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Marijuana-induced Coronary Vasospasm and Myocardial Infarction: A Case Report and Review of Literature

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Abstract

The usage of marijuana and its legalization has been growing rapidly, being abused by a wide range of age groups. Its effects on the heart are well known, but coronary artery vasospasm causing ST elevation myocardial infarction (STEMI) from Marijuana alone is rather lesser known. Herein, we report a case of a middle aged African American man with a significant tobacco smoking history who presented with chest pain typical of myocardial infarction (MI) soon after smoking marijuana. ECG showed ST elevation in inferior leads with first degree AV block and a urine drug screen positive only for marijuana. Coronary angiogram showed mid right coronary artery (RCA) obstruction which was relieved upon injection of intracoronary nitroglycerine. This case report reinstates the significance of considering substance abuse as an etiology of STEMI during initial presentation, ruling out with urine drug samples. We also present a literature review of coronary vasospasm with STEMI, induced specifically by Marijuana and its pathophysiologic mechanisms.

Keywords

marijuana; ST-segment elevation myocardial infarction; cannabinoid receptors; vascular endothelial dysfunction; demand ischemia

1. Introduction

Vasospastic angina as a clinical entity has been known for long with the first case being studied dating back to 1959. According to the COVADIS (Coronary Vasomotion Disorders International Study Group), the diagnosis should include nitrate responsive angina, transient ischemic changes on the electrocardiogram (EKG) and diagnostic evidence of coronary artery vasospasm [1]. Although vasospastic angina is transient, the persistence of the

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inflicting cause can lead to subsequent myocardial infarction. While the risk factors like diabetes, hypertension, smoking [2] and cocaine [3] have been established, the association of marijuana with coronary artery spasm is less frequently reported.

The usage of marijuana in the United States of America (US) has steadily been growing. Marijuana is the most common drug of abuse in the US [4]. According to the National Survey on Drug Use and Health 2018, 19.4% (53.1 million) of the US population aged ≥12 years of age reported using marijuana in the past year which is significantly high from estimates in the past years [4]. The prevalence of marijuana use was 4.1% in 2001–2002 and 9.5% in 2012–2013, showing a significant increase among all demographic subgroups [5]. The use of synthetic cannabinoid drugs has been in the rise as well with increased reporting of adverse effects [6]. As of December 1, 2019, 11 states have legalized marijuana for recreational purposes and 33 states have legalized marijuana for medical use. Given the increasing use, it is crucial to understand the implications of marijuana use in cardiovascular health and disease. We here report a case of ST-segment elevation secondary to coronary vasospasm in the setting of recent marijuana consumption.

2. Case Report

A 64-year old African American male with a history of 20-year tobacco smoking history, gunshot wounds to abdomen, hypertension, no prior cardiac history presented to the ED with a non-radiating retrosternal chest pain lasting for 30 minutes associated with diaphoresis, nausea and vomiting that began after smoking a blunt of marijuana. On arrival at the emergency department his blood pressure was 116/75 mm Hg, heart rate was 57 beats per minute, he was afebrile and respiratory rate was 16 cycles per minute. He was saturating 94% on room air. Complete blood count, electrolytes, liver and renal function tests were within normal limits. Troponin at presentation was 0.08 ng/mL. Electrocardiogram (EKG) showed ST segment elevation in leads II, III and aVF and ST-segment depressions in leads aVL, V4, V5 consistent with inferior myocardial infarction with reciprocal changes and first-degree heart block (Figure 1). Meanwhile, his urine toxicology screen was positive of cannabinoids, negative for opioids, cocaine or amphetamines. A diagnosis of inferior STEMI was established and the patient was emergently taken for coronary angiogram under conscious sedation. Coronary angiogram revealed a focal mid-right coronary artery (RCA) obstruction (Figure 2). Upon injection of 50 mcg intracoronary nitroglycerine normal luminal patency was established (Figure 3). Hence it was established that the patient's inferior STEMI was secondary to RCA spasm likely due to marijuana use. After the procedure, troponin increased to 26.78 ng/mL; subsequently trended down to 13.76 and 6.65 ng/ml. A repeat EKG post coronary angiogram prior to discharge showed normalization of ST-T changes (Figure 4). A transthoracic echo showed normal left ventricle ejection fraction of 55–60% with mild hypokinesia of small area in the inferior wall. Patient was subsequently discharged on aspirin, clopidogrel as a part of dual antiplatelet strategy, high intensity statin (atorvastatin). He was counselled to abstain from marijuana use.

3. Discussion

The psychoactive component of marijuana is delta-tetrahydrocannabinol (δ-THC) [7]. δδ-THC acts on CB1 and CB2 cannabinoid receptors. CB1 receptors are located in the brain, hear, liver and vascular smooth muscle cells and CB2 receptors are present on immune cells. Both receptor types are known to be present in atherosclerotic plaque [8]. CB1 receptors are pro-atherogenic and CB2 are anti-atherogenic [9]. The risk of myocardial infarction increases by 4.8 times within 1 hour of marijuana use [10].

Several mechanisms contribute in pathophysiology of myocardial infarction related to marijuana use. Marijuana at low doses independently causes tachycardia by sympathetic stimulation through the release of norepinephrine [11]. This increased sympathetic tone lasts for 2–3 hours [12] can result in increased afterload and hence result in demand ischemia [13]. At high doses, marijuana inhibits the parasympathetic activity of the heart causing vasodilation, bradycardia, postural hypotension and reflex tachycardia [14].

δ-THC is known to be procoagulant by causing increased platelet aggregation and thrombosis [15]. It has been proposed that marijuana causes plaque rupture due to the hemodynamic stress leading to plaque ruptures, thrombus formation and infarction [15]. Sugamura et al. noted that there was an increased expression of CB1 (prothombotic) in lipid rich atheromatous plaques in patients with unstable angina than in stable angina worsening clot formation and dislodging [16]. Marijuana also leads to slow coronary flow [17] and no coronary flow [18] in the absence of coronary artery stenosis. Marijuana is known to cause vascular endothelial dysfunction [19]. Multiple similar cases of STEMI [20,21,22,23] and NSTEMI related to marijuana induced have been reported in the past [24].

Physicians should be aware of the varied cardiovascular adverse effects of marijuana such as myocardial infarction [9,25], cardiac arrhythmia [26] and myocarditis [27]. Abstinence is the key to prevent recurrent myocardial infarctions secondary to marijuana use [28]. The differential diagnosis for ST segment elevation related to marijuana use includes myocarditis [29] and coved ST-elevation of brugada pattern [30].

4. Conclusion

In summary, we present a case of coronary vasospasm and STEMI secondary to marijuana. This review emphasizes the importance of considering marijuana induced ACS in an emergent clinical setting. With increasing exploitation of marijuana, despite being classified as a Schedule 1 drug by the government, presentation from adverse effects is expected to grow. It is prudent that clinicians stay aware of acute cardiac presentations related to substance abuse such as myocardial infarction, dissections, coronary vasospasm, arrhythmias. Having these as differentials can certainly hasten the focus of management thereby improving cost and work efficiency.

No.	Authors	Age(N)	Gender	Marijuana intake prior to symptoms episode (hours)	Chief complaint	Urine positive for cannabis	ECG	Troponin	CAG finding	Treatment
1	Iftikhar et al. (20)	32	M	1	Radiating chest pain with sweating, nausea	-	ST depression in anterolateral leads	0.97	TIMI II flow in LAD	IV NTG. Flow improved to TIMI III
2	Gunawarden et al. (21)	29	M	4	Radiating chest pain		ST elevation in inferior/ anterolateral	2.8 ng/mL	Slow flow in LAD	Antiplatelets, nitrates, anticoagulants, Ca++ channel blockers
3	J Baskaran et al. (22)	60	M	NA	Syncopal episode, No CPR	+	ST elevation in inferior leads	Negative	80% stenosis of LAD&RCA, ICC in RCA	IV NTG, oral CCB
4	Casier et al. (23)	52	M	2	Syncope, asystole with ROSC	+	ST elevation in antero-inferior leads	6.35 mcg/L	Marked vasospasm of LAD	IV NTG – vasodilation was achieved

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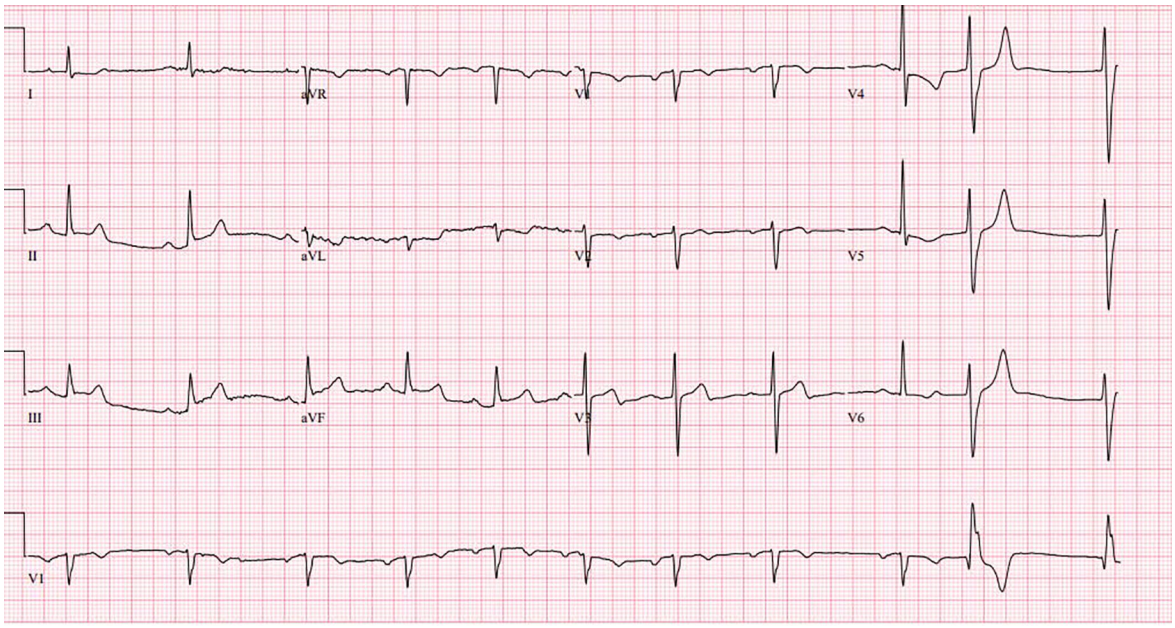

Figure 1.

ST elevation in leads II, III and aVF and ST segment depression in leads aVL, V4, V5 consistent with inferior infarction with reciprocal changes, premature ventricular complex, and first-degree heart block

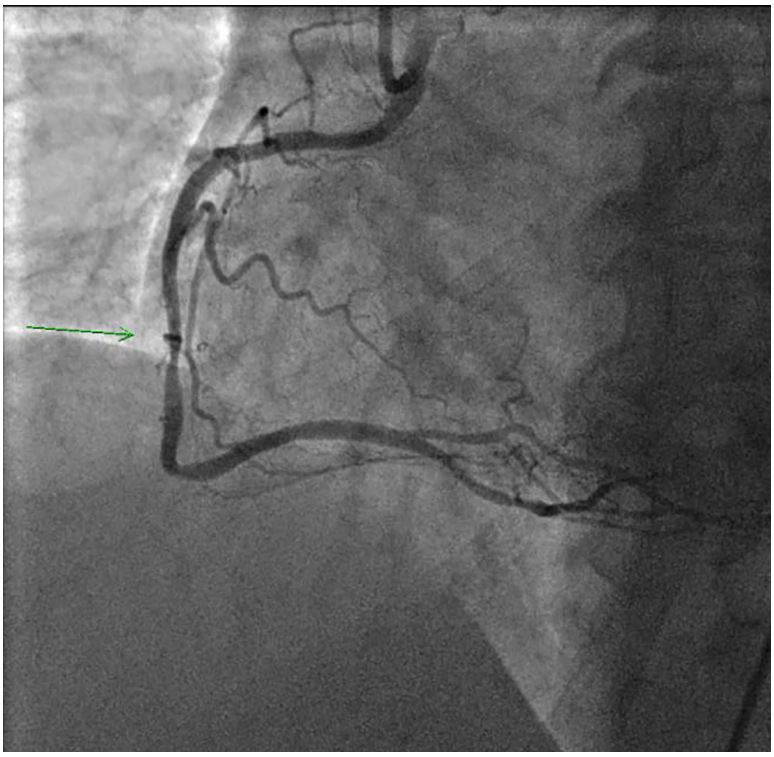

Figure 2.
Coronary angiogram of right coronary artery (RCA) showing mid-RCA tubular lesion with 60% obstruction

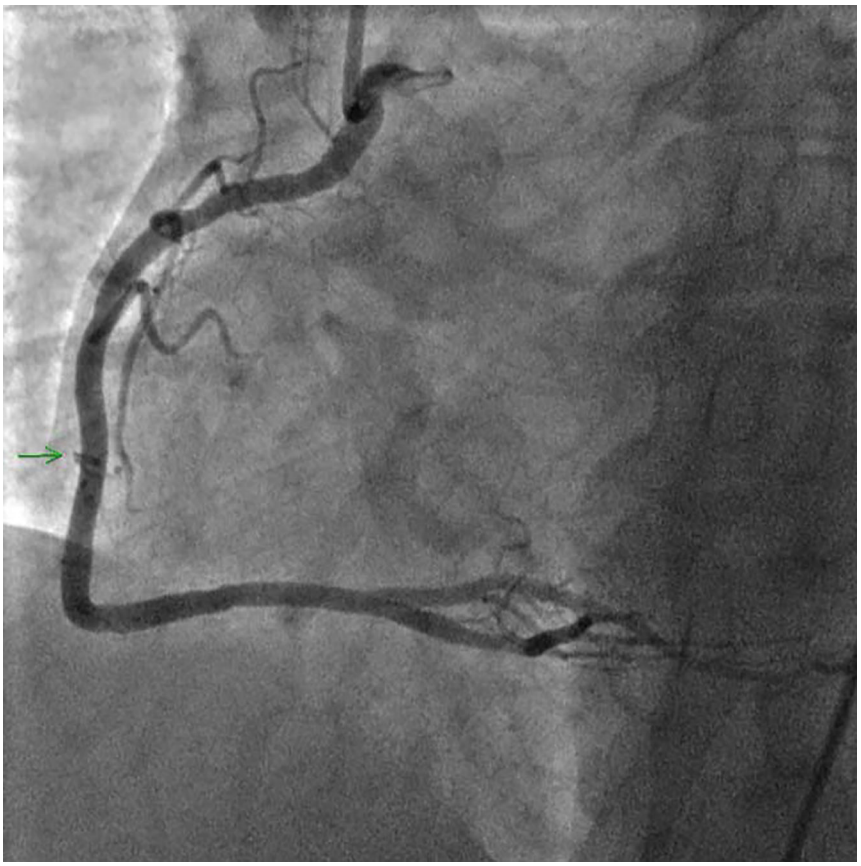

Figure 3.
Coronary angiogram of right coronary artery (RCA) showing reestablished patency following intracoronary nitroglycerine injection

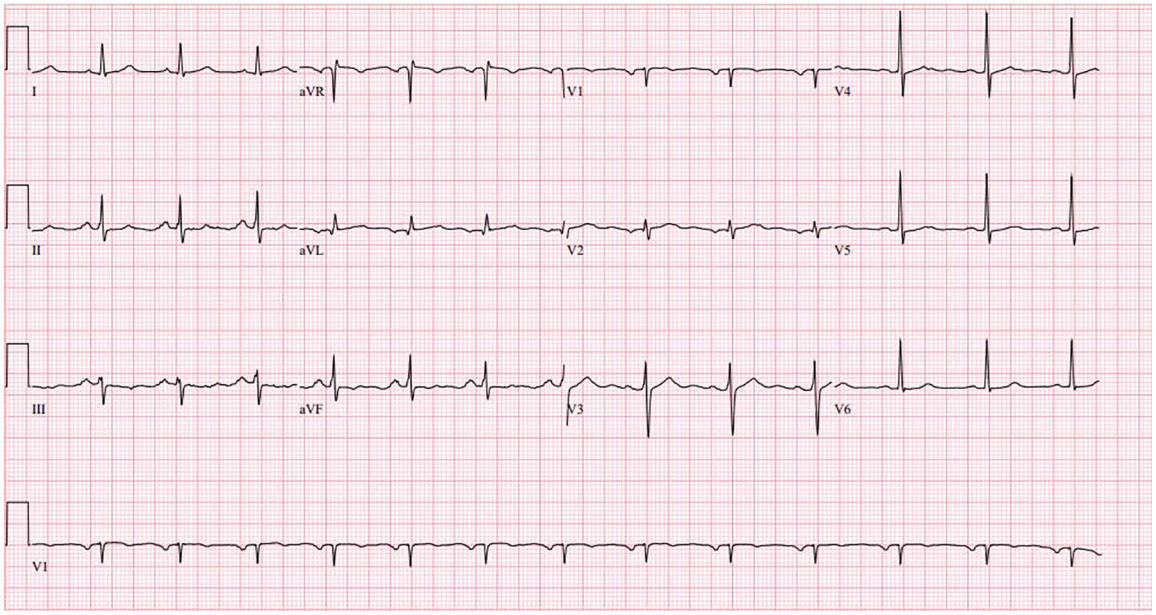

Figure 4.
Repeat EKG after coronary angiogram showing normalization of ST/T wave changes