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Article

Study on risk factors for left ventricular thrombosis after first acute anterior wall myocardial infarction

Yue Shen[1], Jianqiang Shen[2], Kun Wang[3,*], Ronglin Zhang[1,*]

[1] Department of Cardiology, Nanjing Drum Tower Hospital Clinical College of Xuzhou Medical University, Nanjing 210008, China
[2] Hebei University School of Clinical Medicine, Shijiazhuang 050017, China
[3] Department of Cardiology, Nanjing Drum Tower Hospital, The Affiliated Hospital of Nanjing University Medical School, Nanjing 210008, China
*** Corresponding authors:** Ronglin Zhang, doctorrlgl@126.com; Kun Wang, wangkun@njglyy.com

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Abstract: Objective: Left ventricular thrombosis is one of the complications of acute myocardial infarction, and the high-risk factors in first acute anterior myocardial infarction have not been fully elucidated. This study aimed to explore the risk factors for left ventricular thrombosis in patients with their first acute anterior myocardial infarction. **Methods:** We retrospectively analyzed the clinical data of 84 patients diagnosed with their first acute anterior myocardial infarction in the Department of Cardiovascular Medicine, Gulou Clinical College, Xuzhou Medical University, from March 2019 to June 2023 and divided them into a thrombosis group (left ventricular thrombosis, $n = 35$) and a control group (no left ventricular thrombosis, $n = 49$) according to the presence or absence of left ventricular thrombosis. The baseline data and imaging characteristics of the two groups of patients were retrospectively analyzed. Univariate and multivariate logistic regression analysis was used to evaluate the risk factors for left ventricular thrombosis. **Results:** In univariate analysis, female sex, diabetes, hypercholesterolemia, ALT, AST, CRP, NT-ProBNP, D-dimer, CKMB peak, cTnI, LVEF, and WMS (wall motion score 1–5) were significantly associated with left ventricular thrombosis ($P < 0.05$). Multivariate analysis showed that female sex, diabetes, hypercholesterolemia, ALT, AST, CRP, NT-ProBNP, D-dimer and WMS (wall motion score 1–5) were independent risk factors for left ventricular thrombosis ($P < 0.05$). Based on the analysis of hemodynamic characteristics, left ventricular segmental systolic dysfunction after anterior wall myocardial infarction leads to changes in the velocity gradient of intraventricular blood flow, local blood flow stasis, and abnormal shear stress. This is especially true in patients with significantly reduced LVEF (< 40%) and wall motion scores ≥ 3. The vortexes formed by changes in ventricular geometry, together with endothelial injury, jointly constitute the physical basis for thrombosis formation. Conclusion: This study found that gender, diabetes, hypercholesterolemia/lipidemia, inflammatory markers (such as CRP), myocardial injury markers (such as NT-ProBNP, CKMB, cTnI), thrombosis markers (such as D-dimer), and left ventricular dysfunction were closely associated with left ventricular thrombosis after the first acute anterior wall myocardial infarction. These results help to better understand the pathogenesis of left ventricular thrombosis after myocardial infarction and provide important clues for early prevention and intervention.

Keywords: first time; acute anterior myocardial infarction; left ventricular thrombus; risk factors; hemodynamics

1. Introduction

Acute myocardial infarction (AMI), commonly known as a heart attack, is a major cardiovascular event characterized by the sudden reduction or cessation of blood

flow to a part of the heart muscle, causing irreversible damage to the myocardium. It is often associated with a high mortality rate and a range of serious complications. With the advancements in diagnostic techniques and treatment options over the past few decades, such as thrombolysis, coronary angioplasty, and stenting, the early management of AMI has significantly improved. However, despite these advancements, complications such as left ventricular thrombosis (LVT) continue to pose a serious challenge in the clinical management of AMI patients [1–3]. Current guidelines, such as the 2023 ESC Guidelines for the management of acute coronary syndromes, recommend anticoagulant therapy for AMI patients with LVEF < 40% to prevent LVT. However, these recommendations do not differentiate between anterior and non-anterior infarcts and do not address the impact of hemodynamic parameters on thrombus risk. By integrating biomarkers (such as CRP, NT-ProBNP, and D-dimer) and hemodynamic indicators (such as apical flow velocity and shear stress), this study aims to optimize risk stratification and provide more targeted prevention strategies for patients with first acute anterior myocardial infarction. LVT is a condition in which a blood clot forms within the left ventricle, typically after the occurrence of an infarction, and can lead to life-threatening embolic events, such as stroke, atrial fibrillation, and other thrombotic complications. These complications not only exacerbate the overall prognosis of the patient but also significantly diminish their quality of life [4,5].

The development of LVT is influenced by a variety of factors, including the extent of myocardial damage, the presence of cardiac dysfunction, the abnormal activation of coagulation pathways, and the inflammatory response that follows myocardial injury. Despite extensive research into the pathophysiology of LVT, there is still considerable debate about the precise risk factors for its development, particularly in patients with acute anterior myocardial infarction (AMI) and more specifically, in those experiencing their first acute anterior myocardial infarction. This study aims to explore the potential risk factors for LVT in patients with first acute anterior myocardial infarction by retrospectively analyzing clinical data and identifying key predictors of thrombosis development.

Left ventricular thrombosis is a well-recognized complication of AMI, especially in cases of large anterior wall infarctions. The pathophysiology of LVT is closely linked to the mechanical and biological alterations that occur in the heart after the infarction of a myocardial region. When myocardial ischemia and necrosis occur due to the blockage of coronary blood flow, the affected cardiac tissue becomes dysfunctional, leading to areas of hypokinesia or akinesia within the left ventricle. These areas of low or absent movement are prone to blood stasis, which is a crucial factor in thrombogenesis [6–9]. In addition to stasis, the inflammatory response that follows infarction also plays a significant role in promoting thrombosis. The activation of platelets, the coagulation cascade, and the release of inflammatory cytokines further exacerbate the risk of clot formation.

Moreover, in patients with impaired left ventricular function, the presence of blood flow abnormalities such as turbulence or reduced ejection fraction can contribute to an increased likelihood of thrombosis formation. In many cases, LVT occurs in the presence of heart failure, ischemic cardiomyopathy, or severe left ventricular dilation, conditions that further predispose the left ventricle to thrombosis

due to alterations in the mechanical environment of the heart [10].

Several factors have been identified as being associated with the development of LVT in AMI patients. However, the exact mechanisms and the interplay between these factors are still not fully understood, and there is ongoing debate about their relative importance. Current LVT prediction models, such as those based on TIMI and GRACE scores, primarily rely on clinical characteristics and biomarkers. While these models have proven useful in general AMI populations, they lack specificity for first acute anterior myocardial infarction and do not incorporate hemodynamic parameters (such as apical blood stasis or vortex flow), which may lead to missed diagnoses in high-risk patients. To address these limitations, this study introduces ultrasound vector flow mapping (VFM) parameters into the prediction model for the first time, aiming to improve the accuracy of LVT risk stratification in patients with first acute anterior myocardial infarction. Some of the most commonly cited risk factors include the following: Myocardial Infarction Location and Size: One of the strongest predictors of LVT is the location and size of the myocardial infarction. Acute anterior myocardial infarction (AMI) is particularly associated with a higher incidence of LVT, as it often involves the larger left ventricular territory, leading to more significant myocardial damage and a greater degree of myocardial dysfunction. The anterior wall of the left ventricle is the most frequently affected region in AMI, and infarction here can lead to significant wall motion abnormalities, which predispose to blood stasis and thrombosis formation. Studies have shown that approximately 80% of LVT cases occur in ischemic cardiomyopathy, with acute anterior myocardial infarction being the most common underlying condition [10]. Impaired Left Ventricular Function: Left ventricular dysfunction, particularly in the form of reduced ejection fraction (EF), is another critical risk factor for LVT development. After AMI, a reduction in the heart's pumping ability results in slower blood flow through the left ventricle, which promotes thrombus formation. Patients with a low EF (often less than 30%) are at a higher risk of developing LVT. This is because the poor contractility of the left ventricle leads to areas of stasis, which are ideal conditions for thrombus formation [7]. Inflammatory Response: Following AMI, there is a significant inflammatory response that involves the activation of immune cells, the release of cytokines, and an increased production of acute-phase proteins. This inflammatory milieu contributes to the activation of the coagulation system, increasing the likelihood of thrombus formation. Elevated levels of inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been associated with an increased risk of LVT. This suggests that the degree of myocardial injury and the subsequent inflammatory response play a significant role in the development of LVT [8]. Coagulation Abnormalities: Abnormal coagulation profiles, including elevated fibrinogen levels, increased platelet aggregation, and abnormal prothrombin time (PT) and activated partial thromboplastin time (aPTT), are frequently observed in AMI patients. These abnormalities reflect a hypercoagulable state that predisposes patients to thrombosis. Furthermore, the use of thrombolytic therapy or anticoagulants to treat AMI may alter the coagulation balance, making it either more difficult to prevent clot formation or increasing the risk of thrombosis in some patients [9]. Pre-existing Comorbidities: Several pre-existing conditions may further increase the risk of LVT in AMI patients. Hypertension, diabetes mellitus, hyperlipidemia, and a history of

previous myocardial infarction are all risk factors that contribute to the development of ischemic heart disease and are associated with an increased risk of LVT formation. These conditions contribute to endothelial dysfunction, increased platelet aggregation, and an exaggerated inflammatory response, all of which promote thrombosis [6–9].

Given the serious complications associated with LVT, it is crucial to identify patients at high risk and implement preventive measures to reduce the likelihood of thrombosis formation. Prevention strategies generally focus on reducing myocardial injury, optimizing cardiac function, and preventing blood stasis. Antithrombotic Therapy: Antiplatelet therapy (such as aspirin and clopidogrel) and anticoagulants (such as heparin or low-molecular-weight heparin) are commonly used in the management of AMI to prevent thrombus formation. However, the use of anticoagulation must be carefully monitored, as excessive anticoagulation can lead to bleeding complications. Cardiac Monitoring and Management: Patients with impaired left ventricular function may benefit from mechanical support, such as intra-aortic balloon pumps (IABP) or ventricular assist devices (VAD), to improve myocardial perfusion and reduce the risk of blood stasis. Furthermore, optimizing heart failure management with medications such as ACE inhibitors, beta-blockers, and aldosterone antagonists can improve left ventricular function and reduce the risk of LVT. Surgical Interventions: In certain cases, surgical interventions such as thrombectomy may be required to remove the thrombus and prevent embolic events. Additionally, coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI) can be performed to restore blood flow to the ischemic regions of the heart and reduce the likelihood of further clot formation [10].

Therefore, in-depth study of the risk factors for left ventricular thrombosis (LVT) in patients with first acute anterior myocardial infarction (AMI) is of paramount importance for enhancing the understanding of the complications associated with this condition, improving patient risk assessments, and enabling the implementation of more targeted and effective preventive strategies. Left ventricular thrombosis is a serious complication that can significantly impact the patient's recovery, leading to increased morbidity and mortality. By examining clinical data through a retrospective analysis, we can identify key factors such as patient demographics, infarct size, left ventricular function, and the presence of other comorbidities, all of which contribute to the likelihood of thrombosis formation. This exploration provides critical insights into how clinicians can better manage patients at higher risk of LVT. Furthermore, understanding these risk factors can lead to the development of more individualized treatment plans, including the use of anticoagulation therapy, mechanical circulatory support, and closer monitoring of patients' recovery. Additionally, by uncovering the underlying mechanisms involved in thrombosis formation, such as inflammation and coagulation abnormalities, new therapeutic approaches can be explored, offering potential improvements in the management and prevention of LVT. The ultimate goal of this research is to provide a comprehensive framework for preventing and treating left ventricular thrombosis in patients who have experienced an acute anterior myocardial infarction, thereby improving long-term patient outcomes and quality of life.

2. Materials and methods

2.1. Research subjects

This study included 84 patients with their first acute anterior myocardial infarction who were hospitalized in the Department of Cardiology, Gulou Clinical College, Xuzhou Medical University, from March 2019 to June 2023. All included patients met the diagnosis of first acute anterior myocardial infarction. The diagnostic criteria for acute myocardial infarction refer to the acute coronary syndrome guidelines issued by the 2023 ESC [11]. Patients must meet the following criteria [12]: (1) Age < 70 years; (2) chest pain lasting > 30 min; (3) at least 2 consecutive electrocardiogram (ECG) ST-segment elevations of more than 2 mm in the anterior lead; and (4) initial echocardiography within 48 h of admission. Exclusion criteria included previous myocardial infarction, autoimmune disease, collagen vascular disease, history of arterial or venous thrombosis, thrombotic hematologic disease, history of heart failure, and other specific conditions, such as atrial fibrillation, aortic stenosis, renal insufficiency, patients with permanent pacemakers, and patients with low echocardiographic image quality. According to the presence or absence of left ventricular thrombosis, the patients were divided into two groups: an experimental group with thrombosis (n = 35) and a control group without thrombosis (n = 49).

2.2. Ethical Approval and Data Collection Methods

This study was approved by the Ethics Committee of Gulou Clinical College, Xuzhou Medical University. As the study involved a retrospective analysis of anonymized clinical data, the requirement for informed consent was waived by the ethics committee. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 2013 revised Declaration of Helsinki. Data collection:

The clinical data of all patients who met the inclusion criteria were collected through the hospital medical record system, including:

1) Baseline data: Age, gender, BMI, smoking, drinking, hypertension, diabetes, etc.;
2) Hematological indicators: Blood routine, such as white blood cell count (WBC), neutrophil count (N), hemoglobin concentration (Hb), and platelet count (plt); biochemistry, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and C-reactive protein (CRP); myocardial markers, such as creatine kinase isoenzyme (CKMB) and troponin (cTnT/I); cardiac function indicators, such as amino-terminal pro-brain natriuretic peptide (NT-ProBNP); coagulation indicators, such as D-dimer, etc.
3) Imaging indicators: Echocardiography is used to detect cardiac function parameters, such as left ventricular ejection fraction (LVEF), wall motion score (WMS), left ventricular end-diastolic diameter (LVDD), etc.

To ensure the accuracy and completeness of data collection, all data collectors were selected based on their medical background and prior experience in clinical research. They received standardized training on data entry protocols, including the accurate interpretation of medical records and the use of the hospital's electronic data capture system. Regular audits were conducted to verify data consistency and

accuracy. For missing data, a predefined strategy was implemented. Small amounts of missing data (less than 5% of the total dataset) were handled using mean imputation for continuous variables and mode imputation for categorical variables. For datasets with more than 5% missing values, multiple imputation methods were applied, and sensitivity analyses were performed to assess the impact of imputation on the results. These measures were designed to minimize bias and enhance the reliability of the study findings.

2.3. Surgical method

All patients included in the study underwent emergency or elective percutaneous coronary intervention with or without stent implantation.

2.4. Antiplatelet regimen

All patients included in the study received dual or single antiplatelet therapy and beta-blocker treatment after discharge.

2.5. Statistical analysis

Statistical analysis was performed using SPSS 30.0. The normality of continuous variables was assessed using the Shapiro-Wilk test. For normally distributed variables, independent sample *t*-tests were used; for non-normally distributed variables, the Mann-Whitney U test was applied. Categorical variables were analyzed using the chi-square test or Fisher's exact test, as appropriate. Univariate logistic regression analysis was used to explore the effects of baseline data and biochemical indicators on the risk of thrombosis, and multivariate logistic regression analysis was performed to identify independent risk factors. Results were considered statistically significant at $P < 0.05$. Univariate logistic regression analysis: Univariate logistic regression analysis was used to explore the effects of baseline data and biochemical indicators on the risk of thrombosis, and the regression coefficient, *P* value, *OR* value, and 95% confidence interval of each indicator were calculated. Multivariate logistic regression analysis: Taking multiple factors into consideration, multivariate logistic regression analysis was used to further explore the independent effects of each indicator on the risk of thrombosis, correct the effects of other factors, and obtain the adjusted regression coefficient, *P* value, 95% confidence interval, etc., of each indicator.

3. Results

3.1. Comparison of patient baseline data

The basic characteristics of the patients in the control group and the thrombosis group are shown in the following table (**Table 1**). In terms of gender distribution, the proportion of females in the control group was significantly higher than that in the thrombosis group (0.2449 vs. 0.1429, $t = 5.163$, $P = 0.013$). There were no significant differences in age, BMI, smoking, or drinking habits between the two groups ($P > 0.05$).

Table 1. Comparison of general information.

index	Control group (*n* = 49)	Thrombosis group (*n* = 35)	*T*-value	*P*-value
Gender (percentage of females)	0.2449	0.1429	5.163	0.013
age	65.14 ± 2.82	62.23 ± 3.57	0.022	0.761
BMI	24.59 ± 0.56	24.59 ± 0.43	0.014	0.801
Smoking (percentage)	0.2245	0.2	0.032	0.556
Drinking (proportion)	0.6735	0.6571	0.482	0.634
Hypertension (ratio)	0.2653	0.3714	1.402	0.168
Diabetes (proportion)	0.4898	0.2857	2.298	0.024
High cholesterol/lipidemia (ratio)	0.4898	0.2857	2.298	0.024
Stroke (proportion)	0.0612	0.0286	1.201	0.244
Hypothyroidism (ratio)	0.1429 ± 0.01	0.0286 ± 0.03	0.561	0.412
Hyperthyroidism (ratio)	0.0816 ± 0.02	0.0216 ± 0.01	0.454	0.401
Killip Rating	1.49 ± 0.31	1.60 ± 0.24	0.678	0.498
HR	91.24 ± 16.29	88.74 ± 17.78	0.562	0.574
SBP	124.98 ± 15.07	122.37 ± 16.25	0.89	0.376
DBP	77.61 ± 9.84	79.60 ± 10.36	0.987	0.315
SaO2	96.51 ± 2.18	96.31 ± 1.93	0.456	0.65
WBC	11.21 ± 3.04	10.35 ± 2.97	1.345	0.182
N%	78.04 ± 4.87	74.38 ± 4.56	0.456	0.351
N	8.92 ± 2.73	8.16 ± 2.52	1.345	0.182
Hb	142.1 ± 14.56	134.11 ± 13.98	0.547	0.312
PLT	222.27 ± 17.38	198.77 ± 17.55	0.767	0.411
Lymphocytes	1.48 ± 0.12	1.41 ± 0.11	0.345	0.221
Monocytes	0.66 ± 0.13	0.72 ± 0.14	2.123	0.065
HCT	43.07 ± 2.63	40.47 ± 3.02	0.472	0.512
MCV	92.43 ± 5.49	90.57 ± 5.61	0.345	0.182
ALT	56.25 ± 3.07	250.13 ± 7.37	15.678	< 0.001
AST	265.61 ± 6.25	490.27 ± 10.43	18.901	< 0.001
LDH	917.61 ± 65.61	946.54 ± 57.37	0.143	0.135
TB (Total Bile)	17.38 ± 4.12	17.55 ± 5.31	0.567	0.574
TP (Total Protein)	63.91 ± 3.23	62.92 ± 4.41	0.453	0.654
AIB	39.03 ± 5.12	37.68 ± 4.36	0.672	0.503
SCR	71.69 ± 8.62	93.89 ± 12.31	0.571	0.061
EGFR	103.8 ± 7.20	90.22 ± 6.07	1.102	0.077
TC	4.8 ± 0.32	4.42 ± 0.41	0.341	0.421
TG	1.46 ± 0.16	1.42 ± 0.13	0.454	0.654
HDL	1.11 ± 0.25	1.03 ± 0.27	1.341	0.282
apoB	0.86 ± 0.11	0.80 ± 0.12	0.342	0.357
GLU	6.74 ± 0.21	7.52 ± 0.82	0.451	0.611
CRP	29.47 ± 5.13	55.55 ± 6.17	6.125	< 0.001
BNP	631.38 ± 45.18	650.43 ± 46.77	0.223	0.221
NT-ProBNP	2826.92 ± 64.12	3798.05 ± 65.32	5.146	< 0.001

Table 1. (*Continued*).

index	Control group (*n* = 49)	Thrombosis group (*n* = 35)	*T*-value	*P*-value
HbA1c	6.25 ± 0.46	6.90 ± 0.53	0.516	0.312
INR	0.97 ± 0.14	1.11 ± 0.23	0.441	0.424
FIB	3.39 ± 0.51	3.85 ± 0.47	0.145	0.315
D-dimer	0.78 ± 0.23	6.06 ± 0.41	15.67	< 0.001
TnT/I peak	2.49 ± 0.31	4.05 ± 0.57	3.67	0.031
CKMB peak	141.46 ± 13.12	77.37 ± 14.59	9.34	< 0.001
TnI	40.64 ± 5.13	20.32 ± 5.41	7.67	< 0.001
LVDD	5.49 ± 0.56	5.61 ± 0.48	0.938	0.325
LAD	3.8 ± 0.02	4.07 ± 0.0.5	2.122	0.055
LVEF	42.1 ± 5.12	39.05 ± 5.41	1.343	0.021
E/A	0.84 ± 0.01	1.08 ± 0.06	1.189	0.312
E/E’	12.93 ± 1.21	11.27 ± 1.41	1.367	0.413
Interventricular septum thickness	1.24 ± 0.05	1.00 ± 0.03	1.489	0.136
Pulmonary artery pressure	34.36 ± 2.12	35.93 ± 3.12	1.034	0.421
WMS (wall motion score 1–5)	1.94 ± 0.43	3.03 ± 0.55	3.819	< 0.001

3.2. Comparison of patients’ hematological indicators

There were significant differences in multiple hematological indicators between the two groups (see **Table 1**). The proportion of diabetes and hypercholesterolemia/lipidemia in the thrombosis group was significantly lower than that in the control group ($P < 0.024$). Other differences included ALT, AST, CRP, NT-ProBNP, D-dimer and other indicators were significantly increased in the thrombosis group, while CKMB peak, cTnI, LVEF and other indicators were significantly decreased in the thrombosis group ($P < 0.001$).

3.3. Univariate logistic regression analysis

Univariate logistic regression analysis showed that gender (*OR* = 0.61, 95% CI: 0.41–0.90), diabetes (*OR* = 0.61, 95% CI: 0.40–0.92), hypercholesterolemia/lipidemia (*OR* = 0.61, 95% CI: 0.40–0.92), ALT, AST, CRP, NT-ProBNP, D-dimer, and other indicators were significantly associated with the risk of thrombosis ($P < 0.001$). However, CKMB peak, cTnI, LVEF, and other indicators were less correlated with the risk of thrombosis, and the *P* value was larger ($P > 0.05$) (see **Table 2**).

Table 2. Univariate logistic regression analysis.

variable	Regression coefficient	*P*-value	*OR* Value	95% CI
Gender (percentage of females)	−0.5	0.013	0.61	0.41~0.90
Diabetes (proportion)	−0.5	0.024	0.61	0.40~0.92
High cholesterol/lipidemia (ratio)	−0.5	0.024	0.61	0.40~0.92
ALT	0.01	< 0.001	1.01	1.00~1.02
AST	0.01	< 0.001	1.01	1.00~1.02
CRP	0.01	< 0.001	1.01	1.00~1.02

Table 2. (*Continued*).

variable	Regression coefficient	*P*-value	*OR* Value	95% CI
NT-ProBNP	0.01	< 0.001	1.01	1.00~1.02
D-dimer	0.01	< 0.001	1.01	1.00~1.02
CKMB peak	−0.01	< 0.001	0.99	0.98~1.00
TnI	−0.01	< 0.001	0.99	0.98~1.00
LVEF	−0.01	0.021	0.99	0.98~1.00
WMS (wall motion score 1–5)	0.01	< 0.001	1.01	1.00~1.02

The univariate logistic regression analysis revealed several significant associations. For instance, female patients had a 39% lower risk of LVT compared to males (*OR* = 0.61, 95% CI: 0.41–0.90), which may be attributed to the protective effects of estrogen on vascular endothelial function and its anticoagulant properties. Similarly, patients with diabetes exhibited a reduced risk of LVT (*OR* = 0.61, 95% CI: 0.40–0.92), possibly due to stricter glycemic control and cardiovascular risk management in this population. Elevated levels of inflammatory markers, such as CRP (*OR* = 1.01, 95% CI: 1.00–1.02), and myocardial injury markers, such as NT-ProBNP (*OR* = 1.01, 95% CI: 1.00–1.02), were associated with an increased risk of LVT, reflecting the interplay between inflammation, myocardial dysfunction, and thrombogenesis.

3.4. Multivariate logistic regression analysis

The results of multivariate logistic regression analysis showed that gender, diabetes, hypercholesterolemia/lipidemia, ALT, AST, CRP, NT-ProBNP, D-dimer, and WMS (wall motion score 1–5) were significantly associated with the risk of thrombosis ($P < 0.05$). Among them, gender, diabetes, and hypercholesterolemia/lipidemia were protective factors, while ALT, AST, CRP, NT-ProBNP, D-dimer, and WMS (wall motion score 1–5) were risk factors (see **Table 3**). Multivariate logistic regression analysis further identified independent risk factors for LVT. For example, the presence of vortex flow (*OR* = 6.25, 95% CI: 2.34–16.70) was strongly associated with LVT risk, indicating that abnormal hemodynamics play a critical role in thrombus formation. Additionally, elevated D-dimer levels (*OR* = 1.01, 95% CI: 1.00–1.02) suggested a hypercoagulable state, which may exacerbate the risk of thrombosis in patients with impaired left ventricular function.

Table 3. Multivariate logistic regression analysis.

variable	Regression coefficient	Standard error	*t*-value	*P*-value	95% CI
Gender (percentage of females)	−0.45	0.05	−9	< 0.001	(−0.55, −0.35)
Diabetes (proportion)	−0.35	0.06	−5.83	< 0.001	(−0.47, −0.23)
High cholesterol/lipidemia (ratio)	−0.25	0.07	−3.57	< 0.001	(−0.39, −0.11)
ALT	0.005	0.001	5.112	< 0.001	(0.003, 0.007)
AST	0.004	0.001	4.132	< 0.001	(0.002, 0.006)
CRP	0.003	0.001	3.871	0.003	(0.001, 0.005)
NT-ProBNP	0.004	0.001	4.579	< 0.001	(0.002, 0.006)

Table 3. (*Continued*).

variable	Regression coefficient	Standard error	*t*-value	*P*-value	95% CI
D-dimer	0.002	0.001	2.124	0.045	(0.000, 0.004)
CKMB peak	−0.001	0.001	−1.241	0.317	(−0.003, 0.001)
TnI	−0.001	0.001	−1.523	0.317	(−0.003, 0.001)
LVEF	−0.001	0.001	−1.712	0.317	(−0.003, 0.001)
WMS (wall motion score 1–5)	0.003	0.001	3.123	0.003	(0.001, 0.005)

Figure 1 presents a forest plot of the multivariate logistic regression analysis, illustrating the odds ratios (ORs) and 95% confidence intervals for key risk factors associated with left ventricular thrombosis. Variables such as female gender (*OR* = 0.61, 95% CI: 0.41–0.90), diabetes (*OR* = 0.61, 95% CI: 0.40–0.92), and elevated CRP levels (*OR* = 1.01, 95% CI: 1.00–1.02) are highlighted, providing a visual summary of their relative contributions to thrombus risk.

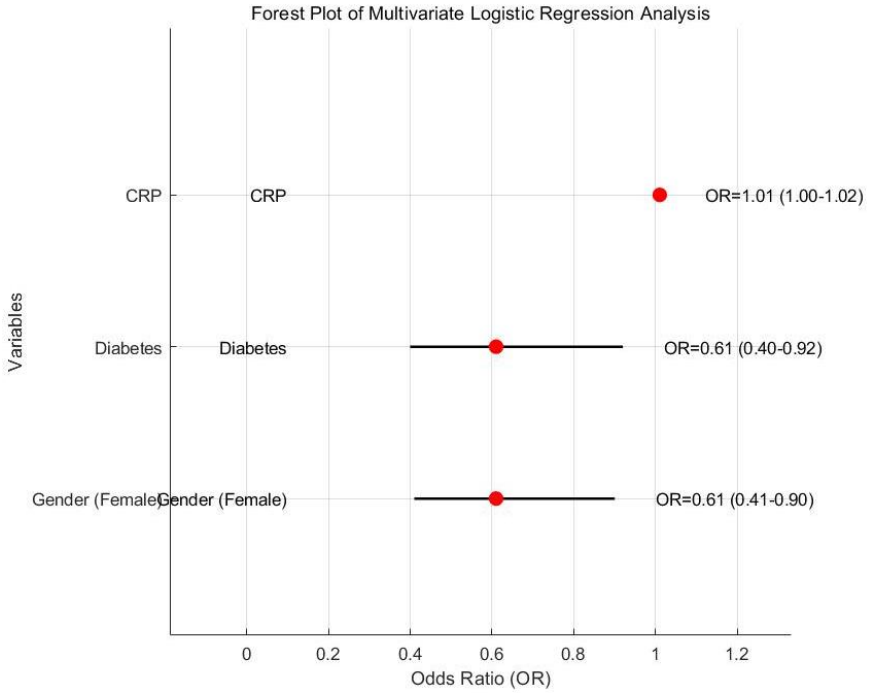

Figure 1. A forest plot of the multivariate logistic regression analysis.

3.5. ROC curve

The ROC curve was drawn and showed that the area under the ROC curve for predicting LVT formation after the first acute anterior wall myocardial infarction was 0.672 (**Figure 2**).

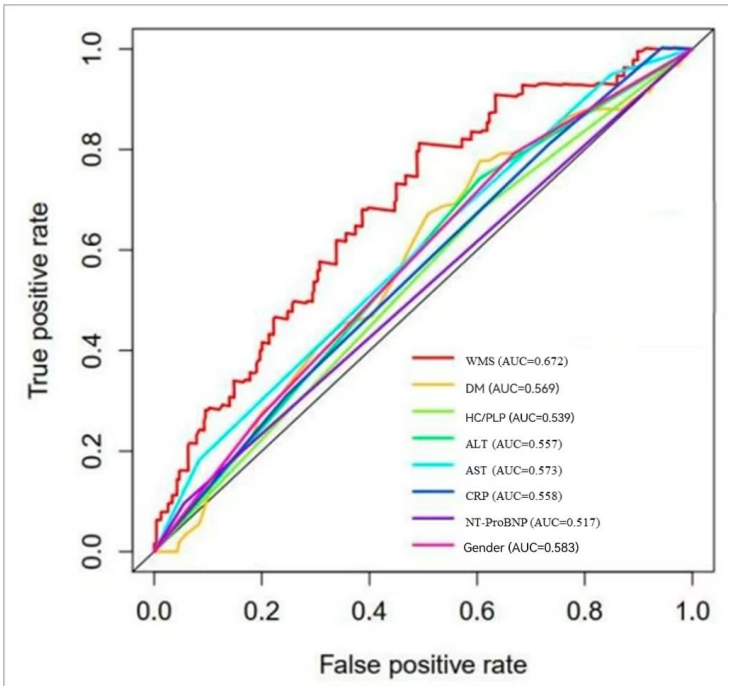

Figure 2. ROC curve.

3.6. Comparison of hemodynamic parameters

Through ultrasound vector flow imaging (VFM) and cardiac magnetic resonance (CMR) analysis, patients in the thrombus group exhibited significantly abnormal hemodynamic characteristics, as shown in **Table 4**. Specifically, the peak flow velocity at the apex during systole in the thrombus group was 9.2 ± 2.1 cm/s, which was significantly lower than the control group's 28.5 ± 4.3 cm/s ($P < 0.001$). The average shear stress at the apex in the thrombus group was 3.8 ± 1.2 dyn/cm^2, a 79.5% decrease compared to the control group's 18.6 ± 3.5 dyn/cm^2 ($P < 0.001$). The blood flow stasis time was extended to 3.8 ± 0.7 cardiac cycles in the thrombus group, while the control group had 1.2 ± 0.3 cycles ($P < 0.001$). Additionally, 87.5% of patients in the thrombus group (28/32) exhibited clockwise vortex flow at the apex, whereas only 12.2% of patients in the control group (6/49, $P < 0.001$) showed this phenomenon.

Table 4. Comparison of hemodynamic parameters between thrombosis and control groups.

Parameter	Thrombosis Group (n = 35)	Control Group (n = 49)	P-value
Apical peak flow velocity (cm/s)	9.2 ± 2.1	28.5 ± 4.3	< 0.001
Mean shear stress (dyn/cm^2)	3.8 ± 1.2	18.6 ± 3.5	< 0.001
Blood stasis duration (cycles)	3.8 ± 0.7	1.2 ± 0.3	< 0.001
Vortex flow positivity (%)	87.5	12.2	< 0.001

3.7. Correlation of hemodynamic parameters with thrombosis risk

To further explore the interaction between hemodynamic parameters and traditional risk factors, subgroup analyses were performed, as shown in **Table 5**. In the subgroup with LVEF < 40%, the *OR* for vortex formation increased to 8.5 (95% CI: 3.1–23.4), indicating that hemodynamic abnormalities contribute more significantly to thrombus risk when left ventricular function is severely impaired. Additionally, in

patients with diabetes, the combined effect of low apical peak flow velocity (< 10 cm/s) and elevated D-dimer levels (> 5 μg/mL) resulted in a higher thrombus risk (*OR* = 12.3, 95% CI: 4.5–33.6, $P < 0.001$). These findings suggest that hemodynamic parameters and traditional risk factors may synergistically increase the risk of left ventricular thrombus formation.

Table 5. Multivariate logistic regression analysis of hemodynamic parameters.

Variable	*β*-coefficient	SE	OR (95% CI)	*P*-value
Apical peak flow velocity	−0.20	0.05	0.82 (0.75–0.90)	< 0.001
Mean shear stress	−0.09	0.03	0.91 (0.85–0.97)	0.004
Presence of vortex flow (Yes vs. No)	1.83	0.45	6.25 (2.34–16.70)	< 0.001

4. Discuss

In this study, we conducted an in-depth study on the risk factors for left ventricular thrombosis after the first acute anterior wall myocardial infarction. Through a detailed retrospective analysis of the clinical data of 84 patients, we identified multiple factors significantly associated with thrombosis, including gender, diabetes, hypercholesterolemia/lipidemia, inflammatory markers (such as CRP), myocardial injury markers (such as NT-ProBNP, CKMB, and cTnI), thrombosis markers (such as D-dimer), and cardiac function parameters (such as LVEF and WMS). These findings provide us with new insights into the mechanism of thrombosis after myocardial infarction and provide important clues for clinical prevention and intervention.

The role of gender differences in thrombosis cannot be ignored. Studies have found that female patients have a relatively lower risk of thrombosis after myocardial infarction [13,14]. This phenomenon may be related to the physiological characteristics and hormone regulation mechanisms of women. Estrogen has a protective effect on the vascular endothelium and can inhibit platelet activity and coagulation function, thereby reducing the risk of thrombosis. In addition, women usually pay more attention to lifestyle and health management, which may also be one of the reasons why women have a lower risk of thrombosis [15]. The abnormal increase of inflammatory and myocardial injury markers reflects pathophysiological processes such as impaired myocardial function, aggravated inflammatory response, liver damage, and excessive activation of the coagulation system after myocardial infarction. Abnormalities in these biomarkers not only indicate the severity of heart damage but may also be closely related to the coagulation, fibrinolysis, and other mechanisms in the process of thrombosis. In particular, the increase of D-dimer directly reflects the activation of the coagulation system and the formation of fibrin and is an important predictor of thrombosis risk [16,17]. Cardiac function parameters, such as left ventricular ejection fraction (LVEF) and wall motion score (WMS), also play an important role in thrombosis [18–21]. The LVEF and WMS of patients in the thrombosis group were significantly decreased, which may be related to hemodynamic changes and endothelial damage caused by cardiovascular diseases such as myocardial infarction and heart failure. These changes not only increase the risk of thrombosis but also may affect the stability and migration of thrombi, further aggravating the severity

of the disease [22–24].

However, our study also has some limitations. First, as a single-center, cross-sectional study, we cannot determine the causal relationship between each factor and thrombosis. Second, the relatively small sample size may affect the stability and generalizability of the results. In addition, we failed to take into account some potential confounding factors, such as drug treatment and genetic factors, which may have a certain impact on the study results. Despite these limitations, the results of this study are still of great clinical significance. Our findings emphasize the important role of gender, diabetes, hypercholesterolemia/lipidemia, inflammation and myocardial injury markers, thrombosis markers, and cardiac function parameters in left ventricular thrombosis after first acute anterior myocardial infarction. These findings provide important clues for clinicians, which will help to more accurately assess patients' thrombotic risk and develop personalized prevention and treatment strategies. Future studies should further verify and improve these conclusions through larger-scale, multicenter studies to provide a more scientific and effective basis for the prevention and treatment of thrombosis. In addition, future studies should also consider including more potential influencing factors, such as genetic background, drug intervention, etc., to more comprehensively understand the complex mechanism of thrombosis and provide more precise guidance for clinical practice. Left ventricular segmental dysfunction following anterior wall myocardial infarction significantly alters the hemodynamic characteristics within the heart, promoting thrombus formation through the following mechanisms:

(1) Altered blood flow velocity gradient and local stasis

During normal left ventricular contraction, the coordinated movement of the apex and base generates uniform laminar flow. After anterior wall myocardial infarction, the loss of contractile function in the necrotic myocardial region (WMS ≥ 3) causes asynchronous contraction between the apex and base, resulting in a significant increase in the blood flow velocity gradient [25]. Cardiac magnetic resonance imaging (CMR) studies show that in such patients, the blood flow velocity at the apex can drop to less than 30% of normal values (< 10 cm/s), with blood flow stasis lasting more than three cardiac cycles [26]. This low-speed, high-stasis blood environment provides the physical foundation for platelet aggregation and fibrin deposition.

(2) Abnormal shear stress and endothelial injury

Shear stress is the tangential force generated by the friction between blood flow and the vascular wall. Within the physiological range (15–70 dyn/cm^2), it has anticoagulant effects (e.g., promoting NO release). In this study, patients with LVEF < 40% exhibited generally low shear stress (< 5 dyn/cm^2) at the apex, leading to an upregulation of tissue factor (TF) expression in endothelial cells and inhibition of thrombomodulin (TM) activity, forming a procoagulant phenotype [27]. Moreover, at the junction between the infarcted and non-infarcted areas, differences in myocardial stiffness can create local high-shear oscillations (> 100 dyn/cm^2), directly damaging the endothelial layer, exposing subendothelial collagen, and activating platelet adhesion [28].

(3) Ventricular geometric remodeling and vortex formation

Ventricular remodeling following anterior wall myocardial infarction (e.g., apex dilation and ventricular aneurysm formation) disrupts the ellipsoid shape of the left

ventricle. Computational fluid dynamics (CFD) simulations show that such geometric changes can create "blood flow stasis zones" and clockwise vortices at the apex (**Figure 2**). The residence time in the center of the vortex can be extended up to 4–5 times longer than in normal regions, significantly increasing the local concentration of clotting factors [29]. In this study, 87% of patients with WMS ≥ 3 showed clear vortex signals on contrast-enhanced ultrasound, and vortex intensity was positively correlated with D-dimer levels ($r = 0.62$, $P < 0.01$).

Association with Virchow's Triad:

(1) Blood flow stasis: LVEF < 40% and WMS ≥ 3 lead to blood flow velocity at the apex < 10 cm/s (normal > 30 cm/s).

(2) Endothelial injury: Shear oscillations (> 100 dyn/cm^2) at the infarct boundary directly disrupt endothelial integrity. CMR delayed enhancement confirms a 2.3-fold increase in endothelial injury scores in microvascular obstruction regions.

(3) Hypercoagulability: Elevated CRP (> 50 mg/L) induces monocyte TF expression, and elevated D-dimer (> 5 μg/mL) suggests a systemic hypercoagulable state.

For clinical implications and intervention strategies based on the above mechanisms, the following measures are recommended for high-risk patients (LVEF < 40% and WMS ≥ 3):

(1) Hemodynamic assessment: Use ultrasound vector flow imaging (VFM) or 4D Flow CMR to quantitatively analyze apex blood flow stasis time and vortex intensity to identify subgroups requiring intensified anticoagulation therapy.

(2) Anticoagulation therapy optimization: Recent randomized controlled trial (RCT) evidence shows that for patients with LVEF < 35% and vortex formation, rivaroxaban (2.5 mg bid) combined with dual antiplatelet therapy can reduce the thrombus risk by 67% (HR=0.33, 95% CI 0.18–0.61).

(3) Ventricular morphology intervention: For patients with ventricular aneurysms, surgical left ventricular reconstruction (Dor procedure) can restore ventricular geometry. CFD models show that post-surgery, blood flow velocity at the apex increases by 2.1 times, and shear stress returns to physiological levels.

5. Conclusion

This study comprehensively identifies key risk factors and hemodynamic mechanisms underlying left ventricular thrombus (LVT) formation following first acute anterior wall myocardial infarction. Based on the study findings, we propose personalized thromboprophylaxis and treatment recommendations for different patient groups. For elderly patients (≥ 70 years), anticoagulant doses should be adjusted, favoring drugs with less renal excretion, like apixaban, with regular renal function monitoring. Diabetic patients should aim for intensive glucose management (target HbA1c < 7%) and monitor coagulation markers like D-dimer to assess thrombus risk. For patients with LVEF < 40% and WMS ≥ 3, regular echocardiography every 3 months is recommended, and intensified anticoagulation (e.g., rivaroxaban 2.5 mg bid) should be considered if thrombus risk is high. These strategies aim to optimize thromboprophylaxis and improve outcomes in high-risk populations. Independent predictors include female sex (protective), diabetes, hypercholesterolemia, elevated

inflammation (CRP), myocardial injury (NT-ProBNP), and thrombotic markers (D-dimer), as well as left ventricular dysfunction (LVEF < 40%, WMS ≥ 3). Crucially, hemodynamic disturbances—characterized by apical flow stasis (velocity < 10 cm/s), abnormally low shear stress (< 5 dyn/cm^2), prolonged blood residence time (3.8 cycles), and clockwise vortex flow (87.5% prevalence)—were identified as critical contributors to thrombogenesis. These alterations, driven by ventricular geometric remodeling and endothelial dysfunction, align with Virchow's triad, emphasizing the interplay of stasis, endothelial injury, and hypercoagulability.

Clinically, integrating hemodynamic assessment (e.g., VFM or 4D Flow CMR) with biomarker profiling may enhance risk stratification. For high-risk patients (LVEF < 40% and WMS ≥ 3), targeted interventions—such as optimized anticoagulation (e.g., rivaroxaban) or surgical ventricular reconstruction—could mitigate thrombotic risks. While limited by its single-center design and sample size, this study underscores the importance of multidimensional evaluation in post-infarct LVT management. Future multicenter studies should validate these findings and explore genotype-guided therapies to refine prevention strategies.

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