Transient peaked T waves during exercise stress testing: an unusual manifestation of reversible cardiac ischemia

Marian F. Manankil, MD,* Theodore Wang, MD, Pachalla K. Bhat, MD
Section of Cardiology, Advocate Illinois Masonic Medical Center, Chicago, IL, USA

Abstract
A 57-year-old man presented with atypical upper body pain, initially attributed to musculoskeletal etiology. After analgesic failure, an exercise myocardial perfusion imaging was performed. During stress testing, patient’s pain was reproduced, accompanied by prominent T-wave peaking with minor J-point elevation. T-wave amplitude decreased at the end of the recovery phase when his chest pain completely resolved. The myocardial perfusion imaging revealed extensive reversible ischemia of the septum and apical walls. Subsequent coronary arteriography demonstrated a 99% stenosis of the left anterior descending artery that was stented. Patient has remained asymptomatic since. We conclude that transient peaked T waves with minor J-point elevation during exercise may be an unusual electrocardiographic manifestation of reversible cardiac ischemia.

Keywords: Peaked T-waves; Electrocardiogram in stress test

The case
A 57-year-old Hispanic man with no significant medical history presented to his physician with intermittent right-sided pain, numbness, and weakness for the past 2 weeks, reproducibly occurring 4 minutes after walking with prompt resolution with rest. The pain started from his right parietal area and usually radiated down to the right neck, shoulder, and upper extremity. Occasionally, it radiated to the right hemithorax and left shoulder. On physical examination, the patient had mild tenderness of the right neck and partial reproduction of pain after shoulder abduction. His symptoms were initially thought to be musculoskeletal in origin, and he was prescribed naproxen that was ineffective. An exercise myocardial perfusion imaging was then performed.

The resting electrocardiogram (EKG) showed sinus bradycardia and was otherwise normal (Fig. 1A). The patient underwent exercise stress testing using the Bruce protocol for 10 minutes and 6 seconds and reached a peak heart rate of 145 beats per minute (89% of the maximum predicted heart rate). At the end of stage 3, the patient complained of right-sided chest tightness similar to his presenting symptoms. At stage 4, the chest pain became more severe and symptom limiting, and radiated to include the left side of the chest. The treadmill test was then terminated. His chest pain resolved within 3 minutes into recovery. Occasional isolated supraventricular ectopic beats were seen during exercise. Overall, the stress and recovery EKGs were not consistent with reversible ischemia using current criteria. However, the patient developed prominent peaked T waves with minor J-point elevation, which started during stage 3 concurrent with the onset of chest pain that gradually resolved within 10 minutes after exercise was stopped. The T-wave amplitude in leads V2 through V4 increased to 10 to 12 mm; and in the inferior leads, 7 to 9 mm (Fig. 1B-D). The electrocardiogram subsequently normalized (Fig. 1E) in close concert with the time course for resolution of his symptoms. The myocardial perfusion imaging demonstrated extensive reversible ischemia in the septum and apical walls of the left ventricle. The left ventricular ejection fraction was preserved (61% at stress and 59% at rest, Fig. 2).

The patient was advised to see his primary care physician as soon as possible. At his physician’s clinic a week later, the patient again developed chest tightness and was sent to the emergency department. Serial cardiac enzymes were normal, and the serum potassium was 4.2 mEq/L. An admission EKG was obtained when the pain had resolved and was normal.
Coronary arteriography was performed on the second day of admission. There was a 99% stenosis at the mid left anterior descending (LAD) artery just distal to the takeoff of the first diagonal branch (Fig. 3). The left main, left circumflex, and right coronary arteries were angiographically normal. The left ventriculography revealed normal ejection fraction with no regional wall motion abnormalities, and the left ventricular end-diastolic pressure was 20 mm Hg. Percutaneous coronary intervention of the LAD lesion was then performed using 2 drug-eluting coronary stents (Fig. 4). There were no procedural complications, and he was discharged the following day. He has remained asymptomatic during 7 months of follow-up.

Discussion

We have presented a patient in whom compelling evidence suggests that transient peaked T waves and minor J-point elevation, and not ST-segment depression, proved to be electrocardiographic manifestations of reversible cardiac ischemia, inasmuch as his clinical symptoms were reproduced with exercise, the onset and resolution of chest pain and EKG abnormalities were temporally related, and his symptoms have resolved after successful coronary intervention.

The current American College of Cardiology and American Heart Association requirements for a positive exercise test result is greater than or equal to 1 mm of horizontal or down-sloping ST-segment depression or elevation for at least 60 to 80 milliseconds after the end of the QRS complex. Typically, the ST segment of patients with myocardial ischemia becomes more horizontal as the severity of the ischemic response worsens. With progressive exercise, the depth of ST-segment depression may increase and involve more EKG leads; and the patient may develop angina. In the immediate postrecovery phase, the ST-segment depression may persist, with down-sloping ST segments and T-wave inversion, gradually returning to baseline after 5 to 10 minutes.
To date, there have been relatively few studies that have analyzed the T-wave changes that may occur with stress testing. In 1995, Lee et al. evaluated the predictive power of exercise-induced increased T-wave amplitude in the right precordial leads (V_1 through V_4) in detecting myocardial ischemia. Patients with LAD lesions had higher T-wave amplitudes after exercise than during rest in V_1 through V_4, but the greatest T-wave amplitude increase was observed in lead V_2. An exercise-associated T-wave increase of at least 2.5 mm was found to have a 95% specificity for reversible ischemia.

Fig. 2. Myocardial perfusion imaging with gated stress, stress, and rest images. A, Short-axis view of the left ventricle showing reversible defect in the septal wall. B, Vertical long-axis view revealing apical reversible ischemia. C, Horizontal long-axis view showing reversible defect in the apical and septal walls. Areas with reversible ischemia are denoted with arrows.

Fig. 3. Diagnostic coronary angiography images shown in right anterior oblique (RAO) caudal (A, B), left anterior oblique (LAO) caudal (C) and LAO cranial (D). Various projections of the left coronary artery showing the subtotal mid-LAD occlusion (arrows). The rest of the LAD showed mild luminal irregularities. The left main and left circumflex arteries were angiographically normal.
ischemia. They suggest that, although this finding may occur only occasionally, it may aid in the diagnosis of patients who develop this abnormality, in the absence of ST-segment depression. Ellestad observed peaked T waves in the precordial leads, most pronounced in leads V2 through V4, during treadmill exercise testing in a patient who had an anterior wall acute myocardial infarction several days later. Madias and Agarwal described a patient with coronary artery disease who developed transient ST-segment depression, right bundle-branch block, left anterior hemiblock, ST-segment elevation, and “giant” T waves that persisted late in the recovery period during the exercise stress test. An angiogram revealed a stenotic LAD. Our patient exhibited similar findings with peaking of the T waves in V2 through V4 associated with significant stenosis of the LAD.

The mechanism of this observation may be extrapolated from the finding of hyperacute T waves (defined as an amplitude ≥50% of the R wave in the same lead in ≥2 contiguous leads) seen in the earliest stage of an evolving transmural infarct. Studies involving ligation of a coronary artery have shown that hyperacute T waves may develop as soon as 2 minutes after disrupting coronary flow. The explanation for this is unclear, although it has been proposed that tall T waves may reflect a localized increase in plasma potassium concentration secondary to myocardial ischemia or possibly due to prolonged repolarization in ischemic tissues. In the very early phases of myocardial ischemia, the first area to be affected is the subendocardium, being farthest from the blood supply. This may lead to a delay in repolarization of the subendocardium that may appear on the surface EKG as tall hyperacute T waves.

We conclude that, in some patients, transient peaked T waves with minor J-point elevation during exercise may be an unusual manifestation of reversible cardiac ischemia.

References