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Original Article

Platelet count to lymphocyte count ratio may predict mortality in Stanford type B acute aortic dissection

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Abstract: Background: Acute aortic dissection (AAD) is a common disease with a high death rate. It has many relationships with inflammation activated by the tearing of the aortic wall. Our aim was to determine whether platelet-to-lymphocyte ratio (PLR) can predict the mortality of patients with Stanford type B AAD. Methods: We collected patients with Stanford type B AAD from 2007 to 2012. We measured the basic information for all patients such as race, sex, age, past medical history, white cell count, platelet count, lymphocyte count, treatment and so on. All of the patients were followed up for complications and survival status for years after admission. We used receiver-operating characteristic (ROC) analyses for end point events for all-cause mortality in order to determine the cut-off points. We divided the patients into high and low PLR groups. Logistic analysis and Kaplan-Meier curves were compared between the two groups. The relationship of the platelet count to lymphocyte count ratio and mortality was assessed for patients with Stanford type B AAD evaluate the prognosis of patients in these two groups by assessing the relationship of the PLR and mortality. Results: This study included 134 patients with Stanford type B AAD (men 73.9%). The study's primary end-point was all-cause death. The median time from symptom onset to the end of follow-up was 30.93 months. There were 19 deaths and 115 survivors. The best cut-off value was 260.68 (10^9). The area under the curve was 0.711 (95% confidence interval 0.58-0.84. $P < 0.05$, sensitivity and specificity 63% and 88%). These patients were divided into high-PLR (n = 24) and low-PLR (n = 110) groups. The survival rate of low-PLR patients was significantly lower than that of the high-PLR group (58.3% vs 91.8%; $P < 0.001$). Correlation analyses and logistic analysis found that the following variables were independently associated with mortality: PLR (OR, 6.14; 95% CI, 1.401-26.895; P = 0.02). Conclusions: We found that the PLR was an independent risk factor for type B AAD and the PLR could be used to evaluate patient prognosis and assist physicians in selecting treatments.

Keywords: Platelet-to-lymphocyte count ratio, mortality, type B acute aortic dissection

Introduction

Acute aortic dissection (AAD) is an emergent vascular disease with the clinical characteristics of high morbidity and mortality [1]. Approximately 48.6% of patients with AAD die before admission, and approximately 30% die in the hospital [2]. Aortic dissection is characterized by high morbidity, fatality and misdiagnosis rates; 5-10 out of every 100,000 people experience aortic dissection, and in a preliminary assessment of aortic dissection, a foreign study reported that the misdiagnosis rate of AAD was as high as 40% [3].

Stanford type B AAD without complications is usually treated medically and has a lower in-hospital mortality rate. The 5-year mortality rate is 30-40% [4]. However, to identify high-risk factors for type B AAD, several predictors of short-term adverse events in AAD have been investigated. These predictors, including older age [5], female sex [6], hypotension [7], and severe emotional stress [8], are associated with a higher risk of adverse events in AAD patients. Investigators have also found that maximum aortic diameter [9] and false lumen closure status [10] can predict outcomes in AAD. Many methods can be used to diagnose AAD, including magnetic resonance imaging (MRI) and especially computed tomographic angiography (CTA). However, these tools are unavailable at the bedside, are time-consuming or have low sensitivity and specificity for diag-

nosing AAD, and consequently, AAD has high mortality and misdiagnosis rates. Therefore, there is an interest in identifying additional simple biomarkers that could be used to predict outcomes of type B AAD at admission. Biomarkers that are rapid, noninvasive, accurate and helpful for diagnosis and treatment can be used to guide clinicians' decision making.

Previous studies have proven that thrombotic biomarkers such as platelet count [11] and D-dimer have been associated with mortality in AAD patients [12]. Platelets are a pivotal factor in the development of AAD. At the site of the aortic wall injury, platelets gather and form a thrombus within the false lumen, which expands the rupture of the AAD. There is a significant negative correlation between the platelet count and outcomes of AAD [13]. AAD patients with a high MPV/platelet count ratio have been reported to have more frequent onset of in-hospital adverse complications and poor survival [14]. T lymphocytes enter the peripheral blood and immune organs, an acute inflammatory response occurs, and then T lymphocytes are activated and peripheral lymphocytes decrease; thus, the platelet count to lymphocyte count ratio (PLR) increases significantly. During the chronic inflammation stage, lymphocytes play a major role. Lymphocytopenia indicates a generalized state of immune depression, and survival appears to be adversely influenced by the depressed immune function. Studies have reported that AAD patients have low lymphocyte levels. However, the value of the PLR for predicting mortality in type B AAD has not been reported. To the best of our knowledge, this study is the first to evaluate the potential prognostic value of the PLR in patients with type B AAD.

Methods

This is a single centre prospective study to evaluate the potential prognostic value of the PLR in patients with Stanford type B AAD. The study was approved by the medical ethics committee of the First Affiliated Hospital of Xinjiang Medical University (No. 20070826-8). All of the participants signed the informed consent.

Diagnosis of Stanford Type B AAD and study population

For this study, we primarily used CTA and typical clinical symptoms for diagnosis. Any dissections that did not involve the ascending aorta, regardless of the location of the initial intimal tear or dissection range, was defined as Stanford type B AAD (Stanford classification). According to the time of symptom onset, aortic dissection can be divided into four stages: super acute (< 24 h), acute (2-7 days), subacute (8-30 days), and chronic (> 30 days) [15]. The exclusion criteria were as follows: refusal to participate in the study; past aortic surgery, infectious diseases, haematological system diseases, and liver or spleen diseases; and patients who had coronary heart disease, systemic inflammatory disease, heart failure or Marfan syndrome were also excluded. All of the subjects were treated with surgery and drugs according to their wishes, or only by conservative medication. According to the patient's condition, we gave angiotensin II receptor (ARB), angiotensin-converting enzyme inhibitor (ACEI), calcium-channel blocker (CCB), β-blockers, and diuretics selectively.

Blood collection and assays

Intravenous blood samples were collected using standardized EDTA blood vessels within 1 hour after admission. White blood cell (WBC), platelets, and lymphocytes were counted using an automated blood analysis system by Beckman Coulter LH750 (USA).

Other definitions

The laboratory and clinical features of patients, such as age, sex, previous diagnosis of diabetes, hypertension, hypercholesterolemia, hypertriglyceridemia, smoking, weight, family history of cardiovascular disease, and height and weight were obtained from medical records. Hypertension was considered in patients with repeated blood pressure measurements if the systolic BP (SBP) was ≥ 140 mmHg and/or the diastolic BP (DBP) was over 90 mmHg and/or they used antihypertensive medication. Type 2 diabetes was diagnosed when plasma fasting glucose was more than 7.0 mmol/L (or 2 h postprandial glucose ≥ 11.1 mmol/L) and/or if there was current use of diabetes medication. Smoking was defined as current smoking.

End-points and follow-up

The study's primary end-points were all-cause death, including those due to recurrence of an aortic dissection or aortic rupture. Patients

Table 1. General characteristics of the project participants by male and female groups

Patient characteristics	Men (n = 99)	Women (n = 35)	P
Age (year)	50.59 ± 13.70	52.17 ± 11.55	0.213
Race [n (%)]	77 (77.8)	28 (80.0)	0.794
Smoking [n (%)]*	56 (56.6)	1 (2.9)	0.000
Hypertension [n (%)]	83 (83.8)	27 (77.1)	0.375
Admission SBP (mmHg)	154.01 ± 32.22	146.43 ± 27.47	0.734
Admission DBP (mmHg)	94.03 ± 24.07	85.26 ± 19.50	0.590
PLR	179.69 ± 95.96	181.26 ± 66.35	0.095
Medical treatment [n (%)]	24 (24.2)	14 (14)	0.075
Surgery [n (%)]	4 (4.0)	1 (2.9)	0.751
Interventional therapy [n (%)]	71 (71.7)	20 (20)	0.112

*P < 0.05. PLR, platelet to lymphocyte count ratio. SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2. Clinical background of patients with acute type B aortic dissection by survival and death groups

Variables	Survival (n = 115)	Death (n = 19)	*P*
Men [n (%)]	86 (74.7)	13 (68.4)	0.559
Age (years)*	49.66 ± 12.51	59.11 ± 14.32	0.003
Smoking [n (%)]*	54 (47.0)	13 (68.4)	0.011
Hypertension [n (%)]	95 (82.6)	15 (78.9)	0.700
Admission SBP (mmHg)	152.95 ± 29.09	146.47 ± 42.00	0.525
Admission DBP (mmHg)	92.1 ± 23.61	89.53 ± 21.16	0.633
WBC (× 10^9/L)	10.48 ± 4.06	12.48 ± 5.16	0.122
PLR*	166.34 ± 76.35	263.40 ± 154.89	0.015
Medical treatment [n (%)]*	28 (24.3)	10 (52.6)	0.011
Interventional therapy [n (%)]*	83 (72.2)	8 (42.1)	0.009
Surgery [n (%)]	4 (3.5)	1 (5.3)	0.704
Survival time [n (%)]*	33.87 ± 19.18	13.54 ± 13.85	0.000

**P* < 0.05; SBP, systolic blood pressure; DBP, diastolic blood pressure; PLR, platelet to lymphocyte count ratio; WBC, white blood cell count.

were followed up for 3.5 years after admission. Telephone inquiries and discussions with hospital visitors were used to obtain outcomes and other information.

Statistical analysis

Data are presented as the means ± standard deviations, frequencies and percentages. Receiver-operating characteristic (ROC) analyses were used to determine the Youden index (sensitivity + specificity-1) in order to divide patients into high and low PLR groups. The differences between groups (male and female groups, death and survival groups, high and low PLR groups) were determined using unpaired Student's t tests or chi-square tests. Kaplan-Meier curves for the high and low PLR group patients were compared using the log-rank test. Correlation analyses between variables were performed using Pearson's or Spearman's correlation. Odds ratios (OR) and confidence intervals (CI) were calculated for each factor using stepwise multivariate logistic analysis. A *P* value less than 0.05 was considered statistically significant. All statistical analyses were performed using SPSS 24.0 statistical software (SPSS, Chicago, Illinois).

Results

No different characteristics between male and female patients with Stanford type B AAD

From 2007 to 2012, 244 consecutive patients with AAD were admitted to our hospital. The final study population consisted of the 134 patients who met the inclusion and exclusion criteria. There were 99 men (73.9%) and 35 women (26.1%). The average age was 50.59 ± 13.70 in men and 52.17 ± 11.55 in women. Age, race, PLR, hypertension and treatments did not differ between the male and female patients, as summarized in **Table 1**.

PLR in the death group was significantly higher than that in the survival group. The follow-up period was taken from July to August 2013 of the 134 patients with Stanford type B AAD. The study's primary end-point was all-cause death. In the end, there were 19 deaths and 115 survivors. The median time from symptom onset to the end of follow-up was 30.93 months; for the deceased group, it was 13.54 ± 13.85 months, and for the surviving patients, it was 33.87 ± 19.18 months (P < 0.001). Compared

Table 3. General characteristics of the project participants by H-PLR and L-PLR groups

Patient characteristics	H-PLR (n = 24)	L-PLR (n = 110)	*P*
Men [n (%)]	19 (79.2)	81 (73.6)	0.890
Age (years)	55.08 ± 13.32	50.11 ± 13.00	0.752
Smoking [n (%)]	6 (25.0)	51 (46.4)	0.055
Hypertension [n (%)]	19 (19)	91 (82.7)	0.680
Admission SBP (mmHg)	145.71 ± 37.84	153.41 ± 29.49	0.589
Admission DBP (mmHg)	89.75 ± 21.01	92.17 ± 23.74	0.838
WBC (× 10^9/L)	11.81 ± 4.63	10.54 ± 4.17	0.364
Complication			
Pericardial effusion [n (%)]*	11 (45.8)	21 (19.1)	0.005
Acute kidney injury [n (%)]*	6 (25.0)	8 (7.3)	0.010
New nerve damage [n (%)]*	3 (12.5)	3 (2.7)	0.036
Fever (%)*	3 (12.5)	38 (34.5)	0.034
Hypotension [n (%)]*	1 (4.2)	0 (0)	0.032
Limb movement disorder [n (%)]	3 (12.5)	4 (3.6)	0.077
Survival time (months)	28.03 ± 19.78	31.63 ± 19.83	0.918
Follow-up survival [n (%)]*	14 (58.3)	101 (91.8)	0.000

*$P < 0.05$, H-PLR, high-platelet to lymphocyte count ratio, L-PLR, low-platelet to lymphocyte count ratio. SBP, systolic blood pressure; DBP, diastolic blood pressure.

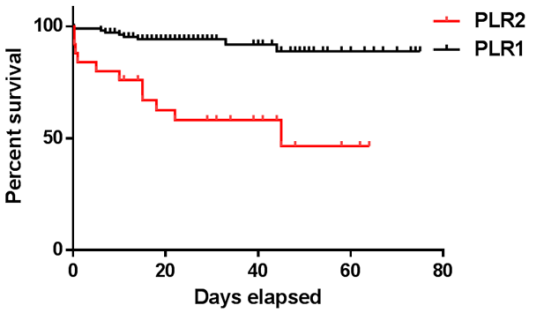

Figure 1. Kaplan-Meier survival analysis of all 134 patients with type B acute aortic dissection according to the cut-off value of PLR. PLR1 stands for low platelet to lymphocyte count ratio, PLR2 stands for high platelet to lymphocyte count ratio.

with the survival group, the death group had a greater average age (59.11 ± 14.32 years vs 49.66 ± 12.51 years; P = 0.003) and more smoking patients (68.4% vs 47.0%; P = 0.011). The mean of PLR in the death group was significantly higher than that in the survival group (263.40 ± 154.89 vs 166.34 ± 76.35; P = 0.015). Sex, history of hypertension and WBC cell count were not statistically significantly different between the death and survival groups. All 134 type B AAD patients either underwent surgery (71.6%) or medical treatment alone (28.4%), and there was a higher mortality rate (66.7%) for the patients who only received medical therapy than for those who received other treatments (33.3%). For details see **Table 2**.

The PLR is valuable in the diagnosis of Stanford type B AAD

The recommended cut-off value for PLR based on the maximum Youden index on the ROC curve was 260.68. The area under the curve was 0.711 (95% confidence interval [CI] 0.58-0.84, P < 0.05) and the sensitivity and specificity were 63% and 88%, respectively.

More death in the high-PLR group than the low-PLR group

The type B AAD patients were divided into high-PLR (n = 24) and low-PLR (n = 110) groups based on the cut-off value of 260.68 (10^9), which was determined by ROC curve analysis. The clinical characteristics of the groups are shown in **Table 3**. The high group had a lower survival time, but the difference was not statistically significant (28.03 ± 19.78 months vs 31.63 ± 19.83 months; P = 0.918). The presence of complications such as pericardial effusion, fever, acute kidney injury, and nervous lesions differed significantly between the two PLR groups (P < 0.05). Fever was objectively observed in approximately one-third of cases of AAD; a previous report showed that the fever was caused by a systemic inflammatory response syndrome and endogenous mediators [16]. Other clinical characteristics did not vary between the type B high- and low-PLR groups. In addition, compared with the medicine-only group, patients with type B AAD who underwent urgent surgery had higher survival rates. The cumulative survival rate of the low-PLR patients was significantly lower than that of the high-PLR group (58.3% vs 91.8%; P < 0.001) shown in **Figure 1.**

All-cause mortality for patients with Stanford type B AAD

Many variables can influence prognosis; logistic analysis indicated that PLR was positively associated with all-cause mortality. After ad-

Table 4. Logistic regression of all-cause mortality for patients with type B acute aortic dissection in the study period

Variable	B	S.E.	Wald	*P*	OR	95% CI
PLR	1.815	0.754	5.794	0.02	6.14	1.401-26.895
Smoking	2.047	0.926	4.885	0.03	7.74	1.261-47.564
Age	0.068	0.031	4.779	0.03	1.07	1.007-1.137
Complication	2.713	0.929	8.539	< 0.01	15.08	2.444-93.081

PLR, platelet to lymphocyte count ratio.

justment for potential con-founders, PLR was an independent predictor of all-cause mortality (hazard ratio 6.14, 95% CI 1.401-26.895, P = 0.02). Other predictors of all-cause mortality were smoking, age and the occurrence of complications (**Table 4**).

Discussion

The progression of aortic dissection is rapid. Patients with type A mainly proceed to emergency surgery, so the role of biochemical markers in Stanford type A is limited. Therefore, we only need biochemical markers for type B, but there is no known definite marker to evaluate the prognosis of type B patients. Therefore, our study aimed to find a new marker for this purpose. A role of PLR in prognostication of colorectal cancer and non-ST-segment elevation acute coronary syndrome has been reported, but the value of PLR for predicting mortality in Stanford type B AAD patients was unclear. The present study investigated whether PLR has prognostic value for predicting short- and long-term mortality in Stanford type B AAD patients. In our study, PLR was an important, independent, potential prognostic factor; this ratio may be able to risk-stratify AAD patients at admission to predict adverse outcomes of type B patients, such as complications and short- and long-term all-cause mortality.

Based on survival status at the end of the follow-up period, the patients were divided into two groups: death and survival. In the death group, the PLR was higher than in the survival group (263.40 ± 154.89 vs 166.34 ± 76.35) and their survival time was shorter (13.54 ± 13.85 months vs 33.87 ± 19.18 months). The patients who died had a higher PLR than the patients who survived.

AAD is the most common life-threatening emergency disease. It has a high misdiagnosis and mortality rate without early diagnosis and appropriate management. Compared with Stanford type A AAD, type B is considered relatively benign, but surgery is still the primary means of improving patient outcomes [17]. Stanford type B AAD without complications is usually treated medically, but the long-term outcome of medical treatment alone has been reported to be sub-optimal, with a high 5-year mortality rate. We also found that medical treatment had high mortality (52.6%) relative to interventional therapy (42.1%) and surgery (5.2%), but the treatments used did not differ significantly between the high- and low-PLR groups in our study.

A systemic inflammation reaction is responsible for the pathogenesis of AAD. It is provoked by aortic intima injury and is reflected by some markers in the serum, including the WBC count, CRP, D-dimer and pro-inflammatory cytokine levels. Studies have reported that D-dimer and CRP levels were related to AAD and were associated with a poor prognosis [18]. When AAD occurs, platelets are activated and adhere to the damaged vessel wall, which leads to thrombus formation within the false lumen. A high level of platelet activation is associated with poor outcomes in aortic dissection [19], and platelet count is a powerful predictor of increased risk of in-hospital death in Stanford type A aortic dissection, even among patients undergoing surgical intervention [17]. Thrombocytopenia is a predictor of late mortality (50.8 months) in Stanford type B aortic dissection according to Cox proportional hazards analysis (P < 0.042) and Kaplan-Meier survival curves. Lymphocytes are related to non-ST elevation acute coronary syndrome (NSTACS) and the immune system [20]. Lymphocyte levels reflect a generalized state of immune depression; in AAD, the lymphocyte count is reduced because of stress-induced steroid exposure. Evidence of whether lymphocyte count alone could be used to diagnose and predict the prognosis of AAD is lacking.

In our study, PLR had a negative relationship with outcomes of patients with Stanford type B aortic dissection; the patients who died had a higher PLR (263.40 ± 154.89) than those who survived (166.34 ± 76.35), and the high-PLR group had a higher mortality rate, including both short-and-long mortality. Smokers and patients who were older had more adverse out-

comes, and their platelet count and lymphocyte levels were lower than in the survival group, but these factors had no statistical significance to evaluate prognosis. Of the other main laboratory parameters, WBC counts (12.48 ± 5.16 vs 10.48 ± 4.06) were significantly higher in the death group than in the survival group. There were no significant differences between the groups in terms of lymphocyte or platelet count.

The latest studies reported that PLR has been widely studied as a prognostic factor in colorectal cancer [21] and non-ST-segment elevation acute coronary syndrome (NSTE-ACS) [22]. However, data regarding the role of PLR in acute aortic syndromes are lacking; thus, our study was the first to evaluate the potential role of PLR in Stanford type B AAD.

Many parameters can be used to predict prognosis, mainly for Stanford type A AAD; for example, the neutrophil-lymphocyte ratio can predict pericardial effusion and in-hospital mortality in patients with AAD type A [23]. This article was limited to the prognosis of Stanford type B AAD; no previous article has shown the predictive value of PLR for Stanford type B AAD. In this article, we used logistic analysis to prove that PLR was an independent prognostic factor for Stanford type B AAD. PLR, smoking, complications, and age are risk factors for poor outcomes of patients with AAD. In terms of risk factors, many articles have reported that platelet counts are correlated with outcomes, possibly because of platelet activation in the formation of thrombi in the false lumen, which increases the rupture in the AAD. Platelet activation also increases bleeding, which influences the prognosis. Regarding lymphocytes, which include T lymphocytes and B lymphocytes, there are currently no articles showing that they can be used to predict the outcomes of AAD. A systemic inflammatory response may be the mechanism through which lymphocytes influence AAD, and anti-inflammatory therapy [24, 25] can improve outcomes after aortic dissection.

The PLR is a novel and inexpensive index of systemic inflammation. It is determined by dividing the absolute platelet count by the absolute lymphocyte count and is more accurate than either platelet or lymphocyte count alone for predicting the outcome of AAD patients. Therefore, an elevated PLR is an easily obtained parameter that reflects thrombo-inflammatory status in Stanford type B AAD and can be used to predict patient outcomes.

Limitations

First, this study has a small sample size and cannot comprehensively reflect all AAD cases. We used a relatively small population to design a single-centre, prospective cohort study. Second, the endpoint of follow-up was limited to all-cause mortality, and complications were not specified. Finally, we did not determine the counts of leukocytes and their sub-types for concomitant evaluation to compare their values for predicting mortality in Stanford type B AAD.

Conclusion

We concluded that a high PLR is a strong predictor of all-cause mortality in patients with Stanford type B AAD. Furthermore, platelet and lymphocyte counts can easily be obtained from routine blood results, and the PLR may be considered by clinicians when choosing a clinical intervention that decreases the mortality of patients with type B aortic dissection.

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Disclosure of conflict of interest

None.

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