Cardiology in the Young

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ST elevation myocardial infarction following a cannabis smoking binge

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Brief Report

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Abstract

Cannabis smoking is considered the most popular illicit drug used worldwide. We present the case of a 26-year-old male with ST elevation myocardial infarction and heart failure subsequent to cannabis smoking abuse. We searched the literature regarding acute myocardial infarction following cannabis smoking and the possible pathophysiologic mechanisms.

A 26-year-old male who has been a regular cannabis smoker for 10 years, abstained from this habit for 3 months. He smoked cannabis extensively in 1 weekend (10 g in 36 hours); the following hours felt left-sided chest pain extending to left shoulder and diaphoresis. The electrocardiogram showed ST elevation in anterolateral leads and in lead II (Fig 1). Primary percutaneous coronary intervention pathway was activated and he had a coronary angiogram immediately (emergency department was bypassed) that showed normal coronary arteries with TIMI 3 flow. The left ventriculogram revealed septal hypokinesia with anterior, lateral and apical akinesia with moderate systolic dysfunction. The ST elevations remained for about 1 hour, gradually resolved, and T wave inversions were noted (Fig 2). The blood test on arrival showed a significant increase in high-sensitivity troponin (Troponin I; 28,820 ng/L); subsequent blood sample taken in the afternoon of Day 1 showed decrease in troponin (20,339 ng/L); at Day 3 troponin was 826 ng/L. At Day 2 he had an echocardiogram that showed an improvement in left ventricle (LV) systolic function (now mildly impaired; anterior, lateral, and apical segments were hypokinetic) with normal size of LV cavity. He was commenced on Ramipril 1.25 mg and Bisoprolol 1.25 mg once a day (due to systolic dysfunction seen in ventriculogram and subsequent echocardiogram), but the latter was stopped due to bradycardia. He was discharged at Day 5 with the diagnosis of ST elevation myocardial infarction due to coronary vasospasm secondary to cannabis smoking. Differential diagnosis involved: (a) Takotsubo syndrome which was ruled out as there was no such pattern in ventriculogram, (b) Myocarditis which was excluded as there were no prior symptoms, spiking temperatures, or increased inflammatory markers, and (c) recanalised thrombotic coronary occlusion either due to plaque rupture or thrombophilia that were ruled out due to patient's age along with completely normal coronary arteries, normal cholesterol levels, and absence of haematology disorders. The patient gave verbal informed consent for publication of the case in a decent way.

Cannabis has been linked to acute myocardial infarction with growing evidence. In the French Addictovigilance Network in a 5-year period (2006–2010) 1.8% of cannabis-related incidents were cardiovascular events.[1]

There are case reports of cannabis-related acute myocardial infarctions that are caused by thrombosis. In a case series of three ST elevation myocardial infarctions, two of them were due to thrombosis.[2] In two other cases with ST elevation myocardial infarction, there was a clot in the left anterior descending artery, and in a case with a non-ST elevation myocardial infarction there was a left main occlusion. In a sudden cardiac death, the autopsy showed thrombosis of the right coronary artery with no continuity between the thrombus and the atheromatous component of a plaque.[3] A 21-year-old male with ST elevation myocardial infarction had intra-vascular ultrasound that did not show any atherosclerotic disease despite the thrombus formation, while in another case intra-vascular ultrasound showed atherosclerotic plaque disruption.[4,5]

On the contrary, there are cases with no thrombosis. In one case, a man in his 40s presented with non-ST elevation myocardial infarction and normal arteries in the coronary angiogram. A case report of an ST elevation myocardial infarction reported normal coronary arteries, while in another ST elevation myocardial infarction case was proved that infarction was due to vasospasm as there was slow flow with no thrombosis which improved with intra-coronary nitrates.[6,7]

Endocannabinoid system is involved in tissue inflammation and injury that could be related to thrombosis. Cannabis can cause lower limb arteritis, and it can be assumed that coronary arteritis could play a role in thrombosis. Platelet activation may also play a key role.[8–10]

We presented the case of a 26-year-old gentleman who was admitted in hospital with ST elevation myocardial infarction with no thrombosis secondary to cannabis smoking. There is a growing evidence of cannabis causing acute myocardial infarction. All mechanisms have been found, including thrombosis with no atherosclerosis, thrombosis with underlying

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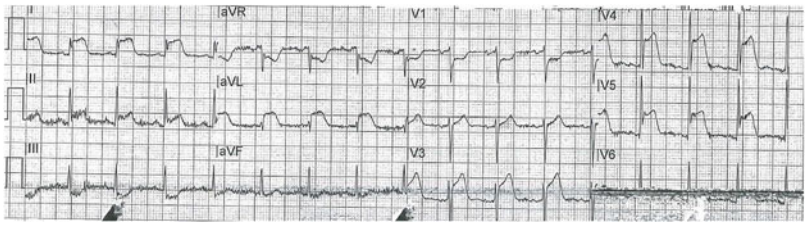

Figure 1. Initial electrocardiogram done by paramedics.

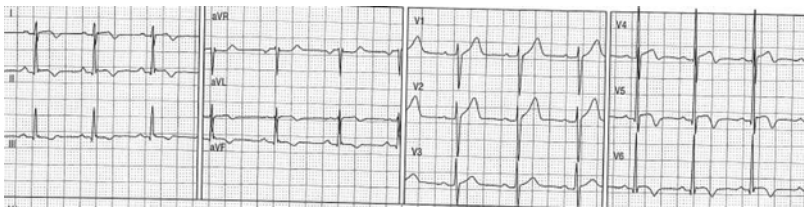

Figure 2. Electrocardiogram on the day of discharge.

atherosclerosis with and without plaque rupture, normal coronary arteries with definite vasospasm, and normal coronary arteries without vasospasm.

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Conflicts of Interest. None.

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