INTRODUCTION

According to the “China Cardiovascular Disease Report 2014” issued by China Cardiovascular Center in 2015, it was pointed out that in 2013, patients with ischemic heart disease (IHD) accounted for 35.82% of the discharged patients with cardiocerebrovascular disease in Chinese hospitals. Non–ST-segment elevation acute coronary syndrome (NSTE-ACS) is a common type of IHD. Compared with ST-segment elevation acute coronary syndrome (STE-ACS), the incidence of NSTE-ACS is higher, accounting for about 75% of acute coronary syndrome (ACS). NSTE-ACS is more complicated and has more complications. Without timely treatment, it may progress to STE-ACS, causing irreversible myocardial damage and even endangering the lives of patients. Early monitoring and intervention of patients with NSTE-ACS are of positive significance for the prognosis and outcome of patients.

Since Morris put forward the concept of P-wave terminal force in lead V1 (PtfV1) in electrocardiogram (ECG) in 1964 and applied it to the study of heart valve disease, it has attracted widespread attention of clinical workers at home and abroad. PtfV1 has been paid more and more attention in the clinical application of cardiovascular diseases, especially in the diagnosis of left ventricular...
dysfunction. In recent years, it has been reported that the increase in the negative value of PtFV₁ in community population is related to the increased risk of cardiovascular mortality. NSTE-ACS can cause a series of intracardiac hemodynamic changes on the basis of myocardial ischemia, which can change the PtFV₁ value. Especially during the hospitalization of patients with NSTE-ACS, PtFV₁ may dynamically change with different conditions, such as underlying diseases, ischemic extent, revascularization, and different treatment methods, which may suggest different long-term clinical prognosis. However, there are no reports to confirm the relationship between the dynamic changes of PtFV₁ in ECG indicator and long-term prognosis in patients with NSTE-ACS.

Therefore, we proposed a hypothesis that the dynamic changes of PtFV₁ in ECG indicator had a predictive value for long-term prognosis in patients with NSTE-ACS. We conducted a prospective observational study to investigate the relationship between dynamic changes in PtFV₁ value and long-term prognosis in patients with NSTE-ACS during hospitalization. This study preliminarily explored the significance of the dynamic changes of PtFV₁ in the long-term prognosis of patients with NSTE-ACS.

2 | MATERIALS AND METHODS

2.1 | Subjects and management during hospitalization

We recruited patients diagnosed as NSTE-ACS (including unstable angina pectoris [UA] and non–ST-segment elevation myocardial infarction (NSTEMI)) at discharge between August 2015 and March 2017 in our study. All patients were diagnosed by coronary angiography. In addition, before the study started, we estimated the sample size to be 1188 patients.

Inclusion criteria were as follows: (a) The ages of the enrolled patients ranged from 18 to 80 years; (b) coronary angiography revealed 50% stenosis in at least one major epicardial coronary artery (anterior descending artery, circumflex artery, or right coronary artery); (c) increased frequency of ischemic chest pain or resting attacks; (d) there were no two consecutive precordial leads in the ECG, or the ST segment of the two limb leads was continuously increased by 0.1 mV; (e) at least one typical angina attack with a rest period of more than 20 minutes; and (f) elevated myocardial damage markers (the levels of myocardial damage markers such as serum troponin I or T, high-sensitive troponin T, or creatinine kinase (CK)-MB were 2-fold the upper limit of normal levels).

Exclusion criteria were as follows: (a) Patients with arrhythmia (atrial fibrillation, atrial flutter, ventricular tachycardia, ventricular fibrillation, ventricular flutter, atrial tachycardia, junctional tachycardia, etc); (b) patients had a history of permanent pacemaker implantation; (c) patients suffered from diseases that can lead to atrial enlargement (such as cardiomyopathy, valvular heart disease, congenital heart disease); (d) patients who were unable to cooperate with follow-up or sign informed consent; and (e) pregnant women and patients with malignant tumors, active hemorrhage, hypokalemia, blood diseases, or severe liver or kidney diseases.

Patient management during hospitalization was conducted in accordance with the ACC/AHA 2014 guidelines to manage unstable angina/non–ST-segment elevation myocardial infarction through conventional treatment. This study was approved by the Medical Ethics Committee of the Hospital. The written informed consents were obtained from all subjects, and the treatment process was not interfered by this study.

2.2 | Measurement of P-Wave Terminal Force in Lead V1

Each subject received an ECG measurement at admission and discharge. All subjects received standard 12-lead ECG (25 mm/s and 1 mV/10 mm) (GE Medical Systems). The PtFV₁ value was measured according to the method proposed by Morris and others. PtFV₁ was defined as follows. When the P-wave of V₁ leads was positive and negative biphasic, PtFV₁ = time (ms) × amplitude (mm) of the negative P-wave of V₁ leads, and the result was negative (mm-ms). When the P-wave of V₁ leads was vertical, it was calculated as 0.

The PtFV₁ value was measured using ECG diagnostic analysis system (Nalong Technology Co., Ltd). The horizontal line of T-P line before the start of the P-wave was extended to the descending branch of P-wave. The horizontal distance between the intersection point and the endpoint of P-wave was the time of negative P-wave. The vertical distance between the intersection point and the lowest point of negative P-wave was the amplitude of negative P-wave.

In order to ensure the reproducibility of the measuring, all measurements of ECG indicators in this study were conducted by two medical staffs independently. For each measurement, it was necessary to repeat the measurement of three sequential waves in the optimal leads and record the median values as the measuring results. The measurement of PtFV₁ value was carried out by specialists in electrocardiogram room of our hospital, and the lead position was marked to ensure that the position of the V₁ lead was consistent.

According to the standard that whether PtFV₁ of ECG was < -0.04 mm-s, the PtFV₁ was divided into PtFV₁(+) and PtFV₁(−). The subjects were divided into four groups according to the changes in PtFV₁ of ECG at admission and discharge. Patients who converted PtFV₁(+) at admission to PtFV₁(−) at discharge constituted the "normalized" group; patients who converted PtFV₁(−) at admission to PtFV₁(+) at discharge constituted the "new positive" group; patients who were both PtFV₁(+) at admission and discharge constituted the "persistent positive" group; and patients who were both PtFV₁(−) at admission and discharge constituted the "persistent negative" group.

2.3 | Collection of other clinical data

The clinical medical history was obtained, and physical examination and clinical laboratory tests were performed in each subject before
and after the study. The basic data were collected, including age, sex, smoking, basic diseases (including hypertension, diabetes, and stroke history), blood pressure, heart rate, revascularization, biochemical indicators (liver function, kidney function, blood lipid level), ejection fraction (EF), left atrial diameter (LA), and medication.

2.4 Follow-up and study endpoint

After the patients were discharged from the hospital, we followed up them from August 2015 to October 2018, with an average of about 19 months. Meanwhile, we recorded the occurrence of clinical events and the use of major drugs. Telephone follow-up was the main form, supplemented by clinical follow-up. The follow-up data in this study were derived from medical staff who participated in follow-up, patients themselves, and their families. All follow-up results were consistent with medical records in our hospital. The agreement and implementation of this study were in line with the Helsinki Declaration and approved by the local ethics committee.

The primary endpoint in this study was a composite endpoint of major adverse cardiovascular events (MACEs), including heart failure (rehospitalization), malignant arrhythmia, non-fatal re-myocardial infarction (including ST-segment elevation and non-ST-segment elevation), angina pectoris or myocardial ischemia requiring re-angioplasty (including PCI or CAGB with the exception of elective staged surgery), and cardiogenic death. The second endpoint was a composite endpoint for all-cause death and all components of the primary endpoint events.

2.5 Statistical analysis

SPSS 20.0 software (IBM Corporation) was applied to analyze all the data. The measured data were expressed as mean ± SD, and the counted data were expressed as percentage. Univariate analysis was used to compare the differences among the four groups. Variance analysis (normal distribution) or rank sum test (non-normal distribution) was used to compare the measurement data, and chi-square test was used to compare the rate and percentage. The Kaplan-Meier method was used to estimate the incidence of endpoint events during the comparison of endpoint events between groups. The log-rank test was used for survival analysis between groups. Factors with P ≤ .10 analyzed by univariate analysis were taken into Cox proportional hazards model, and it was used for survival analysis. Patient-related clinical data were assessed by GRACE score. In the current study, all tests with P < .05 were considered as statistically significant.

3 RESULTS

3.1 Patient characteristics

In our study, there were 1071 patients enrolled, excluding (a) 87 patients with arrhythmia (mainly atrial fibrillation, pacemaker implantation); (b) 6 patients with complicated cardiomyopathy, congenital heart disease, or valvular disease; and (c) 73 patients with incomplete and missing data (mainly missing PtfV1 data). Therefore, there were a total of 905 patients met the standard, and 72.9% of them were males. The average age of the patients was 68.6 ± 12.6 years. There was no significant difference in basic data, including sex, age, smoking, hypertension, diabetes, cerebral infarction, revascularization, proportion of non-ST-segment elevation myocardial infarction, systolic/diastolic blood pressure, heart rate, LDL-cholesterol (LDL-C), medication (Aspirin, Tigrillo/Clopidogrel, Statins, Beta-blockers, ACEI/ARB, Calcium Channel Blockers [CCB], Diuretics), among the four groups (P > .05) (Table 1). Interestingly, there was significant difference in history of diabetes mellitus, ejection fraction (EF), and left atrial (LA) diameter among the four groups (P < .05) (Table 1).

3.2 The results of endpoint events

All patients were followed up by telephone follow-up, supplemented by clinical follow-up. The follow-up time ranged from 1 to 38 months with an average of 19.1 months. The distribution of endpoint events among the four groups is shown in Table 2. The incidence of primary endpoint events was statistically significant in the four groups (P < .05). The highest incidence group was the PtfV1(−)/(+) group, with 25 patients (25.8%). The lowest incidence group was the PtfV1(−)/(−) group, with 35 patients (6.2%). Further analysis showed that the incidence of MACEs was significantly different among the four groups (P < .05). The highest incidence group was the PtfV1(−)/(+) group, with 24 patients (24.7%). The lowest incidence group was the PtfV1(−)/(−) group, with 28 patients (4.9%). However, there was no statistically significant difference in the incidence of cerebral infarction and all-cause deaths (e.g., tumor) in the four groups (P > .05). In addition, for secondary endpoint events, one patient in the PtfV1(−)/(−) group died of massive hemorrhage of the expendable tract (self-condition + antithrombotic agents). The remaining deaths, including all-cause deaths, were due to the combination of various malignancies.

3.3 Univariate analysis of endpoint events

Compared with the PtfV1(−)/(−) group, the risk for the occurrence of primary endpoint events in the PtfV1(−)/(+) group was significantly increased (25.8% vs 6.2%, adjusted HR 5.238, 95% CI 2.965–9.255, P = .000) with Kaplan-Meier survival analysis (P = .000). The risk for the occurrence of a primary endpoint events in the PtfV1(+) group was also increased (9.4% vs 6.2%, adjusted HR 2.105, 95% CI 1.155–3.836, P = .000) with Kaplan-Meier survival analysis (P = .013). Compared with the PtfV1(−)/(−) group, the risk for the occurrence of MACEs in the PtfV1(−)/(+) group was significantly increased (24.7% vs 4.9%, adjusted HR 6.283, 95% CI 3.456–11.418, P = .000) with Kaplan-Meier survival analysis (P = .000). The risk for the occurrence of MACEs in the PtfV1(+) group was also increased (10.1% vs 4.9%, adjusted HR 2.334, 95% CI 1.227–4.440,
P = .000) with Kaplan-Meier survival analysis (P = .008). However, there were no significant differences between the risks for the primary endpoint events and the secondary endpoint events between PtfV1 (+)/(−) group and PtfV1 (−)/(−) group with Kaplan-Meier survival analysis. All results above are shown in Table 3. Survival diagrams are drawn, respectively, in Figure 1.

### 3.4 Multivariate analysis of endpoint events

The primary endpoint events (MACEs) were analyzed using the Cox proportional hazards model. After adjusting for possible confounding factors, the results showed that PtfV1 (−)/(+) and PtfV1 (+)/(+) were still independent risk factors for primary endpoint events when compared with the PtfV1 (−)/(−) group. Their HR (95% CI) was 3.548 (95% CI 2.024–6.219) and 2.133 (95% CI 1.141–3.986), respectively (Table 4).

### 4 DISCUSSION

This study reviewed 905 NSTE-ACS patients’ ECGs recorded at admission and discharge and assessed the prognostic value of dynamic changes in PtfV1(−) ECG indicators. The results of the current study suggested that the dynamic changes of PtfV1 had a predictive value in the long-term prognosis of patients with NSTE-ACS. In addition, persistent PtfV1 (+) during hospitalization and new PtfV1(+) at discharge were significantly associated with an increased risk of long-term MACEs in these patients.

Heart failure is one of the major causes for death in patients with various cardiovascular diseases, including NSTE-ACS.12 With the decrease of ejection fraction (EF) in patients with heart failure, the stroke volume of heart also decreases. The greater the reduction, the more residual blood volume in the left ventricle, which results in an increase in left ventricular end-diastolic pressure. Accordingly, left atrial volume and pressure load also increase excessively. The above changes can cause the left atrial depolarization vector to change, resulting in an increase in the absolute value of the PtfV1.13 When heart failure was corrected, the patient's ventricular contractility was improved, the atrial volume and pressure were reduced, and the PtfV1 value was also reduced. Previous studies have also shown that an increase in negative PtfV1 suggests increased left atrial pressure and left ventricular diastolic dysfunction.14 Therefore, PtfV1 has been widely used in the early diagnosis, treatment, and prognosis evaluation of chronic heart failure.15

**TABLE 1 Characteristics of all patients**

|                         | PtfV1 (+)/(+)| PtfV1 (+)/(−)| PtfV1 (−)/(+) | PtfV1 (−)/(−) |
|-------------------------|--------------|--------------|---------------|--------------|
| Gender (female/male)    | 29/118       | 29/69        | 33/64         | 154/409      |
| Age (y)                 | 68.39 ± 10.51| 69.83 ± 10.36| 68.93 ± 9.28  | 67.25 ± 10.16|
| Smoking (Yes/No)        | 62/85        | 33/65        | 39/58         | 224/339      |
| Hypertension (Yes/No)   | 111/36       | 64/34        | 65/32         | 362/201      |
| Diabetes mellitus (Yes/No) | 48/99      | 34/64        | 40/57         | 153/410      |
| Cerebral infarction (Yes/No) | 14/133    | 15/83        | 12/85         | 50/513       |
| PCI/CABG (Yes/No)       | 106/41       | 76/22        | 70/27         | 436/127      |
| NSTE (Yes/No)           | 31/116       | 18/80        | 24/73         | 125/438      |
| Systolic pressure at admission (mm Hg) | 135.35 ± 19.68 | 136.74 ± 22.88 | 135.49 ± 19  | 133.37 ± 18.97 |
| Diastolic pressure at admission (mm Hg) | 79.03 ± 12.07 | 78.65 ± 10.21 | 78.52 ± 10.70 | 79.37 ± 10.76 |
| Heart rate at admission (BPM) | 74.14 ± 17.98 | 72.87 ± 10.21 | 72.91 ± 11.92 | 71.81 ± 10.2 |
| Systolic pressure at discharge (mm Hg) | 126.35 ± 14.18 | 124.94 ± 12.72 | 126.68 ± 13.67 | 124.95 ± 13.09 |
| Diastolic pressure at discharge (mm Hg) | 75.29 ± 8.83 | 74.01 ± 7.58 | 75.03 ± 8.08 | 74.38 ± 8.04 |
| Heart rate at discharge (BPM) | 70.43 ± 6.88 | 69.58 ± 5.61 | 69.88 ± 7.29 | 69.84 ± 6.59 |
| EF (%)                   | 56.31 ± 15.06| 59.51 ± 11.96| 59.36 ± 12.53 | 63.51 ± 9.54 |
| LA diameter (mm)         | 38.38 ± 5.53 | 35.50 ± 4.72 | 36.02 ± 5.73 | 34.24 ± 4.60 |
| LDL (mmol/L)             | 2.75 ± 1.10  | 2.86 ± 1.17  | 2.98 ± 1.12  | 2.85 ± 1.13  |
| Statin (Yes/No)          | 138/9        | 91/7         | 86/11         | 524/39       |
| Beta-blocker (Yes/No)    | 91/56        | 71/27        | 60/37         | 357/206      |
| ACEI/ARB (Yes/No)        | 101/46       | 61/37        | 60/37         | 319/244      |
| Aspirin (Yes/No)         | 131/16       | 88/10        | 89/8          | 521/42       |
| Tigrillo/clopidogrel     | 138/9        | 93/5         | 85/12         | 513/50       |
| CCB (Yes/No)             | 37/110       | 30/68        | 27/70         | 113/450      |
| Diuretic (Yes/No)        | 41/106       | 24/74        | 18/79         | 119/444      |

Abbreviation: PtfV1, P-wave terminal force in lead V1.
As a critical type of coronary heart disease, non–ST-segment elevation acute coronary syndrome (NSTE-ACS) has a dangerous condition, many complications, and high mortality and disability rate. On the basis of artery atherosclerotic plaques, if NSTE-ACS patients are further combined with some acute conditions, such as coronary spasm, thrombosis, and intraplaque hemorrhage, the stenosis degree of coronary artery will increase rapidly in a short time, resulting in subtotal or total occlusion of coronary artery, which in turn leads to unstable myocardial ischemia and even myocardial infarction. And it will inevitably lead to a series of hemodynamic changes in intracardiac cavity, resulting in changes in atrial volume and pressure load. Accordingly, patients with NSTE-ACS may have abnormal PtVF1, which suggests that it may be related to the severity of condition. In addition, studies have shown that with the increase of PtVF1 value, the prevalence of malignant arrhythmia in patients with cardiovascular disease is significantly increased. Hence, the combined effects of those factors are related to the incidence of cardiovascular events, which may lead to different clinical prognosis.

The abnormalities of PtVF1 during hospitalization in patients with NSTE-ACS indicated that myocardial ischemia had an effect on myocardium, which in turn affected hemodynamics of cardiac chambers, and PtVF1 may also have a dynamic change. The reason may be related to some clinical conditions, such as the extent of myocardial ischemia and the severity of coronary artery disease. It may also be related to the treatment situation and may further indicate the long-term clinical prognosis of patients. However, no relevant studies have reported the relationship between PtVF1 dynamic changes during hospitalization and long-term prognosis in NSTE-ACS patients. Therefore, we conducted this prospective observational study to investigate their correlation.

Our results showed that the incidences of primary endpoint events in the PtVF1 (+)/(+), PtVF1 (+)/(-), PtVF1 (-)/(+), and PtVF1 (-)/(-) group were markedly increased, while those in the PtVF1 (-)/(-) group were memoraably decreased. Univariate analysis showed that there were more risk factors in the PtVF1 (+)/(+) group and the PtVF1 (+)/(-) group for primary endpoint events and MACEs, when compared with the PtVF1 (-)/(-) group. In baseline comparison, there were significant differences in diabetes incidence, EF, and LA among the four groups. In addition, Cox regressive analysis showed that PtVF1 (-)/(+) and PtVF1 (+)/(+) were also independent predictors of primary endpoint events after incorporating age, sex, personal history, revascularization during hospitalization. In conclusion, these

| Table 2 | The endpoint events |
|---------|---------------------|
| | PtVF1 (+)/(+) | PtVF1 (+)/(-) | PtVF1 (-)/(+) | PtVF1 (-)/(-) | P value |
| Number | 147 | 98 | 97 | 563 |
| Primary endpoint events (no. [%]) | 18 (9.4) | 7 (7.1) | 25 (25.8) | 35 (6.2) | .000 |
| Total MACE events (no. [%]) | 16 (10.1) | 6 (6.1) | 24 (24.7) | 28 (4.9) | .036 |
| Cardiogenic death | 4 | 1 | 7 | 8 |
| Heart failure requiring admission | 1 | 0 | 7 | 4 |
| Malignant arrhythmia | 1 | 0 | 1 | 0 |
| Non-fatal re-infarction | 1 | 2 | 2 | 1 |
| Revascularization | 9 | 3 | 7 | 15 |
| Cerebral infarction (no. [%]) | 1 (0.7) | 1 (1.0) | 0 (0) | 2 (0.4) | .286 |
| Other causes of death (no. [%]) | 1 (0.7) | 0 (0) | 1 (1.0) | 5 (0.9) | .376 |

Abbreviations: MACE, major adverse cardiovascular event; PtVF1, P-wave terminal force in lead V1.

| Table 3 | Univariate analysis of endpoint events |
|---------|---------------------|
| | PtVF1 (+)/(+) | PtVF1 (+)/(-) | PtVF1 (-)/(+) | PtVF1 (-)/(-) |
| Number | 147 | 98 | 97 | 563 |
| Primary endpoint events (no. [%]) | 18 (9.4%) | 7 (7.1%) | 25 (25.8%) | 35 (6.2%) |
| HR (95% CI) | 2.105 (1.155-3.836) | 1.160 (0.5-2.692) | 5.238 (2.965-9.255) | 1 |
| P value | .013 | .729 | .000 |
| Total MACE events (no. [%]) | 16 (10.1%) | 6 (6.1%) | 24 (24.7%) | 28 (4.9%) |
| HR (95% CI) | 2.334 (1.227-4.440) | 1.246 (0.502-3.093) | 6.282 (3.456-11.418) | 1 |
| P value | .008 | .635 | .000 |

Abbreviations: MACE, major adverse cardiovascular event; PtVF1, P-wave terminal force in lead V1.
results suggested that the changes of intracardiac hemodynamics induced by acute ischemia can be reversed to some extent by interventions.

Dynamic observation of ECG indicator PtfV₁ can help doctors evaluate the long-term prognosis of patients with NSTE-ACS. Our results were similar to the results of some previous studies. For example, the study by Kobayashi A et al showed that abnormal P-wave terminal force in lead V₁ predicted left main and/or three-vessel disease in patients with non-ST-segment elevation myocardial infarction P-wave abnormality and extensive coronary artery disease.

Furthermore, the study by Li et al also showed that NSTE-ACS patients with persistent PtfV₁(+) ECG indicators at admission and discharge and new PtfV₁(+) at discharge had significantly increased risk of MACEs. It has been reported that after reperfusion therapy in patients with acute myocardial infarction, the performance of myocardial stunning can be improved in a shorter period of time. Therefore, if PtfV₁ continues to be (+) during hospitalization or new PtfV₁(+) appears at discharge in patients with NSTE-ACS (indicating that there is elevated left atrial pressure or left ventricular diastolic dysfunction), it suggests an irreversible left hemodynamics disorder, which may be associated with irreversible myocardial damage or without intervention for residual ischemia of the blood vessels. It has also been reported that left ventricular fibrosis is one of the causes of abnormal PtfV₁. Therefore, long-term repeated myocardial ischemia in patients with NSTE-ACS during hospitalization leads to left ventricular fibrosis, which may also result in persistent PtfV₁(+) or new PtfV₁(+) at discharge.

However, this study has some limitations. First of all, because of the single-center study, there is a certain limit on the sample size. Secondly, the follow-up time is not enough and further large-scale clinical studies are needed.

In conclusion, the dynamic changes of PtfV₁ have important clinical value in predicting the long-term incidence of cardiovascular events in patients with NSTE-ACS. In addition, the PtfV₁ test equipment (mainly for ECG examination) is easy to operate, non-invasive, inexpensive, and reproducible. The measurement of PtfV₁ value can help doctors to early detect, evaluate, and predict the long-term prognosis of patients with NSTE-ACS. Actively taking appropriate treatment measures can reduce the incidence of cardiovascular events and improve the quality of life of patients.

**ETHICS APPROVAL**

This study was approved by the Medical Ethics Committee of Union Hospital, Fujian Medical University [2016KY025].

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