Increased platelet to lymphocyte ratio is related to inflammation in patients with Takayasu arteritis

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Abstract: Objectives: Takayasu arteritis (TA) is a chronic inflammatory disease that involves the aorta and main branches. Platelet to lymphocyte ratio (PLR) has been regarded as an indicator to assess inflammatory state in related rheumatic diseases. However, the association between PLR and TA in the literature has not been fully understood. Thus, the aim of our study is to investigate a relationship between PLR and TA. Methods: This cross-sectional study involved 108 newly diagnosed TA patients who were admitted to the First Affiliated Hospital of Xinjiang Medical University from 2010 to 2015. Results: PLR were higher in active TA patients (a-TA) compared with na-TA (145.4 ± 53.25 vs. 104.4 ± 34.45, P<0.001). PLR was negatively correlated with lymphocyte count in patients with a-TA, and positively correlated with platelet count, hemoglobin and neutrophil to lymphocyte ratio (NLR). Interestingly, a positively correlation between PLR and C reactive protein (CRP) was observed in a-TA. However, there was no correlation between PLR and CRP in na-TA. Stepwise logistic regression analysis showed that PLR was associated with a-TA independently of erythrocyte sedimentation rate (ESR) and CRP. When Receiver operating characteristic (ROC) curves were used to identify the performance of PLR in assessing a-TA, the area under the curve was 0.743 (95% CI: 0.644-0.842, P<0.001) with the sensitivity and specificity of 82.6% and 55.5%, respectively. Conclusions: Our study suggests that increased PLR is associated with TA patients with active disease, and is correlated with CRP in active TA patients. These results indicate that PLR may be used as an indicator to assess disease activity and reflect active inflammation in patients with TA.

Keywords: Takayasu arteritis, platelet to lymphocyte ratio, C reactive protein
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**Table 1.** Baseline information among the active patients, inactive patients and all patients with TA

<table>
<thead>
<tr>
<th></th>
<th>Total TA patients (N=108)</th>
<th>Active group (N=58)</th>
<th>Inactive group (N=50)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>26.9 ± 9.71</td>
<td>24.5 ± 10.16</td>
<td>28.9 ± 8.95</td>
<td>0.020</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>105</td>
<td>7/40</td>
<td>45/13</td>
<td>0.329</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>26.9 ± 9.71</td>
<td>17.3 ± 19.0</td>
<td>5.3 ± 4.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ESR (mm/h)</td>
<td>22.9 ± 16.93</td>
<td>35.9 ± 17.16</td>
<td>12.2 ± 5.01</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Neutrophil count (×10⁹/L)</td>
<td>5.8 ± 2.24</td>
<td>6.1 ± 2.76</td>
<td>5.6 ± 1.72</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lymphocyte count (×10⁹/L)</td>
<td>2.3 ± 0.75</td>
<td>2.1 ± 0.66</td>
<td>2.5 ± 0.79</td>
<td>0.016</td>
</tr>
<tr>
<td>Platelet count (×10⁹/L)</td>
<td>285.3 ± 93.88</td>
<td>332.0 ± 108.91</td>
<td>248.2 ± 56.40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>118.1 ± 16.64</td>
<td>110.6 ± 16.86</td>
<td>124.1 ± 13.93</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NLR</td>
<td>2.5 ± 1.15</td>
<td>2.5 ± 1.18</td>
<td>2.5 ± 1.13</td>
<td>0.995</td>
</tr>
<tr>
<td>PLR</td>
<td>122.9 ± 48.25</td>
<td>145.4 ± 53.25</td>
<td>104.4 ± 34.45</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Figure 1. PLR were higher in a-TA compared with na-TA (P<0.001).

The data analysis was performed by using SPSS16.0 (SPSS Inc, Chicago, IL, USA) statistical software. Continuous variables were showed as means ± SD. The Kolmogorov-Smirnov test was used to determine the normal distribution. Differences between the two groups were assessed by student’s t-test, Mann-Whitney U test and X test. The correlation between the laboratory parameters was analyzed using Spearman correlation. Multivariate logistic regression analysis was performed to determine the factors associated with active TA patients (a-TA). The performance of variables was estimated by receiver operating characteristic (ROC) curves. P<0.05 was accepted as statistically significant.

**Results**

A total of 108 TA patients were included in this study. All clinical data and laboratory data for a-TA and na-TA were outlined in Table 1. At baseline, age, CRP, ESR, platelet count and...
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Hemoglobin were found to be statistically significant between a-TA and na-TA, as shown in Figure 1. PLR was higher in a-TA compared with na-TA (145.4 ± 53.25 vs. 104.4 ± 34.45, P<0.001). PLR was negatively correlated with lymphocyte count, and positively correlated with platelet count, hemoglobin and NLR in patients with a-TA. Interestingly, a positively correlation between PLR and CRP was observed in a-TA, as shown in Figure 2. However, there was no correlation between PLR and CRP in na-TA.

Stepwise logistic regression analysis showed that PLR was associated with a-TA independently of ESR and CRP (Table 2). The area under the curve was 0.743 (95% CI: 0.644-0.842, P<0.001) with the sensitivity and specificity of 82.6% and 55.5% when receiver operating characteristic (ROC) curves were used to identify the performance of PLR in assessing a-TA, as shown in Figure 3.

Discussion

PLR is a useful marker to assess subclinical inflammation in peripheral blood, it is consist of information in regard to platelet and lymphocyte count, which may can avoid an acute alteration compared with single platelet and lymphocyte count for the assessment of inflammation in patients with TA. There were several remarkable findings to this study. We demonstrated that PLR was increased in a-TA compared with na-TA, and was associated with a-TA in logistic regression analysis. Importantly, increased PLR was positively correlated with CRP in a-TA.

Previous studies provided evidences that systemic or chronic inflammation is associated with increased platelet. Elevated PLR has been demonstrated to be associated with ulcers in patients with critical limb ischemia, patients with end stage renal disease and subclinical inflammation in atherosclerosis patients [10, 20, 21]. Thus, we can know the PLR not only shows the state of coagulation, but also reflects the inflammatory status. In fact, PLR as an available parameter in the blood routine tests is an objective measured value, which could be less affected by subjective factors of clinicians. In addition, there are no additional costs for PLR measurement compared with CRP, ESR and imaging findings. In the present study, we suggested that PLR is independently associated with a-TA, indicating increased PLR is an index to reflect disease activity in patients with TA.

Inflammatory cytokine is a potential factor to stimulate thrombocytopenia [22]. It has been demonstrated in previous trials that IL-6 and TNF are associated with disease activity in TA patients [4], and the levels of CRP and IL-18 are

![Figure 2. Scatter plot showed a positively correlation between PLR and CRP in a-TA.](image)

**Table 2. Multivariate logistic regression analysis between laboratory parameters and a-TA**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>p-value</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP (mg/L)</td>
<td>&lt;0.001</td>
<td>2.230</td>
<td>1.145-3.580</td>
</tr>
<tr>
<td>ESR (mm/h)</td>
<td>&lt;0.001</td>
<td>1.278</td>
<td>1.008-2.568</td>
</tr>
<tr>
<td>PLR</td>
<td>0.001</td>
<td>1.820</td>
<td>1.230-2.872</td>
</tr>
</tbody>
</table>

![Figure 3. A receiver operating characteristic (ROC) curves for PLR in estimating patients with a-TA.](image)
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Increased PLR in patients with active TA [23]. Klinge M.H et al. [24] observed that some inflammatory factors such as IL-1, IL-6 and IL-11 were involved in the pathogenesis of TA, which results in increased platelet in patients with TA. Immune and inflammatory factors may also cause elevated platelet in some autoimmune diseases, including rheumatoid arthritis, systemic lupus erythematosus and inflammatory bowel disease [14, 15, 25]. Similarly, increased platelet count has been reported to be associated with the disease severity in psoriasis patients [26]. The mechanism probably explains that aortic lesions may cause higher inflammatory response by aggregating inflammatory cytokines, which produced a relatively large platelet by stimulating megakaryocyte proliferation. In contrast, lymphocyte is reduced in immune disease since inflammatory response and excessive apoptosis simultaneously [27]. Indeed, reduced lymphocyte has been suggested in some diseases such as type 2 diabetes, acute pancreatitis and acute coronary syndrome [28-30]. Moreover, decreased lymphocyte in peripheral circulation has been reported in patients with Behcet’s disease, inflammatory bowel disease and vestibular neuritis [31-33]. Thus, elevated PLR may be explained by active inflammatory status in patients with TA.

There were several limitations in this study. First, our study did not assess the value of PLR in patients undergoing anti-inflammatory treatment. In addition, a small sample size should be noted in the present study. Finally, increased PLR were not estimated in TA subgroups. However, our study suggests that increased PLR is associated with TA patients with active disease, and is correlated with CRP in active TA patients. These results indicate that PLR may be used as an indicator to assess disease activity and reflect active inflammation in patients with TA.

Disclosure of conflict of interest

None.

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References

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