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CASE REPORT

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Marijuana Induced Troponinemia- An Unusual Case of Elevated Troponins

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ABSTRACT

Marijuana (pot, weed, hashish), a very renowned drug, used initially as a medicinal herb, now to ease pain and inflammation is abused worldwide. In the context of the easy availability, easy affordability, the popularity and the rising use of this recreational drug, we review a case where the use of marijuana caused elevated troponins that were alarming and interventional procedures were done to find out the cause, but when the catheterization failed to reveal any evidence of obstruction, marijuana-induced vasospasm was considered the possible culprit of the elevated troponins.

ARTICLE HISTORY

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Introduction

One of the most commonly abused drugs in the United States is marijuana [1].

Cannabis sativa originated from Central Asia but now available around the world [2]. Marijuana (cannabis) has long been used as a medicine to manage health problems and its use has been growing as a mood-altering drug [2]. Marijuana acts on the Cannabinoid receptors and mediates a variety of functions [3]. The effects of marijuana on the cardiovascular system are dose-dependent. Common cardiac manifestations include tachycardia, hypertension, cardiac arrhythmias, AV block, atrial fibrillation, ventricular fibrillation, asystole [3-11]. Despite these reported side effects, there has still been an increase in the use of marijuana. Marijuana can cause elevated heart rate and blood pressure immediately after use, possibly from stimulation of the sympathetic nervous system. Platelet activation, inflammation of the vasculature, and subsequently cannabis causing acute coronary syndrome are some of the other proposed side effects [12]. The recreational use of marijuana has caused an increase in the number of case reports of serious cardiovascular complications across the world. We hereby present a case where marijuana causes elevated troponins possibly by the mechanism of reversible vasospasm. In the context of the rising popularity of recreational cannabis, we hereby discuss the pathophysiological mechanisms and the harmful cardiovascular consequences of the use of marijuana.

Case Presentation

A 46-year-old-man with PMH of HTN, HLD, CAD (with stent placement in 2010 in the left anterior descending artery) presents to the ED with chest pain one day before the presentation. Of note, the patient has not taken any medications for many years. Patient says over the weekend he was at a friend's place, smoking marijuana and drinking beer. He went to bed comfortably, woke up with chest pain that seemed similar but of lesser intensity than the one he had in 2010 when a stent was placed. He felt nauseous and threw up twice, non-bloody, non-bilious vomitus, but after the vomiting, the chest pain resolved. The patient says the pain was retrosternal, non-radiating, no associated sob or diaphoresis. The pain lasted for a couple of minutes. After that, he took a shower, called his relative to drive him to the hospital. At the time of the presentation to the ED, the patient was completely asymptomatic.

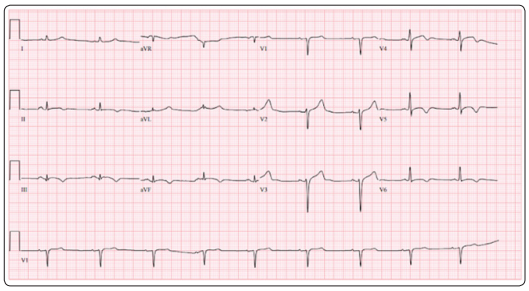

Figure 1: EKG: Showing T Wave Inversion in V4-V6, Sinus Bradycardia, ST Changes in V2-V3

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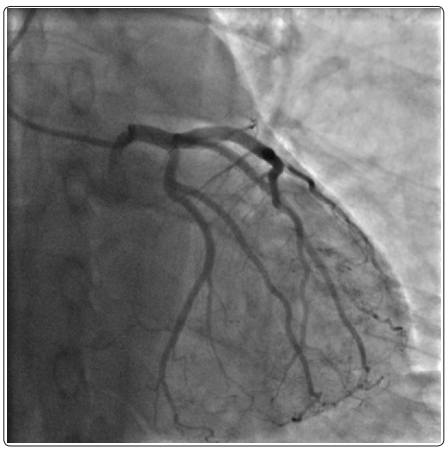

Figure 2: Left Heart Catheterization. No obstructive Lesion Found

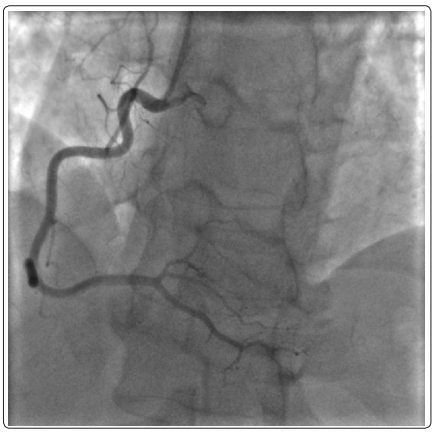

Figure 3: Right Heart Catheterization Showing no Occlusion

EKG [figure 1], showed normal sinus rhythm with a T-wave inversion in V4-V6, sinus bradycardia, and ST changes in V2-V3. All labs normal, except for troponins which were elevated to 28 ng/ml (normal 0.00-0.034 ng/ml), after 6 hours trended down to 24 ng/ml. Risk and benefits were weighed and it was decided based on the patient's risk factors and previous history of stent placement to take the patient to the catheterization lab. Catheterization showed no stenosis. [Figure 2] shows normal left heart catheterization. [Figure 3] shows right heart catheterization without any thrombus or occlusion.

Since catheterization revealed no stenosis and the patient was asymptomatic, he was downgraded to the floors and monitored for a day. Troponins continued to trend down. The patient was discharged home with statins, aspirin, clopidogrel, and a cardiology outpatient appointment. The patient was stressed on medication compliance and to abstain from the use of marijuana. He was counseled on the deleterious effects of marijuana on the body. The patient was followed up every week over the phone to make sure he was refraining from the use of this illicit drug.

Discussion

Serum cardiac troponin I is a sensitive indicator of myocardial necrosis [13]. However, troponin can be elevated in many diseases without clinical evidence of myocardial infarction, like e.g. in our patient troponins were elevated to about 200 times normal, but there was no blockade found on cardiac catherization. Marijuana activates the Cannabinoid-1 receptors (CB1 receptors) within the endothelium which causes reactive oxygen species-mitogen-activated protein kinase (ROS-MAPK) activation [14, 15]. Multiple ROS are generated which provide a vaso-constrictive stimulus to the coronary vessels and also promote endothelial damage [15, 16].

As marijuana induced chest pain and elevated troponins is secondary to vasospasm, the appropriate management would be the abstinence from marijuana. Aspirin, clopidogrel (dual anti-platelet therapy), high intensity statins. Calcium channel blockers and nitrates can also be used because of their anti vaso-constrictive effects [17].

Cannabis induces transient coronary vasospasm [18]. This is supported by many case reports of patients who presented with myocardial infarction but cardiac angiography revealed normal coronary vessels [19]. Marijuana smoking causes an increase in carboxyhemoglobin levels leading to a decrease in oxygen supply. This mismatch oxygen supply-demand causes transient ischemia particularly in the myocardium [20]

Conclusions

Through this case report, we presented a unique case of about a two-hundred-fold increase in troponins in a patient who used marijuana. The most likely mechanism seemed to be reversible coronary vasospasm induced by marijuana use. Cardiovascular complications with marijuana use have been published previously by many authors but such elevated troponins with normal catheterization findings have not been previously documented. There have been cases of marijuana presenting with myocarditis, myocardial infarction, but through our current case, we want to highlight the finding of elevated troponins but normal cardiac catheterization in a patient with marijuana use and the troponins trending down when the causative agent being removed.

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