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Case report

De Winter ST-T syndrome: an early sign of ST segment elevation myocardial infarction

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Abstract

A ST elevation myocardial infarction (STEMI) equivalent electrocardiogram (ECG) pattern reflects an acute thrombotic occlusion of a large epicardial coronary artery without ST-segment elevation. In recent studies, one of these STEMI equivalents, is de-Winter T waves defined by an upsloping ST segment depression (> 1mm) beginning from J-point, and symmetrical, long and significant T waves seen in precordial leads. This syndrome is highly predictive for acute proximal left anterior descending artery (LAD) occlusion. We reported the case of a 45-year-old patient suffering from acute chest pain, presenting in our emergency department with a de Winter" ECG pattern which had evolved in few minutes, after cardiac arrest, into an anterior STEMI. Reperfusion therapy was successful. Prompt recognition of this atypical ECG pattern can ensure immediate detection of evolvement to a STEMI and therefore provide appropriate emergent reperfusion therapy either with percutaneous coronary intervention (PCI) or fibrinolysis if the PCI is not available.

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Introduction

Electrocardiogram (ECG) is an essential tool for the diagnosis of acute myocardial ischemia (MI) and the evaluation of evolving myocardial infarction in the emergency departments (ED). ECG can distinguish between patients with an ST-segment elevation myocardial infarction (STEMI) and those with a non-STEMI, which can guide management along with biomarkers of myocardial necrosis. Although several patients with acute MI caused by occlusion of the epicardial coronary artery do not present with ST-elevation on ECG, they may present with other ECG abnormalities known as STEMI equivalents. The de Winter sign is a STEMI equivalent that was first reported in 2008. It is characterized by an upsloping ST-segment depression at the J-point in leads V1-V6 that continues into a tall positive symmetrical T wave, which signifies occlusion of the proximal LAD artery [1]. This pattern was originally described by de Winter *et al*. [2] as a static sign, but several reports have emerged showing that it can be temporary with dynamic changes which should be detected by repetitive ECGs in order to meet indications for thrombolytic therapy especially if PCI is not available. We herein present a case of de Winter syndrome which evolved into a STEMI, and treated successfully with fibrinolysis in the ED.

Patient and observation

A 45-year-old male presented to the ED with retrosternal chest pain radiating to left arm within the first hour. He had a history of smoking (15 Pack per year) and penicillin allergy. The examination in the triage area showed a patient who was hemodynamically stable with no clinical evidence of heart failure. He scored his pain as 10/10 according to the Numeric Pain Scale (NPS). The first ECG taken 5 min after arrival showed a sinus rhythm at a rate of 83 bpm, ST-segment elevation in right leads (V1, V2, V3R, V4R), 3mm upsloping ST segment depression in leads V3-V6 at the J point which continued into tall, positive symmetrical T-waves and 1.5mm ST-segment depression in leads II, III, aVF, V7-9 (Figure 1). The patient was categorised on a very urgent level of priority and was immediately oriented to the emergency room. During the first assessment, the patient lost consciousness and had seizures. He presented a cardiac arrest with a ventricular fibrillation on the monitor. An immediate shock was delivered. Vital signs after resuscitation were stable: a respiratory rate of 22 breaths/min and the oxygen saturation was 99% on air room, a blood pressure of 120/60 mmHg and a regular pulse rate of 100 beats/min, a Glasgow Coma Scale of 14/15 without any neurological impairment, a pain score of 8/10 according to the NPS.

The ECG performed after the cardiac arrest recovery showed a sinus rhythm at a rate of 100 bpm, ST-segment elevation in leads I, aVL and V1-3, with ST depression in leads II III, aVF and V7-9 (Figure 2). The diagnosis of a STEMI in the anterior territory was made. He was treated with aspirin, clopidogrel, intravenous bolus of low molecular-weight heparin followed by the subcutaneous dose and intravenous titration of morphine according to the pain statement. Reperfusion strategy chosen was fibrinolysis by tenecteplase, as primary PCI was not available and after eliminating the contraindications. Thrombolytic therapy went without any incident and was successful. ECG performed 60 min after administration of the thrombolytic agent revealed ST segment back to baseline (Figure 3). The patient was admitted to the ED for 24 hours then transferred to a cardiac department where a coronary angiography was performed. It showed a subtotal occlusive stenosis in mid-LAD artery (90-99%), a tight stenosis in proximal LAD artery (70-90%) and a significant stenosis in mid circumflex artery (50-70%) (Figure 4, Figure 5). Successful coronary angioplasty was done using a "kissing ballon" on the mid-LAD artery meanwhile a second-generation drug-eluting stent was implanted in the proximal LAD artery. The patient was discharged 24 hours after with favourable outcome.

Discussion

The de Winter ECG pattern, characterized by 1-3mm 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 [3]. It was observed that patients with this ECG pattern were relatively young, predominantly male and had a higher incidence of dyslipidemia compared to patients classical STEMI on ECG [4]. This specific ECG pattern was considered static and persistent from the time of initial recording at symptom presentation up to instant revascularization. However, several studies have showed that this pattern can be temporary with different forms of evolvement [1,3]. De Winter sign was recognized as a STEMI equivalent by Rokos *et al*. [5] in 2010 because it is highly predictive for acute LAD occlusion and required emergent reperfusion therapy with fibrinolysis or a primary PCI. Aihua Wang´s study is the first to report patients with the de Winter ECG pattern who received thrombolytic therapy following the identification of an ST-segment elevation [6]. Fiol Sala *et al*. [7] believed that the stage of de Winter syndrome is earlier than the STEMI hyper acute period, which is equivalent to the change of STEMI hyper acute period. Although, there are two clinical classifications of de Winter syndrome ECG, most of which are resting ECG and basically will not evolve into STEMI dynamic evolution; but a small part are dynamic ECG and evolved into STEMI. In our case, we were dealing with the dynamic form of de Winter syndrome which could benefited from thrombolytic therapy and recovered well with normal left ventricular function, without manifestations of heart failure during follow-up.

Conclusion

According to guidelines for the diagnosis and management of STEMI, they do not recommend fibrinolysis in de Winter ECG pattern. Although, it is considered to be a STEMI equivalent, thrombolytic therapy still targeted at the acute STEMI. Therefore, repetitive ECGs monitoring is necessary for this type of patients in order to track down the moment of ST elevation, especially when catheterization laboratory is not available for PCI.

Competing interests

The authors declare no competing interests.

Authors' contributions

All the authors have read and agreed to the final manuscript.

Figures

Figure 1: first ECG (de Winter complex showed by arrows)

Figure 2: ECG after cardiac arrest recovery (ST-segment elevation showed by arrows)

Figure 3: ECG at 60 min after thrombolytic therapy administration

Figure 4: coronary stenosis in LAD and circumflex arteries

Figure 5: subtotal occlusive stenosis in mid-LAD artery

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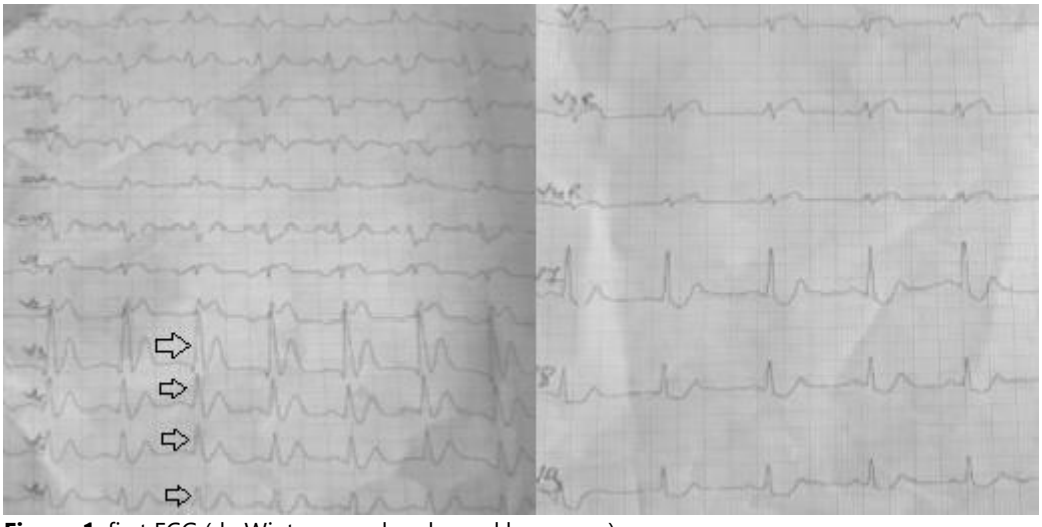

Figure 1: first ECG (de Winter complex showed by arrows)

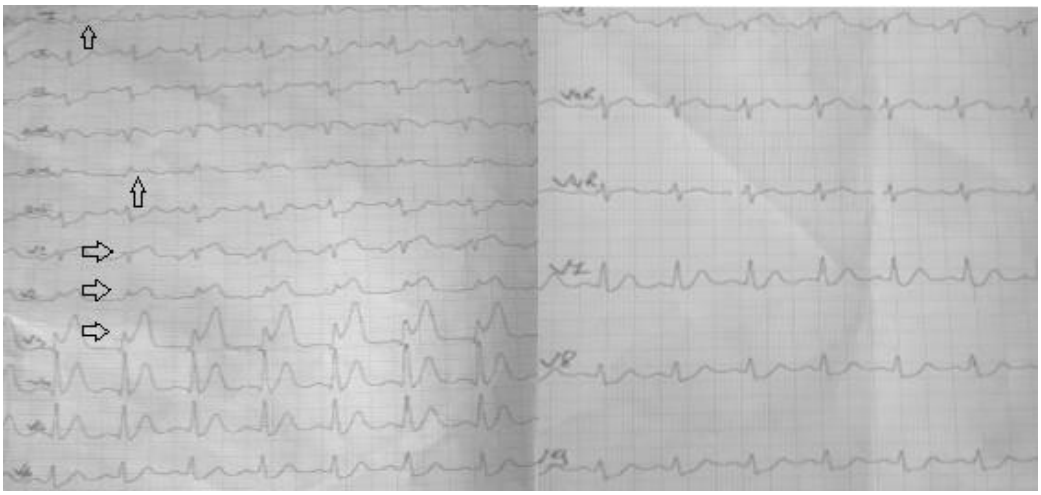

Figure 2: ECG after cardiac arrest recovery (ST-segment elevation showed by arrows)

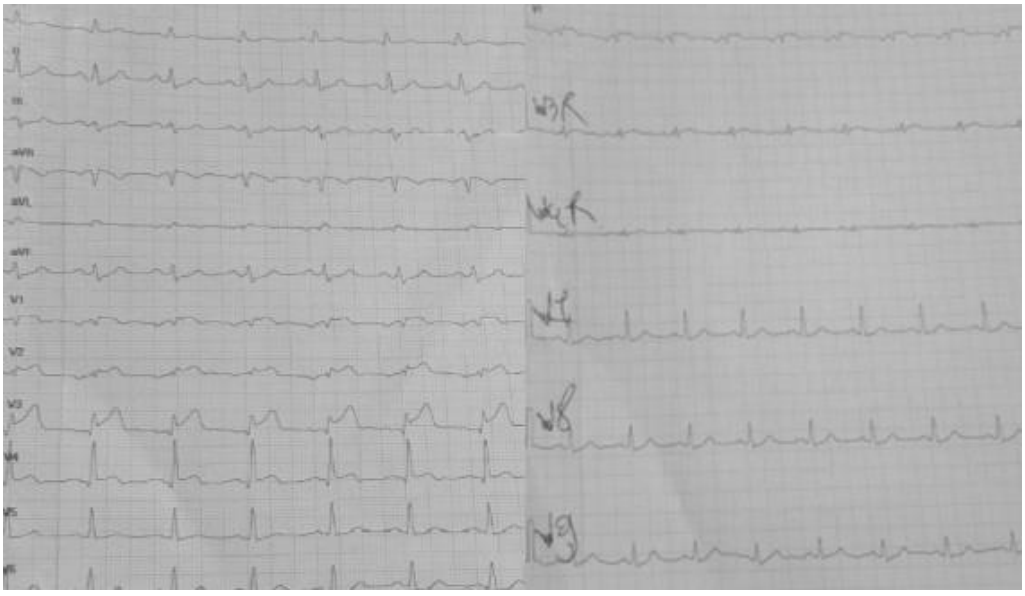

Figure 3: ECG at 60 min after thrombolytic therapy administration

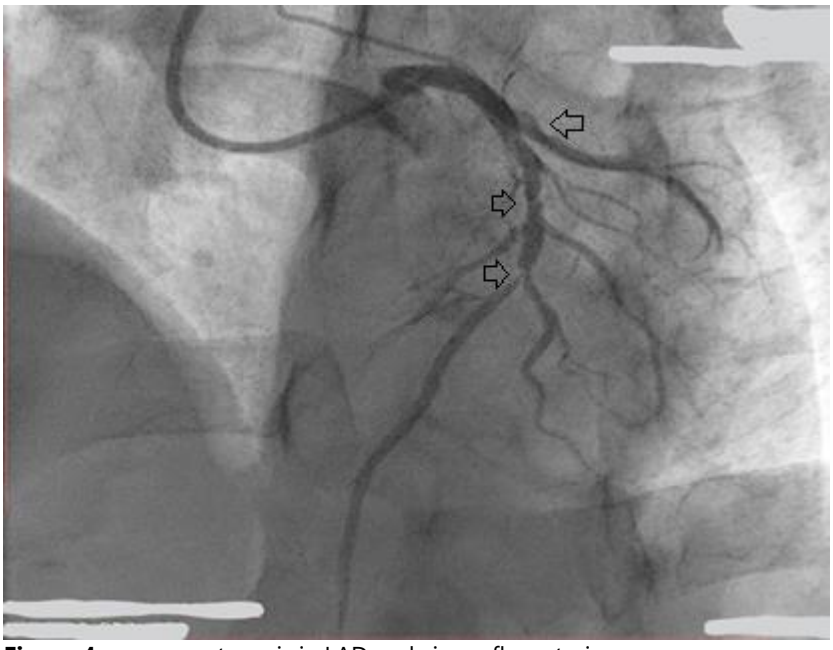

Figure 4: coronary stenosis in LAD and circumflex arteries

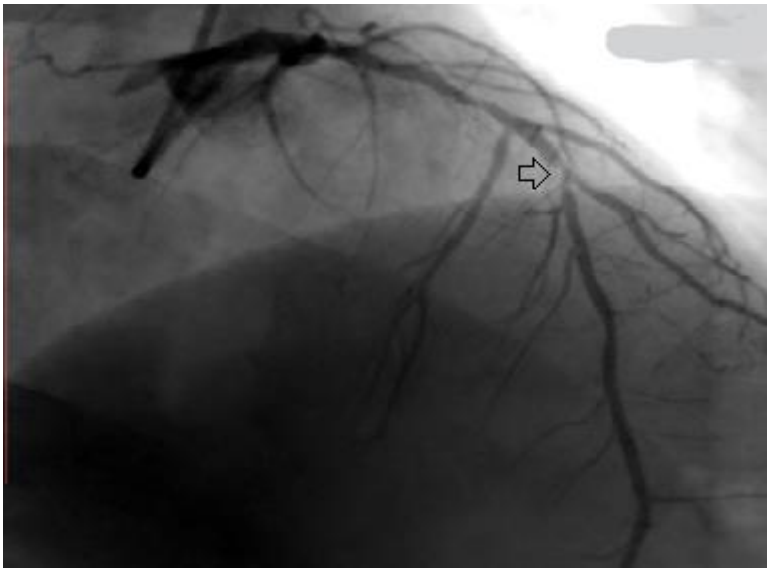

Figure 5: subtotal occlusive stenosis in mid-LAD artery