Marijuana Induced Myocarditis: A New Entity of Toxic Myocarditis

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Abstract Marijuana is the most common drug of abuse in the United States. Marijuana has more than 460 active chemical compounds including δ-9- tetrahydrocannabinol (THC). It acts via the CB1 and CB2 receptors that are distributed in various tissues in the body. Marijuana is known to cause tachycardia, bradycardia, hypertension, to decrease time angina, myocardial infarction and cardiac arrest. Till date, four cases of myocarditis/perimyocarditis associated with marijuana use have been reported. In one such case, it led to the development of heart failure in a young male patient. It is not clear if marijuana in and of itself causes myocarditis/perimyocarditis or if the etio-pathogenesis is actually related to the contaminants in marijuana such as pesticides and heavy metals. We hereby present a young male who with myocarditis related to marijuana use. Clinicians should have suspicion for myocarditis or perimyocarditis in patients presenting with chest pain following marijuana use.

Keywords: marijuana, myocarditis, toxic myocarditis


1. Introduction

In 2016, over 26 million americans reported marijuana use within the last month [1]. Marijuana is the most commonly used drug of abuse in the United States [2]. Despite federal government's classification of the drug as a Schedule one drug, marijuana use is on a steady rise due to the legalization of marijuana in 25 states for medicinal purposes and in 8 states for recreational use [3,4]. Over 460 chemicals including δ-9- tetrahydrocannabinol (THC) have been reported in marijuana [5]. Tachycardia, arrhythmias, hypotension, hypertension, and increase risk of myocardial infarction are adverse cardiovascular effects reported with marijuana use [6]. Potency of marijuana is highly variable and it is often contaminated. Such contamination has little monitoring and is a public health concern [7]. Till date, four cases of myocarditis/perimyocarditis associated with marijuana use have been reported [8,9,10,11]. To the best of our knowledge, we hereby present the fifth case.

2. Case Presentation

A 27 year old male with a history of tobacco and marijuana abuse since the age of 17 presented with a chief complaint of substernal chest pain four hours after he smoked two joints of marijuana. He described the pain as sharp, intermittent, 6/10 in intensity, non-radiating, and was associated with a dry cough. He had an episode of fever a day prior to presentation. On presentation he was afebrile, heart rate was 87 beats per minute, blood pressure was 109/60 mm Hg. On physical examination, chest was clear to auscultation, S1 & S2 heart sounds were heard, no murmur or friction rub were appreciated. Basic laboratory work up including complete blood count, comprehensive metabolic panel, coagulation profile, urine toxicology (DUA8) were obtained. A elevated white blood count of 17,800 white blood cells per microliter (mcL) with granulocyte count of 12,500 per mcL was noted. Electrolyte levels, liver enzymes and kidney functions were within normal limits. Urine toxicology was positive for THC. Chest X-ray was normal. Review of an electrocardiogram (EKG) obtained two years ago revealed normal sinus rhythm and no ST-T changes. EKG obtained during this visit showed normal sinus rhythm, diffuse ST-T segment elevation and PR segment depression (Image 1 and Image 2). Initial troponin value was elevated at 7.3 ng/ml. The patient was diagnosed to have myopericarditis. However, as the troponin levels rose from 7.3 ng/ml to 25.7 ng/ml, considering his history of marijuana use prior to presentation, a coronary angiogram was performed to rule out myocardial infarction. Coronary
angiogram revealed normal coronary arteries. Subsequent troponin trends were as follows: 49.7, 29.4, 73.1, 48.2, 29.4, 33.4, 17.2, and 7.02 ng/ml. Viral and bacterial etiology of myocarditis/pericarditis was ruled out. An echocardiogram (TTE) obtained showed normal left ventricular ejection fraction of 55% and no regional wall motion abnormality or pericardial effusion were noted. Myopericarditis was managed with ibuprofen and colchicine. Counseling was provided for marijuana use. The patient was discharged with a follow up appointment.

Image 1. EKG showing ST segment elevation in leads V2 to V6, I, aVL, T wave inversion in V2-V3, PR segment depression in V3 to V6, I and II and PR elevation and ST depression in aVR

Image 2. EKG obtained after the initial EKG showing ST segment elevation in V4 to V6, I, II, aVL, ST segment depression in leads V1 to V3 and III, PR elevation and ST depression in aVR
3. Discussion

Marijuana exerts various effects on the body via the cannabinoid receptors; CB1 and CB2. [6] CB1 and CB2 receptors are distributed over various tissues such as platelets, vascular smooth muscles and endothelium. CB1 agonism is atherogenic while CB1 antagonism and CB2 agonism is anti-atherogenic. [6]

It is difficult to regulate marijuana potency and contamination [7]. Fungi, bacteria, microbial toxins such as aflatoxins, heavy metals and pesticides are among the contaminants reported in marijuana. Such contamination may carry additional health risks to the marijuana uses [7]. Aspergillus and penicillin specie fungi are known contaminants as well [12,13,14]. In addition, high levels of bacterial contamination have also been noted [15]. Heavy metals such as aluminium in soil may percolate to the marijuana plant and thus may become a contaminant thus, leading to an increase in the heavy metal load in the body upon smoking marijuana [16]. Pesticides used for pest control during marijuana cultivation can be a contaminant. High pesticide levels are noted in indoor grown marijuana when compared to naturally grown marijuana [17,18]. Many substances are added to “bulk up” marijuana [19] and glass has been noted as a contaminant to increase the crystalline appearance similar to resin glands [20].

Case reports of marijuana’s association with pericarditis/myocarditis are rare but have been reported [8,9,10,11] [Table 1]. The pathogenesis of pericarditis/myocarditis related marijuana is unclear. One possible explanation is contaminants as possible cause. Further research to explore marijuana as a cause of myocarditis/pericarditis/myopericarditis is needed.

<table>
<thead>
<tr>
<th>Year of publication, first author, reported from</th>
<th>Age (in years) and sex</th>
<th>Presenting complaint</th>
<th>EKG</th>
<th>Troponin</th>
<th>TTE</th>
<th>Diagnosis</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008, Evangelos Leontiadis, Germany [8]</td>
<td>16 years, Male</td>
<td>Acute heart failure</td>
<td>sinus tachycardia, right axis deviation, slow R-wave progression in leads V1–4 and negative T waves in leads II, III, aVF, and V4–6.</td>
<td>Normal</td>
<td></td>
<td>Severe dilated left ventricle with an ejection fraction of 15% and global hypokinesis, moderate to severe mitral regurgitation, moderate pulmonary hypertension, small pericardial effusion, and a mobile left ventricular apical thrombus.</td>
<td>Myocarditis Recovered</td>
</tr>
<tr>
<td>2014, Carlos E. Rodriguez-Castro, United States [9]</td>
<td>29 years, male</td>
<td>Chest pain</td>
<td>ST-segment elevation in the inferior leads with PR-segment depression</td>
<td>Elevated</td>
<td></td>
<td>Ejection fraction of 70% with moderate concentric hypertrophy consistent with athlete’s heart. No wall motion abnormalities seen.</td>
<td>Myopericarditis Recovered, had a recurrence</td>
</tr>
<tr>
<td>2016, J. Tournebize, France [10]</td>
<td>15 years, male</td>
<td>Chest pain</td>
<td>Not specified</td>
<td>Elevated</td>
<td>Not obtained</td>
<td></td>
<td>Myocarditis Recovered</td>
</tr>
<tr>
<td>2017, Thomas M. Nappe, United States [11]</td>
<td>11 months, male</td>
<td>Chest pain</td>
<td>central nervous system (CNS) depression and then went into cardiac arrest</td>
<td>wide-complex tachycardia.</td>
<td>Not reported</td>
<td>Autopsy revealed a non-dilated heart with normal coronary arteries</td>
<td>Myocarditis Death</td>
</tr>
</tbody>
</table>

4. Conclusion

Potency and contaminants in marijuana are highly variable. Myocarditis/pericarditis/myopericarditis secondary to contaminants in marijuana as a cause should be suspected in young patients with marijuana use presenting with pericarditis. Additional research is needed to further establish the association between marijuana and myocarditis/pericarditis/myopericarditis. Along with the treatment with non-steroidal anti-inflammatory drugs and colchicine, abstinence from marijuana and avoiding passive smoking is of prime importance to avoid the occurrence and recurrence of marijuana related myocarditis/pericarditis/perimyocarditis.

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