



The de Winter ECG pattern occurred after ST-segment elevation in a patient with chest pain

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Case description

A previously healthy 58-year-old male hospital staff member presented to the emergency department (ED) with chest pain, which had started several minutes ago. He was haemodynamically stable with no clinical evidence of heart failure. The first electrocardiogram (ECG) taken 6 min after arrival showed ST-segment elevation in leads I, aVL and V2–6, with ST depression and T wave inversion in lead III, suggestive of an acute anterolateral myocardial infarction (MI) (Fig. 1a). He was treated with aspirin, ticagrelor, nitroglycerin sprays, intravenous morphine and metoclopramide in the ED with relief of chest pain. The initial high-sensitivity cardiac troponin-T level was not elevated. The second ECG taken 28 min after arrival revealed upsloping ST-segment depression > 1 mm at the J point and tall, symmetrical T wave in leads V1–V4, which was compatible with the de Winter ECG pattern (Fig. 1b). The patient underwent transradial coronary arteriography 55 min after the second ECG, which showed total occlusion of the mid left anterior descending artery (LAD) with thrombolysis in myocardial infarction grade 0 coronary flow and no collateral filling (Rentrop class 0) (Fig. 1c). Thrombectomy, followed by insertion of a drug-eluting stent, was performed uneventfully (Fig. 1d).

Discussion

The de Winter ECG pattern, characterized by 1–3-mm upsloping ST-segment depression at the J point in leads V1 to V6 that continues to tall positive symmetrical T waves, is identified in only 2% of the patients with acute MI [1]. It is regarded as a ‘ST-elevation myocardial infarction (STEMI) equivalent’ because it is highly predictive for acute LAD occlusion [2]. This pattern was originally described by de Winter et al. as static, which lasted from the time of the first ECG until procedural ECG and angiographic confirmation of LAD occlusion [1]. However, several reports have emerged showing that this pattern can be temporary. Zhao et al. observe the de Winter pattern after ST-segment elevation in a case of total proximal LAD occlusion, which coincided with spontaneous coronary recanalization [3]. Fiol Sala et al. report a case of the de Winter pattern following ST-segment elevation during thrombolytic therapy [4]. Ayer et al. demonstrate the de Winter pattern temporarily at the time of percutaneous coronary intervention (PCI), with ST-segment elevation evident both before and after reperfusion [5]. Our case is interesting because the ST-segment elevation in the presenting ECG evolved spontaneously to the de Winter pattern before any coronary intervention, indicating that such an evolution does not necessarily coincide with reperfusion.

So far, the electrophysiological mechanism of such ECG changes has remained unknown. De Winter postulates that the absence of ST-segment elevation might be related to ischaemic ATP depletion, leading to the lack of activation of sarcolemmal ATP-sensitive potassium (K_{ATP}) channels [1]. However, it cannot explain the dynamic transition between ST-segment elevation and the de Winter pattern when ATP is depleted in ongoing ischaemia. Some authors believe that the de Winter pattern corresponds to critical subocclusion of LAD, causing subendocardial ischaemia and ST-segment depression, which ultimately progresses to total occlusion, producing ST-segment elevation [3, 4].

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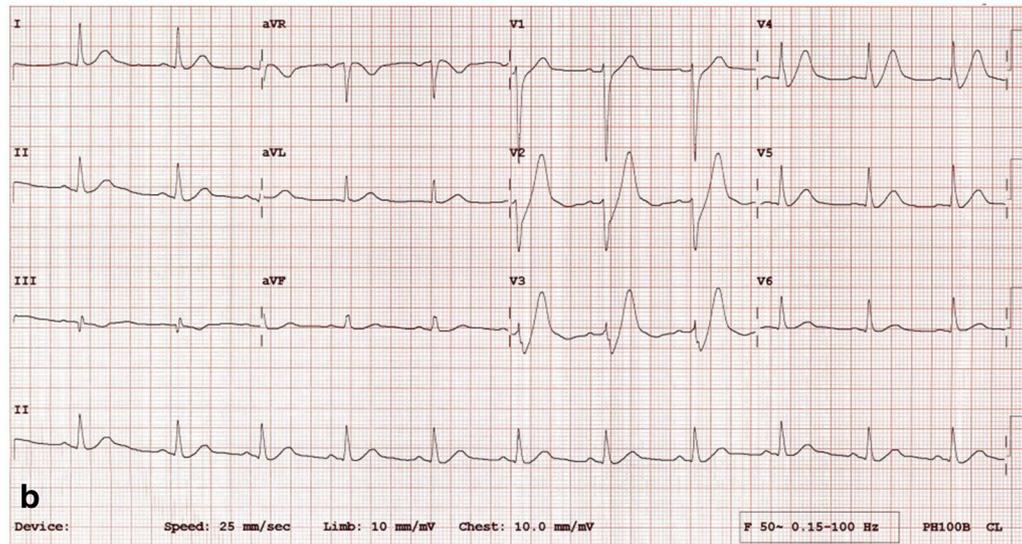
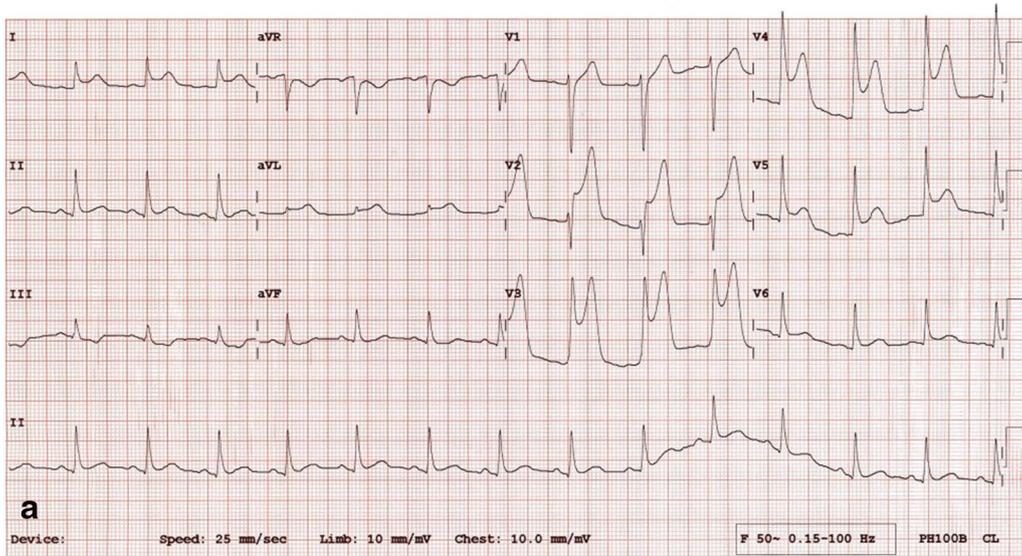


Fig. 1 **a** Presenting ECG showing anterolateral ST-elevation myocardial infarction. **b** A repeat ECG showing upsloping ST-segment depression and tall, symmetrical T wave in leads V1–V4. **c** Cardiac catheterization showing the mid LAD before stent placement. **d** Cardiac catheterization showing the mid LAD after thrombectomy and stent placement

However, de Winter et al. do not identify any association between this ECG pattern and coronary angiographic characteristics, and there was considerable loss of myocardium despite successful PCI, signifying ischaemia beyond subendocardium [1]. In our case, although real-time correlation of ECG changes with angiographic characteristics was not feasible, we believe that an alternation in the severity of occlusion within such a short timeframe was unlikely the cause of the ECG changes.

This case demonstrates that the de Winter pattern might be a temporary phenomenon in the trajectory of STEMI. Should there have been any delay in patient presentation or ECG recording, the de Winter pattern could have been the first ECG feature in our case. Given the stable haemodynamic status, absence of heart failure, relief of chest pain after treatment, and a normal initial troponin assay, many clinicians might have treated the patient with ST depression in the presenting ECG as unstable angina conservatively, especially during night time, weekends and holidays when access to cardiac catheterization is less readily available. In that case, the opportunity of treating the total LAD occlusion with primary PCI could have been missed. Therefore, it is important for emergency physicians to recognize the de Winter pattern as a STEMI equivalent and proceed to the cardiac catheterization laboratory without delay.

Author contributions Cheung ACK, Wai AKC and Tse TS treated the patient and recorded clinical findings. Wong RTM performed literature review. Lam RPK and Cheung ACK wrote the first draft of the manuscript. Wai AKC, Wong RTM and Tse TS critically reviewed the manuscript and revised it. Tse TS provided the pictures of coronary angiogram.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Statement of human and animal rights The authors indicate that all procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal subject was involved in this study.

Informed consent A written informed consent for publication was obtained from the patient. We declare that no work resembling the enclosed article has been published or is being submitted for publication elsewhere. We certify that we have each made a substantial contribution so as to qualify for authorship and that we have reviewed and approved the content of the article. No portion of the text has been copied from other material in the literature.

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