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Clinical Communications: Adults

CANNABIS-ASSOCIATED MYOCARDIAL INFARCTION IN A YOUNG MAN WITH NORMAL CORONARY ARTERIES

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□ **Abstract—Background: The use of cannabis is not usually regarded as a risk factor for acute coronary syndrome. However, several cases of myocardial infarction (MI) associated with cannabis use have been reported in the scientific literature. The etiology of this phenomenon is not known. Objectives: To present a case of cannabis-associated MI in which atherosclerotic coronary disease was excluded as a potential etiology by intravascular ultrasound examination, and briefly review the other possible mechanisms by which this effect may be mediated. Case Report: We present the case of a previously healthy 21-year-old man who regularly smoked cannabis and presented to the Emergency Department with ST-elevation myocardial infarction after participating in a sport. He was also a cigarette smoker, but had no other conventional cardiovascular risk factors. At coronary angiography, a large amount of thrombus was found in the left anterior descending coronary artery. He recovered with medical treatment, and subsequent intravascular ultrasound examination showed no evidence of atherosclerosis at the site of the thrombus. Conclusion: Cannabis-associated MI is increasingly recognized. The etiology is unclear, but we believe this is the first report of the phenomenon where atherosclerotic plaque rupture has been excluded as the cause with a high degree of confidence.** © **2014 Elsevier Inc.**

□ **Keywords—cannabis; myocardial infarction; coronary thrombosis**

INTRODUCTION

Cannabis is the most commonly used illicit drug in the United Kingdom. Adverse effects of cannabis smoking are traditionally thought to be mild. We report a case of acute myocardial infarction (MI) in a previously healthy young man, which we believe was caused by his regular use of cannabis, and discuss other similar published cases and potential mechanisms by which cannabis may trigger MI.

CASE REPORT

A 21-year-old man presented to the Emergency Department (ED) with a 30-min episode of sharp left-sided chest pain that started after a game of soccer. He did not describe any other symptoms and had no past medical history. He was a current cigarette smoker and also smoked cannabis on a regular basis. Aside from this, he had no other cardiovascular risk factors, and though he had used cocaine 4 months previously, strenuously denied any other current or recent illicit drug use. The patient had presented to the same department 1 month prior with similar symptoms, again after a game of soccer. Basic investigations, including an electrocardiogram (ECG), at that time were normal and he was diagnosed with musculoskeletal pain.

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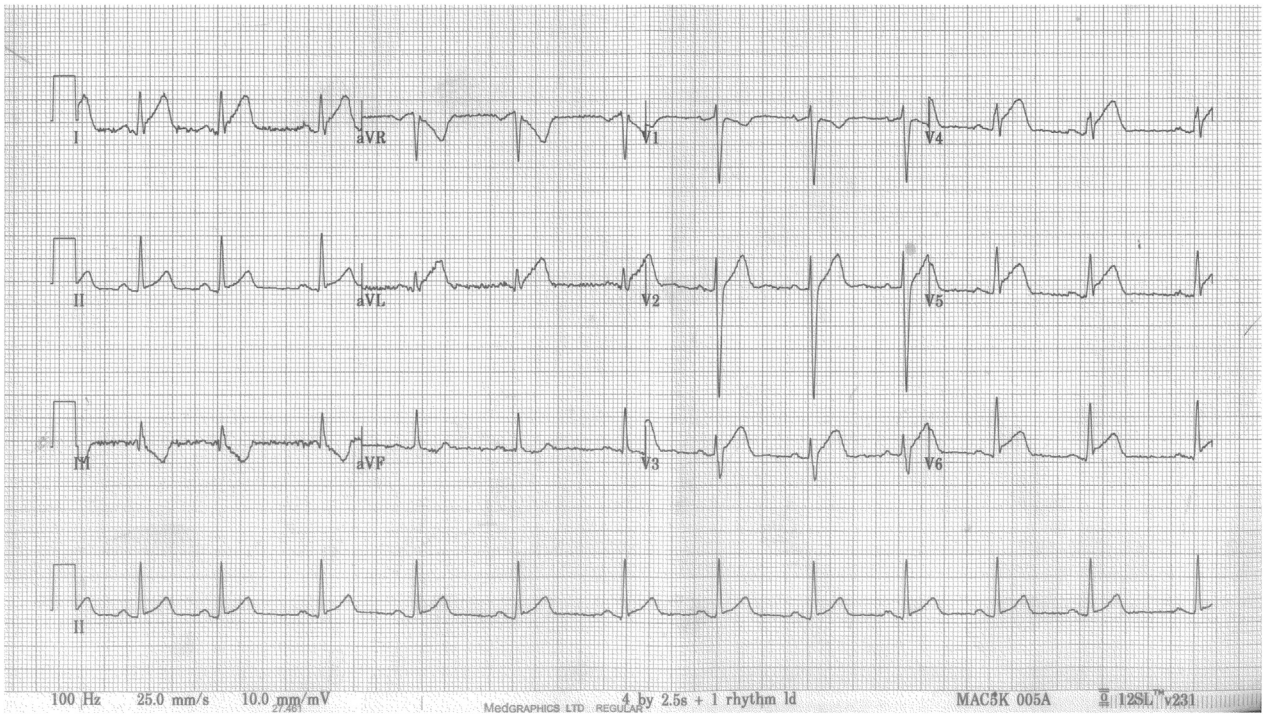

Figure 1. Electrocardiogram on arrival in Emergency Department.

On first assessment he reported that his pain had subsided to a mild discomfort only. His initial ECG (Figure 1) revealed sinus rhythm with 2-mm ST-segment elevation in the anterolateral leads and T-wave inversion in lead III, which were new findings when compared with his ECG 1 month prior. His vital signs were normal and clinical examination was unremarkable. Focused transthoracic echocardiography in the ED demonstrated normal ventricular function with no regional wall motion abnormality and a normal appearance to the aortic root. Shortly afterwards his pain increased again and a repeat ECG (Figure 2) showed worsening anterolateral ST elevation. Repeat echocardiography appeared identical to the first study. Loading doses of aspirin (300 mg) and clopidogrel (600 mg) were administered. Subsequently, blood test results revealed a cholesterol level of 6.5 mmol/L (desired reference range < 5 mmol/L) and triglyceride level of 4.0 mmol/L (desired reference range < 1.7 mmol/L). His initial troponin I level was elevated at 0.18 μg/L. Other tests including full blood count, renal function, and liver function were normal.

The patient was transferred as an emergency to the regional cardiology center, where a coronary angiogram demonstrated a large amount of thrombus with Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow in the proximal left anterior descending (LAD) artery. The remaining coronary vessels appeared normal. Multiple attempts were made to aspirate the thrombus, but a significant amount remained, which was subsequently treated with intracoronary alteplase and abciximab, resulting in improvement to TIMI grade 3 flow, though there was still a significant amount of residual clot. No attempt to dilatate or stent the lesion was made because there was no angiographic evidence of a ruptured plaque, and attempts at intervention were judged to incur a significant risk of causing distal coronary embolization of the thrombus. An intravascular ultrasound (IVUS) examination was not performed for the same reason as well as the additional risk of pulling a clot back into the aorta, potentially causing a stroke. The patient subsequently developed a rash thought to be due to an allergic reaction to abciximab and this was stopped. He was treated with fondaparinux, aspirin, and clopidogrel for 72 h. A repeat angiogram after this period demonstrated residual clot in the LAD but with brisk flow. IVUS was not performed again due to the continued risk of causing embolization. Measurements of the levels of protein C, protein S, antinuclear antibodies, antineutrophil cytoplasmic antibodies, and anticardiolipin antibodies were normal. The plasma homocysteine concentration was mildly elevated at 32 μmol/L (reference range < 16 μmol/L). Testing for evidence of illicit drug use was not performed, though the patient repeatedly denied using any other substance aside from cannabis.

A 3-month course of warfarin was started in addition to aspirin and clopidogrel to provide potent anticoagulation