Risk assessment model for predicting ventricular tachycardia or ventricular fibrillation in ST-segment elevation myocardial infarction patients who received primary percutaneous coronary intervention

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Abstract
Ventricular tachycardia/ventricular fibrillation (VT/VF) is a kind of malignant arrhythmia in ST-segment elevation myocardial infarction (STEMI) patients who received primary percutaneous coronary intervention (PPCI). However, there are no risk assessment tools to anticipate the occurrence of VT/VF.

This study is to build a risk assessment model to predict the possibility of VT/VF onset in STEMI patients undergoing PPCI. A retrospective study was conducted to analyze the patients who underwent PPCI from January 2006 to May 2015. Subjects were divided into VT/VF group and no VT/VF group based on whether VT/VF had occurred or not. In addition, the VT/VF group was further separated into early-onset group (from the time that symptoms began to before the end of PPCI) and late-onset group (after the end of PPCI) based on the timing of when VT/VF happened. Multivariate regression analysis was carried out to distinguish the independent risk factors of VT/VF and an additional statistical method was executed to build the risk assessment model.

A total of 607 patients were enrolled in this study. Of these patients, 67 cases (11%) experienced VT/VF. In addition, 91% (61) of patients experienced VT/VF within 48 h from the time that the symptoms emerged. Independent risk factors include: age, diabetes mellitus, heart rate, ST-segment maximum elevation, ST-segment total elevation, serum potassium, left ventricular ejection fraction (LVEF), culprit artery was right coronary artery, left main (LM) stenosis, Killip class > I class, and pre-procedure thrombolysis in myocardial infarction (TIMI) flow zero grade. Risk score model and risk rank model have been established to evaluate the possibility of VT/VF. Class I: ≤ 4 points; Class II: > 4 points, ≤ 5.5 points; Class III: > 5.5 points, < 6.5 points; and Class IV ≥ 6.5 points. The higher the class, the higher the risk.

The incidence of VT/VF in STEMI patients undergoing PPCI is 11% and it occurs more frequently from the time that symptoms begin to before the end of PPCI, which, in most cases, occurs within 48 h of the event. Our risk assessment model could predict the possible occurrence of VT/VF.

Abbreviations: ACEI = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker, AVIR = accelerated idioventricular rhythms, CAG = coronary angiography, CHD = coronary heart disease, ECG = electrocardiogram, IABP = intra-aortic balloon pump, IRA = infarct-related artery, LAD = left anterior descending, LCX = left circumflex, LM = left main, LMWH = low molecular weight heparin, LVEF = left ventricular ejection fraction, MI = myocardial infarction, PCI = percutaneous coronary intervention, RAAS = renin-angiotensin-aldosterone-system, RCA = right coronary artery, SCD = sudden cardiac death, STEMI = ST segment elevation myocardial infarction, TIMI = thrombolysis in myocardial infarction, VF = ventricular fibrillation, VPBs = ventricular premature beats, VT = ventricular tachycardia.

Keywords: acute myocardial infarction, malignant ventricular arrhythmia, primary percutaneous coronary intervention.
1. Introduction

Malignant arrhythmia, such as sustained ventricular tachycardia (VT) and ventricular fibrillation (VF), is the most common cause of sudden cardiac death (SCD). Coronary heart disease (CHD) is the most frequent etiology of SCD, with approximately 70% of SCD cases due to CHD, and CHD is the most common etiology of ventricular arrhythmia.[1,2] As for the acute stage of ST-segment elevation myocardial infarction (STEMI), VT/VF has brought challenges to reperfusion therapy. There have been numerous studies over the years to detect the risk factors of VT/VF. In the thrombolytic therapy era, ventricular premature beats (VPBs) and accelerated idioventricular rhythms (AVIR) frequently suggested myocardial reperfusion, which is called reperfusion arrhythmia. However, some researchers used AVIR as a marker of myocardial necrosis.[3] Post-thrombolytic VT/VF, no matter when it occurred, has been deemed to be associated with adverse outcomes.[4–7] With the advent of percutaneous coronary intervention (PCI) as the main reperfusion therapy of myocardial infarction (MI), the culprit artery reperfusion rate improved largely. The incidence and prognosis of VT/VF has significantly changed. Rajendra H et al[8] conducted the 1st large retrospective study to seek risk factors of VT/VF during primary percutaneous coronary intervention (PPCI); however, the study only enrolled low-risk patients without renal dysfunction and cardiogenic shock, and it only focused on arrhythmia during PPCI. Some studies[9–15] that focused on pre-PPCI, intra-PPCI, and pro-PPCI have accomplished this as well, with diversified objects and findings. The APEX-AMI study[16] accomplished a related overall examination of VT/VF in STEMI patients; however, inferior myocardial infarction and pre-thrombolytic patients were excluded. In addition, a variety of risk assessment tools are well known, such as thrombolysis in myocardial infarction (TIMI)-score, GRACE-score, CRUSADE-score, STS-score, are required to identify high-risk patients, nevertheless none of the researchers have built a risk assessment model to distinguish the patients at high risk for VT/VF. We wanted to build a scoring system to evaluate the potential occurrence of VT/VF to offer more precise care to STEMI patients.

2. Methods

This study was approved by the Ethical Review Committee of the Second Xiangya Hospital of Central South University (Changsha, Hunan, China). A retrospective study was conducted to analyze patients who underwent PPCI in the Second Xiangya Hospital of Central South University (Changsha, Hunan, China) from January 1, 2006 to May 31, 2015 and who experienced the onset of symptoms (including sustained ischemic chest pain, heart failure, malignant arrhythmia, and unstable hemodynamics) within 12h and the onset of symptoms between 12 and 24h. All of the patients met the diagnosis criteria mentioned in the 2013 AHA/ACC guidelines for STEMI management.[17] The exclusion criteria were: patients who underwent coronary angiography (CAG) and those treated via intra-aorta balloon pump (IABP), temporary pacemaker implantation without the infarct-related artery (IRA) intervention process, rescue PCI after fibrinolytic therapy, and pacemaker implantation before PCI, as well as patients with critical data missing. The patients enrolled in study were divided into 2 groups (the VT/VF group and the no VT/VF group) based on whether VT/VF had occurred or not. In addition, the VT/VF group was further separated into the early-onset group (from the time that symptoms began to before the end of PPCI) and late-onset group (after the end of PPCI) based on when VT/VF happened according to Mehta’s study[16] (Fig. 1).
After data were gathered, SPSS 22.0 was utilized to conduct the statistical analysis. For continuous quantitative data, the K-s normal test is used to check whether the normal distribution is obeyed; the distribution is described by the mean value and standard deviation for the normal distribution data. The difference between groups was determined by the Independent Sample T Test, and the distribution is described by the median and 4-digit spacing for the discrepancy from the normal distribution data. We used the Mann–Whitney U test to compare differences between groups for the classification data. The card-side test was adopted to compare the differences between groups and multifactor logistic regression analysis was used for multifactor analysis. According to the differences and the condition regression tree analysis results, the continuity variable was divided into the classification variable to build the risk assessment model. The risk grading variable was used as test sets for the cross-verification of the model prediction value. All of the tests used in this paper are bilateral tests, with statistical significance set to \( P < .05 \).

3. Results

Out of a total of 938 cases, we enrolled 607 patients in the study. Excluded cases included: 142 patients without IRA treatment, 66 patients with rescue PCI, 26 patients with critical data missing, and 97 for pre-process pacemaker implantation. Of the final 607 included patients, 467 (77%) were males and 140 (23%) were females. There were 67 (about 11%) patients who suffered from VT/VF. Of these VT/VF patients, 55 (82%) were males and 12 (18%) were females. Moreover, 91% (61) of VT/VF cases occurred within 48h and 9% (6) occurred after 48h. According to the methods mentioned above, 50 patients were placed in the early-onset group and 17 patients were placed in the late-onset group. Even in the late-onset group, most of the patients (about 75%) had VT/VF within 48h.

3.1. Demographic and baseline clinical characteristics

Compared with the no VT/VF group, the VT/VF group showed a trend with patients who were elderly and smokers, had pre-myocardial infarction, diabetes, a quick heart rate, low systolic pressure, Killip Class > I, low creatinine clearance, low left ventricular ejection fraction (LVEF), etc (Table 1). The infarct area was also associated with VT/VF onset, and inferior MI has a greater VT/VF occurrence rate (36.7% vs 55.22%, \( P < .001 \)) (Table 1). Moreover, the VT/VF group had a higher CK-MB level (285.8u/L vs 320.9u/L, \( P = .041 \)) and magnitude of ST-segment elevation (Table 1). Comparing the early-onset group and the late-onset group, atrial fibrillation pre-process (6% vs 29.41%,

| Table 1 |
|---|
| **Demographic and baseline clinical characteristics.** |
| **Occurrence situation** | **Occurrence time** |
| **Factors** | **No VT/VF group** | **VT/VF group** | **P** | **Early onset group** | **Late onset group** | **P** |
| **Sex** | | | | | | |
| Male | 412 (76.3%) | 55 (76.9%) | .355 | 39 (78%) | 16 (94.12%) | .270 |
| Female | 128 (23.7%) | 22 (30.04%) | | 11 (22%) | 1 (5.88%) | |
| **Age** | | | | | | |
| 61.99±11.65 | 66.9±9.97 | .001 | 67±9.49 | 66.59±11.56 | .844 |
| **Weight** | | | | | | |
| 65.78±9.93 | 66.51±9.13 | .563 | 65.92±9.04 | 68.26±9.42 | .364 |
| **CABG** | | | | | | |
| 20 (3.7%) | 5 (7.46%) | .144 | 3 (6%) | 2 (11.76%) | .726 |
| **Hypertension** | | | | | | |
| 306 (56.67%) | 42 (62.69%) | .018 | 32 (64%) | 10 (58.82%) | .703 |
| **Stroke** | | | | | | |
| 36 (6.67%) | 1 (1.49%) | .108 | 1 (2%) | 0 (0%) | 1.000 |
| **Diabetes** | | | | | | |
| 116 (21.48%) | 24 (35.82%) | .009 | 17 (34%) | 7 (41.18%) | .594 |
| **Smoking** | | | | | | |
| 281 (52.04%) | 45 (67.16%) | .019 | 33 (66%) | 12 (70.59%) | .728 |
| **Heart rate** | | | | | | |
| 83.29±18.9 | 98.42±20.67 | <.001 | 97.06±19.73 | 102.41±23.41 | .360 |
| **Systolic pressure** | | | | | | |
| 131.09±29.95 | 120.45±27.38 | .006 | 121.16±27.53 | 118.39±27.65 | .718 |
| **Killip class** | | | | | | |
| I | 339 (62.78%) | 32 (47.76%) | <.001 | 21 (42%) | 11 (64.71%) | .366 |
| II | 73 (13.52%) | 25 (37.31%) | 21 (42%) | 4 (23.53%) | |
| III | 83 (15.37%) | 3 (4.48%) | 3 (6%) | 0 (0%) | |
| IV | 45 (8.33%) | 7 (10.45%) | 5 (10%) | 2 (11.76%) | |
| **Top ST elevation>0.3mv** | | | | | | |
| 288 (53.33%) | 50 (74.63%) | .001 | 36 (72%) | 14 (82.35%) | .527 |
| **Sum ST elevation>1.5mv** | | | | | | |
| 108 (20%) | 24 (35.82%) | .003 | 18 (36%) | 6 (33.33%) | .958 |
| **AVF** | | | | | | |
| 36 (6.67%) | 8 (11.94%) | .131 | 3 (6%) | 5 (29.41%) | .021 |
| **Serum K+** | | | | | | |
| 4.44±0.99 | 3.97±0.88 | <.001 | 3.8 (3.1-4.6) | 4.2 (3.8-4.7) | .180 |
| **Creatinine clearance** | | | | | | |
| 73.79±13.92 | 66.18±12.53 | <.001 | 68.6 (55.3-75) | 59.7 (52.5-75.9) | .531 |
| **Myocardial location** | | | | | | |
| Anterior | 250 (46.3%) | 28 (41.79%) | .485 | 22 (44%) | 6 (33.33%) | .530 |
| Extensive anterior | 110 (20.37%) | 10 (14.93%) | .291 | 6 (12%) | 4 (23.53%) | .432 |
| High lateral, anterior-lateral, posterior | 54 (10%) | 9 (13.45%) | .385 | 6 (12%) | 3 (17.65%) | .682 |
| Inferior | 171 (31.67%) | 37 (55.22%) | <.001 | 32 (64%) | 5 (29.41%) | .013 |
| Right ventricular | 45 (8.33%) | 8 (11.94%) | .324 | 7 (14%) | 5 (29.41%) | .448 |
| AV/AB | 77 (14.26%) | 18 (26.87%) | .007 | 14 (28%) | 4 (23.53%) | .000 |
| CK-MB | 285.8 (127.5,438.95) | 320.9 (212.4,450.5) | .041 | 307.3 (197.458.5) | 363.9 (203.3-330.5) | .306 |
| LVEF\% | 46.18±3.71 | 40.35±3.49 | <.001 | 41.74 (37.96-44.62) | 37.71 (36.8-39.06) | .002 |

CABG = coronary artery bypass grafting, MI = myocardial infarction, AVF = three grade atrioventricular block, LVEF = left ventricular ejection fraction, VT/VF = ventricular tachycardia/ventricular fibrillation.
Further demographic and baseline clinical characteristic comparisons between the early-onset-group and non-early-onset group (no VT/VF group and late-onset group) suggested that there was a trend in the early-onset group for patients to be elderly and have pre-MI, a fast heart rate, and low LVEF. Comparisons between the early-onset-group and non-early-onset group (VT/VF group and late-onset group) suggested that there was a trend in the early-onset group and the late-onset group comparisons between the early-onset-group and non-early-onset group within 7 days after reperfusion of occluded artery.

### 3.3. Pharmacotherapy characteristics in hospital

A comparison of each group’s pharmacotherapy features suggested that the VT/VF group used β-blockers, angiotensin converting enzyme inhibitor/angiotensin receptor blockers (ACEI/ARBs), and statins less frequently than the no VT/VF group, while there was no difference between the early-onset group and late-onset group (Table 5). In addition, there was less use of β-blockers in the late-onset group than in the non-late-onset group (52.94% vs 76.1%, $P = .042$) (Table 6).

### 3.4. Risk assessment model building

Logistic multivariate regression analysis for single factors associated with the VT/VF group was used to detect independent factors.
factors of VT/VF occurrence, such as age, diabetes, heart rate, ST-segment elevation magnitude, LVEF, and IRA (Table 7). According to the differences and the condition regression tree analysis results, the continuity variable was divided into the classification variable to build the risk assessment model (Table 8). The model of risk classification was based on the regression tree model with full sample training conditions: Class I ≤ 4; 4 < Class II ≤ 5.5; 5.5 < Class III ≤ 6.5; and Class IV ≥ 6.5. Table 9 demonstrates VT/VF episodes of different risk levels: the higher the level, the higher the risk (Table 9).

### Table 3
Primary percutaneous coronary intervention characteristics.

| Factors          | No VT/VF group (n = 540) | VT/VF group (n = 67) | P       | Early onset group (n = 50) | Late onset group (n = 17) | P       |
|------------------|--------------------------|---------------------|---------|---------------------------|---------------------------|---------|
| IRA              |                          |                     |         |                           |                           |         |
| LAD              | 360 (66.67%)             | 28 (41.79%)         | <.001   | 18 (36%)                  | 10 (58.82%)               | .036    |
| LCX              | 45 (8.33%)               | 3 (4.46%)           |         | 1 (2%)                    | 2 (11.76%)                |         |
| RCA              | 135 (25%)                | 36 (53.73%)         |         | 31 (62%)                  | 5 (29.41%)                |         |
| Multi vessel lesion | 436 (80.74%)          | 55 (82.09%)         | .791    | 41 (82%)                  | 14 (82.33%)               | 1.000   |
| LM lesion        | 46 (8.52%)               | 11 (16.42%)         | .037    | 10 (20%)                  | 1 (5.88%)                 | .266    |
| TIMI grade before PPCI  |                   |                     |         |                           |                           |         |
| 0                | 411 (76.11%)             | 62 (92.54%)         | .018    | 44 (88%)                  | 14 (82.35%)               | .799    |
| 1                | 46 (8.52%)               | 3 (4.48%)           |         | 3 (6%)                    | 1 (5.88%)                 |         |
| 2                | 42 (7.73%)               | 1 (1.49%)           |         | 2 (4%)                    | 1 (5.88%)                 |         |
| 3                | 41 (7.59%)               | 1 (1.49%)           |         | 1 (2%)                    | 1 (5.88%)                 |         |
| TIMI grade after PPCI |                    |                     |         |                           |                           |         |
| 0                | 2 (0.36%)                | 1 (2%)              | .02     | 1 (2%)                    | 1 (5.88%)                 |         |
| 1                | 11 (1.97%)               | 0 (0%)              | .117    | 8 (1.6%)                  | 3 (17.6%)                 | <.001   |
| 2                | 45 (8.08%)               | 1 (2%)              |         | 39 (6.61%)                | 7 (41.76%)                |         |
| 3                | 499 (89.50%)             | 48 (86%)            |         | 541 (91.69%)              | 6 (35.29%)                |         |
| Symptom-balloon time | 450 (300–630)          | 420 (300–720)       | .883    | 420 (300–720)             | 450 (360–570)             | .960    |
| Stent            | 393 (72.78%)             | 50 (74.63%)         | .74     | 37 (74%)                  | 13 (76.47%)               | .716    |
| IABP             | 105 (19.44%)             | 20 (29.85%)         | .051    | 16 (32%)                  | 4 (23.53%)                | .510    |
| Emerging III°AVB | 101 (18.7%)              | 11 (16.42%)         | .69     | 7 (14%)                   | 4 (23.53%)                | .451    |
| ECG recovery >50% | 222 (41.11%)             | 26 (46.81%)         | .717    | 23 (46%)                  | 2 (1.65%)                 | .038    |

ECG = electrocardiogram, IABP = intra aorta balloon pump, III°AVB = three grade atrioventricular block, IRA = infarct related artery, LAD = left anterior descending, LCX = left circumflex, LM = left main, PPCI = primary percutaneous coronary intervention, RCA = right coronary artery, TIMI = thrombolysis in myocardial infarction, VT/VF = ventricular tachycardia/ventricular fibrillation.

### Table 4
Primary percutaneous coronary intervention characteristics for the early-onset group and non-early-onset group, and the late-onset group and non-late-onset group.

| Factors          | Non-early onset group (n = 557) | Early onset group (n = 50) | P       | Non-late onset group (n = 590) | Late onset group (n = 17) | P       |
|------------------|---------------------------------|---------------------------|---------|-------------------------------|---------------------------|---------|
| IRA              |                                 |                           |         |                               |                           |         |
| LAD              | 372 (66.79%)                    | 24 (48%)                  | .001    | 384 (65.08%)                  | 2 (11.76%)                | .525    |
| LCX              | 47 (8.44%)                      | 1 (2%)                    |         | 46 (7.8%)                    | 2 (11.76%)                |         |
| RCA              | 138 (24.78%)                    | 25 (50%)                  |         | 160 (27.12%)                 | 3 (17.65%)                |         |
| Multi vessel lesion | 450 (80.79%)                  | 41 (82%)                  | .835    | 477 (80.85%)                 | 14 (82.35%)               | 1.000   |
| LM lesion        | 47 (8.44%)                      | 10 (20%)                  | .013    | 56 (9.89%)                   | 1 (5.88%)                 | .722    |
| TIMI grade before PPCI  |                     |                           |         |                               |                           |         |
| 0                | 426 (76.42%)                    | 47 (94%)                  |         | 458 (77.63%)                 | 15 (88.24%)               |         |
| 1                | 47 (8.44%)                      | 2 (4%)                    | .028    | 48 (8.14%)                   | 1 (5.88%)                 | .875    |
| 2                | 42 (7.54%)                      | 1 (2%)                    |         | 43 (7.29%)                   | 0 (0%)                    |         |
| 3                | 42 (7.54%)                      | 0 (0%)                    |         | 41 (6.95%)                   | 1 (5.88%)                 |         |
| TIMI grade after PPCI |                    |                           |         |                               |                           |         |
| 0                | 2 (0.36%)                       | 2 (4%)                    | .032    | 2 (4%)                       | 1 (5.88%)                 |         |
| 1                | 11 (1.97%)                      | 0 (0%)                    | .117    | 8 (1.6%)                     | 3 (17.65%)                | <.001   |
| 2                | 45 (8.08%)                      | 1 (2%)                    |         | 39 (6.61%)                   | 7 (41.76%)                |         |
| 3                | 499 (89.50%)                    | 48 (86%)                  |         | 541 (91.69%)                 | 6 (35.29%)                |         |
| Symptom-balloon time | 450 (300–630)               | 420 (300–720)             | .941    | 450 (300–630)                | 450 (360–570)             | .876    |
| Stent            | 406 (72.9%)                     | 37 (74%)                  | .866    | 430 (72.88%)                 | 13 (76.47%)               | .794    |
| IABP             | 109 (19.57%)                    | 16 (32%)                  | .037    | 121 (20.51%)                 | 4 (23.53%)                | .762    |
| Emerging III°AVB | 105 (18.85%)                    | 7 (14%)                   | .397    | 108 (18.31%)                 | 4 (23.53%)                | .751    |
| ECG recovery >50% | 225 (40.30%)                    | 23 (46%)                  | .440    | 244 (41.36%)                 | 3 (17.65%)                | .950    |

IRA = infarct related artery, LAD = left anterior descending, LCX = left circumflex, RCA = right coronary artery, LM = left main, TIMI = thrombolysis in myocardial infarction, PPCI = primary percutaneous coronary intervention, IABP = intra aorta balloon pump, III°AVB = three grade atrioventricular block, ECG = electrocardiogram. Non-early-onset group means no VT/VF group and late-onset group; non-late-onset group means no VT/VF group and early-onset group.
The risk grading score and the total sample training were used as predictor variables; 60% of samples were randomly sampled as training sets and 40% of samples were used as test sets for the cross-validation of model prediction. AUC was 0.9 ($P < .001$) when a specific score was a predictor variable and 0.88 ($P < .001$) when the risk class grade for full sample training was a predictor variable (Figs. 2 and 3).

### 4. Discussion

The study investigated the incidence, episode timing, and risk factors of VT/VF in PPCI patients due to STEMI and built the 1st risk assessment model for predicting the possibility of a VT/VF episode. The incidence of VT/VF in this study was 11%, with approximately 2/3 of cases occurring before the patients were out of the catheter lab, which could be defined as early-onset.

### Table 5

| Factors          | No VT/VF group (n = 540) | VT/VF group (n = 67) | P   | Early onset group (n = 50) | Late onset group (n = 17) | P   |
|------------------|--------------------------|----------------------|-----|----------------------------|---------------------------|-----|
| Aspirin          | 520 (96.3%)              | 62 (92.54%)          | .144| 46 (92%)                   | 16 (94.12%)               | 1.000|
| P2Y12 blocker    | 529 (97.96%)             | 65 (97.01%)          | .645| 49 (98%)                   | 16 (94.12%)               | .446 |
| β receptor blocker | 414 (76.67%)              | 44 (65.67%)          | .049| 32 (64%)                   | 12 (70.59%)               | .621 |
| ACE/ARB          | 415 (76.85%)             | 44 (65.67%)          | .044| 33 (66%)                   | 11 (64.71%)               | .923 |
| statin           | 489 (90.56%)             | 55 (82.09%)          | .032| 40 (80%)                   | 15 (88.24%)               | .716 |
| LMWH             | 448 (82.96%)             | 54 (80.6%)           | .629| 41 (82%)                   | 13 (76.47%)               | .725 |

ACEI=angiotensin converting enzyme inhibitor, ARB=angiotensin receptor blocker, LMWH=low molecular weight heparin, VT/VF=ventricular tachycardia/ventricular fibrillation.

### Table 6

| Factors          | Non-early onset group (n = 557) | Early onset group (n = 50) | P   | Non-late onset group (n = 590) | Late onset group (n = 17) | P   |
|------------------|---------------------------------|---------------------------|-----|-------------------------------|---------------------------|-----|
| Aspirin          | 536 (96.23%)                    | 46 (92%)                  | .253| 566 (95.93%)                  | 16 (94.12%)               | 1.000|
| P2Y12 blocker    | 545 (97.85%)                    | 49 (98%)                  | 1.000| 578 (97.97%)                  | 16 (94.12%)               | .311 |
| β receptor blocker | 423 (75.94%)                    | 35 (70%)                  | .350| 449 (76.1%)                   | 9 (52.94%)                | .042 |
| ACE/ARB          | 426 (76.48%)                    | 33 (66%)                  | .098| 448 (75.93%)                  | 11 (64.71%)               | .388 |
| statin           | 502 (90.13%)                    | 42 (84%)                  | .174| 531 (80%)                    | 13 (76.47%)               | .069 |
| LMWH             | 461 (82.76%)                    | 41 (82%)                  | .691| 489 (82.88%)                  | 13 (76.47%)               | .513 |

ACEI=angiotensin converting enzyme inhibitor, ARB=angiotensin receptor blocker, LMWH=low molecular weight heparin. Non-early-onset group means no VT/VF group and late-onset group; non-late-onset group means no VT/VF group and early-onset group.

### Table 7

| Factors          | OR  | 95% confidence interval |
|------------------|-----|-------------------------|
| Age              | 1.04| 1.001, 1.082             |
| Diabetes         | 4.604| 1.901, 12.654            |
| Heart rate       | 1.027| 1.017, 1.06              |
| Top ST segment elevation (mv) | 162.367| 8,313, 4075.019          |
| Sum ST segment elevation (mv) | 31.705| 4,072, 102.933           |
| Serum K+         | 0.621| 0.394, 0.929             |
| LVEF             | 0.55 | 0.456, 0.642             |
| IRA was RCA      | 1.549| 0.997, 2.425             |
| LM lesion        | 3.624| 1.187, 10.95             |
| Killip Class > 1 | 3.255| 1.421, 7.849             |
| TIMI grade 0 before PPCI | 7.449| 2.09, 37.023             |

RA=intact related artery, LM=left main, LVEF=left ventricular ejection fraction, PPCI=primary percutaneous coronary intervention, RCA=right coronary artery, TIMI=thrombolysis in myocardial infarction.

### Table 8

| Factors          | Classification | Risk grade | Score |
|------------------|----------------|------------|-------|
| Diabetes         | No             | Low        | 0     |
|                  | Yes            | High       | 1     |
| TIMI grade 0 before PPCI | No           | Low        | 0     |
|                  | Yes            | High       | 1     |
| Age              | <65 years old  | Low        | 0     |
|                  | ≥65 years old  | High       | 1     |
| Heart rate       | <75 bpm        | Low        | 0     |
|                  | 76–96 bpm      | Middle     | 0.5   |
|                  | ≥97 bpm        | High       | 1     |
| Sum ST segment elevation (mv) | ≤1mv        | Low        | 0     |
|                  | >1mv           | High       | 1     |
| Serum K+         | >3.2mmol/L     | Low        | 0     |
|                  | ≤3.2mmol/L     | High       | 1     |
| Top ST segment elevation (mv) | ≤0.3–0.4mv      | Low        | 0     |
|                  | >0.5mv         | Middle     | 0.5   |
| LVEF             | ≥42%           | Low        | 0     |
|                  | <42%           | High       | 1     |

LVEF=left ventricular ejection fraction, PPCI=primary percutaneous coronary intervention, TIMI=thrombolysis in myocardial infarction, VT/VF=ventricular tachycardia/ventricular fibrillation.

### Table 9

| Risk grade | Cases | VT/VF cases | Percentage |
|------------|-------|-------------|------------|
| I          | 401   | 7           | 1.75%      |
| II         | 157   | 30          | 19.11%     |
| III        | 31    | 15          | 48.39%     |
| IV         | 18    | 15          | 83.33%     |
| Sum        | 607   | 67          | 11.04%     |

VT/VF=ventricular tachycardia/ventricular fibrillation.
VT/VF. In our study, 91% of patients experienced VT/VF within 48 h, which conforms with other similar studies. Compared with the previous studies of thrombolytic age, the incidence of VT/VF in this study was lower than that of thrombolytic therapy. Although the effect of thrombolysis and PCI on VT/VF has not been confirmed by a large study, it is not difficult to understand the differences considering the superiority of percutaneous coronary intervention (PCI) compared with thrombolysis in rescuing near-death myocardium and the predictive significance of the infarct area to VT/VF.

4.1. Incidence and timing of VT/VF occurrence

The incidence of VT/VF in this study was 11%, which is a little higher than noted in the APEX-AMI study. However, the previous investigations were not exactly the same, such as the PAMI study, which excluded renal dysfunction and cardiogenic shock, and the APEX-AMI study, which did not enroll inferior MI. We found a majority of VT/VF incidences happened within 48 h after an artery event, which conformed with other studies. The reason for this timing is not very clear, but it may be associated with electric instability and altered ION channel function. The patients in early onset group tend to be “sicker”, while the late onset group usually are related to an incomplete reperfusion, a finding that emphasizes the importance of a timely and effective reperfusion.

4.2. Clinical characteristics

This study identified risk factors of VT/VF from clinical characteristics, PCI data, and pharmacotherapy usage. We found that the VT/VF group had more patients who were elderly and smoked and had pre-MI, diabetes, a faster heart rate, lower blood pressure, worse heart function, low creatinine clearance, low LVEF, and higher CK-MB. The VT/VF group also manifested a high proportion of atrial flutter/atrial fibrillation and top ST-segment elevation magnitude of more than 0.3 mv coupled with sum ST-segment elevation magnitude of more than 1.5 mv. Age, MI history, diabetes, and smoking are high-risk factors of SCD or ventricular arrhythmia, while heart function after MI, CK-MB level, and LVEF can reflect the area of infarction. This shows that the patient’s basic health status as well as the ischemic or infarction range may affect the heart’s cardiac electrical activity, followed by inducing VT/VF.

Electrolyte disorders, such as low blood potassium and VT/VF, have been widely accepted. In this study, serum potassium of the VT/VF group was lower than the no VT/VF group, which agreed with earlier studies. It is noteworthy that the serum potassium of both of the groups was still within the normal range, which would have been associated with the sympathetic nervous system activating the renin-angiotensin-aldosterone-system (RAAS) in the acute period instead of the potassium affecting the process of VT/VF onset. Heart rate is an important hemodynamic parameter, which not only determines oxygen demand, but also affects the blood supply of the coronary arteries. When the heart rate increases rapidly, the myocardial oxygen consumption increases, which can aggravate the ischemic injury and increase the likelihood of VT/VF occurrence. Electrocardiogram (ECG) reveals many predictors of VT/VF, such as J wave, QRS wide, Tp-e, and Tp-e/QT ratio. We compared the ST-segment elevation condition between the groups, and the results suggested that the VT/VF group’s ST-segment elevation was higher than the no VT/VF group, which is similar to Demidova’s study. The significance of ST-segment elevation is mainly that it could reflect the infarct area in inferior leads, while the number of anterior leads in ST-segment elevation may be more meaningful.

The peak of CK-MB was positively correlated with the area of MI; there was a bigger infarct area and worse left ventricular function corresponded to a high CK-MB level. During MI, acidosis rendered by anaerobic glycolysis facilitated Na+ flow in through the Na+-H+ exchanger; free radicals mediate lipid peroxidation, which damaged the cell membrane and decreased Na+-K+-ATP enzyme activity, resulting in intracellular Na+ concentration increase, then the Na+-Ca2+ exchanger in reverse transport mode caused intracellular Ca2+ overload, bring about resting potential decline, maximum resting potential, and the threshold potential was approached, automatic rhythmicity was raised, and the likelihood of ventricular arrhythmia is higher.

Inferior wall MI, particularly combined with right ventricle infarction, has a greater possibility for the patient to suffer from VT/VF, which may be associated with there being a rich Ito channel in the membrane of the right ventricle. When there is an inferior wall ischemic injury, most of the Ito channels are quickly inactivated, whereas ICa channels are activated,
extending the phase 2 plateau and thus the action potential duration. The voltage gradients between the different myocardial areas induced by such a channel dysfunction give rise to a phase 2 re-entry which is deemed to be the main arrhythmia mechanism.[29] However, there was no difference in the comparison of right ventricular infarction in this study, which may be related to the subjects with ECG in this study who sometimes failed to execute the right ventricular lead in time, capturing ECG changes of right ventricular leads, and underestimating the number of right ventricular infarction patients. In recent years, the potential relationship between atrial fibrillation and malignant arrhythmia has been established, but the specific mechanism is not clear.[30,31] The results showed that there were more patients with atrial fibrillation/atrial flutter in the late-onset VT/VF group, but no difference was found between the VT/VF group and the no VT/VF group, which was in disagreement with previous studies that showed that atrial fibrillation correlated to VF before PPCI.[10] this may be related to a smaller sample size and insufficient patient’s arrhythmia data pre-admission. As for Killip class, LVEF could directly reflect the heart function, high Killip class, and low LVEF, suggesting a large infarction area, ischemic condition, and a greater possibility of having VT/VF.

4.3. PPCI characteristics

The TIMI grade 0 before PPCI has a little higher incidence rate of VT/VF than those that have partial flow (TIMI grade ≥ 1), which is consistent with previous studies, suggesting that TIMI 0 grade flow means a more serious ischemic injury. There are some studies that have suggested that the time from symptom onset to balloon < 180–360 min is associated with VT/VF.[8,14] The APEX-AMI study[16] also noted that the ischemia time of the VT/VF group was shorter than in the no VT/VF group. Animal experiments demonstrated that the short coronary artery occlusion time is associated with reperfusion arrhythmia and when the occlusion time is beyond 3 h, it will not increase reperfusion arrhythmia but the incidence of ischemia-induced arrhythmia will be raised.[15] Our data were not consistent with these studies; this may be due to insufficient medical records of the accurate balloon time instead of by processing the ending time, which prolonged the total ischemia time. Thus, the results of the study do not contradict the previous conclusions concerning the correlation between short ischemia time and VT/VF occurrence. The mechanism of arrhythmias following a short ischemia time may mainly be based on an electrical instability due to the rapid time-shift from ischemia to reperfusion because the electro activity may transformation frequently in a relatively short time.

The IRA also has a relationship with malignant arrhythmia. It can be seen that an RCA event has the highest VT/VF incidence. The mechanism of this phenomenon is mainly related to the following aspects: First, when the RCA is opened, the excited vagus nerve could compensate for the activated sympathetic nerve.[33] Second, in almost 60% of cases, the sinus node artery arises from the RCA, so its pacemaker function might be impaired after the vessel’s occlusion, making easier for ectopic pacemakers in the ventricular muscle to get activated in a highly sympathetic driven condition such as the myocardial infarction. Third, the RCA occlusion often causes an inferior wall and right ventricular infarction, making more likely to present with ventricular arrhythmias. We also found that the VT/VF group has more patients with LM lesions, but multi-vees lesions are similar between each of the groups. The LM lesions mean more serious MI and a larger area at risk (AAR) may be an appropriate reason.

Reperfusion efficiency also influences cardiac electro activity. The CLARITY-TIMI 28 study[34] enrolled 3491 STEMI patients to investigate the relationship between impaired myocardial perfusion (TIMI grade 0–2) and VT/VF after fibrinolytic therapy. The results even suggested LVEF ≥ 30% or the coronary artery flow rehabilitation impaired myocardial perfusion grade remained associated with an increased incidence of VT/VF. The ST-segment recovery > 50% suggested frequent revascularization. We found that the degree of ST-segment recovery was related to post-procedure VT/VF, and the late-onset ST-segment was more common in recovery in less than 50% of the groups. The late-onset group has a high proportion of TIMI grade < 3 after PPCI, ST-segment recovery of less than 50%, and a low proportion of TIMI grade 3 after PPCI, and ST-segment recovery of more than 50%, suggesting that the reperfusion effect is associated with postoperative VT/VF incidence.

4.4. Pharmacotherapy characteristics

The METOCARD-CNIC study[35] enrolled 270 anterior wall MI cases that were below Killip II; patients were given metoprolol or placebo at random before reperfusion and the infarction size was compared. The results showed that the intravenous infusion of metoprolol before reperfusion could reduce the infarct size and improve LVEF. However, the Early-BAMI study[36] researched 683 STEMI < 12h, Killip I–II patients who randomly received intravenous metoprolol or placebo, and the results suggested that the infarct size made no difference between the 2 groups, but metoprolol could reduce malignant arrhythmia. It can be seen that although β-receptor blockers have different results in reducing the area impacted by MI, the reduction of VT/VF has been confirmed in many aspects. In the treatment of ventricular arrhythmia in acute myocardial infarction, beta receptor blocker is recommended.[37]

The ACEI/ARBs and statins could reduce the ischemia burden, ameliorate ventricular remodeling, stable plaque, inhibit inflammation, and have an antiarrhythmic effect, and they have been widely accepted in the prevention of ventricular arrhythmia in patients with MI.[38] Our results also support the use of beta blockers, ACEI/ARBs, and statins, which were used less in the VT/VF group than in the no VT/VF group; this suggests that beta blockers, ACEI/ARBs, and statins should be used as early as possible to reduce the incidence of VT/VF and significantly improve the patient’s prognosis.

4.5. Risk assessment model

The data mentioned above suggests that there are many risk factors related to VT/VF from baseline clinical characteristics to reperfusion therapy and drug treatments, such as blood pressure, heart function, and reperfusion effect. This suggests we should prevent VT/VF from occurring in multiple ways and use β blockers as early as possible to reduce the onset of this type of malignant arrhythmia.

Multivariate regression analysis showed that there were many factors that were independently related to VT/VF occurrence, such as age, diabetes, heart rate, ST-segment elevation magnitude, LVEF, and IRA. We further constructed a risk assessment model and obtained the scoring system for VT/VF risk, from low to high with 4 grades, using the statistical method: the higher the level, the higher the risk of occurrence of VT/VF. Then, the
predictive value was evaluated by cross-validation with the risk classification as the predictor variable based on the specific score and full sample training (AUC was 0.9 or 0.88 respectively, \( P < .001 \)). It can be seen that the possibility of VT/VF occurrence could be specifically predicted using the risk assessment model and scoring system, but the validation method is purely statistical and requires subsequent trials with larger samples and more accurate data collection to demonstrate its accuracy.

### 4.6. Study limitation

The main limitation of this study is that it is a single-center retrospective study, and the data were not comprehensive. In addition, we only investigated patients who underwent PPCI; patients who had not received PPCI were not included in the study. We are conducting further research through a multicenter prospective study. We also call for further studies to build risk assessment system to predict VT/VF of STEMI patients.

### 5. Conclusion

Incidence of VT/VF in STEMI patients undergoing PPCI is 11% and it occurs more frequently from the time that symptoms begin to before the end of PPCI, which, in most cases, occurs within 48 h of the event. Our risk assessment model could predict the possible occurrence of VT/VF.

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