Acute myocardial infarction in a young man:
A consequence of inherited thrombophilia and marijuana smoking


1Department of Internal Medicine and 2Department of Pathology and Laboratory Medicine, American University of Beirut Medical Center, Beirut, Lebanon

Dear Sir,

Acute myocardial infarction (MI) in young patients is rare. Marijuana smoking increases the risk of acute cardiovascular events (1). Our patient is a 24-year-old man, previously healthy, who presented to the emergency department with oppressive chest pain of four hours duration. The pain was severe in intensity and radiating to the arms and back and was not associated with palpitations or sweating. The patient was a 16 pack year smoker. His grand mother had coronary artery disease at the age of 65 years; there was no family history of stroke or venous thromboembolism. He was physically active and denied any drug intake. His blood pressure was 130/80 mm Hg, pulse was 84, regular, and his temperature was 36.5°C. The electrocardiogram showed sinus rhythm with ST elevation in leads V2 to V6 with reciprocal ST segment depression in II, III and aVF leads. Chest X-ray was normal. Laboratory tests on admission showed elevated troponin-T of 18,090 ng/ml (normal 0.0–0.1 ng/ml) and creatine phosphokinase (CPK) of 7266 IU/l (normal 10–195 IU/l). The patient was diagnosed with antero-lateral MI and he received clopidogrel, aspirin, heparin and thrombolysis with 1.5 million units of streptokinase. Chest cineangiogram done on the same day of admission revealed that the left anterior descending (LAD) coronary artery was totally occluded proximally with a thrombus. Flow was established with a guiding wire and stent at the site of the occlusion.

Drug screen testing was positive for delta9-tetrahydrocannabinol (THC). The patient admitted that he had been smoking marijuana. An extensive genetic testing for acquired and inherited thrombophilia was done. The patient was found to be heterozygous for factor V Leiden, methylenetetrahydrofolate reductase (MTHFR) C677T and A1298C mutations. He was discharged seven days after admission in a stable condition on aspirin, plavix and statins. He was educated about his hypercoagulable state and he is being followed up by his primary physician.

Marijuana smoking is increasing in the United Kingdom and the United States. Data from Lebanon showed that 17% of students admitted to having used marijuana at least once (2). MI as a consequence of marijuana smoking in young individuals is not common (3). The factor V Leiden mutation is the most common established genetic risk factor for venous thrombosis, especially in the young (4). It may also predispose to arterial thrombosis. The risk of suffering an acute myocardial infarction was increased by 40% in heterozygote carriers of factor V Leiden mutation (5). The prevalence of factor V Leiden mutation is around 14.4% in the Lebanese population (6).

Our patient was a moderate-risk young individual who presented acutely with a classical myocardial infarction after smoking marijuana. He suffered a premature myocardial infarction in the presence of normal coronary arteries suggesting a thrombophilic tendency. The combination of factor V Leiden mutation and the MTHFR polymorphism can be expected to increase the risk of coronary artery thrombosis in our case. Smoking may have predisposed this patient to coronary spasm and subsequent thrombosis. Clinicians should consider testing for inherited thrombophilia in young patients with MI, especially when they are known to use marijuana.

References