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CASE REPORT

Evolutionary de Winter pattern: from STEMI to de Winter ECG—a case report

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Abstract

The de Winter electrocardiography (ECG) pattern is a sign that implies proximal left anterior descending coronary artery occlusion in patients with chest pain. We report a case of a 34-year-old man with a history of smoking who presented to the local emergency department with a 49 min history of chest pain. The first ECG of the patient indicated that ST-segment elevation was noted in the lead V2–V4; 57 min later, a second ECG revealed a typical de Winter syndrome when the patient was transferred to the emergency chest pain centre of our hospital. A percutaneous coronary intervention (PCI) was performed approximately 8 h later because the patient initially refused the PCI. Acute coronary artery angiography showed that the proximal left anterior descending coronary artery was completely occluded. Our case suggests that ST-segment elevation myocardial infarction may evolve in the direction of de Winter, which reflects a coronary thrombus in formation, so the de Winter ECG pattern should not be considered static.

Keywords de Winter syndrome; Acute myocardial infarction; Percutaneous coronary intervention; Prognosis

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Introduction

The de Winter sign is a rare electrocardiogram (ECG) manifestation of proximal LAD occlusion. It has become increasingly recognized as an ST-segment elevation myocardial infarction equivalent pattern due to proximal left anterior descending (pLAD) artery occlusion.[1] The previous view was that the de Winter ECG pattern is static. We show that ST-segment elevation myocardial infarction can evolve into the de Winter pattern.

Case presentation

Chief complaints

A 34-year-old male farmer presented with sudden paroxysmal pain in the precardiac area. He had no complaint of abdominal pain, bloody stools, or weight loss.

History of present illness

The patient's symptoms started at 22:30 on 10 May 2021. The patient came to the local emergency department (ED) 49 min later (23:19), when he was referred to our ED at 00:15 on 11 May 2021 and alleviated.

Medical history

The patient had a history of haemorrhoids and no family history of coronary heart disease. He was a current smoker with a smoking history of 18 package-years. No alcohol addiction.

Physical examination

The patient's temperature was 36.5°C, heart rate was 70 bpm, respiratory rate was 18 breaths per minute, and blood pressure was 134/89 mmHg. The S1 and S2 were normal and there was no evidence of a heart murmur on cardiac examination.

Figure 1 The electrocardiogram (ECG) shows ST-segment elevation from V2 to V4 leads.

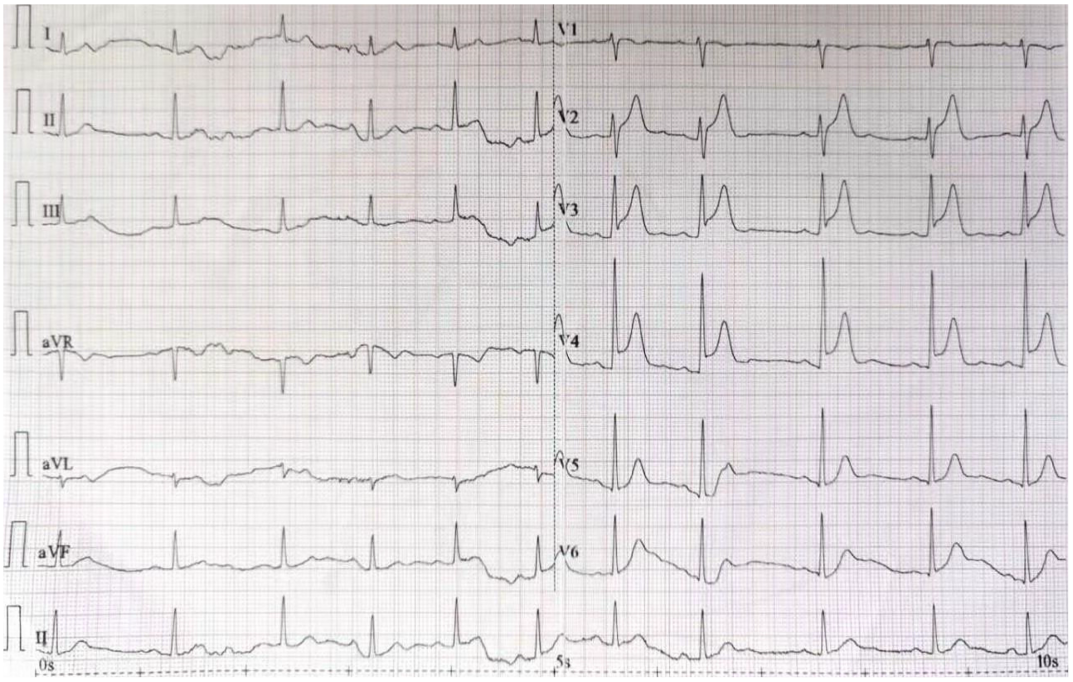
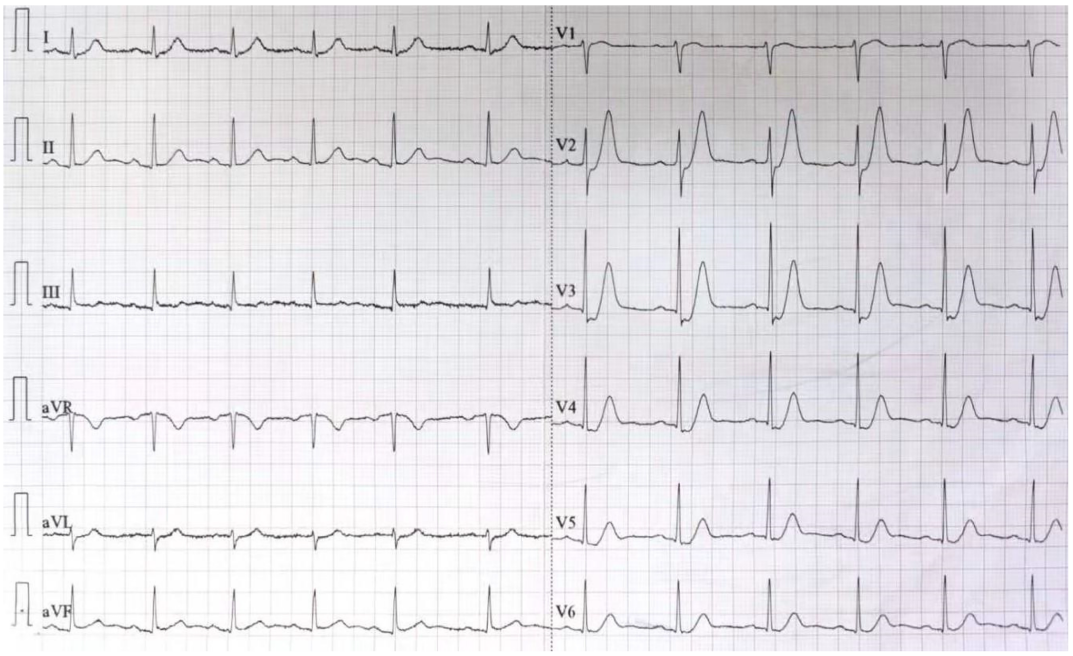


Figure 2 The electrocardiogram (ECG) shows upsloping ST-segment depression at the J-point with tall and symmetrical T waves from V2 to V4 leads.



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Laboratory examinations

His emergency cardiac troponin T (cTnT) level was 47 ng/L (normal value, <40 ng/L), BNP was 124.50 ng/L (normal value: <100), CK-MB was 44.0 U/L (normal value: 0–24), and serum creatinine was 72 μmol/L (normal value: 57–111) (Supporting Information, *Figures S3* and *S4*).

Imaging examinations and management

The first ECG (*Figure 1*), performed immediately in the local ED, showed an acute anterior interwall myocardial infarction. Another ECG (*Figure 2*) was performed 57 min later, showing a typical de Winter syndrome ECG pattern. After admission, the patient was then given a loading dose of aspirin and clopidogrel (300 and 300 mg) for antiplatelet treatment. A percutaneous coronary intervention (PCI) was performed approximately 8 h later and another clopidogrel 300 mg was added, when the patient agreed with the reperfusion treatment. Emergency coronary angiography demonstrated complete occlusion of the pLAD artery after giving off the first diagonal branch and anterior interventricular septum branches [*Figure 3(A)*], and 30% stenosis in the middle segment of the left circumflex artery [*Figure 4(A)*], and about 65% stenosis in the proximal section of the right coronary artery [*Figure 4(B)*]. PCI of LAD was performed after consultation with family members [*Figure 3(B)*]. Echocardiography after PCI indicated myocardial ischaemia below the middle segment of the left anterior septum, but the ventricular ejection fraction was normal. No complications occurred in the hospital. Five days after the operation, the patient was

Figure 3 Coronary artery angiography (CAG) reveals complete occlusion (arrow) of the proximal left anterior descending (pLAD) artery after giving off the first diagonal branch (A). Repeat coronary artery angiography (CAG) after the left anterior descending percutaneous coronary intervention (PCI) (B).

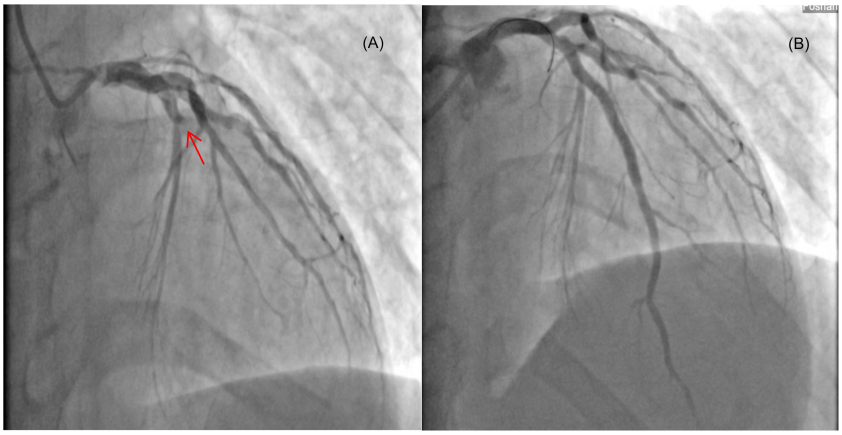
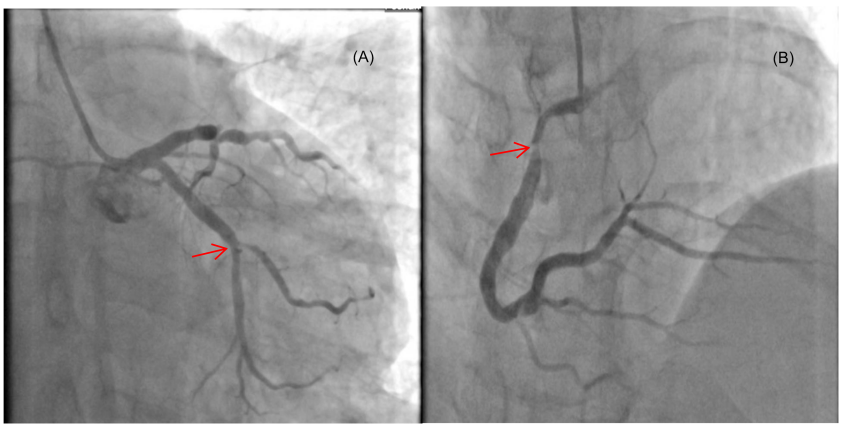


Figure 4 Coronary artery angiography (CAG) reveals 30% stenosis in the middle segment of the left circumflex artery (LCA) (A) and about 65% stenosis in the proximal section of the right coronary artery (RCA) (B).



discharged free of chest pain and the ECG showed persistent negative T waves in V1 to V4 (Supporting Information, *Figure S1*), consistent with the ECG changes after myocardial infarction.

Discussion

The so-called de Winter sign has become increasingly recognized as an ST-segment elevation myocardial infarction equivalent pattern due to pLAD artery occlusion. The diagnostic criteria for de Winter syndrome include (i) a 1 to 3 mm upsloping ST-segment depression at the J point in leads V1 to V6 that continues into tall, positive symmetrical T waves; (ii) QRS complex usually not wide or only slightly widened; (iii) in some patients, a loss of precordial R wave progression; and (iv) a 1 to 2 mm ST-segment elevation in aVR.[1,2]

Different from the static ECG pattern of de Winter syndrome proposed by de Winter *et al.*,[1] our case demonstrated dynamic ST-segment changes. The patient initially showed ST-segment elevation from V2 to V4 leads and then transformed from STEMI to de Winter syndrome after 57 min. A complete occlusion of LAD was confirmed by coronary angiography. This situation has also been reported in the previous literature.[3,4] And interestingly, the emergency cTnT examinations showed that when the ECG pattern of de Winter syndrome had been recorded, the troponin T level was only slightly increased. We consider that the ECG pattern of STEMI changes to the de Winter syndrome owing to autolysis of the intracoronary thrombus and the de Winter ECG pattern reflects a coronary thrombus in formation. This ECG pattern is transient and progresses to STEMI when the thrombus continues to form and completely occludes the coronary arteries.[4] So, the de Winter ECG pattern should not be considered static; the lack of attention and recognition of this special pattern would significantly increase in reperfusion time and various clinical adverse events. The de Winter syndrome should be considered as a high-risk STEMI, for which emergency revascularization is strongly recommended within the time window of intervention following the current guidelines.[5]

Conclusions

As is well known, de Winter syndrome reflects a coronary thrombus in formation, so the de Winter ECG pattern should not be considered static and ST-segment elevation myocardial infarction can evolve into the de Winter pattern.

Conflict of interest

None declared.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Discharge ECG.
Figure S2. Postoperative ECG.
Figure S3. Emergency laboratory examinations.c
Figure S4. Emergency cardiac troponin T.

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