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Predictors of in-hospital heart failure in patients with acute anterior wall ST-segment elevation myocardial infarction☆

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ABSTRACT

Background: Heart failure (HF) is a severe complication of acute ST-segment elevation myocardial infarction (STEMI). Its incidence is associated with myocardial infarction location, and it occurs frequently after acute anterior wall STEMI due to the larger infarct size. However, predictors of in-hospital HF in patients with acute anterior wall STEMI are inadequately defined. We aimed to determine potential predictors of HF in patients with acute anterior wall STEMI during hospitalization.

Methods: A total of 714 consecutive patients who were diagnosed with acute anterior wall STEMI and underwent primary percutaneous coronary intervention (pPCI) between January 2013 to August 2019 were enrolled retrospectively. We assigned the patients to HF and non-HF groups. The clinical parameters were subjected to univariate analysis and logistic regression analysis to obtain the independent predictors.

Results: Among the 714 patients enrolled in the present study (mean age 61.0 ± 13.8 years, men 80.7%), 387 (54.2%) had in-hospital HF. According to a multivariate logistic regression analysis, ventricular fibrillation (VF, OR: 5.66, 95% CI: 2.25–14.23, $P < 0.001$) was the most striking independent predictor of in-hospital HF. Community-acquired pneumonia (CAP, OR: 4.72, 95% CI: 2.44–9.10, $P < 0.001$), age (OR: 1.03, 95% CI: 1.01–1.04, $P < 0.001$), left ventricular ejection fraction (LVEF, OR: 0.96, 95% CI: 0.93–0.97, $P < 0.001$), and peak N-terminal pro-brain natriuretic peptide (NT-pro-BNP, OR: 1.06, 95% CI: 1.02–1.11, $P = 0.006$) were also independently associated with in-hospital HF.

Conclusion: VF, CAP, age, LVEF, and peak NT-pro-BNP were independently associated with in-hospital HF in patients with acute anterior wall STEMI.

1. Introduction

Heart failure (HF) is widely recognized as the significant prognostic factor for ST-segment elevation myocardial infarction (STEMI), and it can occur in the acute or subacute phase of STEMI [1]. Affected by factors such as myocardial infarction type, myocardial infarction site, reperfusion mode, diagnostic criteria, follow-up duration, and medical condition differences, the incidence of new-onset HF after acute STEMI reported in different studies varies considerably, ranging from 10% to 45% [1–7]. Although the prognosis of HF after STEMI has been improved with the development of primary percutaneous coronary intervention (pPCI) and secondary prevention drugs for coronary heart disease, the rate of all-cause mortality and rehospitalization is still high [3,8]. Moreover, it is reported that in-hospital HF occurring after myocardial infarction accounts for the majority of HF cases [9], and it is a strong independent predictor of in-hospital mortality in STEMI patients undergoing pPCI [4]. Thus, it is important to recognize high-risk populations that may develop HF after STEMI.

There are many studies on the predictors of HF after STEMI. However, most studies have reported that the inclusion of patients with

Abbreviations: HF, Heart failure; STEMI, ST-segment elevation myocardial infarction; pPCI, Primary percutaneous coronary intervention; VF, Ventricular fibrillation; CAP, Community-acquired pneumonia; LVEF, Left ventricular ejection fraction; NT-pro-BNP, N-terminal pro-brain natriuretic peptide; RWMA, Regional wall motion abnormality; TIMI, Thrombolysis in myocardial infarction; OR, Odds ratio; CI, Confidence interval; ROC, Receiver operating characteristic; AUC, Area under the curve.

☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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different types of acute myocardial infarction (STEMI or non-STEMI) or with different reperfusion modalities (e.g., thrombolysis, pPCI, or post-thrombolysis PCI) lead to high heterogeneity among the study subjects [10–12]. Moreover, some study subjects have been relatively low-risk due to the exclusion of patients with cardiogenic shock [11,13,14]. In addition, infarct location is closely correlated with clinical outcomes. Anterior wall myocardial infarction is caused by severe lesions of the left anterior descending artery, with greater myocardial necrosis and a more serious effect on left ventricular systolic function compared to other infarct locations. It has been suggested that patients with anterior myocardial infarction have a higher incidence of HF [11,14,15], a lower left ventricular ejection fraction (LVEF) [16], and higher acute and long-term mortality [17] than patients who have other infarct sites. We therefore sought to identify the risk factors associated with this condition for prevention purposes. Currently, predictors of in-hospital HF in patient populations with anterior myocardial infarction remain unclear. Our study was performed retrospectively to search for predictors of in-hospital HF in patients with anterior STEMI.

2. Methods

2.1. Study population

A total of 714 consecutive patients with acute anterior wall STEMI at the First Affiliated Hospital of Jinan University between January 2013 and August 2019 were retrospectively included in this study. Ultimately, 387 patients with in-hospital HF were included in the HF group, and 327 patients without in-hospital HF were included in the non-HF group. The study protocol was approved by the ethics committee at our hospital, and informed consent was obtained from each patient enrolled.

The study included patients with the following conditions: (1) patients first diagnosed with acute anterior wall STEMI; (2) patients who underwent pPCI within 12 h of symptom onset or who underwent pPCI due to clinical and/or electrocardiographic evidence of progressive myocardial ischemia beyond 12 h of symptom onset. Exclusion criteria were as follows: (1) patients with previous myocardial infarction, chronic heart failure, congenital heart disease, cardiomyopathy, or severe heart valve disease; (2) patients with incomplete clinical data.

2.2. Clinical data acquisition

Once enrolled, the following clinical parameters were collected: (1) basic information, such as age, sex, tobacco and alcohol consumption, heart rate, systolic blood pressure and diastolic blood pressure upon admission, and past medical history (hypertension, diabetes, ischemic stroke, or angina pectoris); (2) in-hospital comorbidities and complications, such as sustained ventricular tachycardia, ventricular fibrillation (VF), third-degree atrioventricular block, new-onset atrial fibrillation or community-acquired pneumonia (CAP); (3) laboratory results from the first blood sample, including white blood cell, serum creatinine, cystatin C, low-density lipoprotein, glycosylated hemoglobin and fibrinogen levels. The high peak of biomarkers, including D-dimer, creatine kinase, creatine kinase MB, cardiac troponin I, and N-terminal pro-brain natriuretic peptide (NT-pro-BNP), were obtained according to repeated blood tests during hospitalization. Considering the accumulating effect, the measurement unit for high peak NT-pro-BNP was defined as per 1000 pg/ml; (4) echocardiography data, including LVEF, left ventricular end-diastolic diameter, left atrial diameter, regional wall motion abnormality (RWMA) of the left ventricular wall (including the anterior, apical and rest of the left ventricular wall), mitral regurgitation, aortic regurgitation, left ventricular diastolic dysfunction and left ventricular aneurysm, were obtained from the first transthoracic echocardiography. RWMA was considered present when hypokinesis, akinesia or dyskinesis of regional ventricular wall motion were observed by transthoracic echocardiography; (5) parameters of coronary angiography, such as pre-procedural thrombolysis in myocardial infarction (TIMI) flow in the left anterior descending artery, left circumflex and right coronary artery, post-procedural TIMI flow (< 3 grade) in infarct-related artery, lesions in the proximal left anterior descending artery, single-vessel disease, total artery occlusion, three-vessel disease and angiographically visible collateral blood supply, were also evaluated.

All transthoracic echocardiography examinations were performed within 24 h after admission. The coronary angiograms were analyzed by two independent angiography experts.

2.3. Definitions

The diagnosis of acute STEMI was based on ischemic chest pain, new or presumed new significant electrocardiographic changes in more than two continuous leads (ST elevation and T wave changes), and abnormal cardiac biomarkers (a rise and/or fall in cardiac troponin values with at least one value above the 99th percentile upper limit) [18,19] and was further reconfirmed by angiography. The infarct site was determined initially by electrocardiogram and further confirmed by coronary angiography and transthoracic echocardiography. In-hospital HF was defined as the documentation of clinical symptoms of HF (dyspnea upon exertion or fluid retention) or signs of HF (rales, jugular venous distension, or pulmonary edema) in patients with Killip class≥2 by physicians during hospitalization, and other diseases that can cause the above signs and symptoms were excluded.

2.4. Statistical analysis

All statistical analyses were conducted with SPSS version 25.0. Normal distribution was evaluated according to the Kolmogorov-Smirnov test. Continuous parameters with a normal distribution are presented as the mean ± standard deviation, while continuous parameters with a non-normal distribution are shown as the median (interquartile interval). The independent-sample *t*-test and Wilcoxon W test were used to compare the measurement data between groups, according to normality assumptions. Categorical variables are presented as percentages and were compared by the χ^2 test or Fisher's exact test. The odds ratio (OR) and confidence interval (CI) were obtained by multivariable logistic regression analysis. Variables with a *p*-value <0.05 according to univariate analyses were considered to be significant and were included in an unconditioned logistic regression analysis to select independent predictors ($\alpha = 0.05$). The optimal cut-off point and predictive value of each significant independent predictor were obtained by a receiver operating characteristic (ROC) curve analysis and the area under the curve (AUC).

3. Results

3.1. Univariate analysis results

A total of 714 patients with acute anterior STEMI (mean age 61.0 ± 13.8 years, 80.7% male) were enrolled in this study, including 387 (54.2%) in the HF group and 327 in the non-HF group.

Patients with HF had a high-risk profile including advanced age, more women, higher heart rate, lower blood pressure, lower LVEF, larger left ventricular end-diastolic diameter and more complications (VF, ventricular tachycardia, new-onset atrial fibrillation) and comorbidities (CAP) (Tables 1 and 2). Of the laboratory results, white blood cell, serum creatinine, cystatin C, fibrinogen, peak D-dimer and NT-pro-BNP levels were higher in the HF group than in the non-HF group ($P < 0.05$) (Table 1). In addition, three-vessel disease, pre-procedural TIMI flow≤1 grade in left circumflex and right coronary artery, post-procedural TIMI flow<3 grade in the infarct-related artery, mitral regurgitation, aortic regurgitation, left ventricular aneurysm and RWMA (including the anterior, apical and rest of the left ventricular wall) were associated with in-hospital HF in patients with acute anterior STEMI ($P < 0.05$) (Table 2).

Table 1
Basic information and laboratory results for the HF and non-HF groups.

Variables	HF group (n = 387)	non-HF group (n = 327)	P value
Basic information			
Age	64.5 ± 13.7	56.8 ± 12.7	<0.001
Males	291(75.2)	285(87.2)	<0.001
Hypertension	215(55.6)	169(51.7)	0.301
Diabetes mellitus	123(31.8)	84(25.7)	0.075
Previous angina	74(19.3)	52(15.9)	0.241
History of ischemic stroke	29(7.5)	17(5.2)	0.213
Tobacco consumption	186(48.1)	203(62.1)	<0.001
Alcohol consumption	40(10.3)	55(16.8)	0.011
Heart rate (bpm)	86(75, 100)	80(72, 91)	<0.001
Systolic blood pressure (mmHg)	125.7 ± 26.7	131.3 ± 24.9	0.004
Diastolic blood pressure (mmHg)	76.1 ± 16.3	79.8 ± 14.7	0.002
Community-acquired pneumonia	94(24.3)	12(3.7)	<0.001
Ventricular fibrillation	45(11.6)	6(1.8)	<0.001
Sustained ventricular tachycardia	20(5.2)	1(0.3)	<0.001
Third-degree atrioventricular block	6(1.6)	1(0.3)	0.093
New-onset atrial fibrillation	27(7.0)	7(2.1)	0.003
Laboratory results			
White blood cell (10^9/l)	11.5(8.7, 15.3)	10.7(8.6, 10.7)	0.014
Peak creatine kinase (u/l)	985.0(271.0, 2987.0)	1022.0(280.5, 2471.3)	0.579
Peak creatine kinase MB (u/l)	87.6(31.0, 269.0)	81.0(34.0, 202.5)	0.406
Serum creatinine (μmol/l)	85.0(69.2, 111.1)	74.6(63.0, 87.0)	<0.001
Cystatin C (mg/l)	1.3(1.0, 1.7)	1.1(0.9, 1.4)	<0.001
Low-density lipoprotein (mmol/l)	3.0 ± 1.0	3.7 ± 1.2	0.162
Glycosylated hemoglobin (%)	6.5 ± 1.7	6.5 ± 1.6	0.554
Peak D-dimer (μg/ml)	1154.0(530.0, 1737.1)	490.0 (290.0,1500.0)	<0.001
Fibrinogen (g/l)	4.1 ± 1.6	3.8 ± 1.3	0.012
Peak NT-pro-BNP (1000 pg/ml)	2.2(0.6, 5.8)	0.7(0.2, 2.3)	<0.001
Peak cardiac troponin I (μg/l)	19.2 ± 17.2	17.1 ± 16.6	0.100

Abbreviations: NT-pro-BNP, N-terminal pro-brain natriuretic peptide.

3.2. Unconditioned logistic regression analysis and ROC curve analysis

According to a univariable analysis, parameters with significant differences between the two groups were analyzed further with an unconditioned logistic regression analysis. The results suggest that the most striking risk predictor contributing to HF was VF. The risk of in-hospital HF was 5.66 times greater in anterior STEMI patients with VF than in those without (OR: 5.66, 95% CI: 2.25–14.23, $P < 0.001$). In addition, CAP (OR: 4.72, 95% CI: 2.44–9.10, $P < 0.001$), age (OR: 1.03, 95% CI: 1.01–1.04, $P < 0.001$), LVEF (OR: 0.96, 95% CI: 0.93–0.97, $P < 0.001$) and peak NT-pro-BNP (OR: 1.06, 95% CI: 1.02–1.11, $P = 0.006$) were found to be independent predictors of in-hospital HF among patients with anterior STEMI (Table 3).

3.3. ROC curve analysis

The optimal cut-off points for age, LVEF and peak NT-pro-BNP were 65 years, 50.7% and 1932 pg/dl, respectively, with AUC values of 0.66, 0.68 and 0.70, respectively (Fig. 1). The combined discrimination showed an excellent predictive value reflected by an AUC of 0.74 (Fig. 2).

Table 2
Echocardiography and angiography characteristics of the study population.

Variables	HF group (n = 387)	non-HF group (n = 327)	P value
Coronary angiography			
Pre-procedural TIMI flow≤1 grade in the left anterior descending artery	197(50.9)	166(50.8)	0.970
Pre-procedural TIMI flow≤1 grade in the left circumflex artery	39(10.1)	13(4.0)	0.002
Pre-procedural TIMI flow≤1 grade in the right coronary artery	37(9.6)	14(4.3)	0.006
Single-vessel disease	113(29.2)	123(37.6)	0.017
Presence of total artery occlusion	193(49.9)	154(47.1)	0.460
Presence of collateral blood supply	44(11.4)	25(7.6)	0.093
Lesion in the proximal left anterior descending artery	295(76.2)	233(71.3)	0.131
Post-procedural TIMI flow (< 3 grade) in the infarct-related artery	32(8.3)	12(3.7)	0.011
Three-vessel disease	165(42.6)	103(31.5)	0.002
Echocardiography			
Left ventricular ejection fraction (%)	47(39, 55)	56(48, 60)	<0.001
Left ventricular aneurysm	91(23.5)	41(12.5)	<0.001
RWMA in the apex	277(71.6)	205(62.7)	0.012
RWMA in the anterior wall	330(85.3)	239(73.1)	<0.001
RWMA in the rest of the ventricular wall	203(52.5)	117(35.8)	<0.001
Left ventricular diastolic dysfunction	138(35.7)	135(41.3)	0.123
Left ventricular end-diastolic diameter (mm)	46(42, 50)	45(42, 48)	0.032
Left atrial diameter (mm)	35(32, 40)	35(32, 38)	0.116 +77
Mitral regurgitation	167(43.2)	87(26.6)	<0.001
Aortic regurgitation	72(18.6)	43(13.1)	0.048

Abbreviations: TIMI, thrombolysis in myocardial infarction; RWMA, regional wall motion abnormality.

Table 3
Unconditioned logistic regression analysis results of risk factors.

Variables	*OR* value	95% CI	P value
Age	1.03	1.01–1.04	<0.001
Community-acquired pneumonia	4.72	2.44–9.10	<0.001 +77
Ventricular fibrillation	5.66	2.25–14.23	<0.001
Peak NT-pro-BNP (1000 pg/ml)	1.06	1.02–1.11	0.006
Left ventricular ejection fraction (%)	0.96	0.93–0.97	<0.001

Abbreviations: NT-pro-BNP, N-terminal pro-brain natriuretic peptide.

4. Discussion

Our study showed that VF, CAP, age, LVEF and peak NT-pro-BNP were independent predictors associated with HF after anterior STEMI. These predictors are useful for identifying patients who will require close monitoring during hospital admission due to anterior STEMI to prevent complications and improve outcomes. Furthermore, the combination of independent predictors indicated the excellent predictive ability of HF after anterior STEMI.

VF is an important poor prognostic indicator in patients with STEMI; its presence significantly increases the incidence of short-term adverse cardiovascular events and mortality [20]. The present study showed that VF is the strongest independent predictor of HF in patients with anterior STEMI, increasing the risk of HF by 4-fold; these results are similar to those of previous studies [12,20]. Vicent L et al. studied 1111 acute STEMI patients (89.2% received PCI, and 10.8% received thrombolysis only) and found that VF was independently associated with a high risk of in-hospital HF [12]. Piccini JP et al. found that myocardial infarction patients with ventricular tachycardia or VF had a 3-fold increased risk of developing in-hospital HF [20]. In the condition of myocardial infarction, the mechanism of VF is complex, including abnormal sympathetic

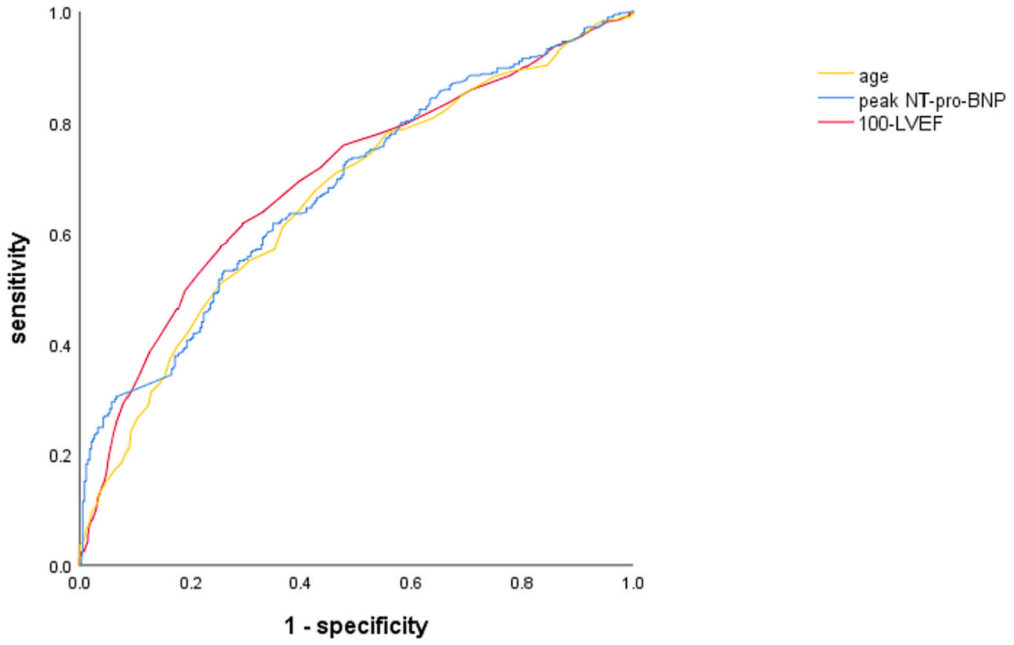

Fig. 1. Receiver operating characteristic curves for age, left ventricular ejection fraction and peak N-terminal pro-brain natriuretic peptide.

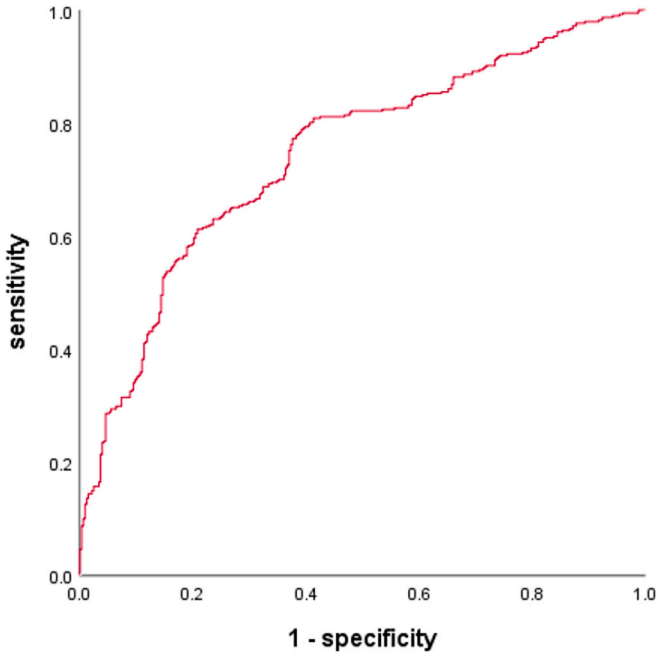

Fig. 2. Combined receiver operating characteristic curve.

activation, parasympathetic dysfunction, neural remodeling, electrophysiological remodeling and neuroendocrine system activation [21]. It has been suggested that patients with acute myocardial infarction who develop VF have the characteristics of larger infarct size [22] and lower LVEF [20], indicating that VF is a manifestation of severe myocardial ischemia. Furthermore, when VF occurs, the heart's blood supply stops suddenly due to rapid and irregular ventricular muscle contraction, which is another serious blow to heart function and easily induces the occurrence of HF. Since most VF occurs within 48 h after myocardial infarction [20,22], it can be considered an early predictor of anterior STEMI patients who have a high risk of HF, enabling clinicians to make timely therapeutic decisions that may improve short-term and long-term clinical outcomes.

It was previously demonstrated that patients with pneumonia have a higher incidence of adverse cardiovascular events, of which new or worsening HF is the most common cardiovascular complication [23,24]. The present study supports that CAP is independently associated with a high risk of HF in patients with anterior STEMI, suggesting that infectious factors play an important role in the development of HF. Currently, data on the association of CAP with HF after STEMI are scarce. Several mechanisms could explain this relationship between CAP and increased risk of HF in patients with anterior STEMI: (1) Circulating inflammatory mediators (cytokines, endotoxins, etc.) and microorganisms can directly lead to nonischemic myocardial injury, which further leads to the loss of viable cardiomyocytes [25,26]. Although lesions caused by pneumonia to the myocardium do not involve coronary arteries, cardiac function can be significantly aggravated and lead to HF in the case of STEMI [27]. (2) Endothelial dysfunction and vascular dystonia due to systemic inflammation increase peripheral vascular resistance, leading to increased left ventricular afterload [26]. (3) Pneumonia can lead to an imbalance between myocardial oxygen supply and demand, further exacerbating myocardial ischemia and expanding the effect of the infarction [24,26]. (4) Microbial infections, systemic inflammatory responses, and some antibiotics (e.g., some macrolides and quinolones) can lead to new or worsening arrhythmias that interfere with the efficient synchronization of the cardiac cycle and the coordinated contraction of cardiomyocytes, thus inducing HF [26]. (5) Microbial toxins and cytokines, such as tumor necrosis factor, interleukin-6, endothelin-1 and nitric oxide, produced by the inflammatory response can inhibit the contractility of the myocardium, further deteriorating the contractile function of the heart, and could thus contribute to the occurrence of HF [24,25]. STEMI complicated by CAP will undoubtedly significantly increase the complexity and severity of the condition. Thus, clinicians need to formulate a precise treatment plan as early as possible to reduce the occurrence of HF.

Theoretically, the older the patient with STEMI is, the poorer their cardiac function is to tolerate ischemia and hypoxia; consequently, the more serious the consequences of acute myocardial ischemia will be. It was previously suggested that age is an independent predictor of short-term and long-term mortality in STEMI patients [14,28]. Indeed, we demonstrated that advanced age is significantly related to HF in our study, which is consistent with the results in the study by Vicent et al. [12].

It has been previously acknowledged that both low LVEF and high peak NT-pro-BNP are important markers of poor left ventricular function [29,30]. NT-pro-BNP is a sensitive index reflecting intraventricular filling pressure and ventricular wall tension, and it is also an important index recognized by clinical guidelines for the diagnosis, severity and prognosis evaluation of HF [31]. In the present study, the high peak of NT-pro-BNP and low LVEF were independently associated with in-hospital HF in patients with anterior STEMI. It has been demonstrated that left ventricular wall tension increases as systolic left ventricular function declines, thus stimulating the natriuretic peptide system to release a large amount of NT-pro-BNP into the blood [32]. Similarly, LVEF is an important indicator for evaluating left ventricular systolic function [31] and is an independent predictor of short-term and long-term cardiac death in patients with STEMI [14]. It has been acknowledged that HF after STEMI is caused mainly by ventricular systolic dysfunction secondary to irreversible ischemic myocardial injury, which is closely related to LVEF decline.

The incidence of HF after acute myocardial infarction varies widely among studies due to different myocardial infarction types, infarct sites, reperfusion treatment methods, HF definitions, and follow-up times [1–7]. Several studies have proven that patients with acute anterior STEMI are at a higher risk of developing HF compared to patients with other infarct sites [11,14,15]. Indeed, the incidence of HF in the present study was 54.2%, significantly higher than the incidence reported in other studies [11,14,15], thus reaffirming that patients with anterior myocardial infarction have a higher risk of developing HF. This complication may be prevented by identifying these patients and promptly initiating aggressive management strategies, including expedited revascularization, more aggressive pharmacological therapies, intensified monitoring for worsening symptoms, more focused hemodynamic management, and early transfer to tertiary care centers with advanced interventional and heart failure facilities.

Several limitations of this study should be acknowledged. First, uncontrollable factors, such as the different times of repeated blood tests, could lead to failure in detecting the real high peak of cardiac biomarker concentrations. Second, selection bias may also exist due to the retrospective design. Third, the indicators included in the analysis were not complete enough, and some important variables were not included in our study, such as door-to-balloon time, hemoglobin levels and secondary prevention drugs, which may have affected the results. Thus, a larger sample size and a more scientific approach should be utilized in further research.

5. Conclusion

In anterior STEMI patients treated with pPCI, HF remains a common complication. VF, CAP, age, LVEF and peak NT-pro-BNP were independently associated with in-hospital HF in patients with acute anterior wall STEMI. Efforts should focus on identifying patients with a high-risk of in-hospital HF to develop tailored aggressive strategies of management.

Disclosure statement

The authors have nothing to disclose.

Declaration of Competing Interest

The authors report no relationships that could be construed as a conflict of interest.

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