

Coronary angiographic significance of hyperacute ST-T changes associated with regadenoson stress

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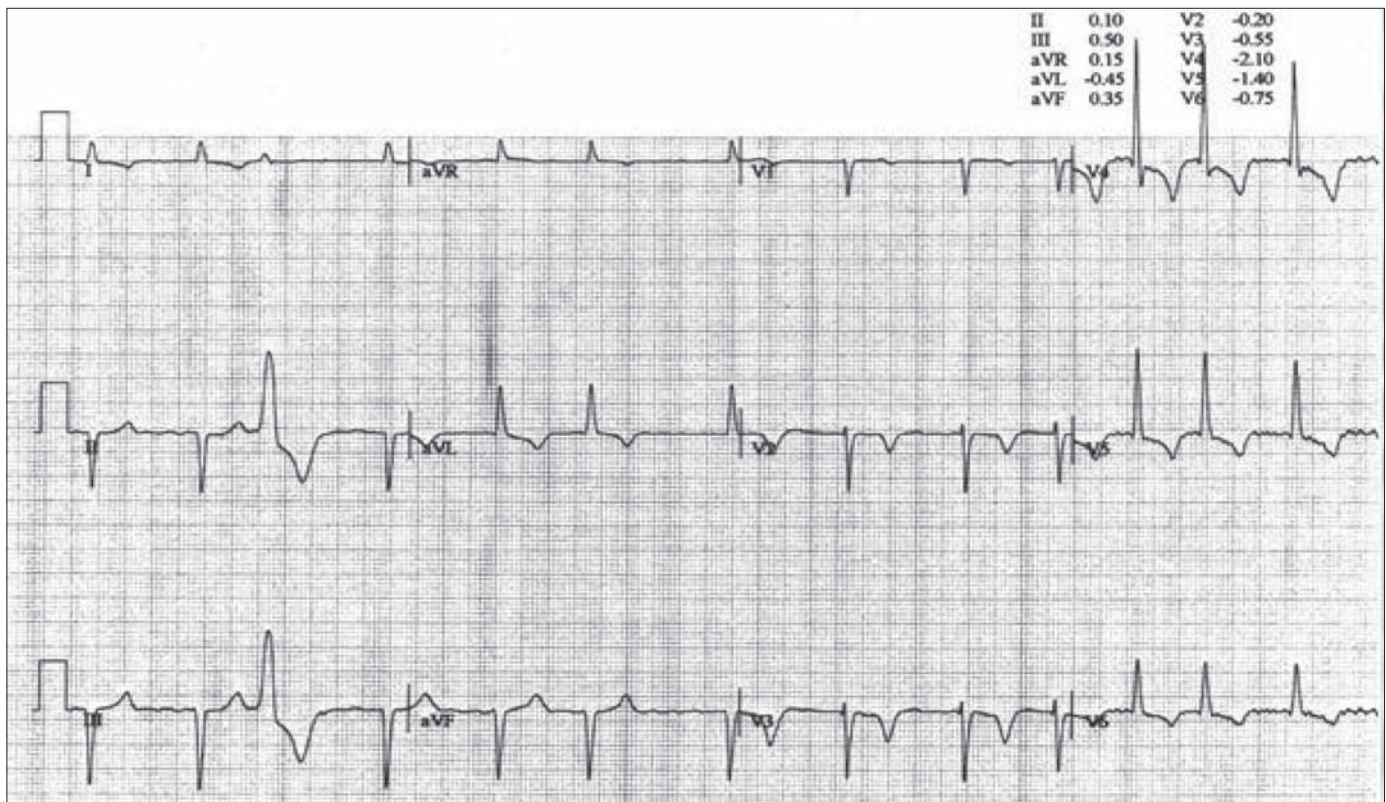


Figure 1. Admission electrocardiogram demonstrating symmetric T-wave inversions across the precordial (V_1 – V_6) leads.

An abnormal electrocardiographic stress test is typically characterized by ST segment depression. In rare cases, ST segment elevation is observed, which, in the absence of diagnostic Q waves, has anatomic specificity for localized myocardial ischemia. Most instances of ST elevation occurring during cardiac stress testing have been observed with exercise, with only six cases reported with pharmacologic stress. Despite different physiologic mechanisms for inducing myocardial ischemia, development of ST segment elevation during pharmacologic stress, as illustrated by the present case, may also be indicative of critical coronary stenoses, warranting urgent coronary arteriography.

CASE PRESENTATION

An 87-year-old man presented with a 1-month history of worsening intermittent “burning” epigastric pain that radiated

to his substernal area. The pain was associated with nausea, eructations, and a bad taste in his mouth, but he denied accompanying dyspnea or diaphoresis. The patient had received a drug-eluting stent approximately 12 years earlier. Subsequent cardiac catheterization 2 years prior to the present episode revealed a patent stent and no new coronary narrowing. His initial troponin I level was <0.05 g/dL, and his electrocardiogram revealed symmetric T wave inversions across the precordium (*Figure 1*). Given the atypical nature of his symptoms, the

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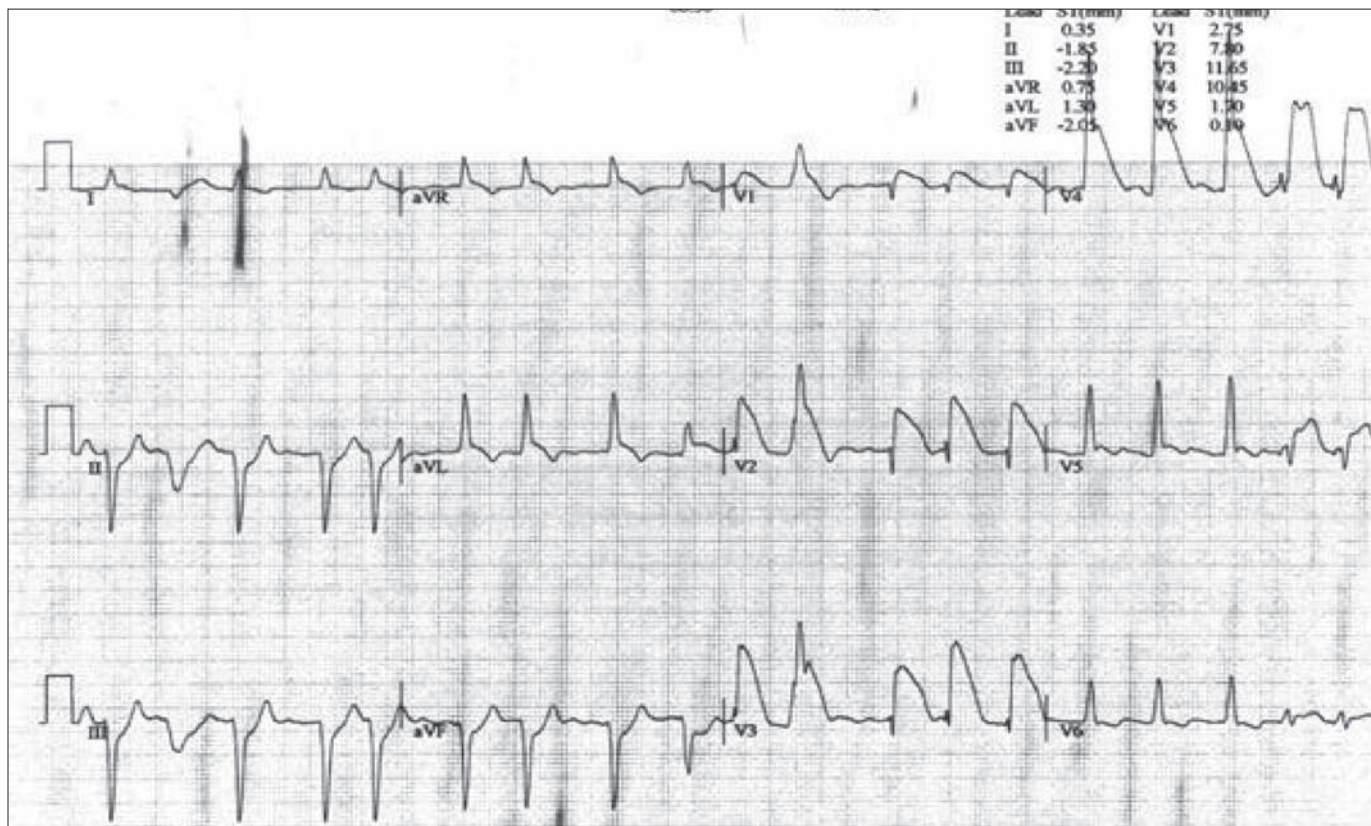


Figure 2. Electrocardiogram following the administration of regadenoson revealing new-onset ST segment elevation and hyperacute T waves in leads V₂ to V₄.

patient underwent a pharmacologic nuclear stress test using regadenoson, a vasodilator similar to adenosine. Following the intravenous administration of 0.4 mg (5 mL) of regadenoson, the patient began complaining of “not feeling well,” and he was given 100 mg (4 mL, 25 mg/mL) of intravenous theophylline. Initial imaging demonstrated a large area of moderately reduced perfusion involving the ventricular septum, left ventricular distal anterior and inferior walls, and apex. Approximately 8 minutes into recovery, sudden severe substernal pain appeared. Electrocardiogram revealed ST segment elevation and hyperacute T waves in leads V₂ to V₄ (Figure 2). The procedure was terminated immediately and the patient was taken to the cardiac catheterization laboratory. Serum drawn at that time revealed that the troponin I had risen to 0.3 ng/mL. Coronary arteriography revealed a long ulcerated lesion in a large left anterior descending coronary artery just beyond the takeoff of the first diagonal. A drug-eluting stent was placed across the stenotic area without incident, and the patient had an uneventful recovery with complete resolution of his symptoms. He was asymptomatic when seen in follow-up a month after the procedure.

DISCUSSION

Exercise stress testing is frequently limited by the inability of individuals to elevate their heart rate to levels likely to induce myocardial ischemia. Accordingly, pharmacologic stress tests, most often using vasodilators, are employed as a substitute for the traditional exercise stress test. Exercise increases myocardial oxygen demand by increasing heart rate and blood pressure and produces ischemia in areas distal to coronary artery stenosis. In

contrast, vasodilator pharmacologic stress tests cause myocardial ischemia by an entirely different mechanism. The pharmacologic agents (dipyridamole, adenosine, or regadenoson) promote coronary vasodilatation through activation of A_{2A} (primarily) and A_{2B} adenosine receptors. Perfusion is increased to healthy well-perfused areas and, conversely, “stolen” away from the stenotic areas, which are already maximally vasodilated.

ST segment depression is the most common electrocardiographic abnormality encountered with stress testing, whether exercise or pharmacologic, and is thought to be indicative of subendocardial ischemia. Conversely, exercise-induced ST segment elevation is rare, occurring in 1.3% of patients (1), and may denote transmural ischemia. Exercise-induced ST segment elevation may also occur in the absence of ischemia in areas of previous myocardial infarction (with diagnostic Q waves and accompanying wall motion abnormalities) (2–4). In contrast to stress-induced ST segment depression, which tends to occur in the inferolateral electrocardiographic leads regardless of the site of coronary artery obstruction, exercise-induced ST segment elevation is usually localized to the area of ischemia (5, 6). Vasodilator stress-induced ST segment elevation appears to be especially rare; review of the medical literature produced only two cases using dipyridamole (2, 7), three cases using adenosine (8–10), and one recently reported case using regadenoson (11). In all but one of these studies, coronary arteriography confirmed $\geq 75\%$ stenoses in at least one major coronary artery. It has been hypothesized that the lone case without significant coronary artery disease may have been due to overexpression of A₁ and A₃ adenosine receptors (in comparison to A₂ receptors), which

have been shown to play an inhibitory role in the regulation of coronary blood flow (12, 13). Our case is consistent with the limited experience in the literature and suggests that ST segment elevation in response to vasodilator stress may indicate a critical lesion requiring emergent coronary intervention. Our experience also emphasizes the need for continued close monitoring of these individuals until definitive measures can be taken.

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