disease severity. Clin Res Hepatol Gastroenterol. 2012;36(5):491–7.
[17] Ahsen A, Ulu MS, Yuksel S, Demir K, Uysal M, Erdogan M, et al. As a new inflammatory marker for familial Mediterranean fever: neutrophil-to-lymphocyte ratio. Inflammation. 2013;36(6):1357–62.
[18] Sen BB, Rifaioglu EN, Ekiz O, Inan MU, Sen T, Sen N. Neutrophil to lymphocyte ratio as a measure of systemic inflammation in psoriasis. Cutan Ocul Toxicol. 2014;33(3):223–7.
[19] Zamora C, Cantó E, Nieto JC, Ortiz MA, Diaz-Torné C, Diaz-Lopez C, et al. Functional consequences of platelet binding to T lymphocytes in inflammation. J Leukoc Biol. 2013;94(3):521–9.
[20] Barnard MR, Linden MD, FRELINGER III AL, Li Y, Fox ML, Furman MI, et al. Effects of platelet

binding on whole blood flow cytometry assays of monocyte and neutrophil procoagulant activity. J Thromb Haemost. 2005;3(11):2563–70.

[21] Linge P, Fortin PR, Lood C, Bengtsson AA, Boilard E. The non-haemostatic role of platelets in systemic lupus erythematosus. Nat Rev Rheumatol. 2018;14(4):195.

[22] Boilard E, Blanco P, Nigrovic PA. Platelets: active players in the pathogenesis of arthritis and SLE. Nat Rev Rheumatol. 2012;8(9):534.

[23] Aletaha D, Neogi T, Silman AJ, Funovits J, Felson DT, Bingham III CO, et al. 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. Arthritis Rheum. 2010;62(9):2569–81.

[24] Salehi-Abari I. ACR/SLICC revised criteria for diagnosis of systemic lupus erythematosus. Autoimmune Dis Ther Approaches. 2015;2(1):1–4.

[25] Gladman DD, Ibanez D, Urowitz MB. Systemic lupus erythematosus disease activity index 2000. J Rheumatol. 2002;29(2):288–91.

[26] Lee J-E, Kim IJ, Cho M-S, Lee J. A case of rheumatoid vasculitis involving hepatic artery in early rheumatoid arthritis. J Korean Med Sci. 2017;32(7):1207–10.

[27] Paley MA, Strand V, Kim AHJ. From mechanism to therapies in systemic lupus erythematosus. Curr Opin Rheumatol. 2017;29(2):178–86.

[28] Ozturk C, Balta S, Balta I, Demirkol S, Celik T, Turker T, et al. Neutrophil–lymphocyte ratio and carotid–intima media thickness in patients with Behcet disease without cardiovascular involvement. Angiology. 2015;66(3):291–6.

[29] Okyay GU, İnal S, Öneç K, Er RE, Paşaoğlu Ö, Paşaoğlu H, et al. Neutrophil to lymphocyte ratio in evaluation of inflammation in patients with chronic kidney disease. Ren Fail. 2013;35(1):29–36.

[30] Sadik CD, Kim ND, Iwakura Y, Luster AD. Neutrophils orchestrate their own recruitment in murine arthritis through C5aR and FcγR signaling. Proc Natl Acad Sci. 2012;109(46):E3177–85.

[31] Uslu AU, Küçük A, Şahin A, Ugan Y, Yılmaz R, Güngör T, et al. Two new inflammatory markers associated with Disease Activity Score-28 in patients with rheumatoid arthritis: neutrophil-lymphocyte ratio and platelet-lymphocyte ratio. Int J Rheum Dis. 2015;18(7):731–5.

[32] Mikhael EM, Ibrahim TN. Neutrophil/lymphocyte ratio is not correlated with disease activity in rheumatoid arthritis patients. Iraqi J Pharm Sci (P-ISSN 1683-3597, E-ISSN 2521-3512). 2013;22(2):9–14.

[33] Imtiaz F, Shafique K, Mirza SS, Ayoob Z, Vart P, Rao S. Neutrophil lymphocyte ratio as a measure of systemic inflammation in prevalent chronic diseases in Asian population. Int Arch Med. 2012;5(1):2.

[34] Bhatnagar M, Agrawal BK, Dubey AB, Aggarwal G. Rheumatoid Arthritis Disease Activity Assessment Using Neutrophil-To-Lymphocyte Ratio and Platelet-To-Lymphocyte Ratio.

[35] Fawzy RM, Said EA, Mansour AI. Association of neutrophil to lymphocyte ratio with disease activity indices and musculoskeletal ultrasound findings in recent onset rheumatoid arthritis patients. Egypt Rheumatol. 2017;39(4):203–6.

[36] Fu H, Qin B, Hu Z, Ma N, Yang M, Wei T, et al. Neutrophil-and platelet-to-lymphocyte ratios are correlated with disease activity in rheumatoid arthritis. Clin Lab. 2015;61(3–4):269–73.

[37] Chandrashekara S, Mukhtar Ahmad M, Renuka P, Anupama KR, Renuka K. Characterization of neutrophil-to-lymphocyte ratio as a measure of inflammation in rheumatoid arthritis. Int J Rheum Dis. 2017;20(10):1457–67.

[38] Zhang Y, Yin Y, Kuai S, Shan Z, Pei H, Wang J. Combination of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio as diagnostic biomarker for rheumatoid arthritis. Int J Clin Exp Med. 2016;9(11):22076–81.

[39] Oehadian A, Suryadinata H, Dewi S, Pramudyo R, Alisjahbana B. The role of neutrophyl lymphocyte count ratio as an inflammatory marker in systemic lupus erythematosus. Acta Med Indones.

2013;45(3):170–4.
[40] Xie S, Chen X. Red blood cell distribution width-to-platelet ratio as a disease activity-associated factor in systemic lupus erythematosus. Medicine (Baltimore). 2018;97(39).
[41] Qin B, Ma N, Tang Q, Wei T, Yang M, Fu H, et al. Neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) were useful markers in assessment of inflammatory response and disease activity in SLE patients. Mod Rheumatol. 2016;26(3):372–6.
[42] Li L, Xia Y, Chen C, Cheng P, Peng C. Neutrophil-lymphocyte ratio in systemic lupus erythematosus disease: a retrospective study. Int J Clin Exp Med. 2015;8(7):11026.
[43] Yolbas S, Yildirim A, Gozel N, Uz B, Koca SS. Hematological indices may be useful in the diagnosis of systemic lupus erythematosus and in determining disease activity in Behçet's disease. Med Princ Pract. 2016;25(6):510–6.
[44] Wu Y, Chen Y, Yang X, Chen L, Yang Y. Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) were associated with disease activity in patients with systemic lupus erythematosus. Int Immunopharmacol. 2016;36:94–9.

Table (1):Description of laboratory data in RA & SLE groups.

Variables		RA group (N=50)	SLE group(N=50)
ESR(mm)	**Range**	10 – 140	15-130
	Mean ±SD	43.2 ± 26.6	45.2±23.5
RF(mg/dL)	**Positive**	34 (68%)	NA
ANA(titre)	**Positive**	NA	41 (82%)
Anti-dsDNA(titre)	**Positive**	NA	38 (76%)
WBCs (x 10^3/ul)	**Range**	2.7 – 15.8	2.2 – 15.9
	Mean ±SD	6.5 ± 2.9	6.1 ± 2.9
Neut. (x 10^3/ul)	**Range**	0.9 – 11.5	0.95 – 9.4
	Mean ±SD	3.6 ± 2.4	3.7 ± 2.01
Lymph. (x 10^3/ul)	**Range**	0.8 – 3.98	0.2 – 5.3
	Mean ±SD	2.07 ± 0.8	1.6 ± 0.8
Hb(g/dl)	**Range**	8.2 – 14.5	7.8 – 14.5
	Mean ±SD	11.7 ± 1.6	11.4 ± 1.6
PLT (x 10^3/ul)	**Range**	169 – 641	104.6 – 442
	Mean ±SD	326.9 ± 100.8	271.3 ± 82.4
NLR	**Range**	0.5 – 8.3	0.4 – 16.1
	Mean ±SD	1.8 ± 1.4	2.9 ± 2.4
PLR	**Range**	72.2 – 567.25	50.2 – 1378
	Mean ±SD	175.9 ± 87.2	232.1 ± 193.9

RA= Rheumatoid Arthritis, SLE=Systemic Lupus Erythematosus , ESR=erythrocytes sedimentation rate, RF= rheumatoid factor, ANA= anti-nuclear antibody, Anti-dsDNA= anti –double strand DNA, WBCS=white blood cells, Hb=hemoglobin, PLT=platelets, Neut = neutrophils, Lymph=lymphocytes, NLR=neutrophils/ lymphocytes ratio ,PLR=platelets /lymphocytes ratio ,SD=standard deviation, NA=not applicable.

Table (2): Correlation between disease activity (DAS28 score) and (NLR & PLR) in RA group.

RA group **Variables** **N(50)**		**Low DAS** **(N = 3)**	**Moderate DAS** **(N = 29)**	**High DAS** **(N = 18)**	**p-value**
NLR	**Mean±SD**	2.6±1.3	1.7±1.03	1.9±1.8	0.52
PLR	**Mean ±SD**	231.4±161.8	163.01±86.8	187.5±73.9	0.345

RA=Rheumatoid arthritis, DAS=disease activity score, NLR=neutrophils/ lymphocytes ratio, PLR=platelets /lymphocytes ratio, SD=standard deviation

Table (3): Correlation between disease activity (SLEDAI score) and (NLR & PLR) in SLE group.

SLE group **Variables** **N(50)**		**Mild SLEDAI** **(N = 19)**	**Moderate SLEDAI** **(N = 24)**	**Severe SLEDAI** **(N = 7)**	**p-value**
NLR	**Mean±SD**	2.42±1.49	3.42±3.14	3.06±1.31	0.408
PLR	**Mean±SD**	201.90±107.02	253.29±255.47	241.57±134.19	0.691
	Median - IQR	186.2 – 144.9	198.2 – 125.8	188.3 – 211.1	

SLE=Systemic lupus erythematosus**,** SLEDAI=systemic lupus erythematosus disease activity index, NLR=neutrophils/ lymphocytes ratio, PLR=platelets /lymphocytes ratio, SD=standard deviation, IQR =Inter quartile range

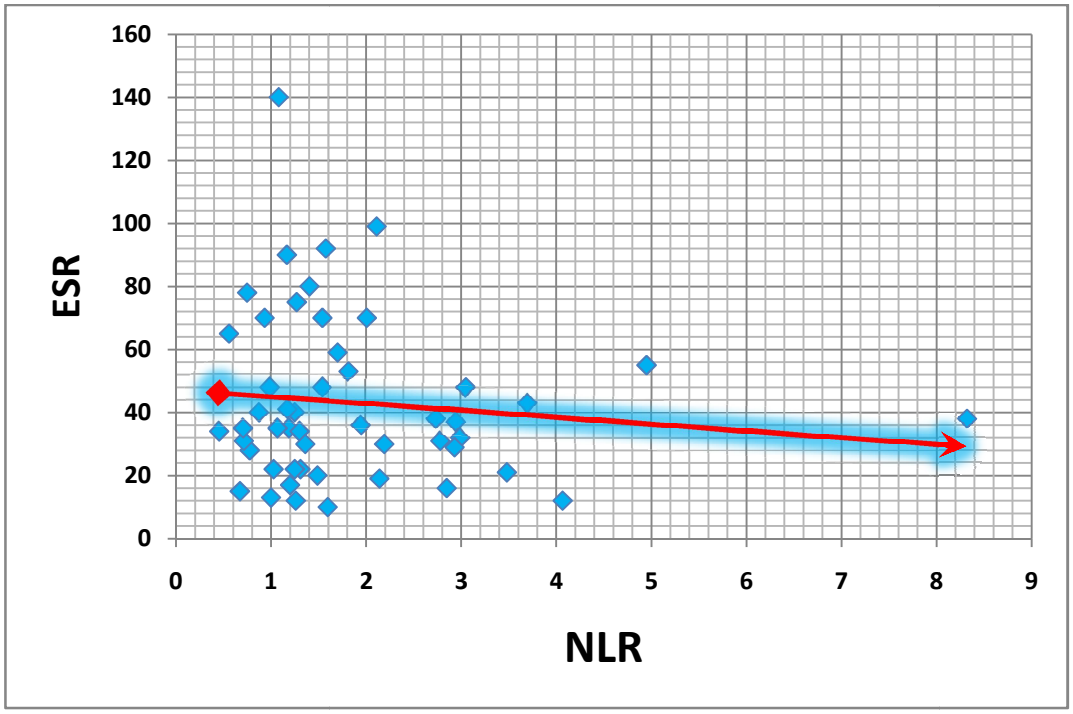

Figure (1): Negative correlation between ESR and NLR in RA group.

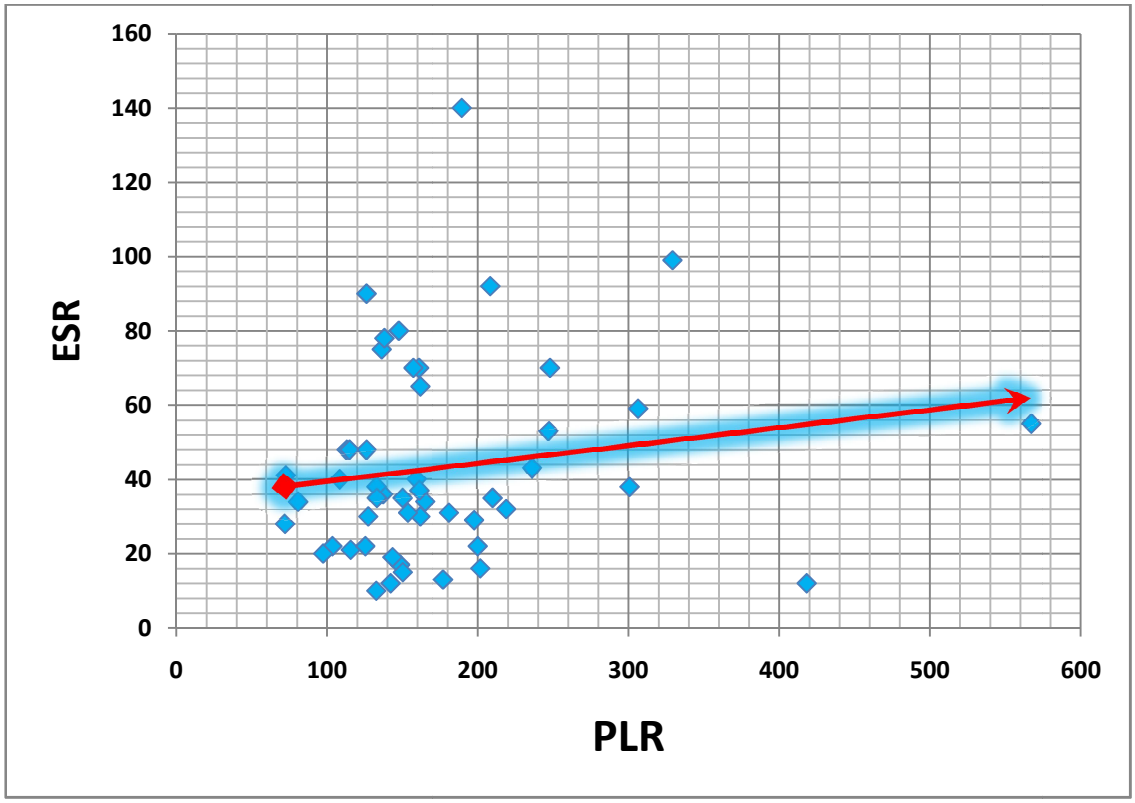

Figure (2): Positive correlation between ESR and PLR in RA group.

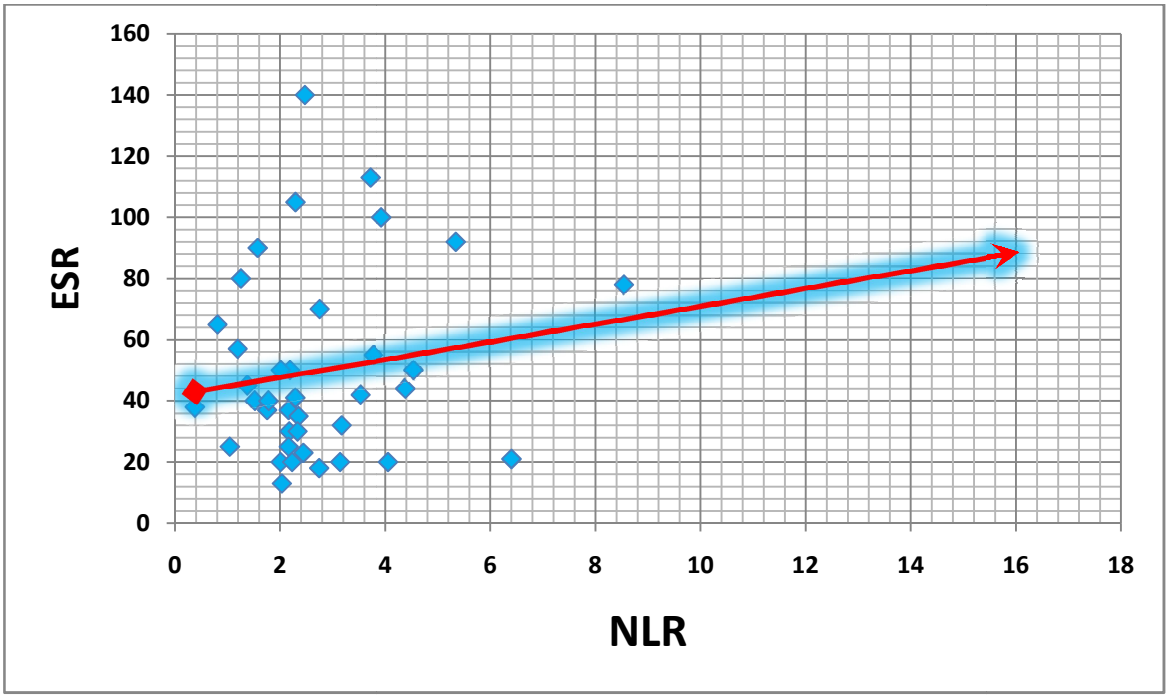

Figure (3): Positive correlation between ESR and NLR in SLE group.

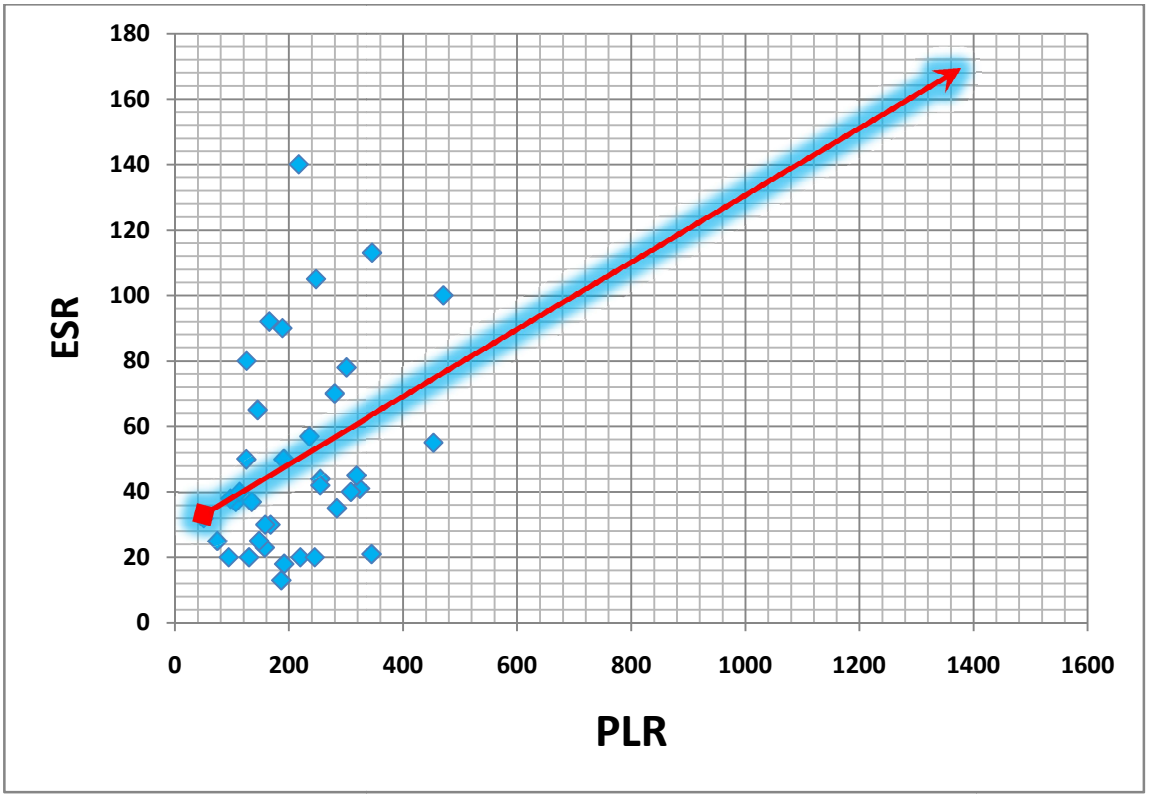

Figure (4): Positive correlation between ESR and PLR in SLE group.