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De Winter Sign as a Precursor to Acute Myocardial Infarction and Post-Streptokinase Dramatic Response; A Case Report

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ABSTRACT

Rationale: *An acute myocardial infarction is uncommonly present with atypical electrocardiographic findings. Left bundle branch block, right bundle branch block, de Winter sign are typical examples for these deviated presentations. De Winter sign is an indicator of acute of the left anterior descending coronary artery occlusion. The sign is extreme rare finding carry highly significant mortality and morbidity rate. Delayed in the diagnosis of this mimic acute anterior myocardial infarction already will have a poor prognostic outcome.*

Patient Concerns: *The current case is middle-aged male patient presented with acute ischemic chest pain with fully developed de Winter T-wave into acute anterior ST-elevation myocardial infarction. The patient received intravenous infusion streptokinase injection.*

Diagnosis: *De Winter sign as a precursor to acute ST-elevation myocardial infarction.*

Interventions: *Electrocardiography and intravenous infusion streptokinase injection.*

Lessons: *The physician should take care of abnormal and unusual electrocardiographic presentations.*
Outcomes: *Dramatic response of de Winter T-wave sign to intravenous thrombolytic therapy.*

Keywords: *De Winter sign, de Winter syndrome, precursor to acute myocardial infarction, post-streptokinase dramatic response*

Abbreviations

CAD: Coronary artery disease

ECG: Electrocardiograph

ICU: Intensive care unit

LAD: Left descending artery

MI: Myocardial infarction

PCI: Percutaneous coronary intervention

STEMI: ST-elevation myocardial infarction

INTRODUCTION

De Winter syndrome is an extreme existence in the cases of acute left descending artery ((LAD) occlusion. It is a signal for very severe coronary artery pathology rather than a higher mortality rate[1]. The syndrome prevalence is nearly 2% of patients of acute anterior myocardial infarction (MI)[2]. Hence, Misdiagnosis is highly associated with increased morbidity and mortality rates[3]. In 2008; the de Winter electrocardiographic (ECG) sign was described in a case report series by both de Winter and Wellens who resulted in 30/1532 patients presented with acute LAD obstructions (2%)[4]. In 2009; Verouden et al. reported this pattern in 35/1890 patients demanding percutaneous coronary intervention (PCI) for LAD obstructions. In this study, the patients were hypercholesterolemic younger, more often male presented with acute anterior STEMI[5]. Diagnostic ECG changes which accompanied by acute occlusion of a coronary artery is a good way for giving reperfusion therapy[3]. As urgent reperfusion therapy is indicated to patients presenting with ST-segment elevated myocardial infarction, patients with ST-segment depression on admission benefit from this urgent therapy only in some cases[1]. This pattern to be an equivalent of anterior ST-segment elevation myocardial infarction (STEMI)[4]. The following are the diagnostic criteria:[6] 1. Tall, prominent, symmetric T-waves in the chest leads. 2. Up sloping ST-segment depression >1mm at the J-point in the precordial leads. 3. Absence of ST-segment elevation in the chest leads. 4. ST-segment elevation (0.5mm-1mm) in a VR. 5. Normal ST-segment morphology may precede

or follow the de Winter sign. De Winter syndrome is a condition accompanied by typical chest pain and a characteristic ECG pattern with no classic STEMI, yet acute total occlusion of the LAD coronary artery[3]. A current guideline described STEMI as a clinical syndrome of typical symptoms of coronary artery disease (CAD) with persistent ECG ST-segment elevation and biomarkers release of myocardial necrosis[7]. Whatever, ECG abnormalities other than ST-segment elevation may guide for transmural myocardial injury[3]. There is strong evidence that the de Winter ECG sign is highly predictive of acute LAD occlusion[6]. Unawareness of the current high-risk ECG sign may result in under-treatment in form of failure of catheter lab activation and strong negative effects on morbidity and mortality[6]. The triage of patients is the main responsibility for cardiologists and emergency care physicians, paramedics, and others who deal with chest pain. They should be able to rapidly diagnose this STEMI-equivalent ECG pattern to ensure appropriate urgent reperfusion therapy with PCI or thrombolysis[3,6]. Several affected leads with ST-segment depressions and ST-segment elevation in a VR may benefit from urgent reperfusion treatment as this has been reported in patients with MI and occlusion of the proximal LA[8]. This sign may be helpful for an invasive strategy in these patients with a better short and long term prognosis[1].

CASE PRESENTATION

A 53-year-old male heavy smoker Egyptian restaurant worker presented with acute severe chest pain and profuse sweating. Chest pain was anginal and compressive. The patient smoked about 50 cigarettes per day for nearly 25 years. He gave a history of angina during the stay in Kuwait three years ago. The patient underwent coronary angiography. There was no detected abnormity. Upon general physical examination, the patient was anxious, severe sweaty, with cold extremities, a regular heart rate of 90 bpm, the blood pressure of 130/70 mmHg, the respiratory rate of 18 bpm, the temperature of 36.4 °C, and the pulse oximeter of O_2 saturation of 96%. No more relevant clinical data were noted during the clinical examination. Urgent ECG tracing was done in the emergency room which showed tall, prominent, symmetric T-waves in the V2-6 leads, up sloping ST-segment depressions in the V5-6 leads, ST-segment elevations in I, and a VL leads, and reciprocal ST-segments depressions in III, a VF, and a VR leads (**Figure 1**). Normal ST-segment morphology in the ECG tracing within 36 hours before this presentation (**Figure 2**). He was admitted into the ICU due to anginal chest pain. Serial ECG tracings in ICU were taken. Within 20 minutes of the above emergency ECG tracing, another one was done which showed tall, prominent, symmetric T-waves in the V2-6 leads, up sloping ST-segment depressions in the V4-6 leads, normalization of ST-segment in I, and aVL leads, reciprocal ST-segments depressions in II, III, and a VF leads, and ST-segment elevation in V1-3 and aVR leads (**Figure 3A**). O_2 inhalation (100%, by nasal cannula, 5 L/min) , and pethidine HCL 100 mg (on intermittent IVB doses), aspirin 4 chewable oral tablet (75 mg), clopidogrel 4 oral tablet (75 mg), and streptokinase IVI (1.5 million units over 60 min) were given. Within nearly 60 minutes of given streptokinase, the ECG tracing showed established ST-segment elevations, pathological Q-wave in V1-6 leads, and normalization of above reciprocal ST-segments depressions in II, III, and a VF leads (**Figure 3B**). Within nearly 16 hours of given streptokinase, the ECG tracing showed gradual resolution of ST-segment elevations, pathological Q-wave in V1-3 leads, and normalization of above reciprocal ST-segments depressions in II, III, and a VF leads (**Figure 3C**). Clinical improvement and electro cardiographic ST-segment (whether elevation or reciprocal ST-depression resolution had happened. The measured random blood sugar was 134 mg/dL. The troponin test was positive (271 ng/L). Later echocardiography was mild anterior hypokinesia with an ejection fraction of 59%. No more workup was done. The patient was continued on captopril tablet (25 mg twice daily), aspirin tablet (75 mg/d), clopidogrel tablet (75 mg/d), bisoprolol tablet (5 mg once daily), nitroglycerin retard capsule (2.5 mg twice daily), and atorvastatin (40 mg/d) until discharged on the 5th day. He maintained on captopril tablet (25 mg twice daily), aspirin tablet (75 mg/d), clopidogrel tablet (75 mg/d), bisoprolol tablet (5 mg once daily), nitroglycerin retard capsule (2.5 mg twice daily), and atorvastatin (40 mg/d). Planning for both possible pacing and catheter revascularization was the future option.

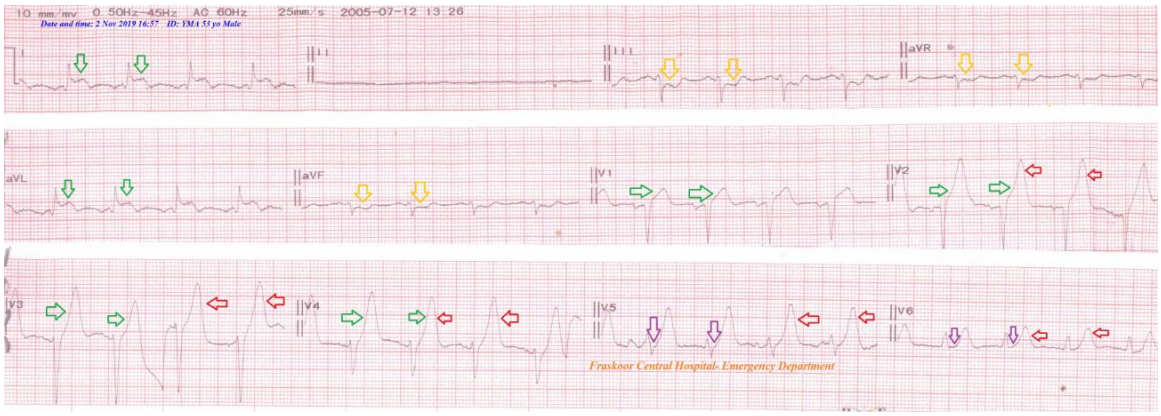

Figure1. *Urgent ECG tracing in the emergency room showing tall, prominent, symmetric T-waves in the V2-6 leads (red arrows), up sloping ST-segment depressions in the V5-6 leads (purple arrows), ST-segment elevations in I, and aVL leads (green arrows), and reciprocal ST-segments depressions in III, aVF, and aVR leads (yellow arrows).*

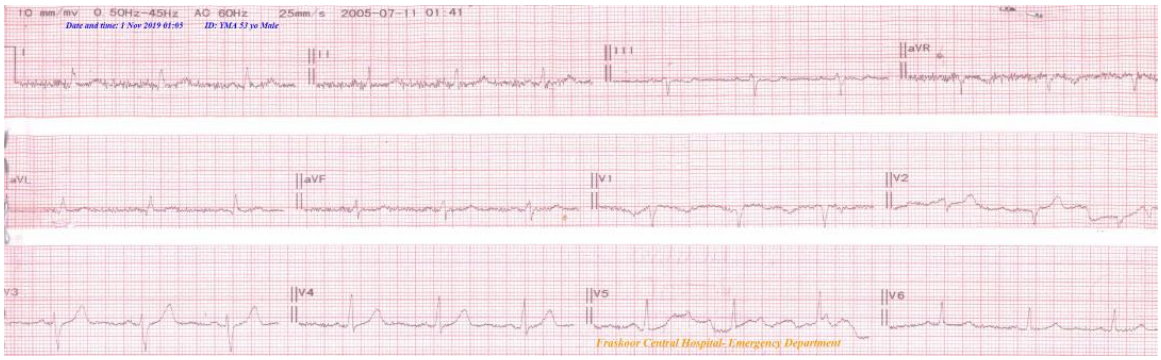

Figure2. *ECG tracing 36 hours before the above presentation in figure 1. Otherwise the ECG artifacts, there are no more abnormalities.*

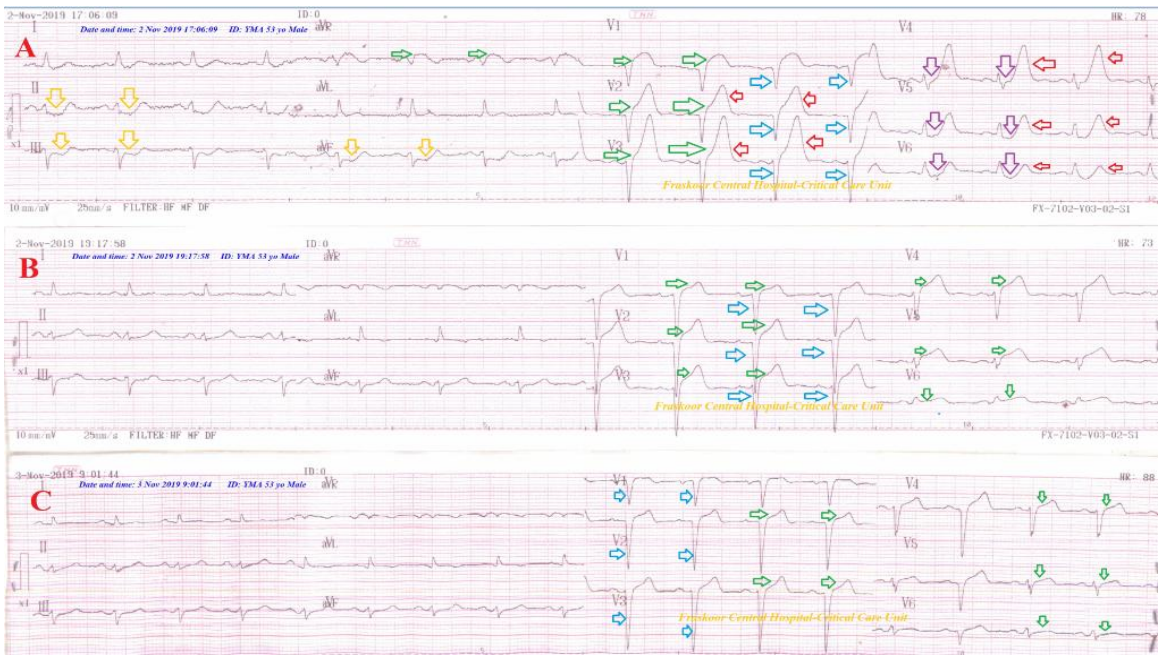

Figure3. *ICU serial ECG tracings; A. tracing showing tall, prominent, symmetric T-waves in the V2-6 leads (red arrows), up-sloping ST-segment depressions in the V4-6 leads (purple arrows), ST-segment elevations in aVR, and V1-3 leads (green arrows), pathological Q in V-3 leads (blue arrows), and reciprocal ST-segments depressions in II, III, and aVF leads (yellow arrows). B. tracing showing established ST-segment elevations (green arrows), pathological Q-wave in V1-3 leads (blue arrows), and normalization of above reciprocal ST-segments depressions in II, III, and aVF leads. C. tracing showing gradual resolution of ST-segment elevations (green arrows) and pathological Q-wave in V1-3 leads (blue arrows).*

DISCUSSION

- **Overview:** A middle-aged male patient presented with acute fully developed de Winter T-wave into acute anterior ST-elevation myocardial infarction.
- I can't **compare** the current case with similar conditions. There are no similar or known cases with the same management for near comparison.
- **Study question here;** How the physician expects atypical electrocardiographic acute myocardial infarction presentations?
- **The primary objective** for my case study was developing of acute anterior ST-elevation myocardial infarction post de Winter T-wave.
- **The secondary objective** for my case study was the dramatic response of de Winter T-wave syndrome to intravenous thrombolytic therapy.
- **Limitations of the study:** There are no known limitations for the study.
- **Recommendations**: It is recommended for the physicians to be oriented with atypical electrocardiographic myocardial infarction presentations like de Winter T-wave or syndrome.

CONCLUSIONS

The physician should take care of the abnormal and unusual electrocardiographic presentation of acute myocardial infarction like de Winter T-wave or syndrome.

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