Cross-sectional Study

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The mean platelet volume (MPV) in patients with systemic lupus erythematosus (SLE) and its correlation with disease activity: a cross-sectional/case–control study

Mariam Khadra, MD[a], Tasneem S. Drie, MD[b,*], Maysoun Kudsi, PhD, MD[c]

Introduction: Systemic lupus erythematosus (SLE) is a systemic disease, with unknown etiology. The authors aimed in this study to determine the connection between mean platelet volume (MPV) and disease activity of SLE. Although it has been studied in other rheumatological conditions like rheumatoid arthritis, its role in adult patients with SLE needs to be defined, especially in Syria.

Materials and methods: The authors have included in a cross-sectional study, 80 patients with SLE and 80 controls. The SLE group was divided into two groups based on their disease activity index: the active disease group and the non-active disease group. In all groups, MPV and erythrocyte sedimentation rate (ESR) were analyzed. Clinical findings and Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) were evaluated in all patients.

Results: MPV was significantly lower in SLE patients compared to the control group (8.49 ± 1.2 fl and 10.0 ± 0.5 fl, respectively) ($P = 0.001$). A decrease in MPV below the cut-off value (7.2 fl) increased the risk of active disease by an odds ratio of 9.79 (95% CI: 3.4–27.9) ($P < 0.001$).

Conclusion: MPV may be a disease activity indicator in patients with SLE. MPV is reduced in patients with active SLE and presents an inverse correlation with SLEDAI.

Keywords: biomarkers, disease activity index, mean platelet volume, systemic lupus erythematosus

Introduction

Systemic lupus erythematosus (SLE) is a chronic systemic immune condition, with various clinical presentations. Its incidence is 5 per 100 000 of the population, with higher rates reported in adult women, Asians, African blacks, and Hispanics[1].

SLE has a wide range of clinical manifestations and presentations, but the diagnosis is made according to the American College of Rheumatology (ACR)/European League of Rheumatism (EULAR) criteria[2].

Considering the remitting relapsing nature of most cases of SLE, it is essential to have a biomarker to monitor its disease activity. Although the most effective and reliable tool to measure SLE disease activity is still open to debate, there are fortunately many validated measures, including the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) and others[3]. These indexes are lengthy and time consuming. The SLEDAI-2K has an important limitation due to the dichotomous scoring of each item, which causes a ceiling effect. On the contrary, the SLEDAI can identify differences in cases with diverse levels of activity in individual parameters within the same SLEDAI-2K score[4].

Mean platelet volume (MPV) is a parameter detected during routine blood count and to which clinicians do not usually pay much attention. Platelet volume is a marker determined by megakaryocytes during platelet production, which is associated with platelet function and activation.

Under normal circumstances, there is an inverse relationship between platelet size and number[5]. MPV has been reported as a simple inflammatory indicator in many inflammatory diseases, such as rheumatoid arthritis (RA), scleroderma, rheumatic fever,

HIGHLIGHTS

- We aimed in our study to determine the connection between mean platelet volume (MPV) and the disease activity of systemic lupus erythematosus (SLE).
- MPV may be a disease activity indicator in patients with SLE, as its value decreased in patients with active disease.
- MPV is an easily measurable parameter for activity biomarkers in SLE.

[a]Faculty of Medicine, [b]Rheumatology Department, Faculty of Medicine and [c]Damascus University, Syrian Arab Republic

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*Corresponding author. Address: Al-Mouwasat Hospital: Al-Mouwasat University Hospital, Damascus, Syrian Arab Republic. Tel.: +963 948 958 318.
E-mail: samsomadrei@gmail.com (T.S. Drie).

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ankylosing spondylitis, and even chronic obstructive pulmonary disease[6,7].

MPV is an easily accessible and valuable marker of SLE disease activity[8]. Established cardiovascular risk factors, such as smoking, hypertension, dyslipidemia, and diabetes, can influence MPV, depending on confounding factors. Low-grade inflammation is one such factor[9].

In the present study, we aimed to find out whether MPV does or does not correlate with SLEDAI and whether it can be used as a predictor of lupus activity.

Patients and methods

Study design

A cross-sectional study was conducted in Syria between January 2021 and January 2022. Our study was approved by the Ethics Review Committee of the Faculty of Medicine (approval number 220345/2021-A/M), and written informed consent was obtained from every participant. This case is submitted on the research registry dashboard.

Our study is compatible with the STROCSS Guideline Checklist.

Inclusion criteria

Patients older than 16 years who can sign the informed consent by themselves, diagnosed with SLE according to ACR/EULAR criteria 2019[2], and healthy participants as a control group who were accompanied by their patients in surgical clinics matching with age and sex.

Exclusion criteria

Smoking, infectious conditions, thyroid diseases, other inflammatory articular conditions, antiphospholipid syndrome, hypertension, cardiac ischemia, recent cardiac infarction, thrombosis, diabetes mellitus, acute and/or chronic renal failure, hemoglobin level more than 16.5 g/dl, and platelet level more than 150 000 mm^3.

All the above-mentioned diseases influence platelet number and/or size, so their inclusion would have caused a selection bias in our results.

Sample size

The sample size was calculated by using the frequency of low MPV in patients with actively flaring lupus from the hospital records, along with 95% confidence interval (CI) using (http://www.who.iznt/chp/steps/resources/sampling/en/). A total of 80 patients were assessed and were divided into two equal groups: 80 SLE patients (40 subjects each in the active-SLE and the inactive-SLE groups) and 80 healthy participants as a control group.

Laboratory tests

The study was conducted on all patients attending the rheumatic diseases clinic and admitted to the rheumatic diseases department at Al-Assad and Mouwasat University Hospital in Damascus during the aforementioned study period.

Patients underwent thorough clinical and laboratory evaluation, including a complete medical history. The following data were collected: sex, age, white blood cell (WBC) count, neutrophil count (NEU), lymphocyte count (LYM), platelet count (PLT), MPV, erythrocyte sedimentation rate (ESR), C3, anti-dsDNA, and urine analysis. All analyses were performed within an hour of collecting the samples and in the laboratory of Al-Assad University Hospital, and therefore the same laboratory equipment was used for all patients.

Complete blood count (CBC) was done with a Cell-Dyn Ruby analyzer (Abbott Diagnostics, USA), while creatinine, blood urea nitrogen, and serum albumin were measured using a DxC800 Synchron analyzer (Beckman Coulter, USA). ESR determinations were performed by the Wintrobe method, whose upper normal limit was 20 mm/h.

Table 1

Characteristics of the study group

Variables	SLE group	Control group	*P*
Age, years	32.5 ± 10.7	31.9 ± 11.1	0.798
Sex, *n*, %			
Female	72 (90%)	74 (92.5%)	0.512
Male	8 (10%)	6 (7.5%)	
Disease duration, years	6.2 ± 3.8	–	–

Table 2

Clinical findings and laboratory variables of the active/inactive SLE group

Variables	Active SLE patients (40)	Inactive SLE patients (40)
Clinical manifestations		
Fever	13 (32.5%)	8 (20%)
Fatigue	29 (72.5%)	–
Polyarthritis	36 (90%)	–
Myalgia	28 (70%)	12 (30%)
Oral ulcers	24 (60%)	–
Butterfly rash	12 (30%)	–
Photosensitivity	19 (47.5%)	16 (40%)
Alopecia	6 (15%)	–
Serositis	12 (30%)	–
Neuropsychiatric presentations	22 (55%	–
Edema	20 (50%)	–
Gastrointestinal manifestations	5 (12.5%)	–
Anorexia	–	7 (17.5%)
Vomiting	–	2 (5%)
Arthralgia	–	8 (20%)
Nonspecific pain	–	21 (52.5)
Laboratory findings		
Low WBC	12 (30%)	4 (10%)
Lymphopenia	9 (22.5%)	1 (2.5%)
C3	12 (30%)	–
C4	16 (40%)	–
Anti-dsDNA antibodies	18 (45%)	–
Urinalysis		
Hematuria	12 (30%)	–
Pyuria	8 (20%)	–
Proteinuria (> 0.5 gm/day)	17 (42.5%)	–
Cellular cast	8 (20%)	–

Table 3

Mean MPV and other laboratory markers in the three groups

Laboratory variables	Active-SLE patients (40)	Inactive-SLE patients (40)	*t*-test	*P*
Hemoglobin/dl	11.8 ± 1.28 g/dl	12.4 ± 1.2 g/dl	2.162	0.033
White blood cells (10 ×/mm^3)	6.8 ± 1.34	6.7 ± 1.3	0.338	0.735
Neutrophils (10 ×/mm^3)	5 ± 0.097	4.9 ± 0.09	0.478	0.634
Lymphocytes (10 ×/mm^3)	1.8 ± 0.36	1.8 ± 0.47	0.213	0.831
Platelets (10 ×/mm^3)	281 ± 75.1	274 ± 75.8	0.426	0.67
MPV, fl	7.17 ± 1.74	9.86 ± 1.9	6.6	< **0.001***
ESR, mm/h	33.9 ± 9.1	19.17 ± 6.1	8.37	< **0.001***

Bold expresses the values of MPV of the study sample.

SLE activity

Patients were divided into two groups based on their final score from using SLEDAI-2K. The previously published literature demonstrated a mean cut-off score of 5 or higher on SLEDAI-2K as an effective indicator of actively flaring lupus[10] as follows:

(1) Active-SLE patients: SLEDAI score ≥ 5 points.
(2) Inactive-SLE patients: SLEDAI score <5 points.

MPV measurement

Five milliliters of venous blood was drawn into an EDTA tube from each patient for measurement of CBC, including hemoglobin, WBC count, platelet count, MPV, and ESR. Normal values for MPV range from 6.5 to 10.5 fl.

Statistical analysis

The analysis was carried out using Statistical Package for the Social Sciences (SPSS) (IBM Corporation, Armonk, New York, USA) (version 20) and Excel 2010. A predictive value less than 0.05 (P value <0.05) was considered statistically significant.

For categorical variables, it was based on frequency, percentages, graphics, and figures. For continuous variables, measures of mean, standard deviation, and range were used.

Results

There was no statistically significant difference in mean age between patients and controls ($P = 0.798$), and there also was no statistically significant difference in mean age between patients with active SLE and patients with inactive SLE ($P = 0.852$).

A total of 72 (90%) SLE patients were females and 74 (92.5%) of control participants were females. There was no statistical difference in the distribution of males and females between the two groups ($P = 0.512$). In addition, there was no statistically significant difference in the distribution of males and females between the two groups of SLE patients ($P = 0.455$). Demographic details are shown below in Table 1.

The most frequent clinical manifestations were arthritis, fatigue, butterfly rash, and serositis in the active disease group, meanwhile nonspecific pain arthritis, butterfly rash, and anorexia in non-active disease group (Table 2).

There were 20 patients with active renal disease, as renal SLEDAI greater than8 distributed to the histological type of lupus nephritis as: 12 patients with focal proliferative glomerulonephritis, 7 patients with diffuse proliferative glomerulonephritis, and 1 with membranous glomerulonephritis.

The treatment of the SLE group consists of the following:

(1) Active-SLE patients: 28 patients under treatment with predlone (10–30 mg/day), 40 patients under treatment with hydroxychloroquine, 10 patients under azathioprine treatment, 25 patients under treatment with mycophenolate, 2 patients under cyclophosphamide treatment, and 3 patients under rituximab treatment.
(2) Inactive-SLE patients: 20 patients under treatment with predlone (5–15 mg/day), 40 patients under treatment with hydroxychloroquine, 28 patients under azathioprine treatment, 5 patients under treatment with mycophenolate, and 1 patient under cyclophosphamide treatment. Mean MPV was significantly lower in SLE patients compared to the control group (8.49 ± 1.2 fl and 10.0 ± 0.5 fl, respectively) ($P = 0.001$).

There was no statistically significant difference in the average count of WBCs, neutrophils, lymphocytes, or platelets between the two SLE sub-groups. Mean MPV was significantly lower in

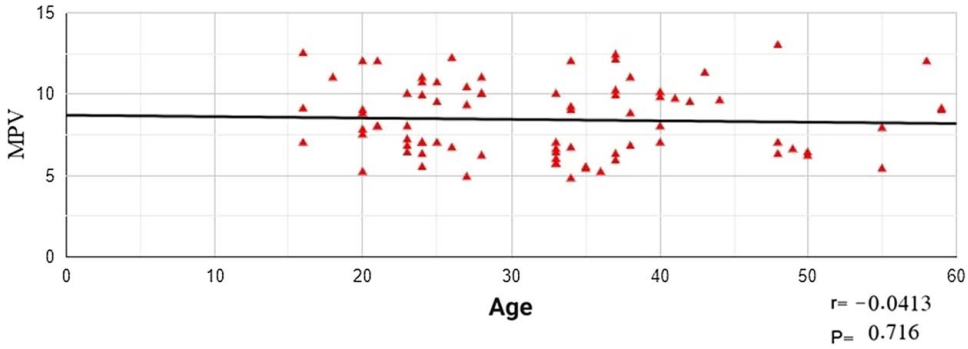

Figure 1. An association was found between the ages of patients with SLE and MPV. MPV, mean platelet volume; SLE, systemic lupus erythematosus.

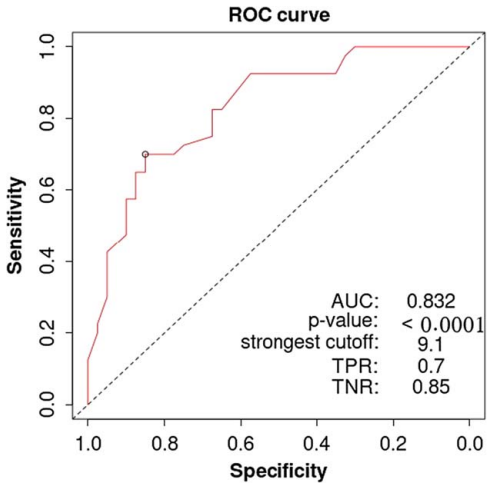

Figure 2. Relationship between MPV and SLE activity. MPV, mean platelet volume; SLE, systemic lupus erythematosus.

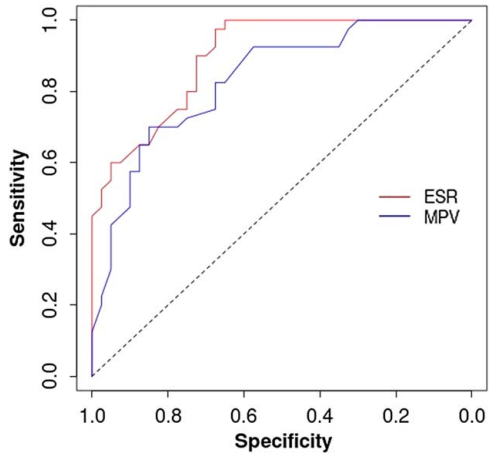

Figure 4. Role of MPV and ESR in predicting the effectiveness of SLE. ESR, erythrocyte sedimentation rate; MPV, mean platelet volume; SLE, systemic lupus erythematosus.

patients with active SLE than in patients with ineffective SLE. The mean ESR was significantly higher in patients with active SLE than in patients with inactive SLE (Table 3).

We found that the mean MPV was significantly lower in patients with active SLE than in patients with ineffective SLE ($P = 0.003$, which is <0.001), as the mean MPV for patients with active SLE was 7.17 ± 1.74 fl with a range of 4.8–12 fl, and for patients with inactive SLE 9.8–12.6 ± 1.9 fl with a range of 6.2–13 fl (Table 3).

A fragile, non-significant inverse association was found between the age of patients with SLE and MPV (Fig. 1).

There was no statistically significant difference in mean MPV between male and female SLE patients ($P = 0.421$).

The area under the receiver operating characteristic (ROC) curve for the role of MPV in diagnosing active SLE was AUC = 0.832, and the best cut-off point for predicting the effectiveness of SLE according to the curve was at MPV 7.2 fl, which was associated with a sensitivity of 67.5%, specificity of 82.5%, and a value of. It has a positive predictive value of 79.4%, a negative predictive value of 71.7%, and a diagnostic accuracy of 75%.

A decrease in MPV below the cut-off value (7.2 fl) increased the risk of active disease by an odds ratio of 9.79 (95% CI: 3.4–27.9) ($P < 0.001$).

Figure 2 shows the relationship between MPV and SLE activity.

The mean ESR for patients with active SLE was 33.9 ± 9.1 mm/h with a range of 21.5–52 mm/h, and for patients with inactive SLE 19.16 ± 6.4 mm/h with a range of 9–34 mm/h.

ESR levels were statistically significantly higher in patients with active SLE than in patients with inactive SLE ($P < 0.0001$). The area under the ROC curve for the role of ESR in diagnosing active SLE was AUC = 0.899, and the best cut-off point for predicting the effectiveness of SLE according to the curve was at an ESR of 23 mm/h, which was associated with a sensitivity of 90% and a specificity of 72.5%. A positive predictive value of 76.6%, a negative predictive value of 87.9%, and a diagnostic accuracy of 81.25%. A higher ESR above the cut-off value (23 mm/h) increased the risk of having active disease by an odds ratio of 23.7 (95% CI: 6.8–82.3) ($P < 0.001$). Figure 3 shows the ROC curve for the role of ESR in predicting the effectiveness of SLE.

Figure 4 shows the ROC curve for the role of MPV and ESR in predicting the effectiveness of SLE.

A statistically significant weak inverse association was found between MPV and disease severity SLEDAI-2K. That is, as SLEDAI-2K increases, there is an associated decrease in MPV.

Table 4 and Figure 5 show the linear relationship between MPV and SLEDAI-2K.

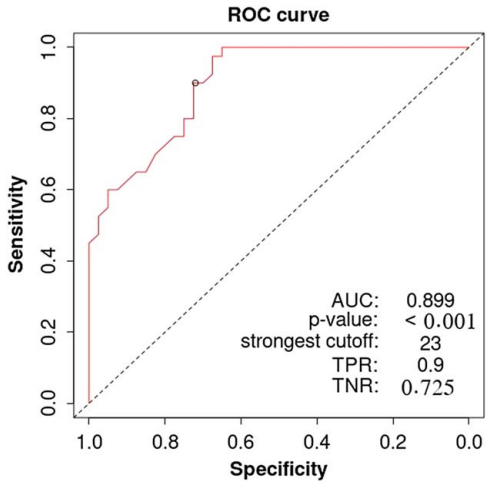

Figure 3. ROC curve for the role of ESR in predicting the effectiveness of SLE. ESR, erythrocyte sedimentation rate; ROC, receiver operating characteristic; SLE, systemic lupus erythematosus.

Table 4

Linear relationship between MPV and SLEDAI-2K

	Pearson's test	*P*	The statistical correlation
MPV	–	>	Inverse correlation
SLEDAI-2K	0.465	0.001	Weak statistically significant correlation

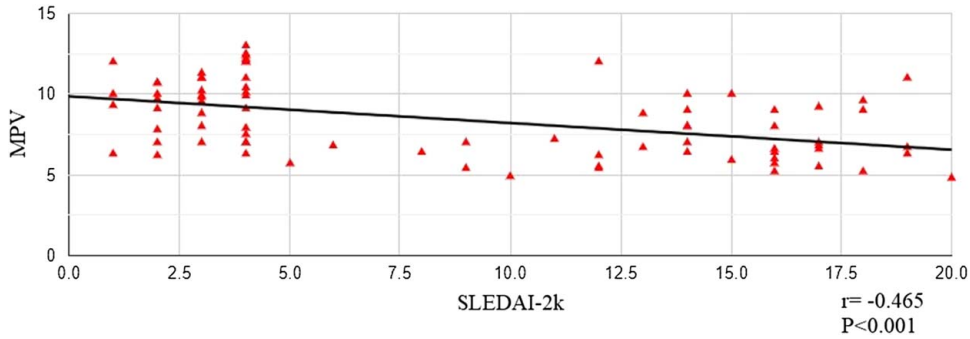

Figure 5. Linear relationship between MPV and SLEDAI-2K. MPV, mean platelet volume; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index.

A statistically significant weak inverse correlation was found between MPV and ESR. That is, the higher the ESR, the lower the MPV (Fig. 6).

Mean MPV was significantly lower in patients with active-renal SLE (20 patients) than in patients with inactive-renal SLE (20 patients). The mean MPV for patients with active-renal SLE was 6.37 ± 1.84 fl, and for patients with inactive-renal SLE 7.1 ± 1.9 ($P < 0.001$).

Mean MPV was significantly lower in patients with active-neuropsychiatric SLE (22 patients) than in patients with inactive-neuropsychiatric SLE (18 patients). The mean MPV for patients with neuropsychiatric manifestations of SLE was 7.0 ± 1.3 fl, and for patients without neuropsychiatric manifestations of SLE 7.3 ± 1.1 ($P = 0.341$).

Discussion

Our study showed that MPV was significantly lower in SLE patients, especially in active disease compared to the control.

In SLE, a decrease in MPV is associated with platelet activation. The immune complexes in SLE are potent activators of platelets through their binding to the FcγRIIA receptor (CD32) on the platelet surface. The immune complexes in SLE can also act through the Toll-like receptor (TLR), whereby they promote platelet activation[11]. The inflammatory cytokines and immune deregulation, which play a role in SLE pathogenesis, can activate platelets, leading to a reduction in MPV value in active disease[12].

We found lower MPV values in actively flaring SLE patients group as well as in other studies that demonstrated lower MPV values in patients with active lupus[13,14].

We found that the higher the ESR, the lower the MPV. That is compatible with some studies[8–14] and disagrees with a study of Hartmann *et al.*[15], which did not find any correlation between MPV and ESR. This may be due to the characteristics of the treatment or study population and the consideration of SLEDAI > 0, as an active SLE. It is known that C-reactive protein (CRP) and ESR are not good biomarkers of SLE activity[16].

The course of the inflammatory state is also accompanied by an increase in the proportion of large platelets, and this may be due to the intracellular synthesis of factors that cause coagulation and enhance inflammation, the dissolution of granulocytes, and the beginning of the aggregation of platelets stored in the spleen. At the same time, these cells rapidly migrate to the site of inflammation where they undergo activation and exhaustion. This seems to explain the decrease in MPV in patients with persistent inflammation[17,18]. In recent years, it has been suggested that MPV may be associated with RA activity; however, data on this topic remain controversial. Some studies showed that RA patients with high disease activity tend to have smaller size of platelets than those at remission. Kisacik *et al.*[19] reported lower values of MPV in patients with active RA than controls, and these values increased significantly after treatment, but remained lower than in control patients.

Another study found that MPV may not be able to predict disease activity in RA patients. And although therapeutic regimens, which improve RA manifestations, can reduce RA activity, they had no effect on MPV[20].

Gasparian *et al.* describe the main reasons for controversy in studies, highlighting that the regulation of platelet function and aging is influenced by the ploidy and maturity of thrombopoietic

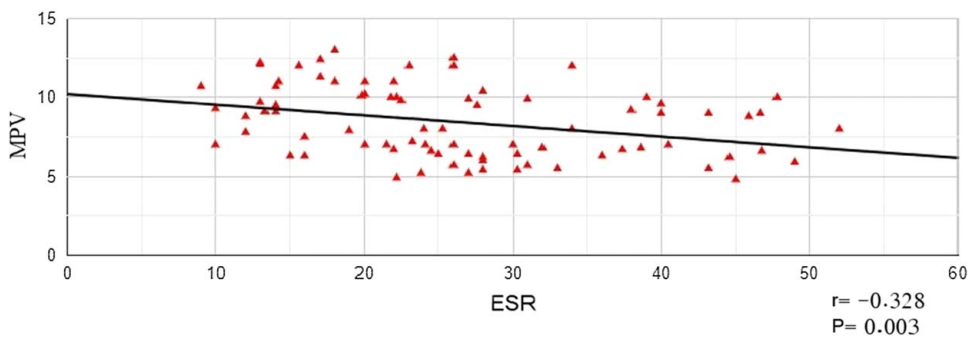

Figure 6. Correlation between MPV and ESR. ESR, erythrocyte sedimentation rate; MPV, mean platelet volume.

progenitors. Additionally, various cytokines and factors in circulation, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-a (TNF-a), affect platelet production. Furthermore, platelet activation in different physiological and pathological situations leads to time-dependent changes in platelet indices.

On other hand, MPV is measured by cell counters using impedance and optical effects. The discordance between the results of different and even the same cell counters limits the interchangeable use of MPV. This can explain, at least partly, why hematological laboratories sometimes do not display the MPV and some other indices of platelet function[9]. Otherwise, previous studies reported the fact that the MPV is highly dependent on the time of storage until the analysis[21].

Antiphospholipid antibodies can mediate platelet activation directly through interaction with a platelet's plasma membrane, by binding diverse platelet receptors and/or by promoting complement deposition on platelets[22]. We did not analyze this antibody.

Treatment such as heparin, corticosteroids, and nonsteroidal anti-inflammatory drugs increase MPV; meanwhile, others may cause low MPV, such as furosemide, gold, penicillin, and quinidine[23]. Our patients were taking nonsteroidal anti-inflammatory drugs from time to time and corticosteroids, but we did not study the effects of these treatments on MPV.

The limitations of our study include the following: the small sample size, the one-center design, the cross-sectional design, which cannot establish a causal relationship, the menstruation during the study, which interferes with the platelet volume, the effect of obesity and lifestyle on MPV, and the effect of anxiety in this patient on MPV. Moreover, patients with antiphospholipid antibody syndrome were not excluded from our study because we did not analyze these antibodies.

Conclusion

In summary, our study findings suggest that MPV can be used as a reliable indicator of disease activity in SLE. We observed a decrease in MPV among patients with active SLE, and this decrease was strongly associated with the SLEDAI. Notably, when the MPV cut-off value was set at less than 7.2 fl, it demonstrated high sensitivity and specificity for determining disease activity in SLE. However, it is important to acknowledge the limitations of our study, including its single-center design and relatively small sample size. To further validate the prognostic and diagnostic value of MPV in clinical practice, larger-scale studies involving diverse SLE populations and molecular biological investigations are recommended.

Ethical approval

Our study was approved by the Ethics Review Committee of the Faculty of Medicine (approval number 220345/2021-A/M), and written informed consent was obtained from every participant.

Consent

Written informed consent was obtained from the patient for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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None.

Author contribution

M.K.: literature review, manuscript writing and editing, and final manuscript review and approval; T.S.D.: obtaining informed written consent, clinical follow-up, manuscript writing, and approval of the final manuscript; M.K.: manuscript writing and approval of the final manuscript. All authors described the case and collected the clinical data, reviewed and edited the manuscript.

Conflicts of interest disclosure

All authors declare no conflicts of interest.

Research registration unique identifying number (UIN)

1. Name of the registry: Mariam Khadra.
2. Unique identifying number or registration ID: research registry 9543.
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): https://www.researchregistry.com/browse-the-registry#homel.

Guarantor

Mariam Khadra, MD/Rheumatologist, Faculty of Medicine, Damascus University; Mariyamkhadra@gmail.com.

Data availability statement

Datasets generated during and/or analyzed during the current study are publicly available, available upon reasonable request.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Protection of human and animal subjects

No experiments were performed.

Confidentiality of data

The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent: The authors declare that no patient data appear in this article.

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