

The de winter electrocardiographic pattern

Summary

We present a clinical case of a patient, without previous cardiac history, admitted to the emergency department with chest pain at low probability for coronary artery disease. The first electrocardiogram showed high-risk changes that were mistakenly interpreted as normal; serial electrocardiograms did not show dynamic changes in ST-segment or in the T wave. The patient remained asymptomatic and with hemodynamic stability. Ultrasensitive troponin was positive, and echocardiography reported a structurally healthy heart. Finally, the patient was submitted to diagnostic coronary angiography, evidencing involvement of the proximal anterior descending artery. After reviewing again the initial electrocardiogram, it revealed a high-risk pattern (the de Winter).

Keywords: electrocardiography, acute coronary syndrome, myocardial reperfusion, chest pain

Volume 11 Issue 5 - 2018

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Received: July 26, 2018 | **Published:** September 12, 2018

Introduction

Chest pain continues to be a major reason for consultation in the emergency room, with heterogeneity in the clinical presentation that ranges from cardiogenic shock or cardiorespiratory arrest to until non-cardiac benign conditions; thus, electrocardiogram (ECG) is a valuable tool for diagnosis and risk stratification. Although it is known that the management of ST-elevation myocardial infarction (STEMI) requires urgent reperfusion, there are other ECG high-risk findings that are associated with adverse outcomes or imminent acute myocardial infarction (AMI); for this reason, the performance of a prompt coronary angiography is beneficial.

Clinical case

47-year-old man, athlete, with a history of dyslipidemia in oral lipid-lowering medication and family history of AMI in first-degree relatives at age 50. He presented to another institution for burning discomfort in thorax; ECG, in presence of chest pain, evidenced high, symmetrical T waves from V2 to V5 with ascending ST depression in V2 to V5, as well as ST elevation in AVR (Figure 1). The patient was referred to our service; serial ECGs were taken without pain, with absence of dynamic changes (Figure 2); ultrasensitive troponin was positive (2592; 0.0-26.4). With the elevation of this biomarker, myocarditis was suspected; a second troponin to perform a curve was requested, along with an echocardiogram. Control biomarker at 3 hours was 7352, and the echocardiogram was reported without pathological findings. In a reanalysis, the initial ECG was interpreted as ST equivalent with the de Winter's pattern; an urgent coronary angiography was performed, finding two lesions in anterior descendente (AD) artery, 80% each. D1 with 95% injury and distal circumflex lesion of 80%. The patient completed his attention in high-dependency unit, revascularized with two coronary stents, and dual anti aggregation for 18 months on an outpatient basis.

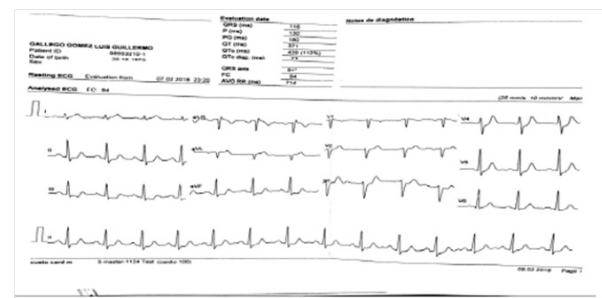


Figure 1 The de Winter pattern. Ascending ST-segment depression in V2 to V5 with high, symmetrical T waves in the same derivatives, associated with 1 mm ST elevation in AVR.

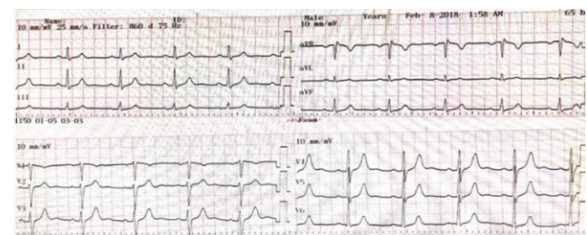


Figure 2 Electrocardiogram in patient without pain. Sinus rhythm is observed without changes in ST-segment or T wave.

Discussion

Acute coronary syndrome (ACS) is the main cause of mortality in our midst.¹ Its diagnosis is based on the probability given by the clinical history, physical examination, electrocardiographic changes and biomarker. ST-segment elevation in the first ECG taken is present in one third of the patients.² The other variations correspond to fluctuations in the T wave, ST-segment depression, branch blocks or normal ECGs. It is estimated that approximately 30% of ACS

diagnoses are lost or delayed by a misunderstanding of high-risk patterns warranting emergency coronary angiography.³ These patterns are: posterior wall infarctions, left bundle branch block with Sgarbossa criteria, Wellens syndrome, ST-segment depression in 8 derivatives associated with ST elevation in aVR (suggesting possible trunk or three vessel disease), hyper acute T waves in early infarctions, and the de Winter pattern,⁴ being highlighted by the presentation and therapeutic implications in the patient described.

The cardiologist Robbert J de Winter describes as a pathognomonic sign of coronary artery diseases (CAD) a ECG pattern that corresponds to proximal occlusion of the DA artery, evidenced by ST-depression from 1 to 3 mm in precordial derivatives V1 to V4, with high and symmetrical T waves in the same derivatives, associated with ST elevation in AVR;³ these changes are physiologically attributed to a variation in the Purkinje conduction fiber system that leads to a delay in intraventricular conduction; with potential wall ischemia, there is an alteration in ATP - dependent potassium channels that contributes to J point depression with prominent T waves in anterior wall.⁵ Other authors suggest that it is due to subendocardic ischemia that leads to a change in transmembrane action potential with progress to the subepicardium, showing a J point depression and slow rise in T wave in the ECG.⁶

Winter et al. in 2008⁷ described this electrocardiographic pattern in a series of 1532 patients; of these, 2% who underwent coronary angiography, exhibited proximal obstruction of DA; Verouden et al.,⁸ reported the same pattern with the same percentage of the findings in the coronary angiography, and the following electrocardiographic criteria were established: ST depression greater than 1 mm at J point in precordial derivatives with continued prominent and symmetrical T waves, ST elevation from 0.5 to 2 mm in aVR, absence of another ST elevation that guides to a coronary anatomy.

It is important to recognize that there are patterns that, even without ST elevation, entail imminently to STEMI, demanding emergent intervention; similarly, subjects with the de Winter pattern tend to be men, with ages between 30 and 40 years and associated hypercholesterolemia.⁹ There are reports with evidence of ECG progression to anterior wall STEMI with DA occlusion.¹⁰

Whether interpreted as a high-risk pattern or a ST equivalent, it is well known that the de Winter pattern is associated with a proximal lesion of DA. Even though little is known about this pattern, these ECG changes in a patient with suspicion of ACS should lead to consideration of urgent coronary angiography and appropriate intervention.

Acknowledgments

None.

Conflict of interest

The author declare

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