Case Report

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Successful Thrombolysis of Dynamic De Winter's Sign: A Case Report

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Abstract

De Winter's T-waves is a rarely reported electrocardiogram (ECG) pattern requiring watchful eyes to recognize it. It was described as static sign until coronary reperfusion. This pattern was on average recorded 1.5 hours after symptom onset. It was considered as à ST-Elevated Myocardial infarction (STEMI) equivalent pattern. We report a dynamic De Winter pattern who attended one hour of the onset of chest pain with rapid ECG changes before reperfusion.

Keywords: De winter sign; Electrocardiography; Myocardial infarction; Thrombolytic therapy; Myocardial reperfusion injury

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Introduction

De Winter's T-waves is a rarely reported Electrocardiogram (ECG) pattern requiring watchful eyes to recognize it. It was described as static sign until coronary reperfusion [1]. This pattern was on average recorded 1.5 hours after symptom onset [2]. It was considered as a ST-Elevated Myocardial Infarction (STEMI) equivalent pattern [3]. We report a dynamic De Winter pattern who attended one hour of the onset of chest pain with rapid ECG changes before reperfusion.

Case Report

A 62 years-old male presented within one hour after the onset of chest pain with radiation to the neck. He had no medical history. The physical exam found a regular heart rate=65 beats per minute (bpm), blood pressure=120/50 mm Hg, respiratory rate=22 cycles per minute (cpm), oxygen saturation=95% on room air. Pain intensity was evaluated seven on the 0 to 10 analog scale.

Five minutes upon arrival at the emergency department, the 18-leads initial ECG, showed normal sinus rhythm of 66 bpm, narrow QRS complexes (40 ms), a 4 mm upsloping ST depression in the apicolateral leads (V3-V6) followed by tall, symmetrical T waves and an elevation of 1 mm in aVL. ST depression and positive T waves were maximal in V3 lead **(Figure 1).** Moreover, we noted a 4 mm ST-segment depression at the J point in the inferior leads, 1 mm ST-segment depression in the posterior leads and 1mm ST-elevation in lead aVR. Clinical presentation and first ECG evoked the diagnosis of non ST-segment elevation myocardial infarction (NSTEMI). The patient was given titrated intravenous (I.V.) morphine chlorhydrate, I.V. loading dose of acetyl salicylic acid (250 mg) and oral loading dose of clopidogrel (300 mg). High-sensitivity cardiac troponin (Troponin I Hs) level was of 66.6 ng/L (normal reference for acute coronary syndrome value= 87.5 ng/L).

Eight minutes later, i.e., 73 minutes upon symptoms onset, second ECG revealed ST-segment elevation in the anteroseptal leads (V1-V4) and the high lateral leads (I- aVL). Reciprocal ST Depression was seen in the inferior leads (II, III and aVF) **(Figure 2).**

As immediate Percutaneous Coronary Intervention (PCI) was not available, patient was treated with streptokinase (I.V. 1.5 million units over 45 minutes). Chest pain disappeared and ECG showed decreased ST elevation to 2 mm at 90 minutes post streptokinase infusion **(Figure 3).**

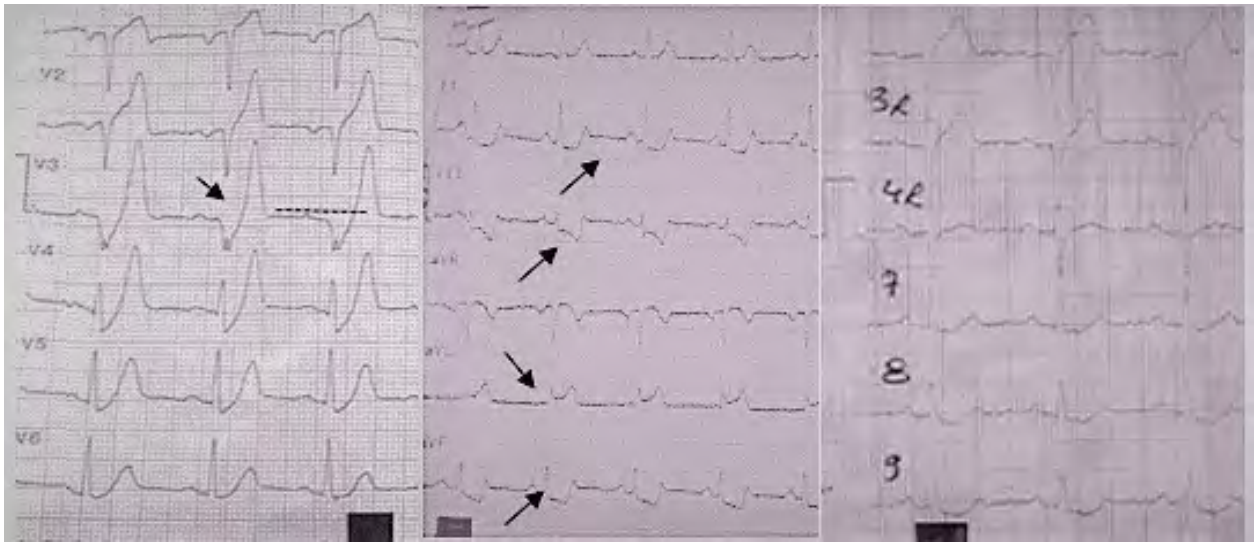

Figure 1 Initial 18-lead electrocardiogram, 5 minutes upon arrival at the emergency department: 4 mm upsloping ST depression in the apicolateral leads (V3-V6) followed by tall, symmetrical T waves; ST-segment depression in the inferior leads; 1 mm ST-elevation in lead aVR and aVL, 1 mm ST-segment depression in the posterior leads.

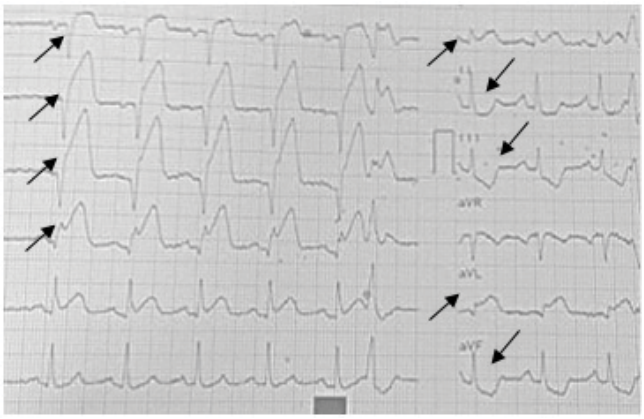

Figure 2 Second electrocardiogram, 13 minutes upon arrival at the emergency department: ST-segment elevation in the anteroseptal leads (V1-4) and the high lateral leads (I-aVL), reciprocal ST depression in the inferior leads (II, III- aVF).

Acute anterior ST elevation myocardial infarction diagnosis with successful thrombolysis was retained. Cardiac biomarker levels peaked at 48 hours (Troponin I Hs= 41956 ng/L). Patient was diagnosed for dyslipidemia with elevated low-density lipoprotein cholesterol of 1.7 g/L (normal=1-1.5 g/L) and high total cholesterol of 2.3 g/L (normal=1.3-2 g/L).

On day 2, echocardiography has showed ischemic cardiomyopathy with left ventricle ejection fraction of 40%. Angiography with PCI performed 72 hours later, noted a tight stenosis in the proximal and middle segment of Left Anterior Descending artery (LAD). The lesion was treated with drug eluting stent. The patient was discharged from hospital on day 5.

Discussion

In 2008, the De Winter ECG pattern was initially described in 2% of patients with anterior myocardial infarction [1]. ECG features show typically a 1- to 3-mm upsloping ST-segment depression at the J point in leads V1 to V6 followed by tall, positive symmetrical T waves [1] with a 1- to 2-mm ST-elevation in lead aVR in most patients [1,2]. It's considered STEMI equivalent [4,5] and has been observed as a static pattern without evolution to STEMI [6]. Only few cases reported dynamic ECG evolvement and modifications may go in the change of De Winter T waves from [7] or into ST-elevation [8]. In a recent review, analyzing ECGs of 70 patients, evolvement into STEMI was assessed in two cases after PCI [2].

Although there are few related cases, De Winter sign was often described in male with a mean age of 53 ± 13 years [2]. There has often been no cardiovascular risk predisposing clinical profile. Our patient had age, sex and dyslipidemia as coronary atherosclerosis risk factors. De Winter sign was recorded 1.5 hours after symptom

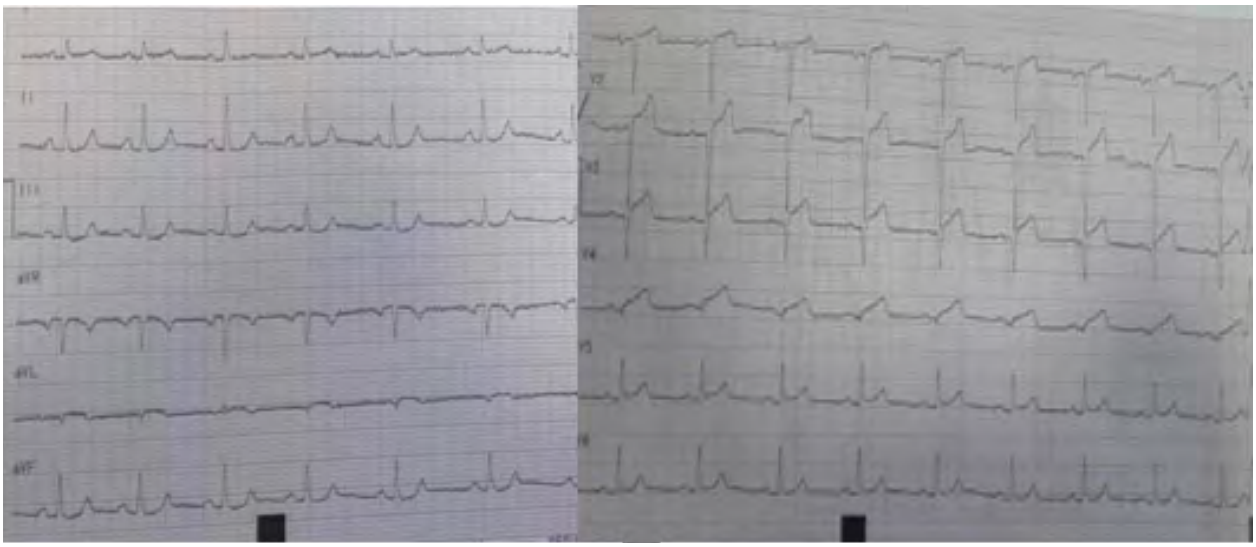

Figure 3 90 minutes post-thrombolysis ECG: ST elevation decreased to 2 mm.

onset [1,2] and persist for hours until coronary reperfusion or aggressive antiplatelet therapy [8]. In our case, De Winter patterns were noted on admission, 65 minutes after chest pain onset. ECG modifications were notified within 73 minutes upon symptoms onset. This ECG change may reflect the time of the complete coronary artery occlusion.

Electrophysiological mechanisms of ECG modifications are unclear. ST depression is related to transmembrane potential gradient. As subendocardial ischemia progressed to full-thickness transmural ischemia, epicardial ST depression gradually increased and ST elevation took place over the ischemic region [9].

ST depression was maximal in V2 lead and evocated an acute LAD disease. In fact, LAD was the main reported culprit artery [10]. While, De winter sign may occur with the others coronary arteries such as first diagonal branch [11], marginal artery [12], right coronary artery [13]. Evolvement of the De winter sign into STEMI was reported only with the LAD lesion particularly before reperfusion [2]. The severe lesion stenosis of LAD may explain the evolvement into ST elevation even before coronary reperfusion. De Winter ECG pattern should indicate an emergency coronary revascularization [10,11].

We have indicated fibrinolysis at appearance of ST elevation with successful outcome. The main question remains if it was safe to administer thrombolytic therapy to the patients with De Winter ECG pattern. The opinions were different in the literature. Rao et al. [14] had considered their De Winter cases at high mortality risk and administered thrombolytic therapy to the two patients with successful outcomes. Therefore, Deng et al. [15] justified his disappointment by the lack of consensus on whether thrombolytic therapy can be used for the treatment of De Winter syndrome and considered this sign related to coronary artery spasm. We cannot consider De Winter sign atypical STEMI presentation but we have to consider it at high risk of development of extensive myocardial infarction and death. In our case, we have not initially detected STEMI, but it seemed more careful to indicate reperfusion even before evolvement into STEMI. While thrombolysis reperfusion was rapid, myocardial damage was considerable as shown by troponin high level and left ventricle ejection fraction. It seemed urgent to indicate primary percutaneous coronary intervention.

Conclusion

De Winter syndrome may be an early and dynamic ECG pattern in the development of acute STEMI. Therefore, repeated ECG must be the rule in patients with acute coronary syndrome. However, there is a real benefit if reperfusion is precociously indicated.

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