Hindawi
Journal of Nanomaterials
Volume 2021, Article ID 9985899, 7 pages
https://doi.org/10.1155/2021/9985899

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

Received 10 March 2021; Accepted 11 May 2021; Published 1 June 2021

Academic Editor: Songwen Tan

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 VAT group than in the non-VAT 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 cardiogenicity caused by malignant VTA with hemodynamic disorders [5]. In recent years, the treatment and prediction of VTA have been continuously developed, and many new technologies 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 incidence 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]. Sanjuan 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 electrolyte and autonomic imbalance concomitant with declined pH leading to increased tissue excitability, enhanced automaticity, and finally in electrical instability [8]. According to the available data, predictors independently associated with ventricular tachycardia and ventricular fibrillation are continuously 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 concentration, 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 hospital from February 2018 to February 2020 were initially selected into this retrospective study. Each patient had coronary angiography, and subsequent PCI was performed without 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, classifying as anterior wall involvement and nonanterior wall involvement. After surgery, antiplatelet therapy was performed. 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 cardiopulmonary resuscitation, infectious diseases, or myocarditis 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 antiarrhythmic 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 Cardiology 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 segment 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: ventricular tachycardia, defined as three or more consecutive ventricular complexes at a rate of greater than 120 beats/min; premature ventricular contractions (PVCs) including frequent (>5 isolated unifocal beats/min), bigeminy (alternate sinus and ventricular beats), multifocal (multifocal beats in the same hour of recording), couplets (two consecutive ventricular beats, R-on-T according to $\text{R-R}'/\text{R-T} < 0.85$), 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 analysis was performed on the following indicators: sex, age, tobacco use, alcohol consumption, diabetes mellitus, hypertension, 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 cholesterol (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 interval from the peak to the end of the T wave (TpTe), Killip classification 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 ascertained using a high-sensitivity immunoradiometric assay (Shionogi, Osaka, Japan). The concentration of cTnT was examined by immunoassay (Elecsys 1020, Boehringer

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

Variable	VTA group ($n = 60$)	Non-VTA group ($n = 140$)	χ^2/t	P
Age (years)	68.95 ± 10.68	57.64 ± 10.36	5.67	<0.001
WBCs ($\times 10^9$ cells/L)	15.73 ± 2.68	11.84 ± 3.49	7.71	<0.001
Neutrophils ($\times 10^9$ 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.

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 estimate 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 2: Comparison of baseline variables between AMI patients with or without VTA occurrence.

Variable	VTA group ($n = 60$)	Non-VTA group ($n = 140$)	χ^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)	81.15 ± 28.24	83.09 ± 37.43	0.360	0.719
SUA (mmol/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 ($\times 10^9$ 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

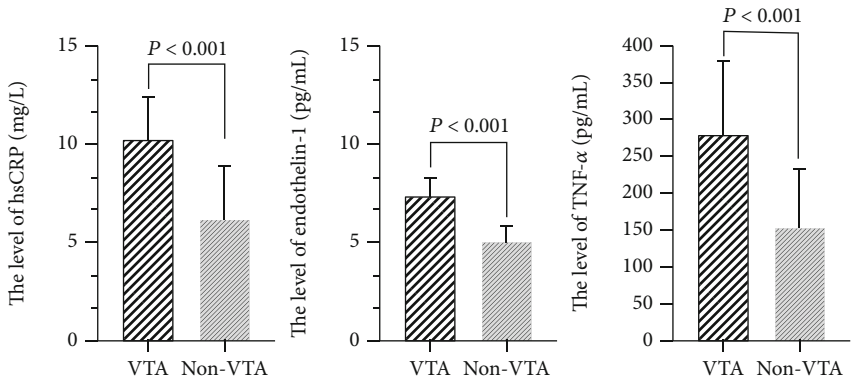

Figure 1: The levels of hsCRP, endothelin-1, and TNF-α between the AMI patients with or without VTA attack.

Table 3: Multivariate logistic regression analysis for independent risk factors of VTA complicating AMI.

Independent variable	OR (95% CI)	P
Serum potassium	3.012 (1.356-4.578)	<0.001
cTnT	2.434 (1.410-3.766)	0.004
CK-MB	1.968 (1.211-3.112)	0.009
hsCRP	1.593 (1.120-2.491)	0.019
Endothelin-1	2.742 (1.714-4.157)	0.027
TpTe	7.224 (2.613-17.97)	<0.001
Killip classification ≥ class II	1.874 (1.033-3.174)	0.034

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).

Table 4: Association between hospitalization complications and VTA occurrence.

Complications	VTA group ($n = 60$)	Non-VTA group ($n = 140$)	χ^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 ($n = 140$)	χ^2	P
All-cause deaths in the hospital	2 (3.3%)	1 (0%)	1.396	0.163
All-cause deaths within 1 year outside the hospital	8 (13.3%)	1 (0.7%)	3.945	<0.001

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 in VTA patients. A meta-analysis pointed out that frequent ventricular premature beats can increase the risk of adverse events in patients with nonstructural heart disease [16]. For structural heart disease, especially after MI, frequent ventricular premature beats are significantly associated with increased mortality [17]. 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

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.

References

[1] G. Arora and V. Bittner, "Chest pain characteristics and gender in the early diagnosis of acute myocardial infarction," *Current Cardiology Reports*, vol. 17, no. 2, p. 5, 2015.

[2] G. W. Reed, J. E. Rossi, and C. P. Cannon, "Acute myocardial infarction," *Lancet*, vol. 389, no. 10065, pp. 197–210, 2017.

[3] A. H. Bui and J. W. Waks, "Risk stratification of sudden cardiac death after acute myocardial infarction," *The Journal of Innovations in Cardiac Rhythm Management*, vol. 9, no. 2, pp. 3035–3049, 2018.

[4] T. M. Kolettis, "Coronary artery disease and ventricular tachyarrhythmia: pathophysiology and treatment," *Current Opinion in Pharmacology*, vol. 13, no. 2, pp. 210–217, 2013.

[5] V. P. Kuriachan, G. L. Sumner, and L. B. Mitchell, "Sudden cardiac death," *Current Problems in Cardiology*, vol. 40, no. 4, pp. 133–200, 2015.

[6] D. M. Henkel, B. J. Witt, B. J. Gersh et al., "Ventricular arrhythmias after acute myocardial infarction: a 20-year community study," *American Heart Journal*, vol. 151, no. 4, pp. 806–812, 2006.

[7] R. Sanjuan, M. L. Blasco, H. Martinez-Maicas et al., "Acute myocardial infarction: high risk ventricular tachyarrhythmias and admission glucose level in patients with and without diabetes mellitus," *Current Diabetes Reviews*, vol. 7, no. 2, pp. 126–134, 2011.

[8] J. P. Piccini, J. S. Berger, and D. L. Brown, "Early sustained ventricular arrhythmias complicating acute myocardial infarction*," *The American Journal of Medicine*, vol. 121, no. 9, pp. 797–804, 2008.

[9] T. S. Podolecki, R. K. Lenarczyk, J. P. Kowalczyk et al., "Risk stratification for complex ventricular arrhythmia complicating ST-segment elevation myocardial infarction," *Coronary Artery Disease*, vol. 29, no. 8, pp. 681–686, 2018.

[10] J. S. Alpert, K. Thygesen, E. Antman, and J. P. Bassand, "Myocardial infarction redefined–a consensus document of The Joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction," *Journal of the American College of Cardiology*, vol. 36, no. 3, pp. 959–969, 2000.

[11] K. Thygesen, J. S. Alpert, A. S. Jaffe et al., "Fourth universal definition of myocardial infarction (2018)," *Global Heart*, vol. 13, no. 4, pp. 305–338, 2018.

[12] E. E. Brodie, D. Allan, D. N. Brooks, J. McCulloch, and W. S. Foulds, "Flash and pattern reversal visual evoked responses in normal and demented elderly," *Cortex*, vol. 28, no. 2, pp. 289–293, 1992.

[13] C. M. Tompkins, V. Kutyifa, A. Arshad et al., "Sex differences in device therapies for ventricular arrhythmias or death in the multicenter automatic defibrillator implantation trial with cardiac resynchronization therapy (MADIT-CRT) trial," *Journal of Cardiovascular Electrophysiology*, vol. 26, no. 8, pp. 862–871, 2015.

[14] M. Hayashi, W. Shimizu, and C. M. Albert, "The spectrum of epidemiology underlying sudden cardiac death," *Circulation Research*, vol. 116, no. 12, pp. 1887–1906, 2015.

[15] P. A. Pellikka, A. Arruda-Olson, F. A. Chaudhry et al., "Guidelines for performance, interpretation, and application of stress echocardiography in ischemic heart disease: from the American Society of Echocardiography," *Journal of the American Society of Echocardiography*, vol. 33, no. 1, pp. 1–41.e8, 2020.

[16] V. Lee, H. Hemingway, R. Harb, T. Crake, and P. Lambiase, "The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review," *Heart*, vol. 98, no. 17, pp. 1290–1298, 2012.

[17] J. T. Bigger Jr., J. L. Fleiss, R. Kleiger, J. P. Miller, and L. M. Rolnitzky, "The relationships among ventricular arrhythmias, left ventricular dysfunction, and mortality in the 2 years after myocardial infarction," *Circulation*, vol. 69, no. 2, pp. 250–258, 1984.

[18] S. Rasoul, M. J. de Boer, H. Suryapranata et al., "Circumflex artery-related acute myocardial infarction: limited ECG abnormalities but poor outcome," *Netherlands Heart Journal*, vol. 15, no. 9, pp. 286–290, 2007.

[19] H. Tikz, Y. Balbay, R. Atak, T. Terzi, Y. Gençl, and E. K. Utüuk, "The effect of thrombolytic therapy on left ventricular aneurysm formation in acute myocardial infarction: relationship to successful reperfusion and vessel patency," *Clinical Cardiology*, vol. 24, no. 10, pp. 656–662, 2001.

[20] M. G. Colombo, I. Kirchberger, U. Amann, L. Dinser, and C. Meisinger, "Association of serum potassium concentration with mortality and ventricular arrhythmias in patients with acute myocardial infarction: a systematic review and meta-analysis," *European Journal of Preventive Cardiology*, vol. 25, no. 6, pp. 576–595, 2018.

[21] D. R. Anderson, J. T. Poterucha, T. R. Mikuls et al., "IL-6 and its receptors in coronary artery disease and acute myocardial infarction," *Cytokine*, vol. 62, no. 3, pp. 395–400, 2013.

[22] H. Blangy, N. Sadoul, B. Dousset et al., "Serum BNP, hs-C-reactive protein, procollagen to assess the risk of ventricular tachycardia in ICD recipients after myocardial infarction," *Europace*, vol. 9, no. 9, pp. 724–729, 2007.

[23] M. Jankowich and G. Choudhary, "Endothelin-1 levels and cardiovascular events," *Trends in Cardiovascular Medicine*, vol. 30, no. 1, pp. 1–8, 2020.

[24] G. Novo, A. Sansone, M. Rizzo, F. P. Guarneri, C. Pernice, and S. Novo, "High plasma levels of endothelin-1 enhance the predictive value of preclinical atherosclerosis for future cerebrovascular and cardiovascular events: a 20-year prospective study," *Journal of Cardiovascular Medicine (Hagerstown, Md.)*, vol. 15, no. 9, pp. 696–701, 2014.