Research Article

Risk Factors Associated with the Incidence of Ventricular Arrhythmias Complicating Acute Myocardial Infarction and Prognosis Analysis

Guibin Li, Shengxin Liu, Jiali Jin, Kejun Ding, and Caizhen Qian

Department of Cardiology, Zhuji People’s Hospital, China

Correspondence should be addressed to Caizhen Qian; wsygr1988@163.com

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Ventricular arrhythmias (VTA) usually occur following acute myocardial infarction (AMI). However, risk factors for VTA attack after AMI have been not well-recognized. The purpose of the study is to identify risk factors associated with the incidence of VTA complicating AMI. A total of 200 patients with AMI who were admitted to our hospital from February 2018 to February 2020 were retrospectively analyzed. These 200 patients were classified into a non-VTA group (n = 140) and a VTA group (n = 60) based on the occurrence of VTA within 24 after AMI. Patients in the VTA group were older than those in the non-VTA group. The VTA group had more numbers of WBCs and neutrophils than the non-VTA group. The level of serum potassium was lower, but the levels of cTnT and CK-MB were higher in the VTA group than in the non-VTA group. The VTA group presented an increase in proportions of anterior MI, TpTe, and proportions of Killip classification ≥ class II but a decline in LVEF when comparable to the non-VTA group. The two groups were not significantly different concerning other variables including sex, tobacco use, alcohol consumption, diabetes mellitus, hypertension, heart rate, Scr, SUA, BUN, PTL counts, TC, TG, HDL-C, LDL-C, D-dimer, BNP, LVS, LVP, and LVEDd. The levels of hsCRP, endothelin-1, and TNF-α were remarkably higher in the VTA group than in the non-VTA group (P < 0.001). Multivariate logistic regression analysis was performed, with clinical variables including age, WBCs, neutrophils, serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TNF-α, anterior MI, TpTe, proportions of Killip classification ≥ class II, and LVEF as an independent variable and with the occurrence of VTA as a dependent variable. It was revealed that serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TpTe, proportions of Killip classification ≥ class II, and LVEF were independent risk factors of VTA complicating AMI. Compared with the non-VTA group, the incidence rate of simple left heart failure, total heart failure, stroke, and dyslipidemia in the VTA group was significantly higher than those in the non-VTA group (P < 0.05). It was found that the proportion of all-cause deaths within one year outside the hospital was higher in the VTA group than in the non-VTA group (P < 0.05). Collectively, the study demonstrates serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TpTe, proportions of Killip classification ≥ class II, and LVEF were independent risk factors of VTA complicating AMI.

1. Background

Acute myocardial infarction (AMI) is myocardial necrosis caused by acute and persistent ischemia and hypoxia of coronary arteries [1]. AMI is accompanied by increased serum myocardial enzyme activity and progressive changes in an electrocardiogram, which can be complicated by arrhythmia, shock, or heart failure, and can often be life-threatening [2]. Sudden cardiac death results from sustained ventricular arrhythmia (VTA) and ventricular fibrillation complicating AMI in approximately 20-50% of cases [3]. VTA is a serious sequela of ventricular remodeling after MI. It is often replaced by weak fibrous tissue scars and necrotic myocardium in the infarct area. The diseased ventricular wall abducts and bulges, causing the myocardium in the necrotic area to lose its contractile function, and the local ventricular muscle compliance decreases and abnormal movement occurs, forming a left ventricular aneurysm. Owing to the high incidence of coronary artery disease, the number of sudden cardiac deaths each year in the general population is
estimated at 250/million, with rates remaining stable during the past decade [4]. Deaths of AMI patients are often sudden, which is closely related to VTA attack, especially the cardio-
genicity caused by malignant VTA with hemodynamic disor-
ders [5]. In recent years, the treatment and prediction of VTA have been continuously developed, and many new tech-
nologies have been continuously applied in this field.

The incidence and mortality of VTA are high in the early stage of AMI. Therefore, it has become the focus of clinical research to find an effective predictor of VTA and to carry out risk stratification. Henkel et al. reported that the inci-
dence of malignant VTA in patients with acute myocardial infarction was 1.9%-10.2%, and the risk of death was 6 times higher than that in patients without VTA after AMI [6]. San-
juan et al. reported that the incidence of VTA in the early stage of AMI was 20.0%, and its mortality was 3.3 times of that in the non-VTA patients [7]. The main mechanisms underlying VA attack during the acute stage of AMI are elec-
trolyte and autonomic imbalance concomitant with declined pH leading to increased tissue excitability, enhanced automa-
ticity, and finally in electrical instability [8]. According to the available data, predictors independently associated with ven-
tricular tachycardia and ventricular fibrillation are continu-
ously characterized, such as atrial fibrillation, cardiogenic shock, baseline heart rhythm more than 70 beats/min, chronic kidney disease, family history of sudden cardiac death, left main stenosis, low serum potassium concentra-
tion, and ST resolution less than 70% [9]. Of note, there are still significant challenges in risk stratification for VTA.

2. Materials and Methods

2.1. Patient Population. A total of 213 patients with AMI who were admitted to the Department of Cardiology of our hospi-
tal from February 2018 to February 2020 were initially selected into this retrospective study. Each patient had coro-
ary angiography, and subsequent PCI was performed with-
out any delay. The culprit vessels of MI involve the left main coronary artery, left anterior descending coronary artery, left circumflex coronary artery, and right coronary artery, classi-
fying as anterior wall involvement and nonanterior wall involvement. After surgery, antiplatelet therapy was per-
formed. Finally, the study encompassed 200 participants with informed content per patient and with the approval of the Ethics Committee of our hospital, as we excluded 13 patients considering the following exclusion criteria: history of car-
diopulmonary resuscitation, infectious diseases, or myocard-
ditis symptoms; severe skeletal muscle injury or trauma; previous history of AMI; AMI diagnosed in other hospitals and transferred into our hospital for further treatment; MI lasted more than 24 hours; history of PCI and coronary artery bypass grafting; history of rheumatic disease and nephropathy; and oral administration of arrhythmic drugs within 2 weeks. These 200 patients were classified into a non-VTA group and a VTA group based on the occurrence of VTA within 24 after AMI.

2.2. The Diagnosis of AMI. The diagnosis of AMI was made in accordance with a consensus document of The Joint European Society of Cardiology/American College of Cardi-
ology Committee for the redefinition of myocardial infarction [10, 11]: significant elevations of sensitive and specific biomarkers, such as cardiac troponin T (cTnT) and creatine kinase-myocardial band isoenzyme (CK-MB); ischemic symptoms, such as chest pain lasting at least 20 min; ST seg-
ment elevation in two or more limb or precordial leads.

2.3. Detection of VTA. All these patients were monitored by the standard 12-lead 24-hour electrocardiogram (EGG) at admission to the hospital, and the electrocardiographic T wave and Q wave (QT) intervals were measured by the authors without the knowledge of any outcome values. The diagnostic criteria considered for VTA were as follows: ven-
tricular tachycardia, defined as three or more consecutive ventricular complexes at a rate of greater than 120 beats/min; premature ventricular contractions (PVCs) including fre-
fquent (>5 isolated unfocal beats/min), bigeminy (alternate sinus and ventricular beats), multifocal (multifocal beats in the same hour of recording), couplets (two consecutive ven-
tricular beats, R-on-T according to R-R2/R-T < 0.85), and overall frequency (total number of PVCs in the recording divided by the number of analyzable hours and expressed as the number per hour).

2.4. Data Collection and Outcome Measures. Outcome analy-
sis was performed on the following indicators: sex, age, tobacco use, alcohol consumption, diabetes mellitus, hyper-
tension, heart rate, white blood cells (WBCs), neutrophils, glycosylated hemoglobin (HbA1C), blood glucose, serum potassium, serum creatinine (Scr), serum uric acid (SUA), blood urea nitrogen (BUN), platelet (PTL) count, total cho-
sterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), blood-D-dimer, B-type natriuretic peptide (BNP), cTnT, CK-MB, high-sensitivity C-reactive protein (hsCRP), endothelin-1, TNF-α, anterior or nonanterior MI, the inter-
val from the peak to the end of the T wave (TpTe), Killip clas-
sification of cardiac function at admission, ventricular septal thickness (LVS), left ventricular posterior wall thickness (LVP), left ventricular end-diastolic diameter (LVEDD), and left ventricular ejection fraction (LVEF).

2.5. Laboratory Analysis. Blood samples were drawn from each patient at admission into the hospital and collected into tubes supplemented with ethylenediaminetetraacetic acid. The levels of HbA1C, GLA, blood glucose, serum potassium, Scr, SUA, BUN, TC, TG, LDL-C, and HDL-C were measured by an automatic biochemistry analyzer (Hitachi 7150, Hitachi Ltd., Tokyo, Japan). Peripheral blood platelets were counted by an automated hematology analyzer (Hemato-
Flow, Beckman Coulter, USA). D-Dimer was evaluated by an immunoturbidimetric assay using the Advanced D-
Dimer assay (Dade-Behring, Deerfield, IL, USA) and CRP using BeckmannAssay360 (Beckman, Bera, CA, USA). Plasma B-type natriuretic peptide (BNP) levels were ascer-
tained using a high-sensitivity immunoradiometric assay (Shionogi, Osaka, Japan). The concentration of cTnT was examined by immunoassay (Elecsys 1020, Boehringer
Mannheim Diagnostics, Germany), and the activity of CK-MB was examined by the immune inhibition method (Synchron CX9, Beckman Coulter, USA). TNF-α was measured by the enzyme-linked immunosorbent assay (ELISA) kit (Biosource, Camarillo, CA) and endothelin-1 by the ELISA kit (Enzo Life Sciences, Switzerland).

2.6. Killip Classification of Cardiac Function. Killip classification was performed for clinical estimation of cardiac function [11]. Class I is defined no observation of heart failure with an elevation in pulmonary capillary wedge pressure. Class II is defined as mild and moderate heart failure with rales, S3 gallop and pulmonary venous hypertension, pulmonary congestion, and wet rales in the lower half of the lung fields. Class III is defined as severe heart failure with evident pulmonary edema with rales throughout the lung fields. Class IV is defined as cardiogenic shock with a sign of hypotension (systolic blood pressure < 90 mmHg) and evidence of peripheral vasoconstriction such as oliguria, cyanosis, and diaphoresis.

2.7. Statistical Methods. SPSS22.0 software was employed to perform data management and analysis. Continuous variables are expressed as mean ± standard deviation and compared by the t test. Categorical variables are expressed as proportions and analyzed using the chi-square test or Fisher’s exact probability method. Univariate and multivariate logistic regression analyses were used to determine the independent risk factors of AMI patients with VTA occurrence. P < 0.05 is considered as statistically significant.

3. Result
3.1. Baseline Characteristics. Totally, 200 patients with AMI were finally included into the study; we further split patients into two groups: VTA group (n = 60) and non-VTA group (n = 140). The detailed data regarding comparison of clinical and laboratory characteristics among patients with and without VTA are shown in Tables 1 and 2. As shown in Table 1, patients in the VTA group exhibited remarkable difference from those in the non-VTA group in terms of age, WBCs, neutrophils, serum potassium, cTnT, CK-MB, anterior MI, TpTe, proportions of Killip classification ≥ class II, and LVEF (P < 0.05). Patients in the VTA group were older than those in the non-VTA group. The VTA group had more numbers of WBCs and neutrophils than the non-VTA group. The level of serum potassium was lower, but the levels of cTnT and CK-MB were higher in the VTA group than in the non-VTA group. The VTA group presented an increase in proportions of anterior MI, TpTe, and proportions of Killip classification ≥ class II but a decline in LVEF when comparable to the non-VTA group. As shown in Table 2, the two groups were not significantly different concerning other variables including sex, tobacco use, alcohol consumption, diabetes mellitus, hypertension, heart rate, Scr, SUA, BUN, PTL counts, TC, TG, HDL-C, LDL-C, D-dimer, BNP, LVS, LVP, and LVEDd.

3.2. Levels of hsCRP, Endothelin-1, and TNF-α Were Associated with VTA Attack. It has been reported that hsCRP may serve as a predictor of short-term and long-term mortality after acute coronary syndromes. Endothelin-1 is an important vasoconstricting substance, and its rapid elevation in plasma is related to the onset of AMI. TNF-α has been well-studied for its deleterious cardiovascular effects. Thereupon, we were wondering the relationship between hsCRP, endothelin-1, TNF-α, and VTA attack. As we detected, the levels of hsCRP, endothelin-1, and TNF-α were remarkably higher in the VTA group than in the non-VTA group (P < 0.001, Figure 1).

3.3. Independent Risk Factors of VTA Complicating AMI. In order to find out independent risk factors of VTA complicating AMI, multivariate logistic regression analysis was performed, with clinical variables including age, WBCs, neutrophils, serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TNF-α, anterior MI, TpTe, proportions of Killip classification ≥ class II, and LVEF as an independent variable and with the occurrence of VTA as a dependent variable. It was revealed that serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TpTe, proportions of Killip classification ≥ class II, and LVEF were independent risk factors of VTA complicating AMI (Table 3).

### Table 1: Significant difference concerning clinical variables between AMI patients with or without VTA occurrence.

| Variable          | VTA group (n = 60) | Non-VTA group (n = 140) | χ²/t | P      |
|-------------------|--------------------|-------------------------|------|--------|
| Age (years)       | 68.95 ± 10.68      | 57.64 ± 10.36           | 5.67 | <0.001 |
| WBCs (×10⁹ cells/L) | 15.73 ± 2.68       | 11.84 ± 3.49            | 7.71 | <0.001 |
| Neutrophils (×10⁹ cells/L) | 13.53 ± 1.12   | 9.45 ± 2.89             | 10.59| <0.001 |
| Potassium (mmol/L) | 3.50 ± 0.33        | 4.10 ± 0.33             | 10.28| <0.001 |
| cTnT (ng/mL)      | 6.38 ± 3.88        | 4.79 ± 2.99             | 3.14 | 0.002  |
| CK-MB (U/L)       | 204.69 ± 90.23     | 178.54 ± 8.62           | 3.40 | <0.001 |
| Anterior MI (n (%)) | 40 (66.7%)         | 71 (50.7%)              | 2.08 | 0.037  |
| TpTe (ms)         | 140.52 ± 28.32     | 123.88 ± 32.87          | 3.41 | <0.001 |
| Killip class II (n (%)) | 13 (21.7%)   | 14 (10.0%)              | 2.21 | 0.027  |
| LVEF (%)          | 55.26 ± 13.25      | 58.96 ± 10.35           | 2.12 | 0.035  |

P < 0.05 means significant difference.
3.4. Association between Hospitalization Complications and VTA Occurrence. Among the selected patients, compared with the non-VTA group, the incidence rate of simple left heart failure, total heart failure, stroke, and dyslipidemia in the VTA group was significantly higher than those in the non-VTA group ($P<0.05$). In terms of other complications between the two groups, including atrial fibrillation, ventricular fibrillation, cardiac arrest, pulmonary hypertension, hypotension, cardiogenic shock, left ventricular mural thrombus, lung infection, renal impairment, anemia, hypoproteinemia, and syncope, no statistical difference was exhibited ($P>0.05$, Table 4).

| Variable               | VTA group ($n = 60$) | Non-VTA group ($n = 140$) | $\chi^2/t$ | $P$  |
|------------------------|----------------------|---------------------------|------------|------|
| Sex/male               | 45 (75.00%)          | 100 (71.45%)              | 0.518      | 0.604|
| Tobacco use (n %)      | 33 (55%)             | 85 (60.7%)                | 1.26       | 0.09 |
| Alcohol consumption (n %) | 15 (25%)           | 26 (18.5%)                | 1.87       | 0.16 |
| Hypertension (n %)     | 35 (58.3%)           | 78 (55.7%)                | 1.54       | 0.19 |
| Diabetes mellitus (n %) | 13 (21.7%)          | 40 (28.5%)                | 0.57       | 0.26 |
| Heart rate (time/min)  | 80.69 ± 13.64        | 80.34 ± 14.67             | 0.38       | 0.64 |
| HbA1C (%)              | 6.47 ± 1.54          | 6.28 ± 2.88               | 0.482      | 0.630|
| Blood glucose (mmol/L) | 8.77 ± 3.29          | 8.98 ± 4.99               | 0.299      | 0.765|
| Scr (μmol/L)           | 372.97 ± 28.72       | 381.63 ± 37.48            | 0.247      | 0.805|
| BUN (mmol/L)           | 6.87 ± 2.59          | 7.11 ± 2.57               | 0.604      | 0.547|
| PTL (×10⁹ cells/L)     | 268.41 ± 109.78      | 250.20 ± 144.22           | 0.875      | 0.383|
| TC (mmol/L)            | 4.69 ± 1.44          | 4.40 ± 1.24               | 1.443      | 0.151|
| TG (mmol/L)            | 1.75 ± 0.68          | 1.79 ± 0.67               | 0.385      | 0.701|
| HDL-C (mmol/L)         | 1.32 ± 0.43          | 1.37 ± 0.65               | 0.546      | 0.585|
| LDL-C (mmol/L)         | 2.79 ± 0.67          | 2.71 ± 0.54               | 0.891      | 0.374|
| D-dimer (μg/mL)        | 1.55 ± 0.40          | 1.39 ± 0.83               | 1.423      | 0.156|
| BNP (pg/mL)            | 183.78 ± 89.54       | 179.55 ± 65.29            | 0.373      | 0.710|
| LVS (mm)               | 9.50 ± 0.75          | 9.68 ± 0.53               | 1.931      | 0.055|
| LVP (mm)               | 9.53 ± 0.65          | 9.71 ± 0.82               | 1.509      | 0.133|
| LVEDd (mm)             | 52.33 ± 9.53         | 51.68 ± 6.29              | 0.569      | 0.570|

Figure 1: The levels of hsCRP, endothelin-1, and TNF-α between the AMI patients with or without VTA attack.
3.5. Association between the Short-Term and Long-Term of All-Cause Death and VTA Attack. Patients were followed up one year by clinic or using telephone conducted by trained nurses or doctors who were blinded to the information of patients until all-cause death occurred or through to the last day of the follow-up. All-cause death was defined as death mainly due to AMI, stroke, congestive heart failure, and malignant arrhythmia. There were 2 (3.3%) cases of all-cause deaths in the hospital in the VTA group and 1 case in the non-VTA group. There were 8 (13.3%) cases of all-cause deaths in the hospital in the VTA group and only 1 case in the non-VTA group. It was found that the proportion of all-cause deaths within one year outside the hospital was higher in the VAT group than in the non-VAT group, and the difference was statistically significant ($P < 0.05$). There was no significant difference in the proportion of all-cause deaths in the hospital between the two groups ($P > 0.05$, Table 5). These data suggested that VTA attack following AMI was associated with the long-term of mortality.

4. Discussion

In clinical work, VTA is one of the most common complications of acute MI, which significantly increases the mortality rate. Potential risk factors related to the occurrence of VTA have been confirmed recently, including low LVEF, heart function, NYHA grade ≥ 3, male, persistent electrical asynchrony, and increased transmural repolarization dispersion [12–14]. Among them, the most abundant evidence is recognized as the strongest predictor of LVEF, which is also the strongest predictor of sudden cardiac death [15]. Ventricular premature beats are the most common arrhythmia in clinical practice, often appearing in normal people or patients with structural heart disease. The pathogenesis of ventricular premature beats mainly involves changes in sympathetic nerve tension that cause abnormalities in cardiomyocyte autonomy, microreentry loops, and triggering activities. Another finding of this study is that frequent ventricular premature beats are one of the risk predictors of VTA patients. VTA patients themselves are complicated with severe myocardial ischemia and involve cardiac structure and electrocardiographic remodeling. The heart foundation is poor, and the ventricular transmural negative dispersion increases. Premature ventricular beats, especially R on T ventricular premature beats, are prone to occur. There is a difference in electrical conductance between the scar edge and the surrounding viable myocardium. Premature ventricular beats originating around the scar can easily trigger scar reentrant VTA. Frequent ventricular premature beats increase the chance of VTA to a certain extent. Traditional

### Table 4: Association between hospitalization complications and VTA occurrence.

| Complications                  | VTA group (n = 60) | Non-VTA group (n = 140) | $\chi^2$ | $P$  |
|--------------------------------|--------------------|-------------------------|---------|------|
| Atrial fibrillation            | 5 (8.3%)           | 11 (7.83%)              | 0.114   | 0.909|
| Ventricular fibrillation       | 4 (6.6%)           | 7 (11.7%)               | 0.473   | 0.636|
| Cardiac arrest                 | 2 (3.3%)           | 3 (2.14%)               | 0.494   | 0.621|
| Pulmonary hypertension         | 2 (3.3%)           | 5 (3.5%)                | 0.084   | 0.933|
| Simple left heart failure      | 20 (33%)           | 15 (10.7%)              | 3.858   | <0.001|
| Total heart failure            | 11 (18.3%)         | 2 (1.4%)                | 4.444   | <0.001|
| Hypotension                    | 2 (3.3%)           | 3 (2.1%)                | 0.494   | 0.621|
| Cardiogenic shock              | 3 (5%)             | 6 (4.2%)                | 0.223   | 0.823|
| Left ventricular mural thrombus| 3 (5%)             | 2 (1.4%)                | 1.482   | 0.138|
| Stroke                        | 20 (33%)           | 4 (2.8%)                | 6.078   | <0.001|
| Lung infection                 | 13 (21.6%)         | 16 (11.4%)              | 1.884   | 0.595|
| Renal impairment               | 8 (13.3%)          | 16 (11.4%)              | 1.210   | 0.090|
| Anemia                        | 4 (6.7%)           | 3 (2.1%)                | 1.595   | 0.110|
| Hypoproteinemia                | 3 (5%)             | 2 (1.4%)                | 1.482   | 0.138|
| Dyslipidemia                   | 15 (25%)           | 16 (11.4%)              | 2.430   | 0.015|
| Syncope                       | 1 (1.7%)           | 0 (0%)                  | 1.531   | 0.126|

### Table 5: Association between the short-term and long-term of all-cause death and VTA attack.

| Death                                      | VTA group (n = 60) | Non-VTA group (n = 140) | $\chi^2$ | $P$  |
|--------------------------------------------|--------------------|-------------------------|---------|------|
| All-cause deaths in the hospital           | 2 (3.3%)           | 1 (0%)                  | 1.396   | 0.163|
| All-cause deaths within 1 year outside     | 8 (13.3%)          | 1 (0.7%)                | 3.945   | <0.001|
experience believes that inferior MI is mostly caused by occlusion of the right coronary artery and left circumflex artery, and the branches of the arteries supplying the sinoatrial node and atrioventricular node mostly originate from the right coronary artery, so inferior MI is easily complicated by slowness arrhythmia, including sick sinus syndrome, I-III degree atrioventricular block [18]. Left anterior descending artery occlusion and extensive anterior wall MI are themselves two strong predictors of VTA and further combined with inferior MI, suggesting a large infarct size and severe vascular disease [19]. Myocardial electrical remodeling is more significant with neural remodeling, and its neurohumoral effect activation, increased sympathetic nerve tension, and cardiac electrical homeostasis imbalances, and other related factors that cause malignant VTA, have been further aggravated, which has promoted the formation of ECG reentry loops and ventricular in patients with VTA.

Our results revealed that serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TpTe, proportions of Killip classification ≥ class II, and LVEF were independent risk factors of VTA complicating AMI. Colombo et al. reported that short- and long-term mortality and the occurrence of VTA in patients with AMI were negatively linked with serum potassium concentration [20]. cTnT has been suggested as a new, more specific marker of myocardial cellular damage compared with CK-MB. The secretion of CK-MB seems to be affected by the duration of resuscitation and the presence of cardiogenic shock, which has to be considered when analyzing serum CK-MB levels after cardiopulmonary resuscitation. The elevation of cTnT appears to be only associated with AMI, but not with the duration of chest compressions, or with the number of defibrillations administered. Given that, it is necessary to detect cTnT and CK-MB post-AMI. In a study performed by Anderson et al., they found that hsCRP was increased in AMI patients (4.69 mg/L) compared with controls (2.69 mg/L) [21]. Although our results failed to found a relationship between BNP and the occurrence of VTA, Blangy et al. demonstrated an increased serum BNP and an increased hsCRP were associated with a higher incidence of ventricular tachycardia [22]. The plasmatic levels of endothelin-1 and related peptides produced during the synthesis of endothelin-1 from its precursor molecule preproendothelin-1 were considered as potential risk markers for cardiovascular events. The associations of endothelin-1 with aging, blood pressure, lung function, and chronic kidney disease have been reported, as their association between endothelin-1 levels and evidence of cardiac remodeling, including increased left atrial diameter and left ventricular mass [23]. Novo et al. also supported the role of endothelin-1 in cardiovascular diseases, as its plasmatic levels affected the cardiovascular and cerebrovascular risk profile [24].

In this study, we also compared the hospitalization complications between the two groups. Among the selected patients, compared with the non-VTA group, the incidence of simple left heart failure, total heart failure, stroke, and dyslipidemia in the VTA group was significantly higher than those in the non-VTA group. In terms of other complications between the two groups, including atrial fibrillation, ventricular fibrillation, cardiac arrest, pulmonary hypertension, hypotension, cardiogenic shock, left ventricular mural thrombus, lung infection, renal impairment, anemia, hypoproteinemia, and syncope, no statistical difference was exhibited. Patients were followed up for one year. We found the proportion of all-cause deaths within 1 year was higher in the VAT group than in the Non-VAT group, and the difference was statistically significant. There was no significant difference in the proportion of all-cause deaths in the hospital between the two groups.

Altogether, the study demonstrates serum potassium, cTnT, CK-MB, hsCRP, endothelin-1, TpTe, proportions of Killip classification ≥ class II, and LVEF were independent risk factors of VTA complicating AMI.

Data Availability
The data used to support the findings of this study are included within the article.

Conflicts of Interest
The authors declare that they have no conflicts of interest.

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