CASE REPORT

Myocardial infarction following cannabis induced coronary vasospasm

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SUMMARY

Smoking cannabis is a rare cause of myocardial infarction. We report a 29-year-old man who presented with acute coronary syndrome following consumption of a type of cannabis with the street name 'Kerala Ganja'. KG is smuggled into Sri Lanka from India; it is grown in the south Indian state of Kerala and is much more potent than the local ganja (marijuana). The patient developed dynamic ST-segment elevations in different leads in sequential ECGs, corresponding to different coronary artery territories. Coronary angiogram did not demonstrate evidence of occlusive atherosclerotic disease, but showed slow flow down the left anterior descending artery, which improved with administration of intracoronary nitrates. The patient's cardiac biomarkers were significantly elevated. A diagnosis was made of vasospasm causing myocardial infarction, most likely to have been triggered by cannabis consumption. We highlight the importance of considering this possible aetiology, particularly in patients with ACS with a susceptible social profile.

BACKGROUND

Cannabis is the most common psychoactive drug used worldwide. It is derived from the plant *Cannabis sativa*, and its main active ingredients are δ -9-tetrahydrocannabinol (THC) and other cannabinoids. According to the United Nations Office on Drugs and Crime (UNODC), the amount of THC present in a cannabis sample is generally used as a measure of cannabis potency.¹ Smoking cannabis is known to be a rare cause of myocardial infarction (MI).² The risk of the onset of MI has been shown to be elevated almost fivefold in the first hour after smoking cannabis. Postulated mechanisms for this include complex interactions between increased oxygen demand (due to increased heart rate and blood pressure), decreased oxygen supply (due to increase in carboxyhaemoglobin) and coronary vasospasm.

CASE PRESENTATION

A 29-year-old man presented to the emergency treatment unit in the early hours of the morning with a retrosternal chest pain radiating to left arm and shoulder of 1 h duration. He was a heavy smoker (35 cigarettes per day for 7 years). He had a history of marginally elevated serum lipids, for which he was not on treatment. He had also had a splenectomy 10 years earlier for immune thrombocytopenia (ITP); he was currently on no treatment for ITP, and his platelet

counts had been normal since the procedure. He had no other risk factors for ischaemic heart disease. He had returned home late after a dinner party, and was awoken from his sleep with chest pain. On admission, he was haemodynamically stable, and his physical examination was unremarkable.

INVESTIGATIONS

ECG showed 1–2 mm ST segment elevations in the inferior and lateral leads. His chest pain subsided with glyceryl trinitrate (GTN) sublingual spray. While awaiting primary percutaneous intervention, a repeat ECG was taken 30 min after the first ECG due to recurrence of chest pain. The second ECG showed 2–3 mm ST segment elevations in anterior leads (V2–V5) in addition to previously seen inferior ST elevations. Coronary angiography was performed within 1 h of admission. Slow antegrade flow of contrast in the left anterior descending (LAD) artery was noted. As there was no evidence of atherosclerotic disease, angioplasty was not performed. The patient was treated with intracoronary GTN and abciximab, which resulted in immediate improvement in LAD blood flow. His admission troponin T was <0.01 ng/mL (negative), and troponin I after 6 h was 2.8 ng/mL (positive). Blood counts were normal except for a slightly elevated leucocyte count of $14\,880/\text{mm}^3$. His platelet counts were normal. Other biochemistry was normal, including serum cholesterol. A full thrombophilia screen was subsequently performed, which was normal.

TREATMENT

The patient was treated for acute coronary syndrome with antiplatelets, statins, nitrates, anticoagulants and a calcium channel blocker.

OUTCOME AND FOLLOW-UP

On further questioning, it was revealed that the patient had consumed a form of marijuana with the street name of 'Kerala Ganja' or 'KG' 4 h prior to the onset of symptoms. He also admitted that he had taken a tablet of lysergic acid diethylamide (LSD) (street name 'Superman'), 48 h prior to the onset of chest pain. There were no other risk factors for MI apart from cigarette smoking.

Repeat ECG showed resolution of the ST segments. Echocardiogram performed on discharge was unremarkable, and showed no residual myocardial damage.

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DISCUSSION

MI at a young age is unusual. Common causes include premature atherosclerosis leading to plaque rupture, congenital anomalies of the coronary arteries, coagulopathy and coronary artery spasm (CAS). Drug-induced MI is gaining importance with increased use of recreational drugs. Recreational drugs well known to induce MI include cocaine,³ amphetamines and ecstasy. Their effects on the cardiovascular system are predominantly related to stimulation of the sympathetic nervous system; thus they are known to cause MI secondary to CAS.

The most commonly used recreational drug in Sri Lanka is locally produced cannabis (street name 'Ganja'). There is evidence that cannabis use could trigger MI. Mittleman *et al.*² in a case-crossover study, found that cannabis use increases the risk of MI, especially in the first 60 min after use; the risk was shown to rapidly decrease thereafter. Nonetheless, cannabis is a known rare trigger for MI, with a population attributable fraction of just 0.8 (95% CI 0.38% to 1.67%).⁴ The mechanisms through which cannabis use increases the risk of MI are not fully understood. Marijuana has several well-described effects on the heart, such as dose-dependent increase in heart rate, increase in blood pressure and increased risk of arrhythmias.⁵ Overall, there is a net increase in myocardial oxygen demand with a decrease in oxygen supply, which is due in part to an increase in blood carboxyhaemoglobin levels. This could potentially result in a lower anginal threshold in patients with chronic atherosclerotic narrowing. In-vitro studies have shown that cannabis may have pro-coagulant effects, through increased expression of glycoprotein 11b-111a and P-selectin on platelets.⁶ Vasospasm may also occur, similar to cocaine use.⁷ Several case reports have documented MI triggered by marijuana use. Angiographic findings have been varied. In some, coronary atherosclerosis with superadded thrombosis has been demonstrated, suggesting that cannabis may provoke plaque rupture or promote thrombosis on a ruptured plaque.^{8–12} In others, the coronary arteries have been shown to be normal,^{13–14} while evidence of slow flow within the coronary arteries have been demonstrated in some instances.^{15–16}

Our patient had no evidence of occlusive atherosclerotic plaque disease or evidence of recent plaque rupture in the epicardial coronary angiogram. However, he demonstrated vasospasm in the LAD artery, with slow flow, which improved with the administration of intracoronary nitrates. His ECG changes suggested that a similar vasospasm may have occurred in the right coronary artery as well. The temporal relationship with use of 'Kerala Ganja' made this the most likely inducer of coronary spasm. The patient had also taken LSD 48 h prior to the onset of MI. LSD is a potent psychostimulant, which is known to cause cerebral and lower limb ischaemia; however, there is little evidence in the published literature to suggest that it could cause MI. Furthermore, the long lag period between the exposure and onset of symptoms made LSD unlikely to be the trigger for his acute presentation. Nonetheless, concomitant use of other recreational agents,¹⁶ and also sildenafil,¹⁷ together with cannabis, have been reported to result in MI. The contributory effects of LSD in this instance are difficult to determine.

Learning points

- ▶ Kerala Ganja, produced in Kerala, India, is a more potent preparation than conventional marijuana.
- ▶ Marijuana is potential trigger for acute coronary syndrome in young patients without cardiac risk factors.
- ▶ Marijuana can cause coronary vasospasm.
- ▶ Kerala Ganja may have more potent vasospastic effects than marijuana.
- ▶ A history of substance abuse should be actively sought in patients presenting with acute coronary syndrome and normal coronary arteries.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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