De Winters ST-T syndrome: an early sign of ST-segment elevation myocardial infarction (A case report)

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Abstract

Introduction: 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 the de-Winter T syndrome. It is defined by an upsloping ST-segment depression (>1mm) beginning from the J-point, and is symmetrical, long, with significant T waves seen in precordial leads. This syndrome is highly predictive for acute proximal left anterior descending artery (LAD) occlusion.

Case presentation: 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 a few minutes, after ventricular fibrillation cardiac arrest, into an anterior STEMI. Reperfusion therapy was performed with success.

Conclusion: Prompt recognition of this atypical ECG pattern can ensure the immediate detection of evolvement to a STEMI and therefore provide appropriate urgent reperfusion therapy either with percutaneous coronary intervention (PCI) or fibrinolysis if the PCI is not available.

Keywords: 'De Winter' ECG Pattern; STEMI Equivalent; Left Anterior Descending Coronary Artery.

INTRODUCTION:

The Electrocardiogram (ECG) is an essential tool for the diagnosis of acute myocardial ischemia (MI) and the assessment of evolving myocardial infarction in the emergency departments (ED).

The ECG can distinguish between patients with an ST-segment elevation myocardial infarction (STEMI) and those with a non-STEMI, which can guide their management along with myocardial necrosis biomarkers.

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 the proximal LAD artery occlusion [1]. This pattern was originally described by de-Winter et al. [2] as a static sign However, several reports have emerged showing

that it can be temporary with dynamic changes which should be detected by repetitive ECGs to meet indications for thrombolytic therapy especially if PCI is not available [1-3]. We herein present a case of de-Winter syndrome which evolved into a STEMI and was treated successfully with fibrinolysis in the ED.

Case presentation

A 45-year-old male presented to the ED with retrosternal chest pain radiating to the left arm within the first hour. He had a history of smoking (15 packs per year) and penicillin allergy. The examination in the triage area showed a patient with a stable hemodynamic status and no clinical sign of heart failure. He scored his pain as 10/10 according to the Numeric Pain Scale (NPS).

The first ECG performed 5 min after his arrival showed a sinus rhythm, a rate of 83 bpm, an ST-segment elevation in right leads (V1, V2, V3R, V4R), and a 3 mm upsloping ST-segment depression in leads V3-V6 at the J point which continued into tall, positive symmetrical T-waves and 1.5 mm ST-segment depression in leads II, III, aVF, V7-9 (Figure 1).

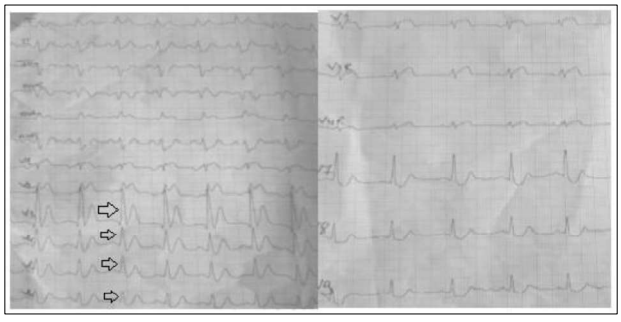

Figure 1. First ECG (de Winter complex showed by arrows).

The patient was categorized 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 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. The pain score was 8/10 according to the NPS.

The ECG realized 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).

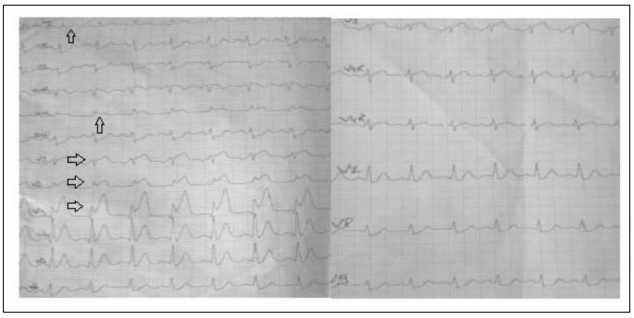

Figure 2. ECG after cardiac arrest recovery (ST-segment elevation shown by arrows).

The diagnosis of a STEMI in the anterior territory was made. He was treated with aspirin, clopidogrel, an intravenous bolus of low molecular-weight heparin followed by the subcutaneous dose and intravenous titration of morphine according to the pain statement. The reperfusion strategy chosen was fibrinolysis by tenecteplase, as primary PCI was not available 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).

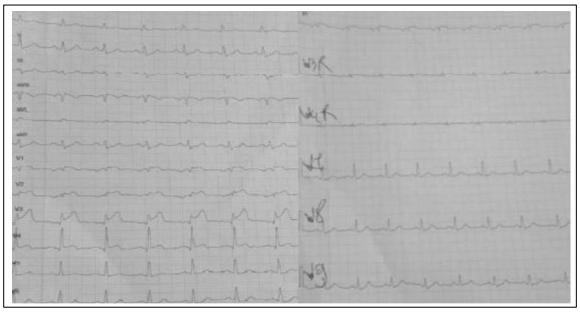

Figure 3. ECG at 60 min after thrombolytic therapy administration.

The patient was admitted to the ED for 24 hours then transferred to the cardiology department where a coronary angiography was performed. It showed subtotal occlusive stenosis in the mid-LAD artery (90-99%), tight stenosis in the proximal LAD artery (70-90%), and significant stenosis in the mid circumflex artery (50-70%) (Figure 4 and 5).

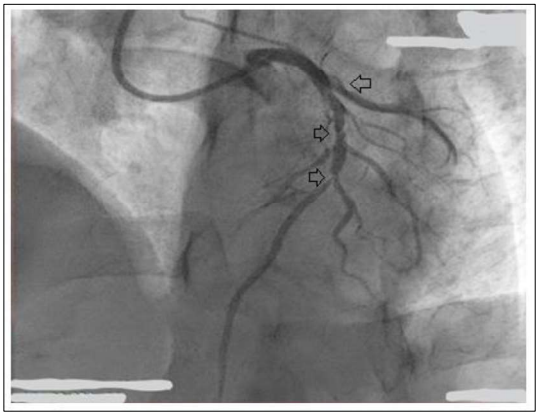

Figure 4. Coronary stenosis in LAD and circumflex arteries.

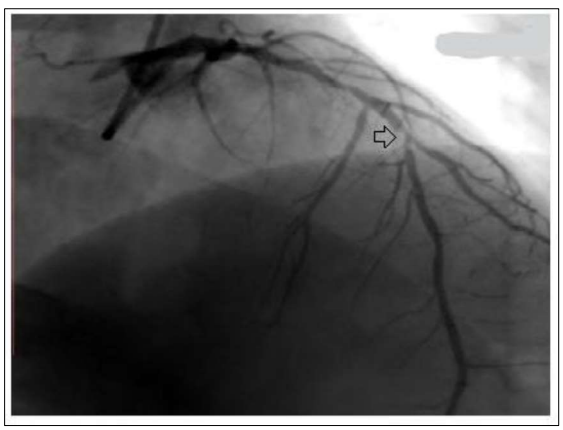

Figure 5: Subtotal occlusive stenosis in the mid-LAD artery.

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 a favorable outcome.

DISCUSSION

The de-Winter ECG pattern, characterized by 1-3mm upsloping ST-segment depression at the J point in V1 to V6 leads that continue to tall positive symmetrical T waves. This pattern 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 with 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 shown 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 urgent reperfusion therapy (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] alleged that the stage of de Winter syndrome is earlier than the STEMI hyperacute period, which is equivalent to the

change of the STEMI hyperacute period. There are two clinical presentations of de-Winter syndrome ECG: (1) resting ECG (the most frequent, and they basically will not evolve into STEMI), (2) and dynamic ECG (they evolved into STEMI). In our case, we were dealing with the dynamic form of the de-Winter syndrome. The patient benefited from thrombolytic therapy and recovered with a normal left ventricular function.

CONCLUSION

According to guidelines for the diagnosis and management of STEMI, fibrinolysis is not recommended in the de-Winter ECG pattern. Although it is considered a STEMI equivalent, thrombolytic therapy is still targeted at the acute STEMI.

Therefore, repetitive ECGs monitoring is necessary for this group of patients to track down the moment of ST-elevation, especially when catheterization laboratory is not available for PCI.

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