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Correlation of neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and mean platelet volume (MPV) with gout activity

A monocentric and retrospective study

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Abstract

To evaluate the correlation of neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and mean platelet volume (MPV) with parameters related to gout activity. The general data of the patients and healthy controls (HCs), including complete blood count, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), serum uric acid (SUA), and the presence of tophi were retrospectively analyzed. NLR, MPV, and PLR were calculated in patients with intercritical gout and gout flares. Correlation of the 3 markers with clinical features, like ESR, CRP, SUA, and the presence of tophi, were analyzed. The results revealed that NLR and PLR were elevated and MPV was markedly decreased in patients with gout compared with HCs (all $P < .05$). In patients with gout flares, NLR, and PLR were higher and MPV was lower than in intercritical gout patients (all $P < .05$). NLR and PLR were positively correlated with ESR and CRP, whereas MPV was negatively correlated with ESR. NLR, PLR, and MPV showed no obvious correlation with SUA and the presence of tophi. The receiver operating characteristic curve showed that NLR was more valuable in assessing gout disease activity. NLR, PLR, and MPV were correlated with inflammatory parameters in gout; they may be used as complementary tools to evaluate gout activity.

Abbreviations: CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, HC = healthy control, IL = interleukin, MPV = mean platelet volume, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio, PLT = platelet, RA = rheumatoid arthritis, ROC = receiver operating characteristic curves, SLE = systemic lupus erythematosus, SUA = serum uric acid, TNF = tumor necrosis factor, WBC = white blood cell.

Keywords: gout, inflammatory mediators, mean platelet volume, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio

1. Introduction

Gout is a common form of inflammatory arthritis.[1] Acute episodes often begin with sudden pain and swelling in the joints of the toes. This condition is always caused by hyperuricemia, which leads to deposition of monosodium urate crystals that accumulate in joints and soft tissues over time.[2] These crystals then induce an acute inflammatory reaction via the massive recruitment of leukocytes and release of cytokines, such as interleukin (IL)-1β, IL-6, tumor necrosis factor (TNF), and chemokines.[3] These proinflammatory cytokines and lysosomal enzymes are released from macrophages and neutrophils.[4] Reversing the overexpression of inflammatory cytokines could attenuate an acute attack of gout.[5] However, in our clinical work, the detection of serum inflammatory factors, like IL-1β, is seldom used as routine detection due to its high cost. Symptomatic episodes, including swelling, pain, tenderness in peripheral joint or bursa, and leukocyte counts, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) are generally used as indicators to evaluate gout disease activity.[6] At present, there are several challenges in the evaluation of gout disease activity: the level of serum uric acid (SUA) and the inflammatory activity are not matched, CRP and ESR are elevated in intercritical gout patients, and both the inflammatory indicators are at normal levels in the gout flare patients. Therefore, relying exclusively on the aforementioned indicators to evaluate gout activity is not comprehensive, and more inflammatory markers related to disease activity need to be identified. Hemogram parameters, such as neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and mean platelet volume (MPV), have been reported to be related to the level of inflammation.[7] Recently, studies have focused on these inexpensive hematological parameters and found that they may be used as indicators to evaluate disease activity in many diseases, such as diabetes mellitus, chronic kidney disease, rheumatoid arthritis (RA), and systemic lupus erythematosus (SLE).[7–10]

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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However, there are few reports of the use of these parameters in gout. Therefore, this study retrospectively analyzed the clinical characteristics of gout patients to evaluate the correlation of NLR, PLR, and MPV with gout activity.

2. Subjects and Methods

2.1. Participants

From May 2020 to May 2021, 118 gout patients from the inpatient department at Xiaoshan Traditional Chinese Medicine Hospital were enrolled in this study, and 3 of the patients were hospitalized twice during that period. In addition, 93 healthy age- and sex-matched subjects who underwent routine physical examinations in the same hospital during the same period were used as healthy controls (HCs). The diagnostic criteria for gout were based on the classification criteria of the American Society of Rheumatology in 1977 or 2015.[11] According to the American Society of Rheumatology 2015 guidelines, gout activity refers to gout patients with gout acute attack, a painful inflammatory arthritis (gout flare). And gout patients without acute arthritis is intercritical gout.

Patients were excluded if they had one of the following characteristics: <18 years of age or >90 years of age; other autoimmune diseases, such as Sjogren syndrome, RA, or SLE; malignant diseases; acute infection or chronic inflammation status; hematological diseases, or had received a blood transfusion in the past 4 months.

2.2. Data extraction

Demographic, clinical, and laboratory data were extracted from electronic medical records, including age, gender, levels of white blood cells (WBCs), neutrophils, lymphocytes, platelets (PLT), MPV, ESR, CRP, SUA, and the presence of tophi. We divided the patients into 2 groups: the gout flare group (patients with gout acute attack, a painful inflammatory arthritis) and the intercritical gout group (patients without an acute attack of gouty arthritis).[6,11] The study protocol was approved by the local ethics committee and conformed to the provisions of the Declaration of Helsinki.

2.3. Statistical analysis

Analyses were performed using SPSS software (version 20.0, SPSS, Chicago, IL). The χ^2 test was used to compare gender differences between groups. The normal distribution of the variables was evaluated with Kolmogorov–Smirnov test. Normally distributed measurement data (MPV, SUA) are presented as mean ± standard deviation. Non-normally distributed measurement data are expressed as median (IQR). All the continuous variables were compared using Student *t* test or the Mann–Whitney *U* test, where appropriate. Spearman correlation coefficient was used to assess the correlation between 2 continuous variables. Receiver operating characteristic (ROC) curves were plotted, and the areas under ROC curves were calculated to assess the predictive value of NLR, PLR, and MPV in patients with gout. Statistical significance was defined as $P < .05$.

3. Results

3.1. Basic characteristics of the study sample

The demographic and clinical characteristics and laboratory data of gout patients and HCs are listed in Table 1. The patients had a median age of 63.00 (IQR 48.75, 71.00), with a gender distribution of 8/110 (F/M). The median age was 62.00 (IQR 53.00, 72.50) years in the HC group with a gender distribution of 9/84 (F/M) women (9). There was no significant difference in age or sex between the gout patients and HCs ($P > .05$).

3.2. NLR and PLR were increased and MPV was decreased in gout patients compared with HCs

As shown in Table 1, NLR and PLR were markedly elevated ($P < .001$) and MPV was significantly decreased ($P < .001$) in gout patients compared with HCs. We found that patients with gout had lower lymphocytes and higher WBC, neutrophil, and PLT counts, and SUA levels than HCs ($P < .05$) (Table 1).

3.3. NLR and PLR were increased and MPV was decreased in gout flare patients

As shown in Table 2, NLR ($P < .05$) and PLR ($P < .05$) were markedly increased, and MPV ($P < .05$) was significantly decreased in the gout flare group compared with the intercritical gout patients. Neutrophil count, WBC, ESR, and CRP were higher in gout flare patients than in intercritical gout patients, whereas lymphocytes counts and SUA levels were lower in gout flare patients than in intercritical gout patients (all $P < .05$). Although PLT counts were increased in gout flare patients, this increase was not statistically significant (all $P > .05$).

3.4. NLR, PLR, and MPV were correlated with clinical inflammatory makers in gout patients

In our study, we found that NLR was positively correlated with ESR ($R = 0.253$, $P = .006$) and CRP ($R = 0.367$, $P < .001$), and PLR was positively correlated with ESR ($R = 0.434$, $P < .001$) and CRP ($R = 0.340$, $P < .001$). MPV was negatively correlated with ESR ($R = –0.183$, $P = .048$), but had no obvious correlation with CRP or SUA. However, all the correlations

Table 1

Comparison between gout patients and HCs regarding demographic and laboratory data.

	Patients (N = 118)	HCs (N = 93)	*P*
Age (yr)	63.00 (48.75, 71.00)	62.00 (53.00, 72.50)	.826
Sex (F/M)	8/110	9/84	.302
WBC ($\times 10^9$/L)	7.89 (6.24, 10.36)	6.07 (5.23, 7.52)	<.001
N ($\times 10^9$/L)	5.75 (4.10, 8.10)	3.70 (3.00, 5.05)	<.001
L ($\times 10^9$/L)	1.40 (0.90, 1.90)	1.60 (1.30, 2.00)	.005
PLT ($\times 10^9$/L)	210.50 (170.25, 254.25)	198.00 (163.00, 232.50)	.034
NLR	3.89 (2.57, 6.81)	2.38 (1.62, 3.12)	<.001
PLR	152.06 (102.99, 237.33)	80.44 (55.65, 119.45)	<.001
MPV (fL)	9.42 ± 1.11	10.11 ± 1.14	<.001
SUA (μmol/L)	467.31 ± 135.78	304.34 ± 77.27	<.001

HCs = healthy controls, L = lymphocyte, MPV = mean platelet volume, N = neutrophil, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio, PLT = platelet, SUA = serum uric acid, WBC = white blood cell.

Table 2

Comparison of blood routine parameters, NLR, PLR, and MPV between patients in gout flares and intercritical gout.

	Intercritical gout (N = 23)	Gout flares (N = 95)	*P*
WBC (×10⁹/L)	7.24 (5.60, 8.34)	8.08 (6.36, 10.69)	.031
N (×10⁹/L)	3.90 (3.00, 5.80)	6.30 (4.80, 8.40)	<.001
L (×10⁹/L)	1.80 (1.20, 2.50)	1.30 (0.90, 1.80)	.013
PLT (×10⁹/L)	211.00 (175.00, 274.00)	209.00 (167.00, 224.00)	.159
NLR	2.16 (1.39, 3.68)	4.67 (3.06, 7.50)	<.001
PLR	95.26 (67.22, 162.31)	165.45 (117.50, 253.00)	.001
MPV (fL)	9.94 ± 1.16	9.30 ± 1.07	.013
ESR (mm/h)	6.00 (3.00, 18.00)	34.00 (15.00, 68.00)	<.001
CRP (mg/L)	7.90 (3.23, 10.60)	46.69 (20.01, 99.05)	<.001
SUA (μmol/L)	543.09 ± 116.17	448.97 ± 134.33	.003

CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, L = lymphocyte, MPV = mean platelet volume, N = neutrophil, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio, PLT = platelet, SUA = serum uric acid, WBC = white blood cell.

Table 3

Correlation analysis of NLR, PLR, and MPV with clinical factors in patients with gout.

	NLR		PLR		MPV	
	R	*P*	*R*	*P*	*R*	*P*
ESR (mm/h)	0.253	.006	0.434	<.001	–0.183	.048
CRP (mg/L)	0.367	<.001	0.340	<.001	–0.122	.189
SUA (μmol/L)	–0.142	.125	–0.158	.088	–0.044	.624
tophi	–0.035	.708	–0.081	.382	0.267	.004

CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, MPV = mean platelet volume, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio, SUA = serum uric acid.

were relatively weak or mild (all $R < 0.5$). Both NLR and PLR showed no correlation with SUA nor with the presence of tophi (Table 3). Applying a Bonferroni correction to the correlations, NLR and PLR was still positively correlated with ESR (both $P < .0083$) and CRP (both $P < .0083$), whereas MPV showed no correlation with ESR ($P = .048$, $P > .0083$) and CRP ($P = .189$, $P > .0083$).

3.5. ROC curves of NLR, PLR, and MPV for their value to predict a gout attack

ROC curve analysis was performed to determine the cut-off value of NLR, PLR, and MPV to predict a gout attack. The area of NLR, PLR, and MPV were 0.765, 0.720, and 0.669, respectively. The optimal threshold for NLR was 3.810, with a sensitivity of 57.9% and a specificity of 78.3%. The optimal cut-off level of PLR was 141.435, with a sensitivity of 60.0% and a specificity of 60.9%. The optimal clinical cut-off level of MPV was 9.350, with a sensitivity of 73.9% and a specificity of 52.6% (Fig.1 and Table 4).

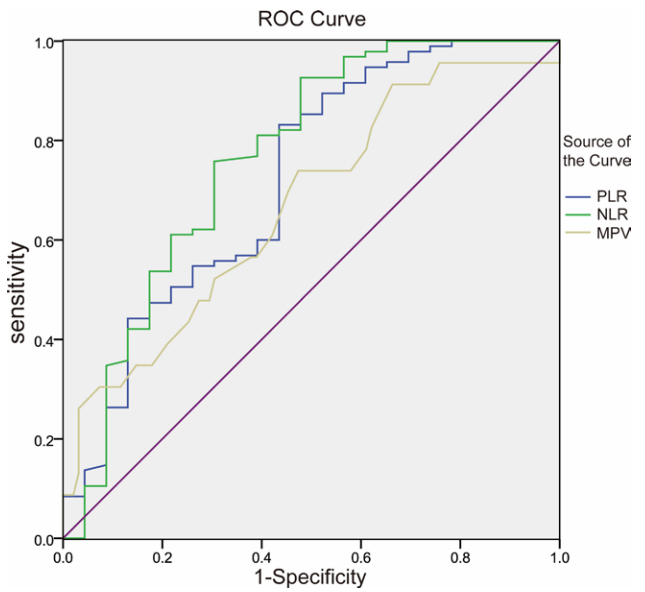

Figure 1. Receiver operating characteristic curves of PLR, NLR, and MPV for differentiating the disease activity in patients with gout. MPV = mean platelet volume, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio.

4. Discussion

The present study demonstrated that NLR and PLR were significantly higher and MPV was lower in the gout group than in the HC group. Furthermore, the gout flare group had higher NLR and PLR and lower MPV as compared with the intercritical gout group. We also found that NLR and PLR were positively correlated with ESR and CRP, whereas MPV was negatively correlated with ESR and was not correlated with CRP. Both NLR and PLR showed no correlation with SUA. The strengths of all the correlations were weak or mild (all $R < 0.5$), indicating these 3 markers could only be used as complementary tools for evaluating disease activity in gout.

A gout attack may develop despite normal or low SUA levels.[12] In this study, SUA levels were higher in the gout group than in the HC group; however, SUA was lower in the gout flare group than in the intercritical gout group. Hyperuricemia is not the only cause of gout flares; monosodium urate crystals could directly recruit neutrophils to aggregate in the area.[13] Neutrophils are the major cell type involved in gout flares and attack development.[14] The massive number of neutrophils leads to an enhancement in blood viscosity and hypercoagulability by causing interactions between the PLTs and endothelium.[15] Platelets adhere to endothelial cells and increases inflammation by causing leukocytes to migrate to the area and adhere. Cytokines and reactive oxygen species increase the release of immature and active PLTs to the peripheral blood.[16] The decreased lymphocytes reflect inflammation.[17] The release of proinflammatory cytokines from leukocytes causes increased

Table 4

ROC of NLR, PLR, and MPV for differentiating gout activity.

					95% CI		
	Cut-off value	Area	Sensitivity	Specificity	Lower bound	Upper bound	*P*
NLR	3.810	0.765	0.579	0.783	0.640	0.891	<.001
PLR	141.435	0.720	0.060	0.609	0.593	0.847	.001
MPV	9.350	0.669	0.739	0.526	0.544	0.793	.012

MPV = mean platelet volume, NLR = neutrophil-lymphocyte ratio, PLR = platelet-lymphocyte ratio.

concentrations of reactive oxygen species in the environment. NLR more effectively indicates inflammation than neutrophil count. NLR is recognized as a biomarker of subclinical inflammation and has been used in combination with other inflammatory markers, like CRP and ESR, to detect inflammation in both autoimmune and nonautoimmune diseases. Previous studies showed that NLR was a powerful independent indicator of psoriasis, RA, and SLE.[18–20] In our study, we found that the WBC and neutrophil counts in gout patients were significantly higher than in HCs, and lymphocyte count was lower in gout patients than in HCs, which reflected the pathological mechanism of acute attack in gout patients. The increase of NLR and PLR may be due to the increase in neutrophils or decrease in lymphocytes, or both. Furthermore, our study found that both NLR and PLR were positively related to ESR and CRP, suggesting NLR and PLR could be potential markers of inflammation in gout.

There are inconsistencies regarding whether MPV is increased or decreased in inflammatory diseases. Increased MPV has been reported in patients with RA,[21] SLE,[22] systemic sclerosis,[23] ankylosing spondylitis,[24] and Behcet disease.[25] In contrast, Kisacik et al[26] reported that MPV was lower in patients with ankylosing spondylitis. Zhang et al[27] found that there was no difference in MPV between patients with gout and HCs. However, our study showed that MPV was lower in gout patients than in HCs. Some research reported that disease activity did not affect MPV in RA and SLE[28]; however, in our study, MPV was markedly lower in the gout flare group than the intercritical gout group. This inconsistency could be explained by differences in the disease activity status of patients. It has been suggested that MPV varies according to the intensity of inflammation: in low-grade inflammation, large PLTs are released from the bone marrow; in high-grade inflammation, the consumption of large PLTs exceeds their release, resulting in a decrease in MPV.[29,30] The decrease in MPV was thought to be regulated by proinflammatory cytokines, such as IL-1β, IL-6, and TNFα, which may explain the findings in this study.

According to this study, NLR and PLR were positively correlated with ESR (R = 0.253 and R = 0.434, respectively) and CRP (R = 0.367 and R = 0.340, respectively) in gout patients. MPV was negatively correlated with ESR (R = –0.183, P = .048), but not with CRP (R = –0.122, P = .189) or SUA (R = –0.044, P = .624). In addition, the ROC curve showed that NLR and PLR had higher areas under ROC curves than MPV. After Bonferroni correction, we found that MPV showed no correlation with ESR and CRP. All these results showed that NLR and PLR would be more useful than MPV in determining disease activity in gout patients.

In conclusion, NLR, PLR, and MPV appear to be markers of systemic inflammation in patients with gout and could serve as complementary markers to evaluate disease activity in gout. The greatest advantage of these results is that these 3 ratios can easily be calculated from routine blood counts and are less costly when compared with other proinflammatory cytokine tests, such as IL-6 and TNFα. Therefore, these parameters are suitable for clinicians to use in diagnosis and treatment.

5. Limitations of this study

There are some limitations of our study. First, this study was retrospective and conducted at a single center; therefore, we could not evaluate a causal relationship between NLR, PLR, MPV, and disease activity in gout patients. Furthermore, all the clinical data and laboratory indicators were collected from electronic medical records, which were missing some clinical data, such as numbers of tophi. Second, the number of the samples was relatively small, and all the patents who were enrolled in the inpatient department were suffering a serious condition, which could have led to selection bias. Therefore, further multicenter and prospective studies are needed to confirm our results.

Author contributions

HW and PC conceived and designed the study. HW and HZ collected and analyzed the clinical data. HW and PC wrote, reviewed, and revised the manuscript.
Conceptualization: Haihua Wu, Panfeng Chen.
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Project administration: Haihua Wu, Panfeng Chen.
Resources: Hui Zhou.
Writing – original draft: Haihua Wu.
Writing – review & editing: Panfeng Chen.

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