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# Right Coronary Artery thrombosis causing Acute Myocardial Infarction in a young and healthy male following marijuana use: A case report and literature review.

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#### Abstract

Marijuana is considered one of the most used recreational drugs worldwide as it is easily accessible and affordable and, even by some individuals is believed to be unharmful compared to other recreational drugs. As example marijuana is legal in some countries, recently the first legal marijuana selling stores opened in the state of Colorado in the USA. It is well known that marijuana has several diverse effects on the cardiovascular system, but it is argued if and how marijuana cause acute myocardial infarction (AMI).

We report the case of a 38-year old healthy male, who suffered ST-elevation myocardial infarction following marijuana use. Coronary angiography confirmed large thrombosis of the right coronary artery (RCA) as the cause of the AMI, which was successfully treated with intracoronary stent placement. The patient was discharged home in a stable condition four days later. Physicians should be aware that marijuana use increases the risk of suffering AMI, and even young and healthy patients presenting with cardiovascular symptoms and concomitant marijuana use should be taken seriously and examined carefully as non-marijuana using patients.

## Keywords

Marijuana - Acute myocardial infarction - literature review

## Introduction

It is proven that marijuana has several hemodynamic consequences such as a dose-dependent increase in heart rate, supine hypertension and postural hypotension<sup>1</sup>. However, it is debated if marijuana can trigger AMI and if so, which mechanism is behind this action.

Several mechanisms have been postulated in the literature. One of the theories is that marijuana causes a net increase in the myocardial oxygen demand with a concomitant decrease in oxygen supply, which is due to an increase in carboxyhemoglobin, resulting in myocardial ischemia<sup>2, 3</sup>.

A literature search on marijuana associated AMI was conducted using PubMed, and only a few earlier reported cases<sup>1, 4-9, 13-24</sup> regarding this issue was found.

## **Case presentation**

A 38-year old and otherwise healthy male, with no overweight (BMI = 24) and without risk factors for coronary heart disease other than tobacco smoking (2 - 3 tobacco cigarettes on a daily basis for the past 10 years), was admitted to our Department of Cardiology at the Regional University Hospital with retrosternal chest pain radiating to the left arm and neck, dizziness and dyspnea.

The symptoms had lasted for two hours and started within 1 - 2 hours of smoking a cigarette containing 0.3 g marijuana. The patient had been smoking marijuana 2 - 3 times a week for the past five years, mostly smoking about 3 g marijuana (10 cigarettes each containing 0,3 g marijuana) during a single day, but had never experienced such symptoms, even during heavy exercise. The patient was not taking any medication and denied abusing alcohol or any other drugs beside marijuana. The patient did not administer marijuana in another route than inhalation by smoking marijuana containing cigarettes.

The patient was hemodynamically stable (blood pressure 116/61 mmHg and pulse 62 beats/min) and physical examination was unremarkable. The patient's electrocardiogram (ECG) showed sinus rhythm, heart rate of 58 beats/min and ST-segment elevation on lead II, III and aVF (figure 1).



Figure 1: Patient's ECG on admission to hospital.

At the cardiology department the patient was given aspirin (a loading dose of 300 mg), Brilique/ticagrelor (a loading dose of 180 mg) and a single intravenous dose of unfractionated heparin (10.000 IU)<sup>10</sup> and subsequently underwent an acute coronary angiography, which revealed large thrombosis of the RCA and otherwise minimal coronary irregularities (figure 2). This was successfully treated with the placement of intracoronary stent and subsequent intravenous infusion of Angiox (Bivalirudin) for the next few hours.



Figure 2: Acute coronary angiography revealing thrombosis and hence occlusion in the RCA (arrow).

An acute bedside echocardiography showed normal ejection fraction and no valvular abnormities. The first troponin I measurements was elevated 31 ng/l (reference interval < 25 ng/l), and the second troponin I values taken few hours later was 900 ng/l. Urine drug test was only positive for marijuana and no other drugs. Other laboratory findings including hemoglobin, electrolytes, hemoglobin 1AC, cholesterol and lipid profile, antithrombin III, prothrombin time, partial thromboplastin time, fibrinogen, homocystein, protein S, protein C and plasminogen activator inhibitor-1 were in the normal range. Lupus anticoagulant, factor V Leiden and prothrombin G20210A tests were negative.

The patient recovered and was discharged from hospital four days later and was asymptomatic on follow-up one month following discharge.

#### Discussion

In the present case, an otherwise healthy 38-year old tobacco cigarette smoking male suffered AMI following the use of marijuana. The patient's cardiac symptoms started within 1 - 2 hours of smoking a cigarette containing 0.3 g marijuana, which may therefore be suspected as the possible cause of the AMI. The patient denied using other illegal drugs or narcotics such as cocaine and explained precisely how much marijuana he used to smoke. Our patient had an insignificant and much less use of tobacco cigarettes compared to patients in similar case reports<sup>7, 8</sup>, as tobacco smoking itself is a risk for coronary heart disease and could play the role of a confounder in such cases.

Conducting a literature search on PubMed, only a few earlier case reports of marijuana associated AMI was found, however almost none of these case reports, precisely mention the exactly dose of marijuana used by the patient, and sometimes the route of marijuana administration (inhaled and/or orally ingested) is not clearly mentioned. It is well known that the effect of marijuana is dependent on the dosage and administration route (the onset of the psychoactive effects is delayed by about an hour when administered orally compared to inhalation <sup>11</sup>). Our patient was considered having a moderate intake of marijuana compared to other healthy individuals<sup>12</sup>.

Marijuana has several hemodynamic consequences such as a dose-dependent increase in heart rate, supine hypertension and postural hypotension<sup>1</sup>. However, it is unclear if and how marijuana can trigger the onset of AMI.

Mittleman et al.<sup>1</sup> carried a study on this issue and concluded that the risk of AMI onset was elevated 4.8-fold within one hour after smoking marijuana, but the risk declined rapidly thereafter.

A possible explanation for the AMI development following marijuana use is that marijuana is associated with an increase in carboxyhemoglobin (resulting in decreased oxygen-carrying capacity) with concomitant increase in the myocardial oxygen demand due to its hemodynamic effects <sup>2, 3</sup>. Thus, some of the marijuana associated myocardial infarctions may be caused by the imbalance between increased myocardial oxygen demand and decreased oxygen supply at the same time.

Aronow et al.<sup>2,3</sup> demonstrated that the anginal threshold, in patients with known chronic stable angina pectoris, was diminished acutely after smoking a single marijuana cigarette, supporting the above mentioned explanation of decreased myocardial oxygen supply together with an increased myocardial oxygen demand.

However, other authors<sup>1, 24, 25</sup> proposed that marijuana induced vasospasm at the site of a vulnerable, but not necessarily stenotic, atherosclerotic plaque, triggering plaque rupture and hence AMI. This fact is supported by the case reported by MacInnes et al.<sup>24</sup>, where a young man suffered coronary artery thrombosis due to plaque rupture following marijuana use.

It has also been proposed, that marijuana may have a direct effect on the coagulation system, including the blood platelets, by inducing increased aggregation of platelets and, a minimal increase in factor VII activity<sup>26</sup>.

Yurtdas et al.<sup>8</sup> proposed that some of the marijuana associated myocardial infarctions may be caused by coronary artery embolism due to marijuana induced atrial fibrillation. However, there is only little information on such an association in the literature.

Marijuana is not only suspected to trigger the onset of AMI, but even cardiac arrest has been reported in young patients using this drug. Sattout et al.<sup>27</sup>reported on a healthy 15-year old male, who suffered cardiac arrest following marijuana use. Marijuana is also believed to have arrhythmogenic potential as investigated by Fisher at al.<sup>28</sup>. There have even been reports of marijuana associated stroke<sup>29</sup>.

The most important message of our case report and literature review is, that marijuana, which is thought to be, one of the most unharmful recreational drugs by many people, is associated with life-threatening cardiac conditions such as AMI, and physicians should pay careful attention to even young and otherwise healthy patients, with no coronary heart disease risk factors, presenting with cardiac related symptoms following the use of this drug.

## Conclusion

In the present case, a healthy 38-year old male, with no relevant risk factors for coronary heart disease besides tobacco smoking, suffered ST-elevation myocardial infarction following the use of marijuana. Marijuana associated AMI has been reported in a few cases in the literature. A literature review was conducted to investigate the association between marijuana and AMI, and the postulated mechanisms behind this. The main result was that marijuana was suspected to be a trigger of AMI in several case reports including our own case report, and physicians should therefore be aware of this, although more evidence of the precise mechanisms behind this action is needed in the future. Our case report is unique in the way that we precisely report the dose and route of marijuana administration in our patient.

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