

Case report of de Winter Syndrome and ST-elevation myocardial infarction

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Abstract

Chest pain is the presenting complaint of a myriad of illnesses, ranging from life-threatening causes such as acute myocardial infarction (AMI) to comparatively benign causes like gastroesophageal reflux. Less than half of patients presented with chest pain suggestive of cardiac origin have classic diagnostic features in the electrocardiogram (ECG). However, identifying the elusive ECG findings of an AMI is a diagnostic challenge.

Subtle ECG changes can easily be missed unless specifically looked for. De Winter syndrome is one such diagnosis, which may evolve into ST-elevation myocardial infarction (STEMI) or vice versa. This is a case of a successfully managed 46-year-old male, who presented with suggestive chest pain and ECG evidence of de Winter syndrome.

Keywords: De Winter syndrome, STEMI equivalents, ST-elevation myocardial infarction, Case report

Introduction

Chest pain is a common symptom with which patients present to the emergency department. But, only less than half of these patients have a diagnostic ECG. Despite the advent of sophisticated medical investigations, ECG remains the cornerstone of the diagnosis of the acute coronary syndrome. It is now evident that a proportion of these patients do not have guideline-stated diagnostic features of ST-elevation myocardial infarction (STEMI), yet have an occlusion that would benefit from early coronary interventions. Such ECG patterns are now known as 'STEMI-equivalents'.

De Winter syndrome is an ECG pattern seen in proximal left descending artery occlusion, which may benefit from management similar to that of STEMI.

Case presentation

A 46-year-old previously healthy man presented to the emergency department of the District General Hospital, Kalutara with sudden onset chest pain which started one hour ago. The pain started while he was walking to his workplace. It was a classical tightening type of chest pain associated with sweating and radiation to the left arm. The pain persisted until he arrived at the hospital.

On admission, he was in pain but hemodynamically stable. He was not breathless and maintained adequate saturation on room air. He had no signs of acute heart failure. His initial ECG showed 2 mm ST segment elevation in leads V2 to V4 with reciprocal ST segment depression in lead III and aVF. The second ECG showed up-sloping ST depressions of more than 2 mm, tall prominent symmetrical T waves in leads V3 to

V5 with ST elevation of 1 mm in aVR and 2 mm in V2. The inferior leads, lead II, lead III and aVF showed more than 2 mm ST depressions (Figure 01).

Bedside echocardiogram showed anteroseptal hypokinesia with an approximate visual ejection fraction of 45-50%.

According to the ECG findings, the first diagnosis was anterior STEMI which later on evolved into de Winter syndrome. As primary percutaneous coronary intervention (PCI) was not feasible within 120 minutes, fibrinolysis was selected as the treatment after excluding contraindications.

After giving loading doses of oral Aspirin 300 mg and Clopidogrel 300 mg, intravenous (IV) Enoxaparin 30 mg bolus was administered. It was followed by intravenous Tenecteplase. Fifteen minutes later, subcutaneous Enoxaparin 60 mg was given. The patient was continuously monitored for electrical or haemodynamic instability and bleeding complications. He continued to have mild chest pain, which was managed with IV morphine. Post-thrombolysis ECG (after 60 minutes) showed resolution of ST segment elevations with frequent ventricular ectopics (Figure 02).

Repeat ECG taken two hours after the thrombolysis showed sinus rhythm with evolving ST elevations in leads V5 to V6. Therefore, the patient was transferred to the National Hospital of Sri Lanka for a rescue PCI. Angiogram revealed a mid-left anterior descending (LAD) artery occlusion with a heavy thrombus burden in the proximal segment. Ballooning and stenting of the mid-LAD artery were done. An ECG resolution was noted along with Q waves in the anterior leads (Figure 03).

Post-procedure ejection fraction was 45-50% with anterolateral hypokinesia. The patient was discharged after two days of hospital stay and arranged for a follow-up at the cardiology unit.

Discussion

De Winter syndrome is an ECG pattern associated with acute LAD artery occlusion. It was first described by De Winter and colleagues in 2008 [1]. It is a non-progressive pattern of ECG changes appearing on precordial leads. This can be seen in about 2% of patients with anterior myocardial infarction [2]. Characteristic ECG features are tall symmetrical prominent T waves in precordial leads and upsloping 1-3 mm ST depressions at J point [1]. QRS complexes are usually narrow. In some cases, there may be a poor progression of the R wave [1]. The patients with this ECG pattern are younger and mostly males compared to those who had anterior STEMI [2]. Though a tall symmetrical T wave was considered an early feature of STEMI, in patients with de Winter pattern of ECG, these tall symmetrical T waves do not evolve until reperfusion [2]. Troponin I can be normal or slightly elevated at the time of the ECG recording. De Winter syndrome and ST-elevation myocardial infarction can evolve into one another.

ECG abnormalities other than ST elevations can be indicators of myocardial injury. Identification of non-diagnostic and subtle ECG changes is crucial in preventing malignant dysrhythmias and cardiac death.

Though primary percutaneous coronary intervention (PCI) is the standard of care in STEMI, in circumstances where primary PCI is not available or delayed for more than 120 minutes, fibrinolysis is recommended within 12 hours of the onset of symptoms, provided there are no contraindications [3,4]. Patients with higher risk, the elderly and who are offered treatment within two hours from symptom onset get the most benefit from thrombolysis. Fibrin-specific agents such as Tenecteplase are preferred. In cases of failed thrombolysis or those with evidence of re-occlusion, rescue PCI is indicated [4].

In this case, a young male patient presented with a history of ischemic-type chest pain and

ECG was diagnostic of de Winter syndrome. He was treated with primary fibrinolysis. Post thrombolysis ECG initially showed resolution, but subsequent ECG was evident of re-infarction. The patient underwent rescue PCI. Due to the heavy thrombus load, post-procedure anticoagulation with enoxaparin was continued.

Conclusion

It is necessary to carefully evaluate the

ECGs for elusive and perilous features to identify possible impending cardiac death. Subtle changes in ECG can be easily missed unless the clinicians especially look for them. De Winter syndrome and STEMI may evolve into one another. Assessment of symptoms and resolution of symptoms after thrombolysis is crucial in diagnosing failed thrombolysis and reinfarction.

Author declaration

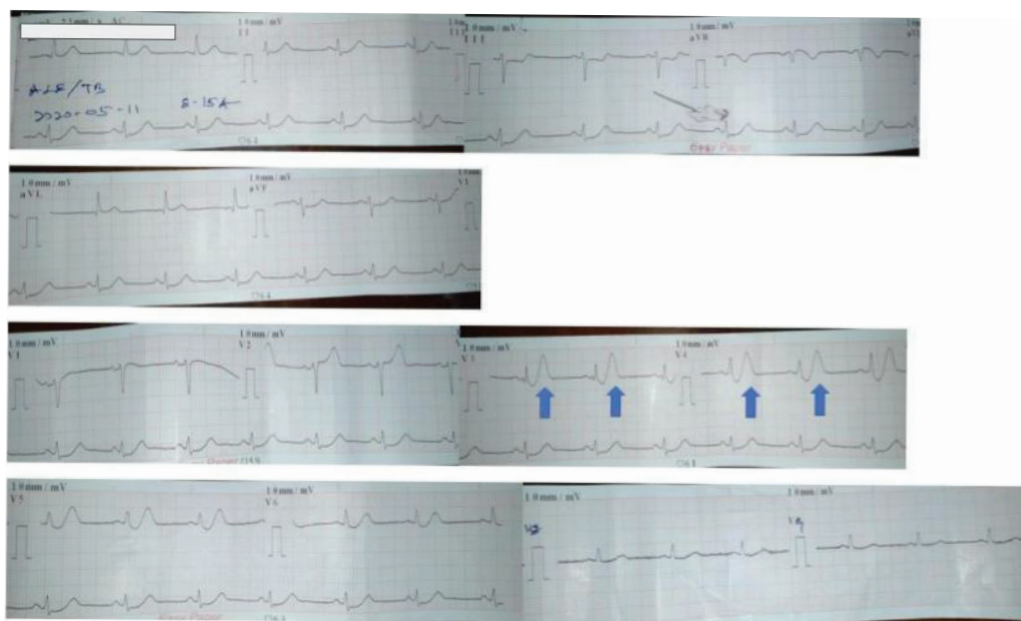
Author contributions: GMS led the clinical management of the patient and reviewed the manuscript. WHM performed a literature survey and wrote and edited the manuscript. Both authors read and approved the final version of the manuscript.

Funding and conflicts of interest: The authors declare that they have no conflicts of interest concerning the research, authorship, and/or publication of this article. This report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics approval and consent to participate: Written informed consent was obtained from the patient for publication of this case report.

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(Arrows show de Winter T waves.)

Figure 01: The ECG findings on admission

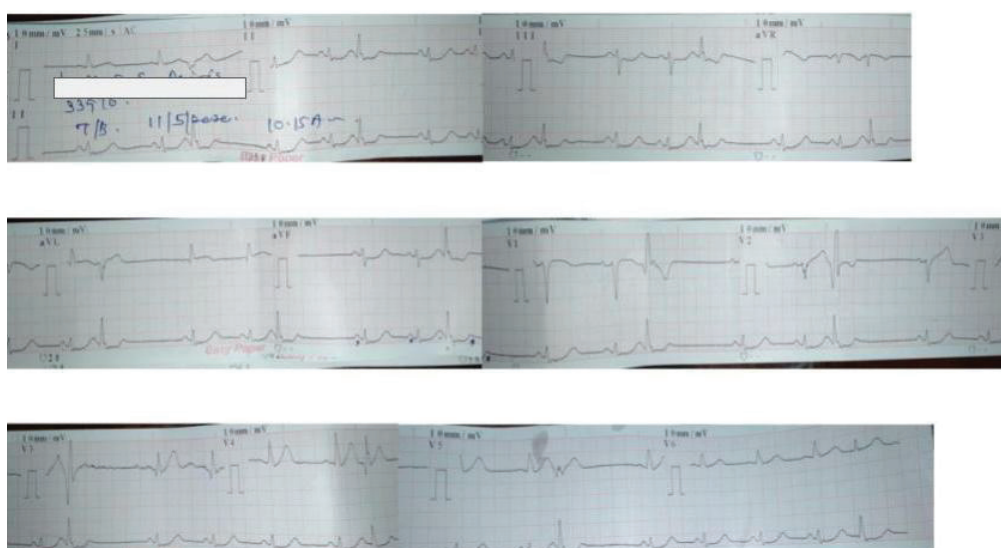


Figure 02: Post-thrombolysis ECG

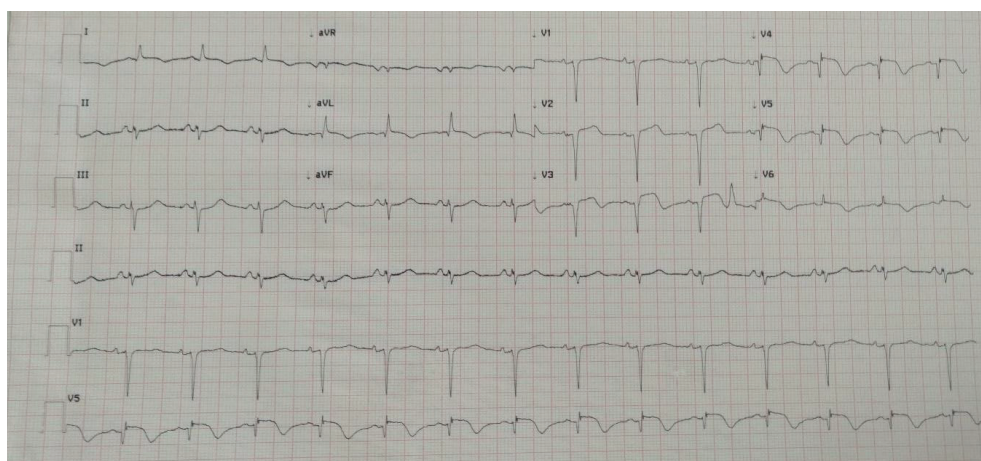


Figure 03: The ECG following rescue PCI