ST-elevation during head up tilt test: a challenging case in syncope unit

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Abstract

A 65-year-old woman, during an elective head up tilt test, after the sublingual nitrate administration, experienced electrocardiogram alteration with ST-elevation in the inferior leads, that returned normal when the patient was laid supine after few minutes. Serial cardiac markers were not elevated and coronary angiography revealed normal epicardial coronary arteries. Paradoxical vasospastic response to nitrates in vasospastic angina patients represents a rare but very challenging condition and the best therapeutic approach in this subgroup of patients remains unclear.

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Key words: Head up tilt test, ST-elevation, electrocardiogram, vasovagal syncope, nitrates.

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Introduction

Head up tilt test (HUTT) is a useful and safe test for the diagnosis of vasovagal syncope (VVS) in patients with suggestive history of reflex neutrally-mediated syncope [1]. Electrocardiographic alterations can rarely occur during HUTT and might be associated to various conditions, pathological or not [2]. In this case report we described an unusual electrocardiographic response to HUTT and discuss the possible physiopathological mechanism.

Case report

A 65-year-old woman with arterial hypertension, dyslipidemia, previous cerebral ischemic stroke was referred to our Syncope Unit for recurrent syncopal episodes of uncertain etiology. She denied any history of chest pain, palpitation or shortness of breath. The medical treatment included Olmersartan 40 mg once daily (OD); Atorvastatin 80 mg OD; Cardioaspirin 100 mg. The twelve-leads electrocardiogram (ECG) was normal. The orthostatic hypotension test and carotid sinus massage were negative. The transthoracic echocardiography showed normal ejection fraction (EF) calculated by the Simpson's biplane method (about 55%) and mild mitral regurgitation. A HUTT was performed according to the Italian protocol [3] with continuous ECG monitoring and blood pressure measurements every 3 min. The baseline value of BP and HR were 115/75 mmHg and 80 bpm, respectively. After 20 min drug-free passive orthostatic phase, 400 microgram of sublingual nitroglycerin spray was administered (BP: 125/70 mmHg, HR: 92 bpm). Three min later, the patient started complaining of dizziness, blurred vision, tingling and angina without experiences syncope. The blood pressure and heart rate were 90/55 mmHg and 72 bpm, respectively. The ECG revealed 2 mm ST-segment elevation (STE) in inferior leads with reciprocal ST depression in the lateral leads (I, aVL) (Figure 1), which returned at isoelectric line when the patient was placed in supine position. High-sensitivity cardiac troponin I (hs-CTnI) and creatine kinase-MB mass concentration (CK-MB mass) were measured baseline and remained stable 6 h after the event. For the high cardiovascular risk, the patient was admitted to the coronary care unit for evaluation of ischemia; coronary angiography revealed normal epicardial coronary arteries (Figure 2). Before discharge, a loop recorder (ILR) was

implanted. At six months follow-up the patient denied further episodes of syncope and no significant electrocardiographic abnormalities or arrhythmias were detected by ILR.

Discussion

The HUTT is generally considered a safe exam utilized in the differential diagnosis of patients with unexplained syncope. Life-threatening complications, such as myocardial infarction , ventricular tachycardia [4] are rare and usually a consequence of coronary artery spasm or marked decrease in arterial blood pressure [5]. The ST segment changes, in particular ST segment depression and T wave inversions, during HUTT were observed at a relatively high incidence (about 18%), primarily in women, with no clinical significance [6]. The primary mechanism driving ST-segment changes in young and otherwise generally healthy patients might be the imbalance of autonomic tone [6] and, in some cases, the improper filter application in single lead ECG monitor [7].

Few case reports showed ST-segment alteration in patients underwent HUTT [2,8]. Colman *et al*. [2] reported a case of ST-elevation in inferior leads, with reciprocal ST-segment depression in the left lateral and anterior leads, in a 56-year-old woman after an uncomplicated HUTT; the nitroglycerine administration

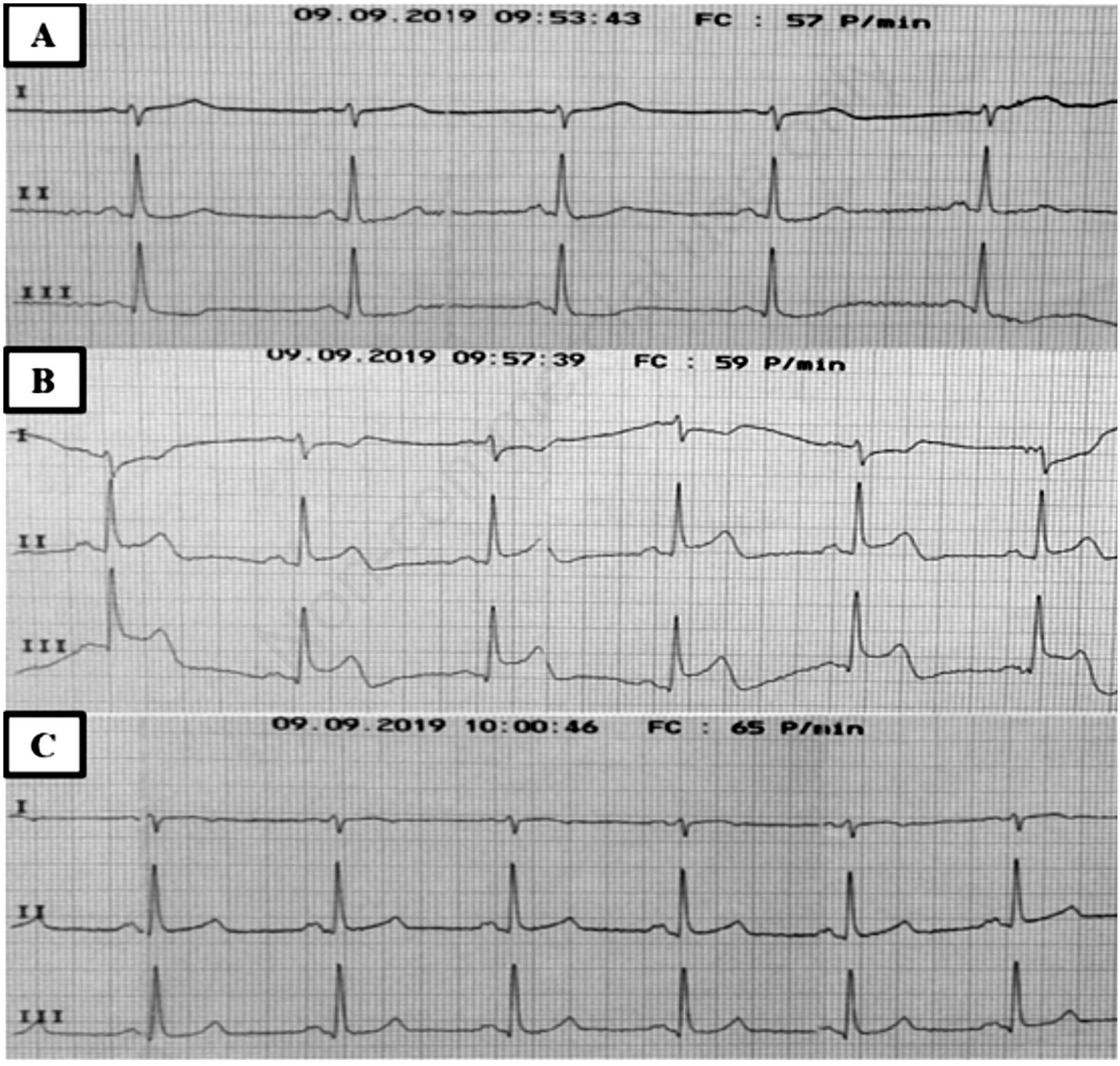

Figure 1. ECG before (A) and after (B) sublingual nitroglycerin administration. C) ECG after the resolution of symptoms.

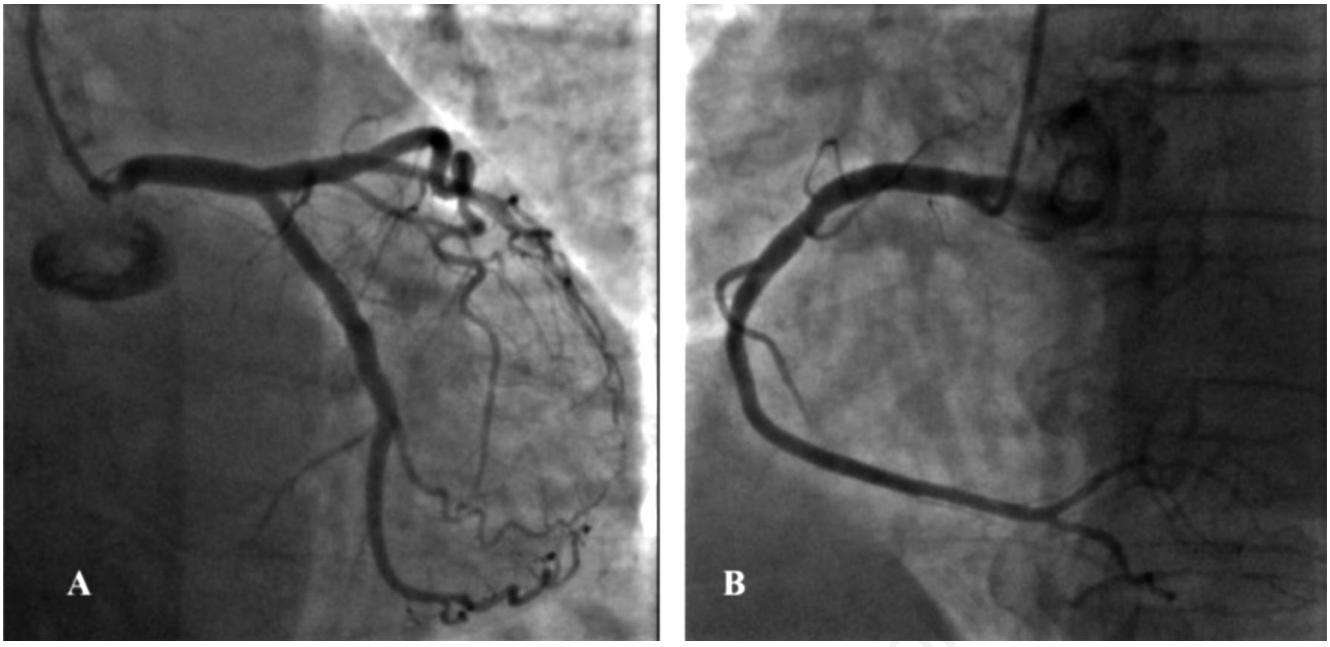

Figure 2. Coronary angiography with normal left (A) and right (B) coronary.

resolved instantly the ST-elevation. The coronary angiography showed no abnormalities. Omar *et al.* [8] described a case of ST elevation in the anterior leads during HUTT in a 57-year-old heavy-smoking man, without prior medical history, rapidly resolved after nitroglycerin therapy; serial cardiac markers were not elevated and coronary angiography revealed a nonocclusive 60% left anterior descending lesion. In both cases, the authors hypothesized that the underling mechanism was a coronary artery spasm [8].

The underling mechanisms of the HUTT-induced ST elevation is still debate, however the most likely cause seems to be the coronary vasoconstriction. HUTT-induced coronary spasm may be caused either by the acetylcholine direct stimulation of vascular smooth muscle of coronary arteries with endothelial damage or stenosis [9]; or by the augmented sympathetic activity which suddenly follows the HUTT induced parasympathetic tone [8]. Vasodilator drugs, as nitrates, usually reverse or prevent coronary artery spasm and its consequents [9].

Our case report is different from the previous reported in literature, because the ST elevation occurs after sublingual nitroglycerin administration during the hypotension and bradycardia related to the vasovagal response and it was accompanied by dizziness and angina.

Nitroglycerin can likewise markedly lower systemic vascular resistance, and may produce an appreciable reduction in coronary perfusion pressure. Such situation should be rare, since there would likely be a corresponding reduction in coronary vascular resistance, resulting in a facilitation of blood flow; however, nitroglycerin might have profound effects on diastolic blood pressure greater than the reduction in coronary vascular resistance, with the net effect of a decline in the transmyocardial coronary flow gradient and *paradoxical ischemia* [10]. Paradoxical vasospastic response to nitrates in vasospastic angina patients represents a rare but very challenging condition [5] and the best therapeutic approach in this subgroup of patients remains unclear [5,10].

Conclusions

ST segment anomalies during HUTT might be related to a different condition, pathological or not. Paradoxical vasospastic response to nitrates during HUTT represents a rare but challenging condition. Rapid acting nitrates are usually effective for a rapid control of anginal episodes, but in rare case might be a trigger for trans-mural ischemia. The underlying mechanisms are not known and any firm conclusion about the best treatment strategy in this situation and the management is not possible.

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