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CASE REPORT

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ST-elevation in patient with vasovagal syncope

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Abstract

We describe a case of a 68-year-old man with known ischaemic heart disease, who passed out after nitroglycerine administration. He had triple-vessel ischaemic heart disease and had suffered from myocardial infarction about 17 years prior to this incident. An ECG revealed ST-segment elevation in the anterior leads. The elevations lasted at least 40 minutes and no longer than 60 minutes. No troponin I level elevation was detected in two subsequent blood samples. The probable causes of ST-segment elevation in this patient are discussed. In our opinion ST-segment elevation was caused by a combination of hypervagotonia and transient myocardial ischaemia. (Folia Cardiol. 2006; 13: 530–533)

Key words: vasovagal syncope, myocardial infarction, ST-elevation

Introduction

Syncope could be a symptom of a potentially life threatening disease. A standard 12-lead ECG should be obtained in each patient after syncope [1, 2]. Syncope or near syncope is one of the four major features of atypical presentation of acute coronary syndrome, the three others being dyspnea, diaphoresis and nausea or vomiting [3]. It should be considered that new ST-elevations in the ECG of an older patient with a history of diabetes mellitus or prior infarctions (in clinical circumstances that indicate a painless acute coronary syndrome) could strongly suggest cardiac ischaemia, and their presence for 20 minutes will lead to the suspicion of myocardial infarction [3, 4].

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Case report

A 70-year-old male had syncope in church. His wife, who accompanied him, revealed that 17 years ago he had a myocardial infarction and that one month previously he had coronary catheterisation and had been qualified for coronary artery by-pass grafting for triple-vessel disease. The echocardiographic assessment revealed no wall motion abnormalities and slightly enlarged left ventricular dimensions. In the past the patient had experienced several spells of syncope in church, the circumstances and clinical features of which were suggestive of vasovagal origin. His medication consisted of 150 mg aspirin, 4 mg perindopril, 40 mg simvastatin and 1.25 mg bisoprolol. In the church he had felt faint and his wife had administered 400 μg nitroglycerine sublingually. Immediately after nitroglycerine administration he had lost consciousness for a short time. On the arrival of the ambulance he regained consciousness but had no palpable pulse on the radial artery and was sweating. Despite profound weakness he was sitting in the pew, having refused lie down. He agreed to lie down on the stretcher

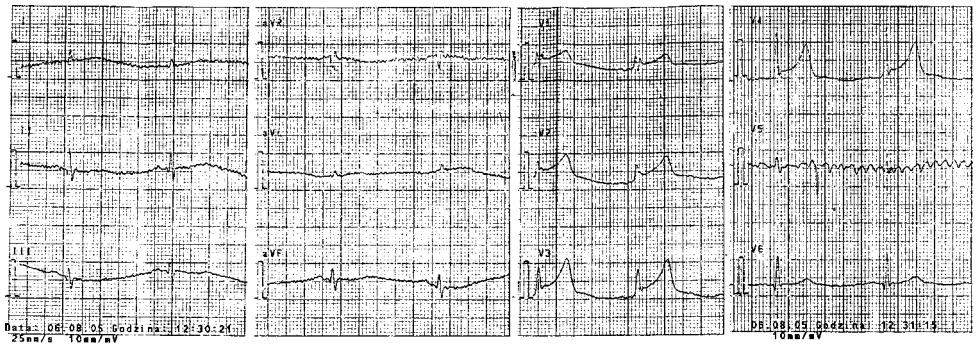

Figure 1. The first 12-lead ECG recording after arrival at the hospital.

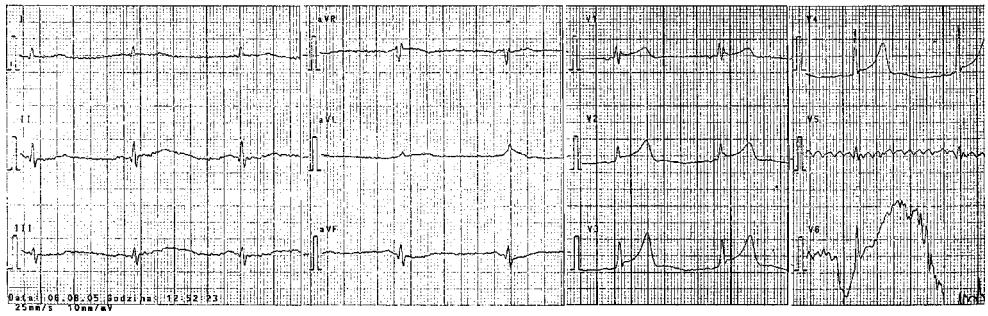

Figure 2. The ECG obtained immediately before transfer to the catheterisation unit.

and in the supine position the pulse on the radial artery was palpable. His blood pressure was 75/50 mm Hg and his heart rate, initially 38–40/min, immediately increased to 46/min. The patient was administered normal saline infusion. After arrival at the hospital a 12-lead ECG reading was obtained, which revealed ST-segment elevations in the anterior leads (Fig. 1). Systolic blood pressure was below 90 mm Hg, the patient hyperventilated up to 30 respirations per minute, suffered from nausea and vomited several times. Low molecular heparin, intravenous fluid infusion and dopamine infusion were administered.

The patient denied pain. The ST-segment elevations were still present when an ECG was obtained immediately before transfer to the catheterisation unit in a tertiary care hospital, approximately 40 minutes after syncope (Fig. 2). A third 12-lead ECG reading was obtained 20 minutes after the patient's arrival at the tertiary care hospital. The ST-segment elevations previously described had disappeared but very high T waves were observed in these leads (Fig. 3). The patient was not admitted to the tertiary care hospital but was referred to the internal diseases department. According to guidelines serial blood samples were taken to assess troponin I concentrations, all of which were within the normal range. Echocardiography revealed no differences in comparison with the study performed one month earlier. The global and local left ventricular systolic function was within the normal range. Unstable angina provoked by vasovagal syncope was diagnosed. One month later the CABG operation and mitral valve replacement were performed. One arterial (LIMA) and two saphenous vein by-pass grafts were performed.

Discussion

Structural heart disease is a major risk factor for sudden death and increases overall mortality rate in patients with syncope. If moderate to severe cardiac disease is found, the evaluation of syncope should be initially directed towards its cardiac cause [1]. Lack of pain does not exclude the presence of acute myocardial infarction, especially in elderly patients with a history of diabetes mellitus or prior myocardial infarctions. The symptoms and signs of dyspnea, weakness, syncope, sweating, hypotonia, nausea, vomiting and confusion may be features of painless myocardial infarction.

In our case, the clinical presentation initially suggested painless acute myocardial infarction with ST-segment elevation. While waiting for the

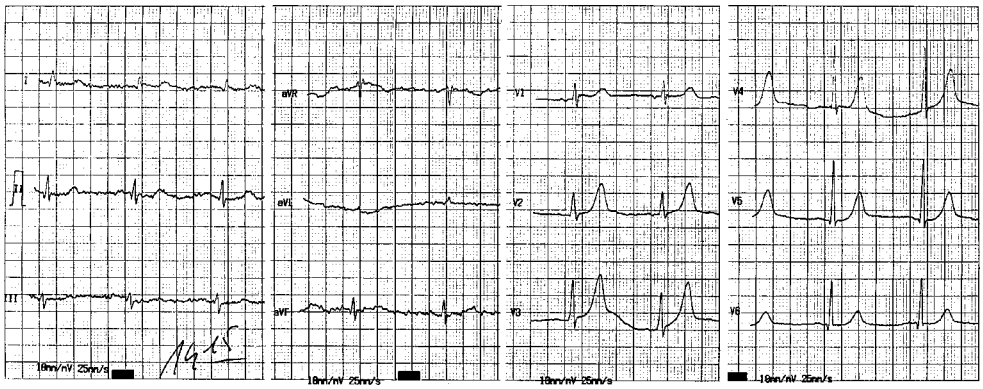

Figure 3. The ECG obtained in the tertiary care hospital — note the disappearance of ST-segment elevation.

ambulance transport to the catheterisation unit of the tertiary cardiac centre a 12-lead ECG was obtained which revealed electrocardiographic changes 40 minutes after syncope. In spite of the difficulties of transportation percutaneous cardiac intervention in this case could, if indicated, have been performed in a timely fashion with a balloon inflation goal within 90 minutes of presentation [5]. Further observation of the patient in the context of his history of previous spells of syncope, the circumstances of the present syncope and the laboratory findings enabled a spell of syncope of vasovagal origin to be diagnosed. The last syncope had provoked an episode of unstable angina.

ST-segment elevations during vasovagal syncope may be the consequence of cardiac ischaemia caused by vasospasm and thrombosis, a critical decrease of coronary flow due to arterial hypotonia or electrocardiographic signs of hypervagotonia. In the pre-syncope phase the reflex activation of the respiratory pump increases venous return and prevents or delays syncope, but hypocapnia and alkalosis due to hyperventilation may lead to coronary vasospasm and subsequent coronary thrombosis [1]. Coronary vasospasm may also be caused by vagal hyperactivity when the endothelium of the coronary arteries has been damaged. In such circumstances the balance between the direct vagal vasoconstrictive effect on the vascular musculature and the indirect vasodilative effect promoted by the endothelium is tipped towards vasoconstriction [6].

Hypervagotonia may also affect the repolarisation process and may lead to ST- segment elevation, as is frequently observed in healthy young people during the night hours. The heart rate and autonomic nervous system may modify the ST-segment elevation in the early repolarisation syndrome [7].

Moreover, it could not be ruled out that the decrease in coronary flow caused by prolonged arterial hypotonia, vascular stenosis and possible local thrombosis had led to cardiac ischaemia. The patient's decision to maintain the vertical position despite arterial hypotonia might have worsened the course of events. Transient electrocardiographic changes and haemodynamic disturbances without cardiac necrosis might have been a manifestation of this process.

The underlying mechanism of ST-segment elevation during myocardial ischaemia may include the transmural voltage gradient caused by depression or loss of the action potential dome or plateau amplitude in the epicardium, but not in the endocardium [7]. Transient ST-segment elevations have also been observed in patients after electrical cardioversion. In these cases it usually lasts shorter than in our patient and may be a consequence of impaired efferent sympathetic responsiveness [8].

Vasovagal syncope provoked by tilt testing may lead to myocardial infarction or transient ST-segment elevations, rapidly disappearing after nitroglycerine administration [9–12]. Myocardial infarction caused by hypotension due to tilt testing in a patient with no known coronary artery disease was described by Goolamali et al. [9], whose patient suffered from multiple episodes of pre-syncope and had no angina symptoms. After nitroglycerine provocation he became profoundly hypotensive. The ECG revealed ST-segment depression and new T-wave inversion. Serial assessment of the troponin T concentrations allowed non- ST-segment elevation myocardial infarction to be confirmed. The patient was without pain during the episode and the myocardial infarction was manifested clinically only by prolonged hypotension (systolic blood pressure below 100 mm Hg) and discomfort. A coronary angiogram revealed amputation of the right coronary artery and moderate stenosis of the left circumflex artery. Subsequent exercise testing was electrocardiographically positive but clinically negative [9].

Wang et al. [10, 11] described a 65-year-old man presenting morning pre-syncopes and chest discomfort. Diagnosis of vasospastic angina was confirmed by the ergonovine provocation test. During tilt testing ST-segment elevation occurred as an incidence of coronary vasospasm. Colman et al. [12] observed ST segment elevations during tilt testing in a 56-year-old female with a history of very frequent episodes of syncope with chest pain. The ECG changes and pain disappeared immediately after nitroglycerine administration. The authors suggested that the syncope episodes had been the consequences of vasovagal reactions and vasospastic angina. Coronary artery spasm may be evoked by carotid sinus stimulation. Such a case was described by Choi et al. [13] in a neck operation and further confirmed by angiography and ergonovine testing.

Arterial hypotension due to vasovagal syncope in a patient with ischaemic heart disease may lead to a critical decrease in the coronary flow through a narrowed coronary artery, local thrombosis and cardiac ischaemia. Maximal shortening of the arterial hypotension period should be the priority in the therapeutical treatment of these patients. Pharmacological treatment should be based on atropine, catecholamines and intravenous fluid infusions.

The case of our patient suggests that long-lasting ST-segment elevations after vasovagal syncope might be caused not only by cardiac ischaemia but also by hypervagotonia. On the other hand, ST-segment elevations after vasovagal syncope in a patient without known ischaemic heart disease may indicate a significant coronary stenosis.

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