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Recreational Marijuana Use Leading to Thrombosis in a Patient with Minimal Cardiovascular Risk Factors

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Abstract Marijuana is one of the most commonly used psychotropic drugs in the United States. Recent legalization of cannabis and decriminalization in multiple jurisdictions in the United States has contributed to its increased availability and social acceptance, especially in young adults. The overall prevalence of cardiovascular disease in young adults has been increasing as a consequence of marijuana use. However, there remains a paucity of knowledge on its adverse effects, especially with regards to the cardiovascular system. Here, we present a case of a young adult with a history of marijuana use and minimal cardiovascular risk factors who presents following a seizure episode and was found to have coronary artery thrombosis. A high degree of suspicion, along with a better understanding of the biochemical pathways involved in cannabis associated myocardial ischemia is extremely important to treat this condition promptly and prevent fatal outcomes. After the legalization of cannabis in various states in the United States, there is an increasing need to understand and educate the public regarding health hazards, especially on the cardiovascular system.

Keywords: coronary artery thrombosis, cannabis, cardiovascular risks, marijuana

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1. Introduction

Marijuana is the most commonly used psychotropic drug in the United States especially among the youth, after alcohol. It is known to have potentially severe socioeconomic as well as health implications. The cardiovascular effects of cannabis are not well understood however its use has been shown to increase the risk of cardiac arrhythmias, myocardial infarction, and potentially sudden death [1]. This raises concern given its legalization in several states and push towards legalization in many more states.

2. Case Presentation

A 31-year-old male with a history of seizure disorder and marijuana use was brought to the ED following an episode of witnessed generalized tonic clonic seizure. Upon presentation to the emergency department, the patient endorsed having constant, dull and nonradiating substernal chest pain but denied shortness of breath, nausea, vomiting, palpitations, dizziness, diaphoresis or any other symptoms. He did not have any prior similar episodes of chest pain. He reported smoking marijuana prior to presentation and has been smoking it for the past 2 years. Denies consuming alcohol, tobacco or any illicit drug use. He did not have any family history of cardiac

disease. He was afebrile, blood pressure 126/80mmHg, pulse 99/min, respiratory rate 20 breath/min and oxygen saturation 99% on room air. The initial troponin level was 0.05. EKG revealed normal sinus rhythm with no ST changes. CT Head was unremarkable. He did not have any other episode of seizures after getting treatment with Levetiracetam. At this point, the patient reported complete resolution of his chest pain without any intervention. A six-hour repeat troponin was markedly elevated at 12.89 with peak level of 35. The patient was promptly started on dual antiplatelet therapy with an emergent EKG performed. Repeat EKG revealed a new biphasic T-waves in V1 and peaked T-waves in anterior leads as shown in Figure 1. A 2-D Echocardiogram done at this time showed an LVEF of 40%-45% with basal anteroseptal, basal inferoseptal, and basal inferior hypokinesis.

Given these new findings, the patient was taken for emergent cardiac catheterization which showed a moderately large thrombosis localized in the mid-left anterior descending (LAD) with no compromise of flow and evidence of with distal embolization to the apical inferior portion of the LAD.

Post cardiac catheterization, the patient was transferred to the ICU for close monitoring. Once medically stable, he was discharged home on dual antiplatelet therapy and Atorvastatin with instructions to follow up in the cardiology clinic. Repeat echo in 8 months as he lost to follow up meanwhile, showed normalization of LV systolic function.

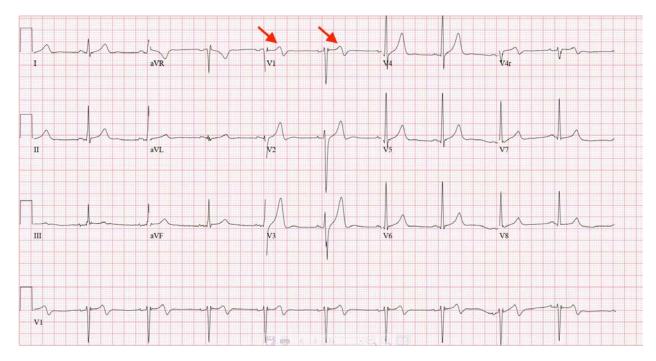


Figure 1. EKG showing biphasic T-waves in V1 and peaked T-waves in anterior leads

3. Case Discussion

Marijuana has been in use for many centuries. However, it has gained a lot of popularity over the past decade stemming from media promotion and its subsequent legalization in several states in recent times. The increase in use has brought to light side effects that were previously unidentified. These side effects include pancreatitis, chronic bronchitis, cancers and most importantly its effects on the cardiovascular system [1].

Although not fully understood, Marijuana use has been shown to have different effects on the cardiovascular system including coronary vasospasm, thrombosis, cardiac arrhythmias, and acute myocardial infarction [2,3]. Typically, the average age of cardiovascular disease in the United States is 50 years for men and 72.0 years in women. However, with Marijuana, most commonly used amongst the young adult population, the prevalence of these cardiovascular conditions has increased in the younger age group as a consequence. Several mechanisms have been identified through which this occurs.

There are multiple cannabis species, each with a differing amount of the two major active ingredients, Δ -9-tetrahydrocannabinol (THC) and cannabidiol (CBD). Research studies show that Δ -9-THC acts through two receptors on the platelet surface; CB1 and CB2 receptors can potentiate platelet activation and platelet aggregation. Δ -9-THC has been found to stimulate a significant amount of arachnoid production in platelets which subsequently increases the production of thromboxane A2 and prostaglandins. These findings are important as the arachnoid acid and cyclooxygenase are proinflammatory molecules that lead to endothelial injury, platelet activation, and consequentially increase in the risk of a cardiovascular event [1,2].

Additionally, marijuana can modify the coronary blood flow and heart rate promoting ischemia and possible infarction in young adults with minimal atherosclerotic risk factors [4]. This is in line with the case of our patient who had no cardiovascular risk factors but was found to have elevated cardiac enzyme and subsequent ECG changes. His coronary angiography showed no evidence of atherosclerotic disease however he had thrombosis in the mid LAD with embolization to the apical inferior portion of the LAD.

Understanding these biochemical pathways involved in THC-associated myocardial infarction can help guide management. Antiplatelets, beta-blockers and statins, and angiotensin-converting enzyme inhibitors remain the mainstay therapy for management and prevention of complications.

4. Conclusion

In light of the rapidly shifting landscape regarding the legalization of marijuana for medical and recreational purposes, the prevalence of cardiovascular disease is increasing especially in young adults. Its clinical presentation appears to be less severe compared to the typically affected age group. A high degree of suspicion is necessary amongst clinicians to treat promptly and avert fatal complications. The majority of the public use this for recreational purposes, thinking it is a safe drug, especially teenagers. Public awareness about the ill-effects of marijuana is the need of the hour and all physicians should always recognize those effects and advise their patients properly.

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