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ORIGINAL ARTICLE

Antiphospholipid Antibodies as Potential Prognostic Indicators of Recurrent Myocardial Infarction

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SUMMARY

Background: **Inflammation and autoimmunity are pivotal contributors to myocardial infarction (MI) risk, with emerging evidence linking autoimmune disorders to MI pathogenesis. To further elucidate this relationship, we conducted a prospective cohort study investigating the prognostic impact of antiphospholipid antibodies (aPLs), antinuclear antibodies (ANA), and anti-extractable nuclear antigen antibodies (anti-ENA) on recurrent MI.**
Methods: **In this single-center study, 458 patients with acute MI (AMI) were enrolled and prospectively followed for 3 years. Autoantibody profiles, including aPLs (anti-cardiolipin antibodies [ACA], anti-β2-glycoprotein I [anti-β2GPI], lupus anticoagulant [LA]), ANA, and anti-ENA, were assessed and compared between recurrent AMI and non-recurrent AMI groups. The primary endpoints were all-cause mortality and recurrent AMI at 3-, 12-, and 36-months post-enrollment.**
Results: **Compared to non-recurrent AMI patients, those with recurrent AMI exhibited significantly higher positivity rates for ACA IgG (p = 0.034), ACA IgM (p = 0.039), anti-β2GPI IgG (p = 0.025), anti-β2GPI IgM (p = 0.035), and PLs + ANA/anti-ENA (p = 0.001). Multivariate analysis identified aPLs + ANA/anti-ENA (HR: 2.84, 95% CI: 1.45 - 6.12, p = 0.033), hypertension (HR: 2.83, 95% CI: 1.23 - 5.65, p = 0.027), and hyperlipidemia (HR: 2.67, 95% CI: 1.34 - 5.37, p = 0.039) as independent risk factors of AMI recurrence. The cumulative recurrence rates at 3, 12, and 36 months were 0.7%, 2.6%, and 6.8%, respectively, while the cumulative mortality rates were 6.5%, 12.0%, and 15.9%.**
Conclusions: **Concurrent positivity for aPLs and ANA/anti-ENA serves as an independent risk factor for recurrent AMI and is associated with increased mortality in AMI patients. These findings underscore the prognostic significance of autoimmune dysregulation in AMI outcomes.**
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KEYWORDS

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INTRODUCTION

According to the latest research from the Writing Committee of the Report on Cardiovascular Health and Diseases in China, about 330 million patients suffered from cardiovascular disease (CVD) by 2019 [1]. Despite advances in acute-phase management, survivors of acute myocardial infarction (AMI) remain at substantial risk

of recurrence, with an 8.2% recurrence rate within 90 days post-discharge [2]. Notably, patients experiencing recurrent AMI face a 1-year mortality rate of 2.5%, a risk 25.42-fold higher than non-recurrent cases [3]. Early identification of high-risk individuals is therefore critical for implementing targeted surveillance and therapeutic interventions to mitigate recurrence.

Emerging evidence implicates autoimmune dysregulation in the pathogenesis of MI. Autoimmune disorders such as antiphospholipid syndrome (APS) [4], systemic lupus erythematosus (SLE) [5], and rheumatoid arthritis (RA) [6] are established risk factors for myocardial infarction (MI). However, the significance of subclinical autoimmunity - defined by detectable autoantibodies in the absence of overt autoimmune disease - for the prognosis of MI remains poorly characterized. Antiphospholipid antibodies (aPLs), including anticardiolipin antibodies (ACA), anti-β2-glycoprotein I (anti-β2GPI), and lupus anticoagulant (LA), are heterogenous autoantibodies targeting phospholipid-protein complexes [7]. aPLs are associated with both arterial and venous thromboembolic disease [8-10], and they occur independently of traditional cardiovascular risk factors [11]. Notably, aPLs are detectable in ~11% of MI cases [12], with higher prevalence among young patients [13]. Similarly, antinuclear antibodies (ANA) and anti-extractable nuclear antigen antibodies (anti-ENA) - key biomarkers in autoimmune diagnostics - are linked to atherosclerosis [14,15], and predict adverse cardiovascular events, including MI [16]. Furthermore, anti-ENA positivity correlates with anti-oxidized LDL and aPLs in SLE [17], suggesting synergistic prothrombotic effects. Nevertheless, the collective impact of aPLs, ANA, and anti-ENA on AMI recurrence remains unresolved.

This study aimed to elucidate the association between aPLs/ANA/anti-ENA and recurrent AMI, evaluating their prognostic utility in predicting post-AMI outcomes.

MATERIALS AND METHODS

Study population

This single-center, prospective cohort study enrolled 458 patients with first-episode MI from the First Affiliated Hospital of Hebei North University between August 2019 and May 2021.

Diagnosis was established in accordance with the 2014 Chinese Guidelines for Rapid Diagnosis and Treatment of Acute Coronary Syndromes. Exclusion criteria comprised: 1) comorbid cardiovascular/cerebrovascular diseases or recent major surgery; 2) infectious disease at admission (diagnosed via clinical history, physical examination, or routine clinical laboratory tests); or 3) chronic inflammatory or autoimmune disease. The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Hebei North University, and informed consent was obtained from all participants. The flow chart of the study is shown in Figure 1.

Data collection

Clinical, demographic, and laboratory data were systematically collected and anonymized by trained physicians using standardized case report forms. Key variables included: gender, age, hypertension (systolic blood pressure ≥ 140 mmHg or diastolic pressure ≥ 90 mmHg in 2 separate measurements after the acute phase or use of antihypertensive drugs before recruitment), hyperlipidemia (cholesterol serum levels ≥ 5.7 mmol/L or use of cholesterol-lowering drugs), diabetes (history of diabetes mellitus, use of hypoglycemic agent or insulin, or fasting glucose ≥ 7.0 mmol/L), smoking habits (whether a patient was a current smoker or former smoker who had quit smoking for 6 months before the index event), drinking habits (whether weekly consumption > 14 drinks for men or > 7 drinks for women), and known family history of coronary heart disease (coronary heart disease recorded in first-degree relatives by interviewing probands or family members).

Laboratory indicators associated with MI collected at enrollment included: total cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, hypersensitive C-reactive protein (hs-CRP), fibrinogen, creatinine, hemoglobin, platelets, and leukocytes.

Autoantibody measurements

Autoantibody profiling was performed on peripheral blood samples obtained within 24 hours of acute event onset prior to therapeutic intervention. Analytical methods followed our previously published protocol [18]. IgG/IgM/IgA ACA, IgG/IgM/IgA anti-β2GPI, and anti-ENA levels in serum were quantified via chemiluminescent immunoassay (Bio CLIA 6500 analyzer; HOB Biotech, Jiangsu, China). The levels of autoantibodies higher than 20 RU/mL were considered positive, which were based on the recommendations by the manufacturer. Anti-ENA includes the following autoantibodies: anti-AMA-M2, anti-CENP-B, anti-dsDNA, anti-Jo-1, anti-nRNP/Sm, anti-PM-Scl, anti-Ro-52, anti-Scl-70, anti-Sm, anti-SS-A, and anti-SS-B. For LA determination, plasma samples were analyzed by dilute Russell's viper venom time (dRVVT) assay according to the guidelines [19] using the Sysmex CN6000 coagulation system (Siemens, Marburg, Germany). A ratio of LA1/LA2 higher than 1.2 was considered LA positive. Finally, ANA in serum was determined by indirect immunofluorescence (Euroimmun, Hangzhou, China). All assays were measured according to the manufacturers´ protocol.

Outcome assessment

The outcomes of our study were all-cause death and recurrent AMI at 3, 12, and 36 months of follow-up. Recurrent AMI was defined as a new AMI event meeting the diagnostic criteria per the 2014 Chinese Guidelines for Rapid Diagnosis and Treatment of Acute Coronary

Table 1. Clinical characteristics and laboratory indicators of AMI patients.

Characteristics	Recurrent AMI (n = 31)	No recurrent AMI (n = 427)	HR (95% CI)	p-value
Clinical characteristics				
Female, n (%)	12 (38.7)	159 (37.2)	1.12 (0.98 - 1.34)	0.861
Age, years (mean ± SD)	61.34 ± 8.44	59.76 ± 11.12	1.01 (0.92 - 1.14)	0.341
Hypertension, n (%)	14 (45.2)	91 (21.3)	3.30 (1.96 - 7.33)	**0.009**
Hyperlipidemia, n (%)	13 (41.9)	89 (20.8)	2.79 (1.55 - 6.13)	**0.021**
Diabetes, n (%)	9 (29.0)	101 (23.7)	1.67 (0.72 - 3.01)	0.329
Renal dysfunction, n (%)	5 (16.1)	66 (15.5)	1.23 (0.55 - 3.91)	0.812
Smoking habits, n (%)	18 (58.1)	159 (37.2)	2.21 (0.93 - 5.73)	**0.016**
Drinking habits, n (%)	17 (54.8)	245 (57.4)	0.89 (0.66 - 3.51)	0.412
Family history of known AMI, n (%)	11 (35.5)	155 (36.3)	0.89 (0.35 - 3.11)	0.421
Mean laboratory results (mean ± SD)				
Total cholesterol, mmol/L	4.67 ± 1.04	4.21 ± 1.01	1.31 (0.46 - 2.01)	0.552
HDL cholesterol, mmol/L	1.24 ± 0.41	1.29 ± 0.33	0.86 (0.51 - 1.78)	0.231
Triglycerides, mmol/L	1.31 ± 0.87	1.25 ± 0.64	1.22 (0.64 - 3.01)	0.533
hs-CRP, mg/L	8.11 ± 4.32	7.13 ± 3.16	1.56 (1.02 - 2.02)	0.552
Fibrinogen, g/L	3.02 ± 0.65	3.15 ± 0.91	0.82 (0.72 - 1.91)	0.671
Creatinine, µmol/L	71.23 ± 21.66	68.51 ± 20.34	1.02 (0.99 - 1.81)	0.762
Hemoglobin, g/L	138.41 ± 16.22	131.22 ± 19.21	1.12 (0.72 - 2.14)	0.766
Platelets, × 10^9 cells/L	231.55 ± 48.21	224.46 ± 51.89	1.01 (1.00 - 1.02)	0.435
Leukocytes, × 10^9 cells/L	6.10 ± 1.76	6.23 ± 1.54	0.97 (0.91 - 1.73)	0.332

Continuous variables are expressed as mean ± SD. Categorical variables are expressed as cases (percentage). HRs (95% CI) and p-values were obtained from a univariate Cox model. HR, hazard rate, CI - confidence interval, AMI - acute myocardial infarction, HDL - high density lipoprotein, hs-CRP - hypersensitive C-reactive protein.

Syndromes, including the extension of the original MI, the occurrence of new infarcts adjacent to the original infarct area or away from the original infarct area. Patients without recurrent events through a 36-month follow-up were defined as no recurrent AMI.

Statistical analysis

Hazard ratios (HRs) and 95% confidence intervals (CIs) were assessed by Cox proportional hazards models. A univariate Cox model was used to compare demographic variables and risk factors at baseline, and multivariate analysis was used to detect the independent risk factors for recurrence. Pearson's chi-squared test was applied for statistical comparison of categorical variables, and Fisher's exact test was used when one of the theoretical frequencies was less than 5. All test results were considered to be statistically significant if their two-sided p-values were < 0.05. All analyses were performed using SPSS Statistics 22.0.

RESULTS

Baseline characteristics of AMI patients

The study cohort comprised 458 patients with first-onset AMI who completed 3-, 12-, and 36-month follow-up assessments. During the observation period, 31 patients (6.7%) experienced recurrent AMI events, with temporal distribution as follows: 1) very early recurrence (0 - 3 months): 3 cases (9.7%); 2) early recurrence (4 - 12 months): 9 cases (29.0%); and 3) late recurrence (13 - 36 months): 19 cases (61.3%). There were no significant differences in gender (female: 38.7% vs. 37.2%) or age (61.34 ± 8.44 vs. 59.76 ± 11.12) between

Table 2. Autoantibodies of patients with recurrent AMI and without recurrent AMI.

Characteristics	Univariate analysis			Multivariate analysis	
	Recurrent AMI（n = 31)	No recurrent AMI (n = 427)	p-value	HR (95% CI)	p-value *
ACA IgG, n (%)	9 (29.0)	65 (15.2)	0.034	2.56 (1.33 - 5.25)	0.115
ACA IgM, n (%)	7 (22.6)	58 (13.6)	0.039	1.55 (0.81 - 2.94)	0.581
ACA IgA, n (%)	2 (6.5)	22 (5.2)	0.231	not selected	not selected
anti-β2GPI IgG, n (%)	8 (25.8)	58 (13.6)	0.025	1.32 (0.55 - 2.29)	0.883
anti-β2GPI IgM, n (%)	6 (19.3)	49 (11.5)	0.035	1.12 (0.38 - 2.01)	0.561
anti-β2GPI IgA, n (%)	3 (9.7)	25 (5.9)	0.101	not selected	not selected
LA, n (%)	3 (9.7)	35 (8.2)	0.722	not selected	not selected
ANA, n (%)	4 (12.9)	36 (8.4)	0.085	not selected	not selected
Anti-ENA, n (%)	6 (19.3)	56 (13.1)	0.112	not selected	not selected
aPLs + ANA/anti-ENA, n (%)	6 (19.3)	23 (5.4)	0.001	2.84 (1.45 - 6.12)	0.033
Hypertension, n (%)	14 (45.2)	91 (21.3)	0.001	2.83 (1.23 - 5.65)	0.027
Smoking habits, n (%)	18 (58.1)	159 (37.2)	0.011	1.43 (1.08 - 2.88)	0.231
Hyperlipidemia, n (%)	13 (41.9)	89 (20.8)	0.023	2.67 (1.34 - 5.37)	0.039

Categorical variables are expressed as cases (percentage). p-values were obtained by Pearson's chi-squared test. HRs (95% CI) and p-values * were obtained from a multivariate Cox model. HR - hazard rate, CI - confidence interval, aPLs + ANA/anti-ENA - one or more aPLs and ANA or ENA were positive.

the patients with recurrent AMI and no recurrent AMI. Out of all the indicators, only three factors were associated with recurrent AMI: hypertension (HR: 3.30, 95% CI: 1.96 - 7.33, p = 0.009), hyperlipidemia (HR: 2.79, 95% CI: 1.55 - 6.13, p = 0.021), and smoking habits (HR: 2.21, 95% CI: 0.93 - 5.73, p = 0.016). Table 1 presents comprehensive baseline characteristics and laboratory parameters for the study population.

The autoantibodies in AMI patients

While previous studies have established associations between aPLs/ANA and AMI pathogenesis, their prognostic value remains poorly characterized. We compared the positive rates of aPLs, ANA, and anti-ENA as well as "aPLs + ANA/anti-ENA" (when both aPLs and ANA or anti-ENA were positive) between the recurrent AMI and no recurrent AMI groups, and the positive rates of ACA IgG (p = 0.034), ACA IgM (p = 0.039), anti-β2GPI IgG (p = 0.025), anti-β2GPI IgM (p = 0.035), and aPLs + ANA/anti-ENA (p = 0.001) were all significantly higher in patients with recurrent AMI compared to in patients with no recurrent AMI. Then, we explored the influencing factors of recurrent AMI using multivariable Cox proportional regression analysis, where all factors with a p-value < 0.05 from the univariate analysis were included. After this, only aPLs + ANA/anti-ENA (HR: 2.84, 95% CI: 1.45 - 6.12, p = 0.033), hypertension (HR: 2.83, 95% CI: 1.23 - 5.65, p = 0.027), and hyperlipidemia (HR: 2.67, 95% CI: 1.34 - 5.37, p = 0.039) were found to be the independent risk factors for recurrence (Table 2).

Recurrence rate and mortality of AMI patients

Recurrence rate and mortality are important indicators to evaluate the prognosis of AMI patients, so we next compared the cumulative recurrence rate and mortality between patients with aPLs + ANA/anti-ENA (-) and aPLs + ANA/anti-ENA (+). The cumulative recurrence rates (Figure 2A) of patients with AMI at months 3/12/36 were 0.7%, 2.6%, and 6.8%, and the cumulative mortality (Figure 2B) at months 3/12/36 were 6.5%, 12.0%, and 15.9%, respectively. In addition, the cumulative recurrence rate (Figure 2A) and mortality (Figure 2B) at month 36 in AMI patients with aPLs + ANA/anti-ENA (+) were both higher than those in AMI patients with aPLs + ANA/anti-ENA (-), but there was no significant difference at months 3/12 between the two groups.

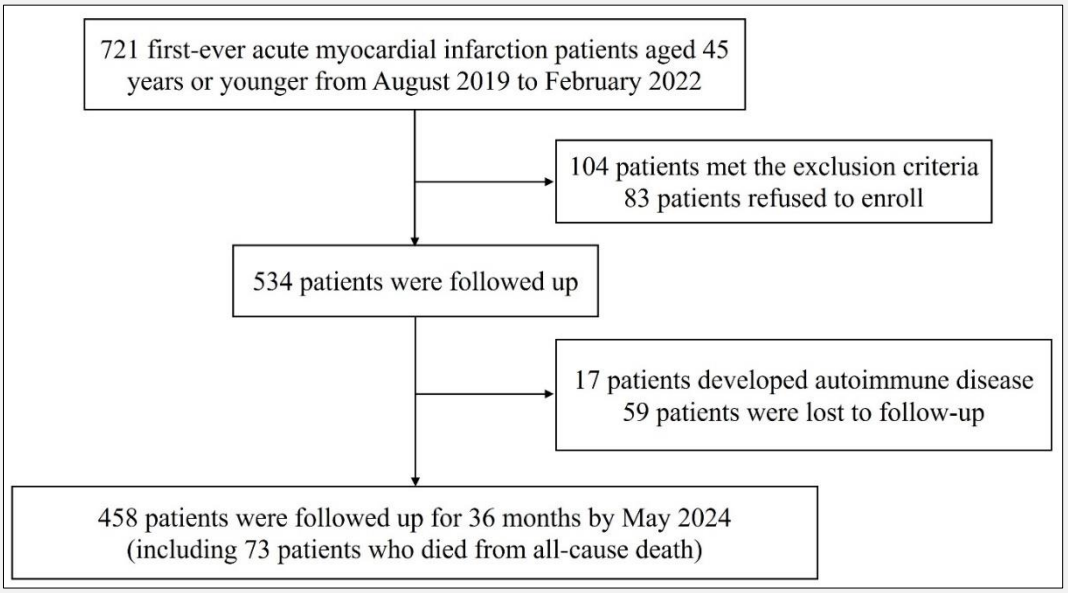
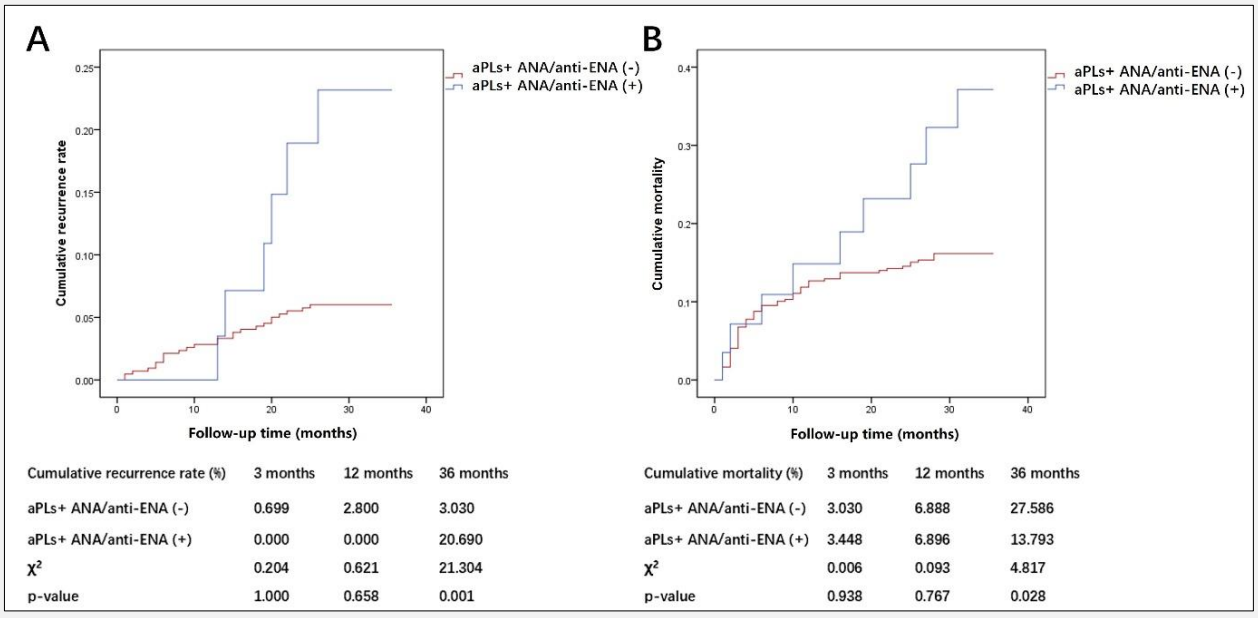


Figure 1. Flow chart of the study.



Cumulative recurrence rate (%)	3 months	12 months	36 months
aPLs+ ANA/anti-ENA (-)	0.699	2.800	3.030
aPLs+ ANA/anti-ENA (+)	0.000	0.000	20.690
χ^2	0.204	0.621	21.304
p-value	1.000	0.658	0.001

Cumulative mortality (%)	3 months	12 months	36 months
aPLs+ ANA/anti-ENA (-)	3.030	6.888	27.586
aPLs+ ANA/anti-ENA (+)	3.448	6.896	13.793
χ^2	0.006	0.093	4.817
p-value	0.938	0.767	0.028

Figure 2. Cumulative recurrence rate and cumulative mortality of AMI patients.

A) Cumulative recurrence rate and B) cumulative mortality of AMI patients, defined as aPLs + ANA/anti-ENA (-) and aPLs + ANA/anti-ENA (+).
χ^2 and p-values were obtained by Pearson's chi-squared test (Fisher's exact test was used when one of the theoretical frequencies was less than 5).
aPLs + ANA/anti-ENA (+): aPLs were positive and ANA or ENA was positive; aPLs + ANA/anti-ENA (-): patients who were not aPLs + ANA/anti-ENA (+).

DISCUSSION

Our study provides important new evidence regarding the prognostic value of autoantibodies in patients with acute myocardial infarction (AMI), particularly in those without clinically diagnosed autoimmune diseases. The key findings demonstrate that co-positivity for antiphospholipid antibodies (aPLs) combined with antinuclear antibodies (ANA) or anti-extractable nuclear antigen antibodies (anti-ENA) represents a novel independent risk factor for recurrent AMI (HR: 2.84, 95% CI: 1.45 - 6.12), while also confirming the established role of traditional cardiovascular risk factors.

Several important observations emerge from our data. First, we found significantly higher rates of multiple autoantibody positivity (including ACA IgG/IgM, anti-β2GPI IgG/IgM, and combined aPLs + ANA/anti-ENA) among patients experiencing recurrent AMI compared to those without recurrence. This finding extends previous reports linking autoimmune diseases with cardiovascular risk [20,21] by demonstrating that subclinical autoimmunity, as evidenced by autoantibody positivity, may similarly impact prognosis. Notably, while single autoantibody positivity showed differential expression between groups, only combined aPLs + ANA/anti-ENA positivity achieved independent prognostic significance. This observation aligns with emerging concepts of an "autoimmune continuum" in cardiovascular disease and is supported by prior work showing that multiple persistent autoantibodies, but not single autoantibodies, predict thrombotic events [22,23].

The temporal pattern of risk in our autoantibody-positive patients warrants particular attention. We observed that the increased risk associated with combined aPLs + ANA/anti-ENA positivity manifested primarily in long-term outcomes (13 - 36 months) rather than in short-term outcomes (0 - 12 months). This delayed emergence of risk suggests that conventional 1-year follow-up periods may be insufficient to capture the full risk profile of autoantibody-positive AMI patients and supports consideration of extended monitoring for this high-risk subgroup.

Several potential mechanisms could explain our findings. The combination of aPLs and ANA/anti-ENA may reflect more generalized immune dysregulation that promotes chronic vascular inflammation and thrombosis. This is consistent with previous reports showing particularly high thrombotic risk in SLE patients with specific aPLs profiles [24] and with the identification of β2GPI-dependent aCL IgG as a predictor of vascular events [25]. The synergistic effect of multiple autoantibodies may involve activation of complementary pro-thrombotic pathways or more profound endothelial dysfunction.

Several limitations of our study should be acknowledged. First, the single-center design and relatively small number of recurrence events may limit generalizability. Second, single-timepoint autoantibody measurements prevent assessment of persistence, which, as prior work suggests, may be clinically important [23]. Third, we lacked data on potential confounders including medication adherence and lifestyle factors. Finally, our study population was drawn primarily from underserved regions, which may account for the higher mortality rates observed compared to previous reports [3].

It is worth noting that the study subjects included in this research were not restricted by region or ethnicity, and the guidelines used for disease diagnosis also did not impose any regional or racial limitations. Therefore, we believe that the findings of this study are equally applicable to populations other than Chinese individuals.

These findings have important clinical implications. They suggest that comprehensive autoantibody screening in AMI patients could improve risk stratification and identify candidates for enhanced monitoring. Future research should focus on multicenter validation studies, longitudinal autoantibody profiling, and exploration of targeted therapeutic strategies for high-risk, autoantibody-positive patients.

In conclusion, our study identified combined aPLs and ANA/anti-ENA positivity as a novel prognostic marker in AMI and highlights the importance of considering autoimmune mechanisms in cardiovascular risk assessment. These results support the incorporation of autoantibody testing into routine post-AMI evaluation and suggest potential opportunities for personalized management approaches based on autoimmune profiling.

Data Availability Statement:
The original contributions presented in the study are included in the article. Further inquiries can be directed to the corresponding author.

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Declaration of Interest:
The authors declare that they have no potential conflict of interests.

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