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ORIGINAL RESEARCH

Impact of Pan-Immune Inflammation Value on Short-Term Outcomes and Long-Term Prognosis in Patients with Type A Aortic Dissection

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Background: Inflammatory responses are closely linked to the onset and progression of aortic dissection. The Pan-Immune Inflammation Value (PIV), a composite index derived from peripheral blood cell counts, has demonstrated prognostic relevance in multiple clinical conditions. However, its predictive value in acute Type A Aortic Dissection (TAAD) has not been well established. This study aims to investigate the relationship between preoperative PIV and both short-term and long-term outcomes in patients with acute TAAD.

Methods: This retrospective study included acute TAAD patients who underwent surgical repair between September 2017 and December 2020. The optimal cutoff value for PIV was determined using receiver operating characteristic (ROC) curve analysis. Patients were then divided into low and high PIV groups based on this threshold. Short-term outcomes, including prolonged ICU stay (>7 days) and postoperative complications were compared between groups using univariate and multivariate logistic regression analyses. Cox regression analyses were performed to identify independent predictors of long-term survival.

Results: A total of 171 acute TAAD patients were included and stratified into low (n = 75) and high (n = 96) PIV groups based on preoperative values. In the high PIV group, patients had significantly longer surgery time, increased blood loss, greater volumes of red blood cell and plasma transfusions, longer ICU stays, and higher incidence of both overall and major complications (p=0.007, p=0.010, p=0.001, p=0.040, p=0.048, p<0.001, p=0.003, respectively). Multivariate logistic regression analysis identified high preoperative PIV as an independent risk factor for prolonged ICU stay (>7 days) (OR = 0.686, 95% CI, 0.303–0.954, p = 0.027), overall complications (OR = 0.037, 95% CI, 0.005–0.210, p = 0.002), and major complications (OR = 0.085, 95% CI, 0.026–0.173, p = 0.017). Additionally, lower preoperative PIV levels were significantly associated with improved long-term survival (HR = 0.757, 95% CI, 0.378–0.859, p = 0.020).

Conclusion: PIV was identified as an independent predictor of prolonged ICU stay, overall and major postoperative complications, and long-term survival in patients with acute TAAD. As an integrative biomarker reflecting systemic immune-inflammatory status, PIV may serve as a valuable tool for early risk stratification and prognostic management.

Keywords: type A aortic dissection, inflammatory response, overall survival, pan-immune inflammation value, prolonged ICU stay, postoperative complications

Introduction

Aortic dissection is a life-threatening vascular emergency associated with a high mortality rate. According to the Stanford classification, Type A aortic dissection (TAAD), which involves the ascending aorta and may extend to other parts of the aorta, typically has a worse prognosis compared to Type B, which is confined to the descending aorta.[1] Data indicate that the pre-hospital mortality rate for patients with TAAD is as high as 48.6%, while the 30-day mortality rate for those who survive to admission is 47.4%,[2] making it one of the leading causes of death within 24 hours of onset.[3] Recent years have seen an increase in the incidence of Type A aortic dissection, particularly among younger patients.[4] Currently, surgery

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remains the primary treatment for TAAD. However, the prevention of postoperative complications and ensuring comprehensive care are equally essential for improving patient outcomes. Therefore, early assessment and identification of high-risk TAAD patients are critical for improving survival outcomes and optimizing resource allocation.

Several biomarkers have been identified with potential diagnostic and prognostic value in aortic dissection. However, their clinical application has been limited due to various challenges. Smooth muscle myosin heavy chain (smMHC) is a protein specific to smooth muscle, with serum levels increasing up to 20-fold within 24 hours of disease onset.[5] Within the first 3 hours, elevated circulating smMHC levels exhibit a sensitivity of 90.9% and a specificity of 98%.[6] Other biomarkers, such as serum amyloid A1 (SAA1)[7] and matrix metalloproteinases (MMPs),[8] have also been investigated, but their clinical integration has been hindered by technological support, equipment, and cost limitations. Therefore, there is an urgent need for a reliable, easily accessible biomarker to support risk stratification in TAAD patients.

Inflammation is central to the progression of TAAD, contributing to aortic wall damage and alterations in endothelial cell function.[9] Additionally, elevated inflammation levels are closely associated with the postoperative complications, including acute kidney injury, cerebral complications, and liver dysfunction.[10] Previous studies have identified several inflammation-related biomarkers with potential prognostic value in patients with aortic dissection, including the neutrophil-to-lymphocyte ratio (NLR),[11] C-reactive protein (CRP),[12] and interleukin-6 (IL-6).[13] However, these biomarkers primarily reflect specific aspects of the inflammatory response and may fail to capture the overall immune-inflammatory status. Chen et al further noted the limited predictive value of individual indicators and emphasized the need for more comprehensive assessments to improve prognostic accuracy.[14] Therefore, composite indices derived from multiple immune and inflammatory cell counts—such as the Pan-Immune Inflammation Value (PIV)—have emerged as promising candidates for more reliable and comprehensive risk stratification.

PIV, a novel systemic inflammation marker, combines platelets, monocytes, lymphocytes, and neutrophils counts. Easily obtained from routine blood tests, PIV has demonstrated promising prognostic value across various disease settings. Wang et al reported that elevated PIV levels were associated with an increased risk of postoperative recurrence in patients with colorectal cancer.[15] In cardiovascular research, PIV has also been linked to adverse events in patients with myocardial infarction undergoing percutaneous intervention, demonstrating superior predictive performance compared to conventional inflammatory markers in assessing coronary artery stenosis.[16] However, its prognostic significance in patients with TAAD remains unexplored, particularly regarding its ability to predict long-term survival, ICU stay, and postoperative complications.

Given the prognostic potential of PIV, this study aims to retrospectively analyzes the clinical data of TAAD patients to evaluate its utility to predict both short-term and long-term outcomes. The objective is to refine risk stratification, supporting personalized decision-making and enhancing perioperative management.

Materials and Methods

Study Design and Participants

This study was designed as a retrospective cohort study and conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines (Table S3). Consecutive patients with acute Stanford TAAD admitted to the Department of Cardiothoracic Surgery at the First Affiliated Hospital of Chongqing Medical University between September 2017 and December 2020 were included. Patients were categorized into high and low PIV groups for comparative analysis. Patients were eligible for inclusion if they met the following criteria, (1) a diagnosis of Stanford TAAD confirmed by aortic computed tomography angiography (CTA); (2) patients who underwent surgical repair for TAAD. Exclusion criteria included, (1) absence of clinical or laboratory data exceeding 20%; (2) presence of hematological disorders, infections, or treatments that could affect biomarkers; (3) death from causes unrelated to TAAD; (4) TAAD patients with chronic phase. Given the relatively low incidence of TAAD, all eligible cases were consecutively included in accordance with predefined inclusion and exclusion criteria to ensure data integrity and reduce selection bias. Ultimately, 171 patients were included based on these criteria (Figure 1). The final sample size is comparable to previous studies investigating prognostic markers in TAAD,[17] and reflects the number of eligible patients available during the study period. The study was approved by the Independent Ethics

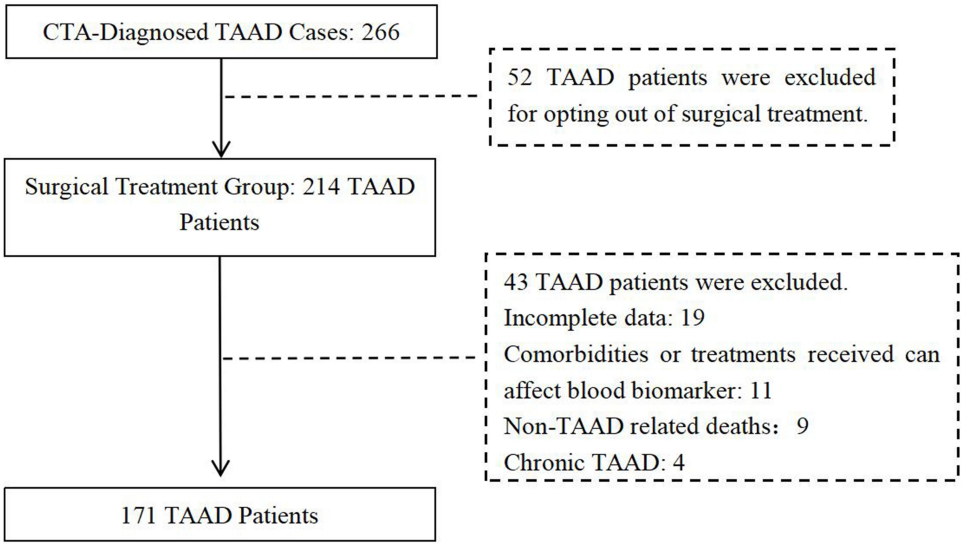

Figure 1 Workflow for Patient Inclusion.

Committee of the First Affiliated Hospital of Chongqing Medical University (2025–114-01) and conducted in accordance with the ethical principles of the World Medical Association Declaration of Helsinki.

Data Collection

Clinical information of the patients were retrospectively reviewed from the electronic medical record system, including gender, age, smoking history, history of hypertension, diabetes mellitus, cardiovascular disease, blood pressure at admission, and heart rate. Blood indicators were measured within 24 hours of hospital admission and prior to surgery. The measured biomarkers included absolute neutrophil count (ANC), white blood cell (WBC) count, red blood cell count (RBC), hemoglobin (Hb), platelet count, absolute lymphocyte count (ALC), monocyte count, serum albumin, uric acid, urea nitrogen, alanine aminotransferase (ALT), aspartate aminotransferase (AST), cardiac troponin T (cTnT), myoglobin (Mb), D-dimer, fibrinogen and D-dimer. PIV was calculated using the following formula, PIV = absolute neutrophil count (10^9/L) × monocyte count (10^9/L) × platelet count (10^9/L) / absolute lymphocyte count (10^9/L).

Follow-up and Treatments

All patients in the study underwent surgical repair. The follow-up period started three months post-discharge and continued until August 20, 2024. Trained interviewers conducted telephone follow-ups at 3, 6, and 12 months post-discharge, with subsequent follow-ups occurring every six months thereafter to ensure continuity of care and monitor patient outcomes. The longest survival duration observed in the study cohort was 2445 days, with a median survival time of 1190 days.

Definition

Aortic survival (AS) was defined as the time from surgery to an aortic-related death, including mortality due to aortic dissection, its complications (eg, surgical complications, aneurysm rupture, thrombosis), or reintervention-related causes. The primary endpoint of the study was mortality within the cohort. Complications were evaluated using the Clavien-Dindo classification system, with major complications defined as those classified as grade III or higher.[18] A prolonged ICU stay was defined as a stay exceeding 7 days.

Statistical Analysis

Random forest imputation was uniformly applied to both survivors and non-survivors for patients with a missing data rate below 20%. The optimal cutoff value of PIV for predicting outcomes was determined through receiver operating characteristic (ROC) curve analysis. Continuous variables are presented as mean ± standard deviation (SD), while categorical variables are

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expressed as n (%). Differences between groups were assessed using the independent samples *t*-test for continuous variables and the Chi-square test for categorical variables. AS was assessed using the Kaplan-Meier method, and survival curves were compared between the two groups. To construct the regression models, preoperative blood indicators with significant differences between PIV groups and intraoperative variables potentially affecting outcomes were included as covariates to control for confounding. Variable selection was based on both statistical significance and clinical relevance for inclusion in the regression models. Univariate and multivariate logistic regression analyses were conducted to identify independent predictors of prolonged ICU stay, overall complications, and major complications. Cox regression analysis was employed to determine predictors of long-term survival. A two-tailed p-value of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS version 27.0 and Python 3.9.

Results

Baseline Characteristics

Initially, 214 patients diagnosed with TAAD via CTA were included in this study. After excluding 19 patients with incomplete data, 11 patients with comorbidities or treatments affecting blood parameters, 9 patients who died of causes unrelated to TAAD, and 4 patients with chronic phase, 171 patients were ultimately included in the analysis. This cohort consisted of 53 deaths and 118 survivors. Missing data for key variables ranged from 0% to 11.11%. Random forest imputation was applied to cases with less than 20% missing data, while those exceeding this threshold were excluded from the analysis. The baseline characteristics of the patients are detailed in Table S1. The "Outcome Count (%)" column in the table shows the frequency of deaths in each subgroup and their proportion to the total number of deaths in that category.

The mean age of the included patients was 48.82 ± 9.61 years, with 136 male patients (79.53%) and 35 female patients (20.47%). Among the cohort, 118 patients (69.01%) had a history of hypertension, and 102 patients (59.65%) had a history of smoking—both of which are well-established risk factors for TAAD.

Significant statistical differences were observed between survivors and deceased patients in terms of clinical features such as shock, abdominal pain, and Pericardial effusion ($p<0.05$ for each). Although the incidence of shock was low (11 cases, 6.43%), its presence was associated with a significantly higher mortality rate compared to patients without shock (63.64% vs 28.75%). Pericardial effusion, a common complication in acute TAAD patients, was observed in 73 patients (42.69%) in our study. Those with pericardial effusion upon admission exhibited a significantly higher mortality risk, with a mortality rate of 43.84% compared to 21.43% in patients without effusion. This may reflect a more complex clinical condition, such as aortic dissection rupture or other severe complications, often accompanied by organ dysfunction or acute circulatory issues, further increasing the risk of mortality.

Sensitivity and Specificity of PIV at Admission

PIV demonstrated strong predictive performance for long-term survival in acute TAAD patients. The area under the ROC curve (AUC) was 0.816 (95% CI, 0.750–0.882, $p < 0.001$). The optimal cutoff value for PIV, determined using the maximum Youden's Index, was 1782. At this threshold, PIV exhibited a sensitivity of 68.5% and a specificity of 89.8% (Figure 2). Subsequently, PIV was divided into two groups based on the optimal cutoff value, the low PIV group (<1782) and the high PIV group (≥1782).

Association of Preoperative Blood Indicators with PIV Groups in TAAD Patients

The optimal cutoff value for PIV, determined through ROC curve analysis, was 1782. Based on this cutoff, TAAD patients were divided into two groups, those with low PIV (<1782) and those with high PIV (≥1782). Preoperative blood indicators for both groups are summarized in Table 1.

Significant differences were observed between the two groups in several key preoperative blood indicators, including WBC, ANC, ALC, PLT, albumin, creatinine, lactate dehydrogenase, D-dimer, and fibrinogen degradation products ($p < 0.05$ for all). These findings suggest that patients in the high PIV group exhibit a more severe pathophysiological state, characterized by increased inflammatory responses, greater tissue damage, renal dysfunction, and an elevated risk of thrombosis and fibrinolysis abnormalities. Such pathological changes are likely contributing factors to the poorer survival outcomes observed in this group.

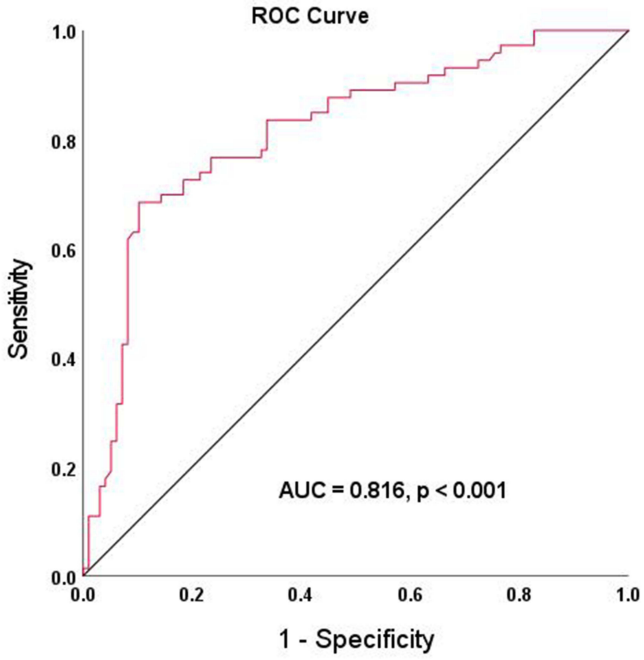

Figure 2 Optimal Cutoff Value of PIV for Predicting Long-Term Survival in TAAD Patients.
Abbreviations: AUC, Area Under the Curve; ROC, Receiver Operating Characteristic.

To account for potential confounding effects and better evaluate the independent prognostic value of PIV, we included the above-mentioned variables with intergroup differences in subsequent logistic and cox regression analyses. Notably, WBC, ANC, ALC, and PLT were excluded from the models as they are directly incorporated into the PIV calculation. FDP was also excluded, as its role in reflecting fibrinolytic activity is already captured by D-dimer, and including both

Table 1 Preoperative Blood Indicators of TAAD Patients Stratified by PIV

Variables	All Patients (%) 171 Cases	Low PIV Group 75 Cases	High PIV Group 96 Cases	P Value
WBC (×10^9/L)	12.35 ± 4.28	10.67 ± 3.35	15.46 ± 4.07	<0.001
RBC (×10^9/L)	4.02 ± 1.18	4.03 ± 1.20	4.01 ± 1.14	0.915
ANC (×10^9/L)	10.59 ± 4.30	8.88 ± 3.43	13.75 ± 3.95	<0.001
ALC (×10^9/L)	0.98 ± 0.52	1.12 ± 0.52	0.71 ± 0.41	<0.001
Hemoglobin (g/L)	129.37 ± 22.71	129.00 ± 23.36	130.07 ± 21.64	0.763
PLT (×10^9/L)	167.87 ± 58.71	161.06 ± 53.77	180.45 ± 65.53	0.039
Total Protein (g/L)	65.11 ± 7.02	65.04 ± 6.95	65.25 ± 7.20	0.851
Albumin (g/L)	33.71 ± 10.81	32.56 ± 11.54	35.85 ± 9.01	0.041
ALT (U/L)	46.04 ± 124.6	32.75 ± 26.80	70.62 ± 206.03	0.162
AST (U/L)	59.57± 171.30	43.02 ± 93.19	90.18 ± 258.63	0.086
Creatinine (μmol/L)	98.50 ± 57.87	90.28 ± 44.23	113.70 ± 75.09	0.011
Uric acid (μmol/L),	223.36 ± 207.21	218.36 ± 208.51	232.61 ± 206.20	0.669
LDH (U/L)	668.63 ± 974.36	485.50 ± 645.57	822.40 ± 1386.15	0.030
D-dimer (mg/L)	10.28 ± 15.71	6.98 ± 8.66	16.38 ± 22.65	0.003
Fibrinogen (g/L)	3.01 ± 1.69	3.02 ± 1.52	3.00 ± 2.00	0.953
FDP(μg/mL)	41.03 ± 87.78	22.56 ± 24.23	75.20 ± 138.84	0.005
Myoglobin (μg/L)	174.16 ± 478.97	128.87 ± 400.45	257.94 ± 592.50	0.134

(*Continued*)

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Table 1 (Continued).

Variables	All Patients (%) 171 Cases	Low PIV Group 75 Cases	High PIV Group 96 Cases	P Value
CK-MB (μg/L)	3.76 ± 7.01	3.47 ± 5.86	4.28 ± 8.78	0.524
BNP (pg/mL)	962.77 ± 1774.35	997.82 ± 2040.51	897.92 ± 1141.08	0.682
cTnT (ng/mL)	0.22 ± 0.79	0.24 ± 0.86	0.17 ± 0.64	0.519

Abbreviations: WBC, White Blood Cell Count; RBC, Red Blood Cell Count; ANC, Absolute Neutrophil Count; ALC, Absolute Lymphocyte Count; PLT, Platelet Count; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; LDH, Lactate Dehydrogenase; FDP, Fibrinogen Degradation Products; CK-MB, Creatine Kinase-MB; BNP, B-type Natriuretic Peptide; cTnT, Cardiac Troponin T.

may lead to redundancy. Incorporating these variables could introduce multicollinearity, thereby compromising the stability and interpretability of the regression coefficient estimates. Additionally, albumin was excluded due to its limited clinical relevance in the context of postoperative prognosis for TAAD.

Short-Term Outcomes in TAAD Patients

Table 2 presents a summary of the short-term prognostic outcomes of acute TAAD patients stratified by PIV levels. Statistical analysis revealed that operation time was significantly longer in the high PIV group compared to the low PIV group ($p = 0.007$), suggesting a link between elevated PIV levels and more advanced disease severity and surgical complexity. Additionally, ICU stay were significantly prolonged in the high PIV group ($p = 0.048$), supporting the notion that these patients may present with more severe conditions and require more complex postoperative management.

Moreover, patients in the high PIV group experienced significantly greater intraoperative blood loss ($p = 0.010$), higher plasma transfusion volumes ($p = 0.040$), and greater suspended red blood cell transfusion volumes ($p = 0.001$) compared to those in the low PIV group. These findings indicate that elevated PIV levels may be associated with more pronounced intraoperative bleeding and coagulation disturbances, necessitating greater blood product support. Further analysis showed that both the overall complication rate and the major complication rate were significantly higher in the high PIV group than in the low PIV group. These results underscore the potential role of elevated PIV levels as a significant predictor of postoperative complications in acute TAAD patients.

Univariate and multivariate logistic regression analyses demonstrated that preoperative PIV was a significant and independent predictor of prolonged ICU stay (defined as >7 days) in patients with acute TAAD. In the univariate analysis, a lower PIV (<1782 vs ≥1782) was associated with a reduced risk of prolonged ICU stay (OR = 0.420, 95% CI, 0.215–0.810, $p = 0.011$), while elevated creatinine was associated with increased risk (OR = 2.867, 95% CI, 1.101–7.463, $p = 0.031$). After adjusting for confounding factors in the multivariate model, PIV remained an independent predictor (OR = 0.686, 95% CI, 0.303–0.954, $p = 0.027$), as shown in Table 3.

Postoperative complications significantly affect survival outcomes in patients with TAAD. Univariate analysis identified several factors associated with overall complications (Table 4). It was found that Creatinine (OR, 4.486,

Table 2 Short-Term Outcomes Between the High PIV Group and the Low PIV Group

Variables	All Patients (%) 171 Cases	Low PIV Group 75 Cases	High PIV Group 96 Cases	P value
Operation time (minutes)	557.08 ± 133.10	541.21 ± 131.12	575.17 ± 135.90	0.007
Blood loss (mL)	671.50 ± 650.17	577.43 ± 355.33	845.53 ± 967.04	0.010
Plasma Transfusion (mL)	832.88 ± 382.63	788.73 ± 358.18	914.55 ± 414.98	0.040
Suspended Red Blood Cell Transfusion (U)	3.68 ± 2.97	3.15 ± 2.45	4.67 ± 3.58	0.001
ICU stay	11.91 ± 9.04	10.15 ± 7.73	12.85 ± 9.61	0.048
Overall Complications	115 (67.25%)	29 (38.67%)	86 (89.58%)	<0.001
Major Complications	44 (25.73%)	11 (14.67%)	33 (34.38%)	0.003

Abbreviation: PIV, Pan-Immune Inflammation Value.

Table 3 Univariate and Multivariate Logistic Analysis of Prolonged ICU Stay

Variables	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P	OR (95% CI)	P
Creatinine (μmol/L)	2.867 (1.101–7.463)	0.031	1.369 (1.069–2.295)	0.484
PIV (<1782 vs ≥ 1782)	0.420 (0.215–0.810)	0.011	0.686 (0.303–0.954)	0.027
LDH (U/L)	1.062 (0.574–1.966)	0.848	1.124 (0.567–2.229)	0.738
D-dimer (mg/L)	1.461 (0.558–3.822)	0.440	2.049 (0.691–6.078)	0.196
Operation time (minutes)	1.183 (0.612–2.146)	0.671	1.191 (0.604–2.347)	0.614
Blood loss (mL)	1.294 (0.793–2.381)	0.407	1.288 (0.651–2.548)	0.467
Plasma Transfusion (mL)	1.427 (0.772–2.639)	0.257	1.619 (0.833–3.147)	0.156
CBP time (minutes)	1.331 (0.616–2.878)	0.467	1.587 (0.685–3.679)	0.281

Abbreviations: PIV, Pan-immune Inflammatory Value; OR, Odds Ratio; CBP, Cardiopulmonary Bypass; LDH, Lactate Dehydrogenase.

Table 4 Univariate and Multivariate Logistic Analysis of Overall Complications

Variables	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P	OR (95% CI)	P
Creatinine (μmol/L)	4.486 (1.287–15.630)	0.018	2.794 (0.649–12.020)	0.168
PIV (<1782 vs ≥ 1782)	0.017 (0.002–0.129)	<0.001	0.037 (0.005–0.210)	0.002
LDH (U/L)	1.001 (1.000–1.001)	0.174	1.319 (0.527–3.301)	0.554
D-dimer (mg/L)	1.035 (1.000–1.071)	0.048	1.694 (0.290–9.889)	0.558
Operation time (minutes)	1.689 (0.885–3.223)	0.112	1.074 (0.476–2.424)	0.864
Blood loss (mL)	3.232 (1.620–6.448)	<0.001	3.168 (1.331–7.541)	0.009
Plasma Transfusion (mL)	1.398 (0.738–2.651)	0.304	1.049 (0.465–2.367)	0.384
CBP time (minutes)	1.332 (0.573–3.100)	0.506	1.099 (0.360–3.351)	0.868

Abbreviations, PIV, Pan-immune Inflammatory Value; OR, Odds Ratio; CBP, Cardiopulmonary Bypass; LDH, Lactate Dehydrogenase.

95% CI, 1.287–15.630, p = 0.018), PIV (<1782 vs ≥1782, OR, 0.017, 95% CI, 0.002–0.129, p <0.001), D-dimer (OR, 1.035, 95% CI, 1.000–1.071, p = 0.048), Blood loss (OR:3.232, 95%, 1.620–6.448, p < 0.001).

In multivariate analysis, only PIV and Blood loss remained as independent predictors of overall complications. A lower PIV was significantly associated with reduced complication risk (OR = 0.037, 95% CI, 0.005–0.210, p = 0.002), whereas increased blood loss was associated with higher risk (OR = 3.168, 95% CI, 1.331–7.541, p = 0.009).

Consistent results were observed for major complications (Table S2). In the multivariate model, PIV (OR = 0.085, 95% CI, 0.026–0.173, p = 0.017) and blood loss (OR = 2.794, 95% CI, 1.128–6.918, p = 0.026) remained significant. These findings underscore the potential utility of preoperative PIV as a prognostic marker for postoperative complications in patients with acute TAAD.

Long-Term Outcomes in TAAD Patients

In TAAD patients, those with lower preoperative PIV levels had significantly better survival outcomes compared to those with higher levels (Figure 3, p = 0.005). Both univariate and multivariate Cox regression analyses confirmed that PIV was a significant predictor of survival outcomes (Table 5).

Univariate analysis identified several significant associations, including, Creatinine (HR = 3.481, 95% CI, 1.932–6.272, p < 0.001), PIV (<1782 vs ≥1782) (HR = 0.478, 95% CI, 0.280–0.815, p = 0.007), LDH (HR = 1.894, 95% CI, 1.110–3.232, p = 0.019), D-dimer (HR = 2.269, 95% CI, 1.168–4.407, p = 0.016), Operation time (HR = 2.954, 95% CI, 1.486–5.872, p = 0.002), Blood loss (HR = 3.946, 95% CI, 2.197–7.090, p < 0.001), Plasma Transfusion (HR = 2.703, 95% CI, 1.422–5.140, p = 0.002), CBP time (HR = 2.956, 95% CI, 1.660–5.263, p < 0.001).

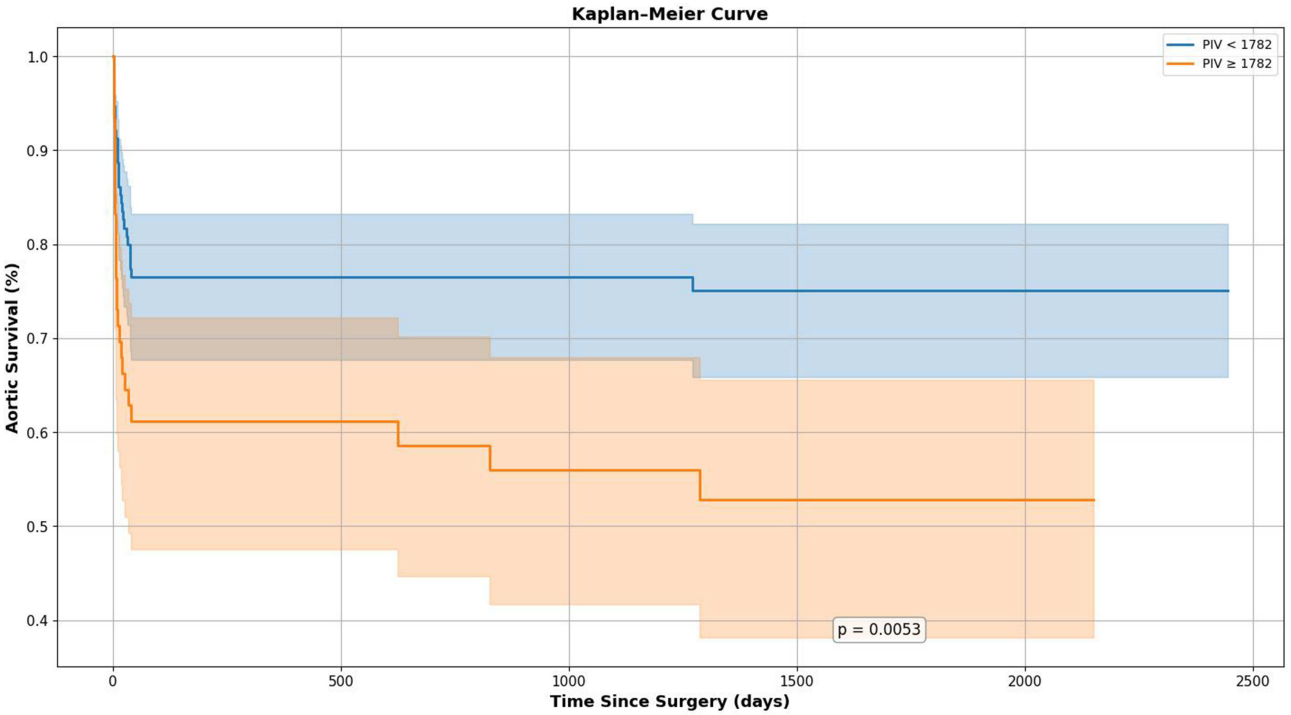

Figure 3 Kaplan-Meier survival curve of overall survival stratified by preoperative PIV in 175 TAAD patients (with Log rank test).
Abbreviations: OS, Overall Survival; PIV, Pan-Immune Inflammation Value.

Moreover, several factors were identified as independent predictors of long-term survival in the multivariate analysis, including Creatinine (HR = 2.367, 95% CI, 1.178–4.759, p = 0.016), PIV (<1782 vs ≥1782) (HR = 0.757, 95% CI, 0.378–0.859, p = 0.020), Blood loss (HR = 3.171, 95% CI, 1.678–5.994, p < 0.001), LDH (HR = 2.080, 95% CI, 1.174–3.688, p = 0.012), CBP time (HR = 2.928, 95% CI, 1.548–5.537, p <0.001).

Discussion

To the best of our knowledge, this study is one of the few to investigate the prognostic value of preoperative PIV levels in the management of TAAD patients, particularly focusing on postoperative complication risks, ICU stays, and long-term survival outcomes. Our findings highlight that elevated preoperative PIV levels are associated with prolonged operation time, increased intraoperative blood loss, higher blood product requirements, prolonged ICU stays, and greater risks of

Table 5 Univariate and Multivariate Cox Regression Analysis of Long-Term Survival

Variables	Univariate Analysis		Multivariate Analysis	
	HR (95% CI)	P	HR (95% CI)	P
Creatinine (μmol/L)	3.481 (1.932–6.272)	<0.001	2.367 (1.178–4.759)	0.016
PIV (<1782 vs ≥ 1782)	0.478 (0.280–0.815)	0.007	0.757 (0.378–0.859)	0.020
LDH (U/L)	1.894 (1.110–3.232)	0.019	2.080 (1.174–3.688)	0.012
D-dimer (mg/L)	2.269 (1.168–4.407)	0.016	1.194 (0.542–2.631)	0.660
Operation time (minutes)	2.954 (1.486–5.872)	0.002	1.857 (0.911–3.785)	0.089
Blood loss (mL)	3.946 (2.197–7.090)	<0.001	3.171 (1.678–5.994)	<0.001
Plasma Transfusion (mL)	2.703 (1.422–5.140)	0.002	1.599 (0.812–3.148)	0.175
CBP time (minutes)	2.956 (1.660–5.263)	<0.001	2.928 (1.548–5.537)	<0.001

Abbreviations: PIV, Pan-immune Inflammatory Value; CBP, Cardiopulmonary Bypass; LDH, Lactate Dehydrogenase; HR, Hazard Ratio.

both overall and major postoperative complications. Importantly, PIV was identified as an independent risk factor for both short-term outcomes, such as ICU stay and postoperative complications, and long-term survival outcomes.

Previous studies have demonstrated the prognostic value of PIV in a range of conditions, including metastatic colorectal cancer, coronary artery disease, and aortic calcification.[16,19–23] Elevated PIV levels have been consistently associated with poorer survival outcomes and shown to outperform traditional inflammatory markers in certain clinical settings.[19,24] However, its predictive role in TAAD—particularly regarding postoperative complications, ICU stays, and long-term survival—had not been previously investigated.

Given the association between elevated PIV levels and adverse outcomes, our findings reinforce existing evidence that intraoperative burden and postoperative complications are major determinants of TAAD prognosis. Prolonged operation time, high demand for blood products, and complications such as cerebral injury, organ hypoperfusion, and renal dysfunction have been recognized as significant predictors of postoperative mortality.[25,26] A large retrospective study reported complications in over 60% of patients, with nearly 20% experiencing three or more, significantly increasing mortality risk.[27] Additionally, ICU stays longer than seven days were linked to severe organ damage and limb disability.[28] In this context, PIV—reflecting systemic inflammation and immune status—may serve as an early, accessible marker of high-risk patients.

In the present study, we identified a preoperative PIV cutoff value of 1782, above which patients exhibited greater intraoperative complexity, higher complication risks, and prolonged ICU stays. Multivariate analyses further confirmed that elevated PIV was independently associated with both short-term outcomes—including postoperative complications and extended ICU stay—and poorer long-term survival. These findings suggest that elevated PIV reflects not only systemic inflammation and physiological stress, but also underlying factors linked to poor outcomes, such as vascular fragility, unrecognized comorbidities, and baseline frailty. It may also indicate heightened responses to pre-hospital stressors like hypoperfusion or tissue injury, or signal a prothrombotic state or endothelial dysfunction. These factors reinforce PIV' s value as a practical tool for early risk stratification and perioperative management in acute TAAD.

To further contextualize these findings, we explored the biological mechanisms by which PIV may reflect disease severity and predict adverse outcomes. This clinical relevance is further supported by mechanistic insights into the inflammatory processes underlying TAAD. The prognostic value of PIV may, in part, stem from its ability to capture systemic inflammatory and immune responses, both of which are central to TAAD progression. Inflammation contributes to smooth muscle cell dysfunction, extracellular matrix degradation, and a loss of aortic wall integrity.[9,29,30] Genomic studies have identified the upregulation of pro-inflammatory genes, such as TLR2, IL1R1, and MMP14, in TAAD tissue, amplifying cytokine signaling and driving tissue damage.[31]

A crucial aspect of inflammation in TAAD progression is the activation of neutrophils, which are rapidly recruited to the dissection site. Here, they interact with activated platelets to form neutrophil extracellular traps (NETs), which directly damage the vessel wall and promote thrombosis.[32] Platelets themselves play a key role in regulating inflammation by expressing TLRs, CD40L, and PD-L1, which activate neutrophils and monocytes, suppress T cell responses, and release chemokines like CCL5 to recruit additional leukocytes.[33] Furthermore, the formation of platelet-neutrophil complexes (PNCs) through P-selectin–PSGL-1 binding amplifies the inflammatory response and enhances leukocyte migration.[34] Studies in mouse models have demonstrated that selective depletion of monocytes/macrophages reduces MMP-9 expression, decreases leukocyte infiltration, and significantly lowers the incidence of dissection.[35]

These cellular interactions exacerbate inflammation and contribute to TAAD progression. By capturing this complex immune-inflammatory crosstalk—including NET formation, monocyte activation, and platelet-driven signaling—PIV provides a holistic proxy for disease severity and vascular injury. Consequently, PIV serves as a valuable indicator of the underlying mechanisms driving TAAD, enhancing its reliability as a prognostic marker for assessing disease outcomes.

While traditional markers such as CRP, PCT, systemic immune inflammation index (SII) and NLR have demonstrated prognostic relevance in various clinical settings, they offer limited insight into the patient' s integrated immune-inflammatory status.[36–39] These markers primarily focus on single aspect of inflammation or immune function, failing to capture the full spectrum of immune cell interactions and inflammatory cascades that drive pathophysiological changes, particularly in complex conditions like TAAD. In contrast, PIV combines parameters from four key circulating cell types—platelets, monocytes, neutrophils, and lymphocytes—offering a more comprehensive view of both inflammation and immune status,

while also capturing aspects of coagulation and the stress response. Compared to traditional markers, this integrative approach provides a broader representation of systemic inflammatory burden, which may explain PIV' s strong association with perioperative outcomes and long-term survival in TAAD, as demonstrated in our study.

Despite providing novel insights into the prognostic role of PIV in TAAD management, this study has several limitations. First, this was a single-center retrospective study, which may limit the external validity and generalizability of the results to broader populations, different healthcare settings, or diverse geographic regions. Second, the relatively small sample size may reduce the statistical power of the analysis and increase the risk of selection bias. Third, dynamic changes in inflammatory biomarkers, including PIV, were not tracked postoperatively due to the lack of longitudinal data, which may influence the accuracy of survival analysis. Future multi-center, prospective studies with larger cohorts are essential to validate our findings and assess the clinical utility of PIV-guided perioperative risk stratification in routine practice.

Conclusion

This study identified PIV as a novel and practical prognostic marker in patients with TAAD. As a composite index derived from routine blood tests, PIV reflects the overall inflammatory and immune status by integrating platelet, neutrophil, monocyte, and lymphocyte counts. Elevated preoperative PIV levels were independently associated with increased postoperative complications, prolonged ICU stays, and reduced long-term survival. These findings highlight the clinical utility of PIV for early risk stratification and individualized clinical management in TAAD patients.

Abbreviations

TAAD, Type A Aortic Dissection; PIV, Pan-Immune Inflammation Value; CTA, Computed Tomography Angiography; ICU, Intensive Care Unit; OR, Odds Ratio; ROC, Receiver Operating Characteristic curve; AUC, Area Under the Curve; CI, Confidence Interval; ANC, Absolute Neutrophil Count; WBC, White Blood Cell Count; RBC, Red Blood Cell Count; Hb, Hemoglobin; PLT, Platelet Count; ALC, Absolute Lymphocyte Count; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; cTnT, Cardiac Troponin T; D-dimer, D-Dimer; FDP, Fibrinogen Degradation Products; CK-MB, Creatine Kinase-MB; BNP, B-type Natriuretic Peptide; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; CRP, C-reactive Protein; PCT, Procalcitonin; IL-6, Interleukin-6; HR, Heart Rate.

Data Sharing Statement

The datasets used in this study are available on request from the corresponding author.

Ethics Approval and Consent to Participate

This study was approved by the ethics committee of Chongqing Medical University (2025-114-01) and followed the ethical standards of the Helsinki Declaration. Informed consent was obtained from all the participants.

Consent to Participate

All patients provided written informed consent.

Author contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have declared that no competing interest exists in this work.

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