Acute Anteroseptal Myocardial Infarction after a Negative Exercise Stress Test

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Abstract: A myocardial infarction is a rare complication which can occur after an exercise stress test. We report a 48-year-old male who was referred to the Mildura Cardiology Practice, Victoria, Australia, in August 2014 with left-sided chest pain. He underwent an exercise stress test which was negative for myocardial ischaemia. However, the patient presented to the Emergency Department of the Mildura Base Hospital 30 minutes after the test with severe retrosternal chest pain. An acute anteroseptal ST segment elevation myocardial infarction was observed on electrocardiography. After thrombolysis, he was transferred to a tertiary hospital where coronary angiography subsequently revealed significant left anterior descending coronary artery stenosis. Thrombus aspiration and a balloon angioplasty were performed. The patient was discharged three days after the surgical procedure in good health.

Keywords: Myocardial Infarction; Exercise Test; Physical Exertion; Coronary Artery Disease; Atherosclerotic Plaques; Case Report; Australia.

Cardiovascular diseases (CVDs) are the leading cause of mortality worldwide, with coronary artery disease (CAD) causing more than 40% of all CVD-related deaths. More than 600,000 Australians are affected by CAD and it is the most common cause of death among people over 45 years old. For the investigation of chest pain, an exercise stress test (EST) is commonly performed and is relatively safe and inexpensive. However, this test can be associated with rare but serious complications, as described in this case.

Case Report

A 48-year-old male was referred for an EST to the Mildura Cardiology Practice, Victoria, Australia, in August 2014 to investigate a single episode of chest pain which had occurred two weeks previously. The chest pain was dull, non-radiating, located in the left anterior region and was not associated with breathlessness. It had resolved spontaneously within 30 minutes and no history of exertional chest pain or dyspnoea was reported. The patient had stable bronchial asthma and was using an inhaled steroid; however, this was the only regular medication taken at the time of referral. He was morbidly obese and had a sedentary lifestyle. Aside from the obesity, an examination of the patient was unremarkable. A resting electrocardiogram (ECG) showed a normal sinus rhythm. The patient underwent an EST utilising the Bruce protocol. He was able to exercise for nine minutes yielding 10.3 metabolic equivalents, indicating a reasonable workload. He reported no chest pain and the test was terminated due to patient fatigue. His peak heart rate was 168 beats per minute (97% of the predicted maximum) and his peak blood pressure was 220/110 mmHg. No ST segment abnormalities occurred during the EST or within the post-exercise recovery period, indicating the absence of ischaemia [Figure 1]. Unfortunately, 15 minutes after leaving the clinic and 30 minutes after completing the EST, the patient...
experienced a sudden onset of severe retrosternal chest pain. He subsequently presented to the Emergency Department of the Mildura Base Hospital. An ECG showed a ST segment elevation of >2 mm in the I, augmented voltage left arm (aVL) and V1–V3 leads, suggesting an anterior myocardial infarction. As such, 50 mg of tenecteplase was administered as thrombolytic therapy. However, the severe chest pain and ST segment elevation persisted 90 minutes post-thrombolysis. The patient was transferred via air ambulance to a tertiary hospital for an emergency coronary angiography. On arrival, he was free of chest pain and the angiography was not immediately performed. An ECG showed the development of Q waves in the anterior leads and persistent ST segment elevation in the I, aVL and V2 leads [Figure 2]. A transthoracic echocardiogram showed that the left ventricle was normal in size with moderate segmental dysfunction. Akinesis of the anterior septum, apex and anterior wall indicated that the infarction was extensive.

Later that night, the patient experienced further episodes of chest pain but did not undergo another ECG. Coronary angiography was performed 20 hours after the occurrence of the post-EST chest pain. This revealed a proximal thrombotic occlusion in the left anterior descending (LAD) coronary artery [Figure 3A] and significant disease in the right coronary artery [Figure 3B]. Thrombus aspiration and a balloon angioplasty with stenting were performed for the proximal and mid-LAD coronary artery. The jailed diagonal branch was balloon-dilated although the right coronary artery was left as it was [Figure 3C]. The final angiographic appearance of the LAD coronary artery.
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Various degrees of coronary artery stenosis underwnt coronary angiography which confirmed of the patients received thrombolytic therapy and occurring within 30 minutes of a negative test; all reported three cases of acute myocardial infarction after a negative EST are extremely rare. Capezzuto et al. found that 4.4% of 1,228 patients with myocardial infarctions underwent heavy physical exercise one hour prior to the onset of the infarction.8 It has a reported sensitivity of 68% and specificity of 77% for detecting haemodynamically-significant CAD.4 While ESTs are usually considered safe, rare complications can occur, including sudden cardiac death (0.005%–0.01%), serious arrhythmias (0.2%) or acute myocardial infarctions (0.04%).7 Previous studies have reported a relationship between strenuous exercise and acute myocardial infarctions; Mittleman et al. found that 4.4% of 1,228 patients with myocardial infarctions underwent heavy physical exercise one hour prior to the onset of the infarction.5

Acute myocardial infarctions occurring shortly after a negative EST are extremely rare. Capezzuto et al. reported three cases of acute myocardial infarction occurring within 30 minutes of a negative test; all of the patients received thrombolytic therapy and underwent coronary angiography which confirmed various degrees of coronary artery stenosis.7 Zargar et al. reported a 51-year-old male who developed an inferior wall myocardial infarction shortly after a negative EST; the patient was treated with thrombolytic therapy and a coronary angiography carried out a few weeks later revealed mid-right coronary artery stenosis of 60–70%.8 Kurata et al. described a 67-year-old man with an acute myocardial infarction occurring 10 minutes after a negative EST with total occlusion of the LAD coronary artery on coronary angiography.11 Intracoronary thrombolysis resulted in the successful opening of the occluded artery without any significant residual stenosis. Kurata et al. speculated that a coronary artery spasm at the site of a minor plaque may have triggered the intimal disruption, leading to intraluminal thrombus formation.11 According to Cadroy et al., only strenuous exercise leads to an increased risk of thrombosis in men, as opposed to moderate exercise; furthermore, patients with underlying CAD have a higher risk compared to patients with normal coronary arteries.12 As such, the authors of the present case report recommend that patients be advised of potential complications and provide informed consent before undergoing an EST. In addition, treating physicians should emphasise the importance of seeking immediate medical help if patients begin to experience any unexpected symptoms after a negative EST, particularly chest pains.

According to the available literature, there are several possible causes of an acute myocardial infarction after a normal EST. Atherosclerotic plaque ruptures can precipitate intraluminal thrombus formation or a patient can haemorrhage into a plaque, leading to luminal occlusion.9,12,13 Coronary artery spasms induced by exercise can occur at the site of a relatively minor atherosclerotic plaque, with subsequent intraluminal thrombosis. Finally, strenuous exercise in sedentary patients can trigger platelet activation, contributing to the occurrence of coronary thrombosis.9,12,13 The coronary angiogram of the patient outlined in the present case report showed complete thrombotic occlusion of the LAD coronary artery with underlying stenoses. One possible explanation for this is that the exercise during the EST caused plaque rupture, which then precipitated an intraluminal clot formation and an acute myocardial infarction. In addition, the patient’s sedentary lifestyle may have facilitated platelet activation.

Discussion

Coronary angiography is the gold-standard method for assessing CAD; however, it is expensive, invasive and carries the risk of significant complications.7 An EST is a recommended screening test for patients with a low or intermediate probability of developing CAD.4 It has a reported sensitivity of 68% and specificity of 77% for detecting haemodynamically-significant CAD.4,5 While ESTs are usually considered safe, rare complications can occur, including sudden cardiac death (0.005%–0.01%), serious arrhythmias (0.2%) or acute myocardial infarctions (0.04%).7 Previous studies have reported a relationship between strenuous exercise and acute myocardial infarctions; Mittleman et al. found that 4.4% of 1,228 patients with myocardial infarctions underwent heavy physical exercise one hour prior to the onset of the infarction.5

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Conclusion

As they are relatively safe, inexpensive and readily available, ESTs remain widely-used non-invasive methods of investigating chest pain and diagnosing CAD. However, a negative test does not exclude significant or minor atherosclerotic CAD. This can
result in a false-negative test or rare but potentially life-threatening complications, as demonstrated in the present case. As a result, patients should be warned about these potential complications and provide informed consent before undergoing the test. Furthermore, patients should be advised to seek immediate medical attention if any unexplained symptoms, particularly chest pains, occur after a negative EST.

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