CASE REPORT

The head-up tilt test — a cause of myocardial infarction

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Abstract

Infarction

A 74-year-old man with no known ischaemic heart disease presented to the Cardiology Department with a history of multiple episodes of pre-syncpe. During a head-up tilt test to investigate a neurocardiogenic cause, after glyceryl trinitrate provocation he became profoundly hypotensive and unwell. Subsequent ECGs and Troponin-T levels confirmed a Non ST-Elevation Myocardial Infarction. Angiography confirmed coronary artery disease. This case highlights a rare complication of tilt testing and emphasises that the test is not without risk.

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Introduction

Head-up tilt testing is generally considered a useful diagnostic aid in unexplained pre-syncopal and syncopal attacks.

Despite its good safety profile, complications have been reported in the literature including malignant ventricular arrhythmias [1,2] and coronary vasospasm [3] although until now, these have only ever been recognized in association with isoprenaline provocation. We present a rare case of myocardial infarction induced by tilt testing potentiated with glyceryl trinitrate.

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Case report

A 74-year-old male was referred to the cardiology clinic by his primary care physician for investigation of four episodes of pre-syncpe over two years. These were not associated with chest pain, palpitations, collapse or neurological disturbance and all occurred whilst standing. He did have risk factors for cardiovascular disease specifically hypertension, hypercholesterolaemia and a history of heavy smoking. However, he was active and had never had symptoms clearly suggestive of myocardial ischaemia. His medication comprised 2.5 mg Bendrofluazide, 10 mg Simvastatin and 150 μg Thyroxine.

Cardiovascular examination and a resting ECG (Fig. 1) were normal. A subsequent 24 h Holter
monitor was unremarkable except for occasional isolated ectopic beats. A head-up tilt test was arranged according to the protocol we use [4]. This involves a supine stage of 10 min followed by a 60° head-up tilt with footboard support for a further 20 min. If negative, 400 μg glyceryl trinitrate (GTN) are then administered sublingually and monitoring is continued for a further 20 min. The heart rate and blood pressure are measured at 2 min intervals throughout the test and any symptoms noted.

He was hypertensive during the supine stage. On tilting, the sinus rate remained on average 85 beats/min but the systolic and diastolic blood pressures started to fall slowly with a trough blood pressure of 106/60 mmHg recorded at 16 min of tilt. The patient remained asymptomatic throughout. GTN was administered after 20 min which precipitated a rapid decrease in blood pressure to a trough blood pressure of 56/46 mmHg (VASIS Type 3) [5]. This was associated with symptoms of pre-syncope and sweating which he described to be similar in nature to his previous episodes and he was placed in a supine position. Precordial pain was not reported during any stage of the test. He maintained sinus rhythm between 70 and 90 beats/min but remained relatively hypotensive with a systolic blood pressure less than 100 mmHg for a further 10 min. An ECG now revealed new ST-segment depression in leads V4–V6 and new T wave inversion in leads I and aVL (Fig. 2). He was treated for an acute coronary syndrome with Aspirin and Low Molecular Weight Heparin. Serial cardiac enzymes revealed a peak CK 974 IU/L and Troponin-T 1.38 ng/ml and an ECG the following day showed the changes to have resolved (Fig. 3). A coronary angiogram demonstrated a totally occluded right coronary artery, mild calcified disease in the left anterior descending artery and a moderate stenosis in the left circumflex artery (Fig. 4). A subsequent exercise stress test was positive for ECG changes only at Bruce protocol stage 2 with asymptomatic ST-segment depression in the lateral leads.

He has been on Aspirin, Ramipril and Simvastatin and has remained free of symptoms of ischaemia and pre-syncope for three months.

Discussion

The head-up tilt test is a common diagnostic procedure to help identify and classify vasovagal syncope. Pharmacological agents such as isoprenaline or glyceryl trinitrate can be used as a provocative stimulus. GTN provocation has demonstrated a high degree of specificity and sensitivity when identifying the vasovagal origin of unexplained syncope [6,7] though the predictive accuracy has been questioned [8,9].
The procedure is non-invasive and generally very safe. However, ventricular fibrillation has been reported as a result of an isoprenaline provocation tilt test in a patient with known ischaemic heart disease [1]. These authors subsequently recommended that patients with ischaemia should not undergo an isoprenaline challenge. Variant angina [10] and inducible arrhythmias [11] secondary to electrolyte disturbances have also been reported with isoprenaline. Wang et al. [2] described a patient who developed severe coronary vasospasm during an isoprenaline tilt test.

To our knowledge myocardial infarction as a complication of tilt testing has not been reported.

**Figure 2** ECG post-tilt test demonstrating new ischaemic changes.

**Figure 3** ECG 24 h post-tilt test revealing resolution of previous ischaemic changes.
previously nor have complications of GTN provocation. We consider that the hypotension induced by tilt testing compounded by GTN administration induced a myocardial infarction in the area supplied by our patient’s diseased left circumflex coronary artery. The spontaneous pre-syncopes observed most likely reflected transient episodes of hypotension induced by myocardial ischaemia. Our case highlights the need for constant monitoring during and immediately after a tilt test. The careful selection of patients for such a test becomes important particularly if known risk factors for ischaemic heart disease are present.

References


Figure 4 Coronary angiogram demonstrating distal right coronary artery filling retrogradely and left circumflex artery stenosis.