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# Coronary Angiographic Features of de Winter Syndrome: More Than Just Occlusion of the Left Anterior Descending Artery

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## ABSTRACT

**Background:** The de Winter electrocardiogram (ECG) pattern is a rare presentation of ST-segment elevation myocardial infarction (STEMI) equivalent. The clinical profile of de Winter syndrome remains to be clarified.

**Methods:** Medical records of 1865 consecutive patients with acute myocardial infarction admitted from November 2018 to July 2023 were screened. Twelve patients (0.6%) with de Winter syndrome were included. STEMI patients whose culprit vessel was the left anterior descending artery (LAD) but without de Winter ECG pattern were selected as controls after 1:2 matching for age and sex.

**Results:** The de Winter syndrome patients were all male, aged 49.0 (46.3–52.5) years. The culprit lesion was the proximal LAD in seven patients (58.3%), the middle LAD in three (25%), the left main coronary artery in one (8.3%), and the ramus intermedius artery in the other. All of their culprit lesions had TIMI Thrombus Grade $< 4$, Cohen-Rentrop Score $\leq 2$, and residual stenosis $\geq 80\%$ after pretreatment with thrombus aspiration or balloon predilatation. Intracoronary imaging data were available in four patients, showing severe atherosclerotic stenosis. Compared with STEMI, de Winter syndrome had a higher prevalence of prior recurrent angina (75.0% vs. 37.5%, $p = 0.034$), better coronary collateralization (Cohen-Rentrop Score: 1 vs. 0, $p = 0.001$), lower thrombus burden (TIMI grade: 1 vs. 2, $p = 0.005$) but more severe atherosclerotic stenosis in the culprit lesions (90% vs. 60%, $p < 0.001$).

**Conclusions:** The culprit lesions of de Winter syndrome have a low thrombus burden, severe atherosclerotic stenosis, and poor collateral circulation protection.

## 1 | Introduction

De Winter syndrome, characterized by an electrocardiogram (ECG) pattern showing a 1–3-mm upsloping ST-segment depression at the J-point followed by tall, symmetrical T waves in precordial leads, was first systematically described and named by de Winter et al. (2008). This distinct ECG pattern was reported to occur in 2.0% of patients with anterior acute myocardial infarction and mostly signified severe stenosis of the proximal left anterior descending artery (LAD) (de Winter

Wenyi Tang and Junwei Xu contributed equally to this study.

et al. 2008; Verouden et al. 2009). It was increasingly recognized by cardiovascular physicians that de Winter syndrome was an anterior ST-segment elevation myocardial infarction (STEMI) equivalent requiring immediate reperfusion therapy, usually referred to as percutaneous coronary intervention (PCI) (Morris and Body 2017; Raja et al. 2019).

As the incidence of de Winter syndrome is relatively low, our current understanding of it mainly relies on analyses from underpowered single case reports or case series. Other than the classical LAD occlusion in the de Winter's pattern, an isolated culprit lesion in the left main or the diagonal branch coronary artery has also been noted recently (Hirase, Wake, and Hirata 2020; Liu et al. 2022). Although urgent PCI is usually advocated, whether thrombolytic therapy is an effective strategy for de Winter syndrome remains controversial (Deng, Liu, and Song 2019; John et al. 2020; Raja et al. 2019; Rao et al. 2018; Xu et al. 2019). In addition, de Winter syndrome was initially thought to be static and persist until LAD revascularization. Whereas, several recent reports indicated that this ECG pattern might be an intermediate stage of myocardial ischemia and it could develop into STEMI or Wellens syndrome (He et al. 2020; Huang et al. 2022; John et al. 2020; Zhao, Wang, and Yi 2016; Zhu, Luo, and Huang 2021). Further investigation is needed to better understand de Winter syndrome. Therefore, we conducted this retrospective study to determine the coronary angiographic imaging features beyond LAD occlusion in patients with de Winter syndrome and see how they differed from acute anterior STEMI.

## 2 | Methods

This study adhered to the ethical guidelines of the Declaration of Helsinki and was approved by the Ethics Committee of the Fifth Affiliated Hospital of Sun Yat-sen University. Informed consent was waived due to the nature of the retrospective observational study. We reviewed the medical records of consecutive patients with acute myocardial infarction admitted to the Fifth Affiliated Hospital of Sun Yat-sen University from November 2018 to July 2023. Patients were included if they had at least one ECG that definitively fulfilled the aforementioned criteria for de Winter syndrome, which is characterized by a 1- to 3-mm upsloping ST-segment depression at the J-point in leads V1–V6, followed by a tall, positive-symmetric T-wave. The QRS complexes were usually not or only slightly widened, and in some, there was a loss of precordial R-wave progression. In most patients, there was a 1- to 2-mm ST-elevation in lead aVR (de Winter et al. 2008). Exclusion criteria were previous myocardial infarction and myocardial revascularization. Baseline characteristics, laboratory results, ECG, echocardiographic parameters, coronary angiographic features, and in-hospital adverse events of the enrolled patients were collected. STEMI patients whose culprit vessel was the LAD during the same period without de Winter ECG pattern were selected as controls after 1:2 matching for age and sex.

Coronary angiograms were independently assessed by two interventional cardiologists (J.C. and L.L.). The extent of coronary collateralization was classified visually using the four grades set by the Cohen-Rentrop Score (CRS): CRS 0, no visible filling of any collateral channels; CRS 1, collateral filling of branches of the vessel to be dilated without any dye reaching the epicardial segment of that vessel; CRS 2, partial collateral filling of the epicardial segment of the vessel being dilated; CRS 3, complete collateral filling of the vessel being dilated. A CRS $\geq 2$ described at least partial retrograde filling of the occluded coronary artery (Rentrop et al. 1985) (Summerer et al. 2021). Angiographic coronary thrombus burden was classified according to the thrombolysis in myocardial infarction (TIMI) thrombus grades: Grade 0, no thrombus present; Grade 1, possible thrombus present, with angiographic characteristics suggestive of thrombus but not diagnostic of thrombus; Grade 2, definite thrombus present, with largest dimensions $\leq 0.5$ the vessel diameter; Grade 3, definite thrombus present, with largest linear dimension $> 0.5$ but $< 2$ vessel diameters; Grade 4, definite thrombus present, with the largest dimension $\geq 2$ vessel diameters; Grade 5, total occlusion, thrombus size cannot be assessed. A TIMI thrombus grade $\geq 4$ was defined as a high thrombus burden, otherwise, it was considered as a low thrombus burden (Gibson et al. 2001). Follow-up was performed through a review of medical records and telephone contact with the patient.

Continuous variables are described as mean $\pm$ standard deviation (SD) or median (interquartile range [IQR]) and categorical variables as number (percentage). Depending on the distribution of the data, data were compared between two groups using unpaired Student's *t*-tests or Mann–Whitney *U* tests for continuous variables and Chi-square or Fisher exact tests for categorical variables. Statistical analyses were performed using SPSS software (version 25.0, SPSS, Chicago, IL). A two-sided $p$ value $< 0.05$ was considered statistically significant.

## 3 | Result

### 3.1 | Demographic and Clinical Characteristics of de Winter Syndrome Patients

A total of 12 patients (0.6%) with de Winter syndrome were included in the study from a screened cohort of 1865 acute myocardial infarction patients. Demographic and clinical characteristics of the de Winter syndrome patients are shown in Table 1. They were all male with a mean age of 49.0 (46.3–52.5) years. Three patients (3/12, 25%) had a known history of hypertension, 2 (2/12, 16.7%) had hyperlipidemia, and 2 (2/12, 16.7%) had diabetes. Five patients (5/12, 41.7%) were current smokers, and 2 (2/12, 16.7%) had a family history of coronary artery disease. All patients presented with angina on arrival, among whom 9 patients (9/12, 75%) had experienced chronic recurrent angina prior to hospitalization with the longest duration of angina being up to 24 months. Three patients (3/12, 25%) presented with Killip class IV. All underwent emergent PCI as soon as de Winter ECG pattern was identified. The median door-to-balloon time was 94.5 (70.0–123.3) minutes.

Cardiac troponin I (cTnI), creatine kinase isoenzyme MB (CK-MB), and N-terminal pro-brain natriuretic peptide (NT-pro BNP) were measured within 24 h after admission and monitored during hospitalization in all enrolled patients. All patients showed significant elevations in cTnI, CK-MB, and

**TABLE 1** | Demographic and clinical characteristics of the included de Winter syndrome patients.

| Variables | Patients with de Winter syndrome ($n=12$) |
|---|---|
| Age, year | 49 (46.3–52.5) |
| Sex, male, (*n*, %) | 12 (100.0%) |
| Preexisting comorbidities | |
| Hypertension, (*n*, %) | 3 (25.0%) |
| Diabetes mellitus, (*n*, %) | 2 (16.7%) |
| Hyperlipidemia, (*n*, %) | 2 (16.7%) |
| Family history of CAD, (*n*, %) | 2 (16.7%) |
| Current smoker, (n, %) | 5 (41.7%) |
| History of recurrent angina, (*n*, %) | 9 (75.0%) |
| Duration of chronic recurrent angina, month | 3.5 (0.1, 10.5) |
| Symptom upon arrival | |
| Angina, (*n*, %) | 12 (100%) |
| Dyspnea, (*n*, %) | 5 (41.7%) |
| Killip class | 1 (1, 3.3) |
| Cardiac markers | |
| Peak cTnI over 500 times the upper limit of normal, (n, %) | 8 (66.7%) |
| Peak CK-MB, U/L | 220 (95.7–459.7) |
| NT-pro BNP, pg/mL | 1110.0 (682.5–1890.0) |
| Lipid profiles | |
| TC, mmol/L | 5.2 ± 1.3 |
| LDL-C, mmol/L | 3.6 ± 1.3 |
| HDL-C, mmol/L | 1.1 ± 0.2 |
| TG, mmol/L | 1.8 ± 0.8 |
| Echocardiographic parameters[a] | |
| Left atrial diameter, mm | 33.2 ± 2.5 |
| LVEDD, mm | 49.2 ± 4.8 |
| RWMA, (*n*, %) | 9 (81.8) |
| LVEF, % | 61.6 ± 12.5 |

Abbreviations: CAD, coronary artery disease; CK-MB, creatine kinase isoenzyme MB; cTnI, cardiac troponin I; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; NT-pro BNP, N-Terminal pro-Brain Natriuretic Peptide; RWMA, regional wall motion abnormalities; TC, total cholesterol; TG, triglycerides.
[a]Data were available in 11 cases with de Winter syndrome.

NT-pro BNP. Eight patients (8/12, 66.7%) had peak cTnI levels over 500 times the upper limit of normal. The mean NT-pro BNP was 1110.0 (682.5–1890.0) pg/mL (reference value, < 125 pg/mL).

All the de Winter pattern ECGs were obtained within 2 h after the onset of chest pain. All patients showed an upsloping ST-segment depression followed by a high, positive-symmetric T-wave in the precordial leads. A few patients (2/12, 16.7%) showed widened QRS complexes, while half (6/12, 50%) had poor precordial R-wave progression. Six (6/12, 50%) had ST elevation in lead aVR and 5 (5/12, 41.7%) had ST elevation in lead V1. A representative example of de Winter ECG pattern is depicted in Figure 1a. As all patients received emergency PCI as soon as the de Winter ECG pattern was identified, we did not capture the dynamic ECG evolution to STEMI before PCI in any of them. However, 10 (10/12, 83.3%) of them developed pathological Q waves, R wave loss, and/or T wave inversions in the precordial leads when de Winter ECG pattern in two patients (2/12, 16.7%) resolved after primary PCI. A total of 11 patients underwent echocardiography within 48 h after emergency PCI, of whom nine (9/11, 81.8%) showed regional wall motion abnormality

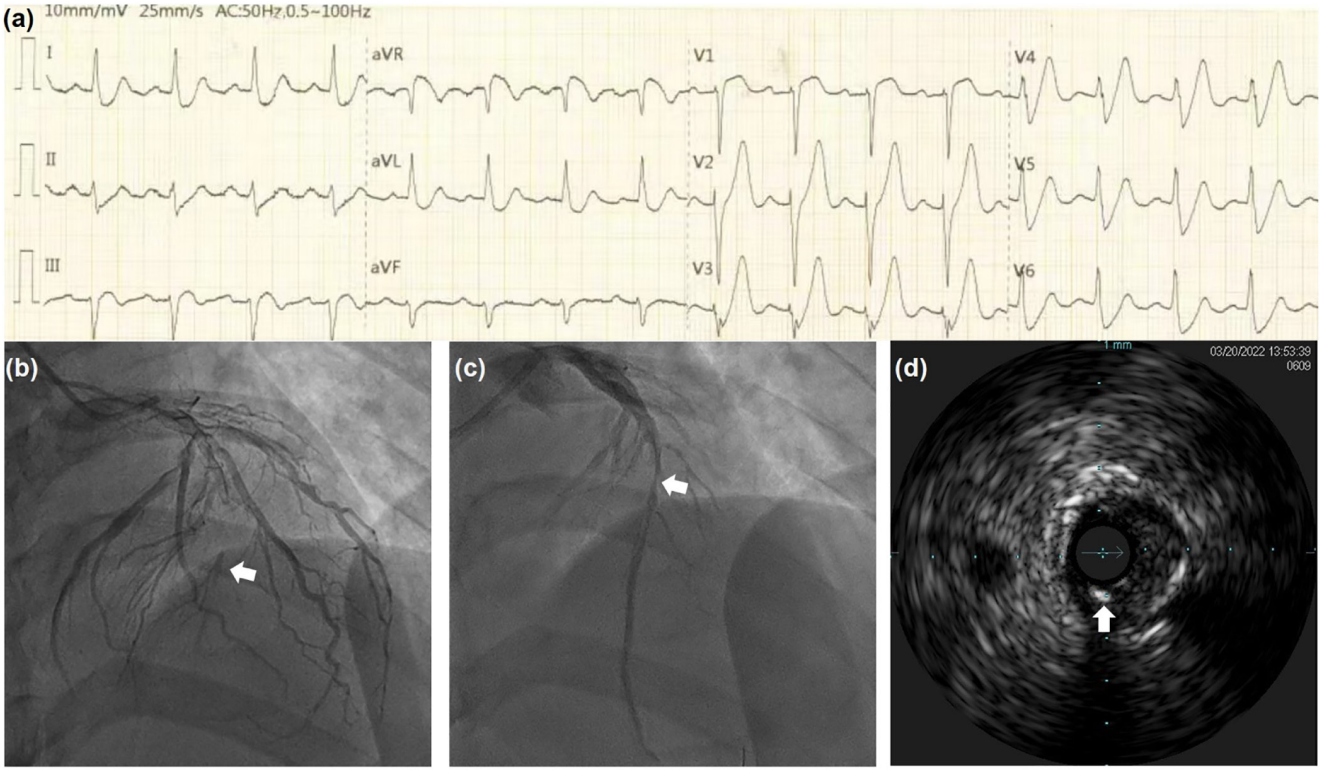


**FIGURE 1** | Representative ECG and coronary angiography of de Winter syndrome patients in this study. (a) The de Winter ECG pattern of a typical eligible patient in the study. (b) Evaluation of coronary collateralization to the occluded left anterior descending coronary artery (LAD) shows a Cohen-Rentrop Score of 2. (c) A coronary stenosis of 90% with a low thrombus burden (TIMI thrombus grade 1) is detected in the middle segment of the LAD. (d) Intravascular ultrasonic imaging of the culprit lesion in LAD shows an atherosclerotic plaque with a calcified nodule (white arrow). The minimum lumen area was 2.17 $mm^2$ and the plaque burden was 67.9%.

(RWMA). The average LVEF was 61.6% ± 12.5%, the mean left atrial diameter was 33.2 ± 2.5 mm and the left ventricular end-diastolic diameter (LVEDD) was 49.2 ± 4.8 mm.

### 3.2 | Angiographic and Procedural Characteristics of de Winter Syndrome Patients

Table 2 shows the angiographic and procedural characteristics of de Winter syndrome patients. A right-dominant coronary pattern was found in nine patients (9/12, 75%). Seven patients (7/12, 58.3%) had the culprit lesion located in the proximal LAD, three (3/12, 25%) in the middle LAD, one in the left main artery, and the other in the ramus intermedius artery. Meanwhile, seven patients (7/12, 58.3%) had at least two- or three-vessel disease. All patients had a TIMI thrombus grade <4 in the culprit lesions, suggesting a low thrombus burden. Regarding collateral flow to the LAD, all patients had a CRS ≤2, indicating poorly developed retrograde filling of the occluded coronary artery. Moreover, all patients had a residual coronary stenosis ≥80% after pretreatment of the culprit lesions. Data of intracoronary imaging were available in four patients and all showed severe fixed atherosclerotic stenosis. Stent implantation was performed in all the 12 patients with de Winter syndrome. Figure 1b–d shows poor coronary collateralization, severe coronary stenosis with low thrombus burden, and obvious atherosclerotic plaque of the infarct-related LAD in a patient with de Winter syndrome, respectively.

### 3.3 | Comparison Between de Winter Syndrome and STEMI Patients With Occluded LAD

Twenty-four age- and sex-matched STEMI patients without de Winter ECG pattern who had the infarct-related artery located in the LAD were included as controls. As shown in Table 3, de Winter syndrome patients had a higher prevalence of prior recurrent angina (75.0% vs. 37.5%, $p = 0.034$), but similar D2B time when compared with STEMI patients (94.5 min vs. 75.5 min, $p = 0.056$). In terms of angiographic characteristics, de Winter syndrome patients had better coronary collateralization (CRS grade: 1 vs. 0, $p = 0.001$), more severe residual stenosis after pretreatment (90% vs. 60%, $p < 0.001$) but lower thrombus burden (TIMI grade: 1 vs. 2, $p = 0.005$) than STEMI patients. Besides, they had similar left atrial diameter, LVEDD, LVEF, and prevalence of RWMA to STEMI patients.

### 3.4 | Complications and Follow-Up of de Winter Syndrome Patients

During hospitalization, four patients (4/12, 33.3%) developed malignant arrhythmias, including three with ventricular fibrillation and one with sympathetic electrical storm. Three patients developed cardiogenic shock. Two patients (2/12, 16.7%) suffered sudden cardiac arrest and underwent successful cardiopulmonary resuscitation. No complications such as cardiac rupture, embolism, ventricular aneurysm, or papillary muscle rupture occurred

**TABLE 2** | Angiographic and procedural characteristics of the included de Winter syndrome patients.

| Variables | Patients with de Winter syndrome ($n=12$) |
|---|---|
| Coronary artery characteristic | |
| Dominant RCA, (*n*, %) | 9 (75%) |
| Two- or three-vessel disease, (*n*, %) | 7 (58.3%) |
| Culprit lesion | |
| Culprit location in proximal LAD, (*n*, %) | 7 (58.3%) |
| Culprit location in middle LAD, (*n*, %) | 3 (25%) |
| Culprit location in left main coronary artery, (*n*, %) | 1 (8.3%) |
| Culprit location in ramus intermedius artery, (*n*, %) | 1 (8.3%) |
| The degree of stenosis in culprit vessel, % | 100 (100–100) |
| TIMI grade of thrombus burden | 1 (1–1) |
| Pre-PCI TIMI flow | 0 (0–0) |
| CRS grade of collateral circulation | 1 (1–2) |
| Interventional therapy | |
| Primary PCI | 12 (100%) |
| Residual stenosis after pretreatment, % | 90 (80–90) |
| Stent implantation, *n* (%) | 12 (100%) |
| Coronary no-reflow | 2 (16.7%) |

Abbreviations: CRS, Cohen-Rentrop Score; LAD, left anterior descending artery; PCI, percutaneous coronary intervention; RCA, right coronary artery; TIMI, Thrombolysis in myocardial infarction.

in the included de Winter syndrome patients during hospitalization. All the patients were eventually discharged in good condition. The average length of hospital stay was 7.0 (5.3–8.8) days.

The median follow-up of these patients was 14.0 (6.0–21.0) months. Although no patients were readmitted for cardiac events, two patients (2/12, 16.7%) developed chronic heart failure. Among the nine patients who underwent repeated echocardiography 6 months after discharge, two showed left ventricular dilatation (LVEDD was 63 mm and 57 mm, respectively) and one developed a ventricular aneurysm despite receiving standard medical therapy.

## 4 | Discussion

The current study focused on the patients with de Winter ECG pattern, an infrequent but potentially high-risk ECG pattern if not recognized in time. In this work, we had two main findings. First, de Winter syndrome was increasingly recognized as an equivalent sign of anterior STEMI, the door-to-balloon time in de Winter syndrome patients was similar to that in STEMI. Second, de Winter syndrome patients tended to have low thrombus burden, significant fixed stenosis, and poor coronary collateral circulation protection in the culprit lesion. Even so, the coronary collateral protection was better in patients with de Winter syndrome than in age- and sex-matched patients with acute anterior STEMI. Based on the previous reports, our study describes a more systematic and comprehensive clinical picture of de Winter syndrome. Also, to the best of our knowledge, this was the first study comparing the differences in coronary angiographic characteristics between de Winter syndrome and anterior STEMI.

Existing studies on de Winter syndrome are still scarce. After first formally naming this particular ECG pattern in a letter to the New England Journal of Medicine in 2008 (de Winter et al. 2008), Dr. de Winter's team added details of the largest study so far on de Winter syndrome in the following year (Verouden et al. 2009). They conducted a retrospective analysis of 1890 patients who underwent PCI for anterior myocardial infarction over a 10-year period, including 35 patients with de Winter ECG pattern. Similar to the findings of de Winter et al., the de Winter syndrome patients in our study were all male, about 50 years old, current smokers, and most commonly had a dominant right coronary artery, a culprit lesion located in the LAD or its major branches and insufficient collateral protection. Nevertheless, our study found that occlusion of the middle LAD, the left main artery, or the ramus intermedius artery could also lead to such ECG manifestations and that two- or more vessel disease was more common in de Winter syndrome, while De Winter et al.'s study suggested that the de Winter ECG pattern was present only in patients with proximal LAD stenosis and that single-vessel disease was more common. Our study additionally revealed that patients with de Winter syndrome had better collateral blood supply than the matched patients with STEMI. Patients with de Winter syndrome also had a higher incidence of chronic recurrent angina, which indicated that they might have undergone ischemic preconditioning of the myocardium prompting a better developed collateral circulation prior to acute occlusion. Coronary collaterals are interarterial connections that have the potential to remodel and expand in case of an epicardial coronary artery stenosis, providing an alternative source of blood supply to jeopardized myocardium (Meier et al. 2012). Our study supported that the extent of coronary collaterals might be one of the factors preventing ST-segment elevation, as the collateral circulation protection was better in de Winter than in STEMI. However, such collateral blood supply was not sufficient enough to preclude persistent severe myocardial transmural ischemia, so the de Winter pattern could evolve into STEMI in some cases.

Currently, evidence for thrombolytic therapy in de Winter syndrome is very limited. Several case reports of de Winter syndrome showed that patients could successfully recover with the resolution of chest pain and the disappearance of the de Winter's pattern on the ECG after thrombolysis (John et al. 2020; Rao et al. 2018; Xu et al. 2019). On the other hand, in a study by Xu et al. (2018), four of 15 patients with de Winter syndrome

**TABLE 3** | Comparison between de Winter syndrome and STEMI patients with occluded LAD.

| | de Winter syndrome ($n=12$) | STEMI ($n=24$) | $p$ |
|---|---|---|---|
| Age, year | 49.0 (46.3–52.5) | 49.5 (47.3–52.0) | 0.704 |
| Gender, male ($n$, %) | 12 (100.0) | 24 (100.0) | |
| History of chronic recurrent angina ($n$, %) | 9 (75.0) | 9 (37.5) | **0.034** |
| Door to balloon time, min | 94.5 (70.0–123.3) | 75.5 (57.3–84.0) | 0.056 |
| Total myocardial ischemia time, min | 180.0 (148.5–263.3) | 243.0 (154.5–331.3) | 0.261 |
| Angiographic characteristics | | | |
| RCA dominance ($n$, %) | 9 (75.0) | 16 (66.7) | 0.715 |
| Two or more vessel disease ($n$, %) | 7 (58.3) | 9 (37.5) | 0.236 |
| Collateral circulation to LAD[a] | | | |
| CRS | 1 (1–2) | 0 (0–0) | **0.001** |
| CRS ≥ 1 ($n$, %) | 9 (81.8) | 5 (22.7) | **0.007** |
| CRS ≥ 2 ($n$, %) | 4 (36.4) | 1 (4.5) | **0.042** |
| TIMI grade of thrombus burden | 1 (1–1) | 2 (1–3) | **0.005** |
| Pre-PCI TIMI flow | 0 (0–0) | 0 (0–0) | 0.758 |
| Residual stenosis after pretreatment, % | 90.0 (80.0–90.0) | 60.0 (50.0–70.0) | **<0.001** |
| Echocardiography within 48 h after admission[b] | | | |
| Left atrial diameter, mm | 33.2 ± 2.5 | 34.1 ± 3.8 | 0.474 |
| LVEDD, mm | 49.2 ± 4.8 | 47.5 ± 4.2 | 0.296 |
| RWMA ($n$, %) | 9 (81.8) | 20 (90.9) | 0.586 |
| LVEF, % | 61.6 ± 12.5 | 57.1 ± 9.2 | 0.249 |

*Note:* Bold value indicates a statistical difference between the two groups.
Abbreviations: CRS, Cohen-Rentrop Score; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; RCA, right coronary artery; RWMA, regional wall motion abnormalities; STEMI, ST-segment elevation myocardial infarction; TIMI, Thrombolysis in Myocardial Infarction.
[a]Data available in 11 cases with de Winter syndrome and 22 cases with STEMI.
[b]Data available in 11 cases with de Winter syndrome and 22 cases with STEMI.

received thrombolytic therapy after they evolved to ST-segment elevation. Among them, failed thrombolysis occurred in two patients and one patient had re-occlusion following successful thrombolysis. Likewise, another study including 14 patients with de Winter syndrome reported that the two patients who underwent thrombolytic therapy both failed (Liu et al. 2020). Our study showed that de Winter syndrome patients had a very low thrombus burden but severe fixed stenosis in the culprit artery, suggesting that patients might respond poorly to thrombolysis and that PCI would be preferable.

Otherwise, although the median door-to-balloon time for de Winter syndrome patients and that for anterior STEMI in our study were not statistically different, there was a trend toward longer door-to-balloon time for de Winter syndrome. A few patients with de Winter ECG pattern experienced malignant ventricular arrhythmias and cardiogenic shock, or later progressed to chronic heart failure. This denoted that de Winter ECG pattern could be a type of fatal myocardial infarction, whereas such an ECG pattern was still under-recognized among noncardiac specialists, for example, the emergency department physicians, while they may see patients suffering from acute chest pain with underlying de Winter's pattern more often in their daily work. Next, we need to do more to further improve understanding of such an ECG pattern among the relevant clinicians involved in the management of patients presented with acute chest pain, so that earlier recognition of ongoing severe myocardial ischemia and earlier reperfusion therapy can be achieved.

### 4.1 | Limitations

There are several limitations to this study. First, it was a retrospective study with a small sample size. Despite screening a cohort of 1865 patients with acute myocardial infarction over 5 years, we had only included 12 patients because of the rare occurrence of de Winter syndrome. Further multicenter studies are therefore needed to confirm these results in larger populations. Second, some diseases that we did not include in this study, such as type A aortic dissection, severe hyperkalemia, myocarditis, and vasospastic angina could also present with de Winter ECG pattern (Ando et al. 2020; García-Izquierdo et al. 2018; Walker, Vivekanantham, and Graf 2021; Zhang

et al. 2022). Such situations are also what we need to keep in the differential diagnosis list as well when coping with de Winter ECG pattern.

## 5 | Conclusions

De Winter syndrome is an anterior STEMI equivalent with the infarct-related artery mostly located in the LAD. De Winter syndrome patients tend to have a low thrombus burden, severe fixed stenosis, and poorly developed collateral circulation of the culprit lesion. Currently, we still need to further improve our understanding of such an ECG pattern to facilitate earlier identification of ongoing severe myocardial ischemia and earlier reperfusion therapy (PCI would be preferable).

### Author Contributions

Jian Chen, Liyun Luo and Wenyi Tang contributed to the study conception and design. Junwei Xu, Fangyuan Cheng, Tianmin Liu, Zijian Lin and Bairong Chen collected and analyzed the data. Jian Chen and Liyun Luo assessed the coronary angiograms. Wenyi Tang, Junwei Xu, Liyun Luo and Jian Chen wrote and revised the manuscript. All co-authors read and approved the final version of the manuscript.

### Ethics Statement

This study adhered to the ethical guidelines of the Declaration of Helsinki and was approved by the Ethics Committee of the Fifth Affiliated Hospital of Sun Yat-sen University.

### Consent

Informed consent was waived due to the nature of the retrospective observational study.

### Conflicts of Interest

The authors declare no conflicts of interest.

### Data Availability Statement

Data are available from the corresponding authors upon reasonable request.

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