A case is presented of significant reversible ST elevation occurring during treadmill testing, and the coronary anatomy and subsequent course are described, indicating that ischemia is a potential cause of this electrocardiographic finding.

Keywords: ST elevation; myocardial ischemia; stress testing; electrocardiography

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Many conditions can cause electrocardiographic (ECG) ST elevation. Significant ST elevation is defined by at least 1 mm elevation in the limb leads or 2 mm elevation in the precordial leads above the isoelectric point (1). With regard to myocardial infarction, diagnostic ST elevation in the absence of left ventricular hypertrophy or left bundle-branch block is defined by the European Society of Cardiology/ACCF/AHA/World Heart Federation Task Force for the Universal Definition of Myocardial Infarction as new ST elevation at the J point in at least two contiguous leads of >2 mm in men or 1.5 mm in women in leads V2–V3 and/or of >1 mm in other contiguous chest leads or the limb leads (2). In addition to ST elevation myocardial infarction, other causes include early repolarization, left ventricular hypertrophy, hypertrophic cardiomyopathy, ventricular aneurysm, left bundle-branch block, pericarditis, myocarditis, aortic dissection, Prinzmetal’s angina, Takotsubo cardiomyopathy, and Brugada syndrome (1, 3). Furthermore, non-cardiac conditions, such as pulmonary thromboembolism, pneumothorax, and atelectasis, can also present with similar ECG changes (1, 3). Thus, ST segment elevations are not specific for myocardial infarction and should be distinguished among the many potential causes. We present a case of exercise-induced ST segment elevation associated with atypical symptoms and caused by ischemia.

**Case**

A 75-year-old Caucasian male with a past medical history of hypertension, hyperlipidemia, and abnormal hemoglobin A1c (6.5) was found to have an abnormal ECG by his primary care physician on routine evaluation. His wife had noted that he appeared to have slowed down a bit, but he denied chest pain or exertional dyspnea.

Baseline ECG revealed poor R wave progression suggesting prior anteroseptal infarction (Fig. 1). The patient had no history of symptoms suggesting myocardial infarction. A stress echocardiogram was performed. During exercise, ST elevation developed in lead V3 and increased progressively over the next 3 min. The test was terminated at 7 min because of dyspnea and the ECG changes, at which time the heart rate had increased to 150 (Fig. 2). No chest pain or arrhythmias were noted. Both dyspnea and ST elevation resolved 4 min after cessation of exercise. No ST changes were noted in other leads. Physiologic pulse and blood pressure responses were noted: blood pressure rose from 130/70 to 160/75, and heart rate dropped from maximal rate of 150 down to 100 within 2 min after exercise.

Resting echocardiography revealed a mildly dilated left ventricle, normal wall thickness, and an ejection fraction of 50%, with mild antero-apical hypokinesis at rest, and dyskinesia immediately post-exercise. No other wall motion abnormalities were noted. Coronary angiography was advised.

The study revealed a 60–70% eccentric stenosis of the left main coronary artery, a 70% long proximal stenosis of the left anterior descending (LAD), followed by a 99% mid-LAD stenosis, with the distal vessel being filled by right-sided collaterals. Additionally, a small LAD diagonal was totally occluded, and the circumflex had a 70% heavily calcified obtuse marginal branch stenosis. The right coronary artery (RCA) revealed minimal
disease. Left ventriculography revealed antero-apical hypokinesis, with an ejection fraction by this modality being 45%.

Coronary artery bypass grafting was performed, with a left internal mammary graft to the LAD and saphenous vein grafts to two obtuse marginal branches. Postoperative course was uneventful. Follow-up echocardiogram 4 months after coronary artery bypass grafting revealed resolution of the resting wall motion abnormality, with normalization of the ejection fraction to 55%.

**Discussion**

ST-segment elevations on ECG are observed in acute myocardial infarction but also in severe ischemia, ventricular aneurysm, Prinzmetal’s variant angina, and abnormal cardiac hemodynamics (4, 5). This patient’s ECG demonstrated ST elevation in one lead only and was reversible after exercise was terminated, thus not fulfilling the criteria for myocardial infarction.

Most patients with exercise-induced ST elevation have significant proximal coronary obstruction supplying the
area subtended by the narrowed vessel (6). However, Specchia et al. described a group of 16 patients with exercise-induced ST elevation, without a history of infarction or ventricular aneurysm, and proposed spasm as a potential cause. Only seven of those patients underwent diagnostic catheterization, however, and they had no significant obstructive disease, only ergonovine-induced spasm (7).

This patient had multi-vessel disease involving the LAD, left main coronary artery, and circumflex, which resulted in marked myocardial ischemia, with subsequent focal ST segment elevation in V3. Miyakoda et al. found ST segment elevation in 93% of their study population, who had 99% stenosis and poor collateral circulation (8). They concluded that ST segment elevation is a good indicator of severe ischemia and poor collateral circulation. Several other case reports also found rare cases of ST elevations in non-Q wave leads during exercise stress testing that accurately predicted the presence of coronary stenoses (9–11).

This patient also had antero-apical hypokinesis at rest, prior to the development of ST elevation during stress testing, and worsening of the wall motion abnormality with exercise. Macieira-Coelho et al. described the concomitant presence of ST segment elevation on stress testing along with left ventricular wall motion abnormalities in 85% of their patient population (5). Miyakoda et al. concluded through their findings that ST segment elevation was not always related to myocardial ischemia but rather to wall motion abnormalities (8).

The patient did have collateralization from the circumflex to the distal LAD, but the presence of significant circumflex stenosis meant that this collateralization was insufficient. The absence of sufficient collateral circulation likely led to severe ischemia, which in turn most likely led to hibernating myocardium, with worsening ischemia occurring with exercise, leading to additional wall motion abnormalities.

Hibernating myocardium has the potential to be reversed once blood flow has been restored to normal, with the possibility of complete return to normal of myocardial function (11, 12). Revascularization appears to have successfully accomplished this in this patient.

In conclusion, we present an example of exercise-induced ST elevation, in a patient with presumably hibernating myocardium, with resolution of wall motion abnormalities with revascularization.

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