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Case Report

Marijuana Induced Coronary Vasospasm – Case Report

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Coronary vasospasm is a well-known condition causing acute chest syndrome and can lead to myocardial infarction, ventricular arrhythmias, and even sudden cardiac death. While there are extensive case series showing the association of coronary vasospasm with drugs like cocaine [1], the association of marijuana with coronary vasospasm has not been reported frequently.

Smoking of marijuana has been reported as a trigger of coronary artery disease and especially acute myocardial infarction (MI). The risk of onset of MI is elevated 4.8 times in the first hour after marijuana use and rapidly decreases thereafter; which may be explained by marijuana induced coronary vasospasm [1]. The effects of the active ingredients in marijuana have been well studied and reported substantially in the literature. When smoked, marijuana results in a rapid, dose-dependent tachycardia by 20-100%, an increase in blood pressure, and an increase in cardiac output by > 30%. This in turn leads to increased oxygen demand, which is augmented by the vasoconstriction endothelial damage by smoking and activation of CB1 receptors by marijuana [2].

Patient description:

38 years old, generally healthy, no underlying diseases, and no family history of heart disease. A year before his current hospitalization, he was involved in a serious traffic accident, and was hospitalized due to fractures in his lower limbs and pelvis. He underwent several surgeries and was then referred to a pain clinic. The patient tried a variety of analgesics including Diclofenac, Oxycodone, Rocket plus, and Trammel with Paracetamol (Zaldiar). Unfortunately, none of the drugs had a significant effect. It was then decided to start treatment with medical cannabis a week before he was admitted to the Emergency Room.

He was admitted to the Emergency Room at our institution due to the sensation of crushing chest pain. He described the pain as intermittent, with three episodes in 24 hours. The pain radiated to the left shoulder and left arm. Additional symptoms included dyspnoea, sweating, nausea, palpitations and dizziness that lasted for about half an hour. At the time of admission, all symptoms had subsided. On examination, he was in good general condition. Vital signs were in the normal range. Physical examination was unremarkable: good air intake, regular heart sounds without murmurs or extra heart sounds, soft abdomen, limbs without edema and no signs of deep vein thrombosis.

ECG on admission showed regular sinus rhythm with no signs of acute ischemia or ST wave changes. Laboratory workup revealed normal complete blood count and chemistry. Troponin test was negative twice. The patient in the Emergency Room was examined by a cardiologist and it was decided he should be admitted to the Cardiology Department for further investigation and monitoring.

An echocardiogram was performed which demonstrated normal systolic function of the left and right ventricles, without wall motion abnormalities or substantial valvar problems. During his treatment in Cardiology, the patient was disobedient to the doctors' instructions and occasionally went to smoke outside the department. On one occasion after the patient returned from smoking a marijuana cigarette, he complained of crushing chest pain. An EKG was performed which demonstrated diffuse ST elevations (photo1). The patient was rushed to the catheterization room and cardiac catheterization was performed, which demonstrated normal coronary arteries. Immediately after the catheterization, an EKG was performed which demonstrated a normal ECG (photo2). Later, another cardiac catheterization was performed with a provocation test with acetylcholine, which was positive (photo3). Treatment with Calcium Channel Blockers and Nitrates was initiated as is customary to treat such cases and the patient remained under observation for 5 days without recurrence of chest pains or changes in the EKG.

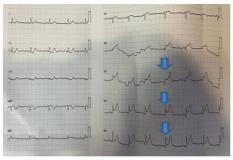

Photo1-ECG showing ST-segment elevation in the anterior leads (*arrows point to ST-elevation in anterior leads)

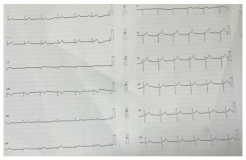

Photo2- normal ECG

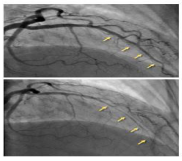

Photo3-Angiogram images of coronary artery showing vasospasm post acetylcholine injection (*arrows point to vasospasm of the artery)

Comment

Drug-induced vasospasm is an important differential diagnosis in younger individuals with cardiac symptoms with no prior cardiac history. Cocaine, amphetamines, and 3, 4-Methylenedioxymethamphetamine (MDMA) are the recreational drugs most commonly associated with vasospasm. Marijuana is well known to cause cardiac effects such as dose-dependent tachycardia, hypertension, and an increased risk of arrhythmias, myocardial infarction, and spontaneous coronary artery dissection [2]. However, coronary vasospasm (Prinzmetal Angina) has rarely been described.

Different mechanisms have been proposed to explain the deleterious effects of cannabis use on the heart. The pathophysiology is thought to be caused by inadequate blood flow through the coronary arteries. It is theorized, high catecholamine release resulting in increased oxygen demand triggers the onset of vasoconstriction and coronary vasospasm [3].

There are two types of Cannabinoid receptors (CB) in humans: CB1 (pro-atherogenic) and CB2 (anti-atherogenic). There is an upregulation of macrophage CB1 receptor and increase in reactive oxygen species (ROS) generation in advanced atheroma's of patients with unstable angina compared to those with stable angina [3]. Rimonabant is a CB1 receptor antagonist that has been shown to decrease the total atheroma volume in the STRADIVARIUS trial (Strategy to Reduce Atherosclerosis Development Involving Administration of Rimonabant - the Intravascular Ultrasound Study) [4]. Further trials are underway to evaluate the usefulness of rimonabant in coronary artery disease.

The workup for such patients with suspected Coronary Vasospasm is identical to the workup for any patient suspected of acute coronary syndrome (ACS). Patients presenting with symptoms of MI, even when substance use is suspected or known, must still be evaluated according to standard protocols based on risk stratification. Several of these patients will require ischemic workup. The international study group of coronary vasomotor disorders known as COVADIS, created a diagnostic criterion to determine the presence of Prinzmetal angina. These include: Clinical response to nitrates during a spontaneous angina episode, transient electrocardiographic changes with concern for ischemia during a spontaneous angina episode, ST-segment elevation or depression = 0.1 mV or new U waves, and finally evidence of coronary vasospasm during angiography. During cardiac catheterization, coronary spasms can be visualized spontaneously or under drug induction. Ergon ovine, acetylcholine, and hyperventilation, can be used in the catheterization laboratory in an attempt to confirm the diagnosis of coronary vasospasm. These tests are warranted only when the diagnosis of vasospastic angina is suspected but not firmly established. Treatment of Prinzmetal angina is focused on decreasing episodes of angina and preventing complications like myocardial injury and arrhythmia. Lifestyle modifications should be promoted, especially smoking cessation. In addition, avoiding medications or drugs that can trigger coronary vasospasm such as cocaine or marijuana should be encouraged as well. Pharmacological therapy includes Calcium Channel Blockers (CCB) as a first-line treatment due to its vasodilation effect in the coronary vasculature and plays an important role in the management of vasospastic angina. Nitrates are also effective in preventing vasospastic events [5].

In relation to our specific patient, he presented complaining of transient chest pain, nausea, and palpitations close to the start of medical cannabis use. ECG changes showed a transient ST elevation. He underwent cardiac catheterization that demonstrated normal coronary arteries. A provocation test with acetylcholine was done to help confirm the diagnosis of vasospasm and it was positive. As noted above the patient received treatment with CCB and Nitrates, and there was significant improvement in his complaining symptoms

Conclusion

With the legalization of marijuana in certain states, marijuana-related hospitalizations and ER visits are likely to increase. It is therefore important for clinicians to know the various effects of marijuana, especially potentially fatal ones like coronary vasospasm. This case illustrates the importance of recognition of vasospasm in patients taking marijuana .Physicians should consider this in their differential diagnosis of patients presenting with chest pain and history of cannabis use.

References

1. Chami T, Kim CH. (2019) Cannabis Abuse and Elevated Risk of Myocardial Infarction in the Young: A Population-Based Study. Mayo Clin proc. 94:1647–9.
2. Ahmad S, Hlaing SW, Haris M, Attar N. (2022) Marijuana: cardiovascular effects and legal considerations. A clinical case-based review. Br J Cardiol. 29:11.
3. Sugamura K, Sugiyama S, Nozaki T, Matsuzawa Y, Izumiya Y, Miyata K, et al. (2009) Activated endocannabinoid system in coronary artery disease and antiinflammatory effects of cannabinoid 1 receptor blockade on macrophages. Circulation. 119:28-36.
4. Nissen SE, Nicholls SJ, Wolski K, Rodés-Cabau J, Cannon CP, et al. (2008) Effect of rimonabant on progression of atherosclerosis in patients with abdominal obesity and coronary artery disease: the STRADIVARIUS randomized controlled trial. JAMA, 299:1547-60.
5. Sueda S, Miyoshi T, Sasaki Y, Sakaue T, Habara H, et al. (2015) Sequential spasm provocation tests might overcome a limitation of the standard spasm provocation tests. Coron Artery Dis.26:490-4.