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## Marijuana Induced Type I Brugada Pattern: A Case Report

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### Abstract

Marijuana is the most common drug of abuse in the United States. Marijuana acts on cannabinoid receptors CB1, CB2 and another distinct endothelial receptor. Marijuana is known to cause tachycardia, hypotension and hypertension. Various arrhythmias including atrial fibrillation, atrial flutter, II degree AV block, ventricular fibrillation, ventricular tachycardia, asystole and brugada pattern associated with marijuana use have been reported. We here present an interesting case of Type I Brugada pattern in electrocardiography (ECG) in a 36 year old healthy African American male who presented after smoking four joints. Urine toxicology test proved marijuana use. Acute coronary syndrome was ruled out, coronary angiogram revealed normal coronaries, 2D echocardiogram showed no evidence of structural heart disease. Upon resolution of Brugada pattern in ECG, procainamide challenge performed in electrophysiology laboratory did not induce Brugada pattern. Patient was asked to return to hospital if he developed fever that did not resolve with antipyretics. Further studies are required to understand the effect of marijuana on cardiac ion channels.

### Keywords

marijuana; cannabis; brugada

### 1. Introduction

Marijuana is the most commonly abused illicit drug in the United States [1]. The Cannabinoid receptors CB1, CB2 and an endothelial receptor distinct from these cannabinoid receptors are present in the heart and mediate a variety of functions [2]. Common cardiac manifestations of marijuana use include a dose-dependent tachycardia, hypotension, orthostatic hypotension, and a supine increase in blood pressure [2,3]. Cardiac

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arrhythmia including atrial fibrillation [4,5,6], atrial flutter [7], II degree AV block [8], ventricular fibrillation [9], ventricular tachycardia [10,11,12], asystole [13,14] have been reported with marijuana use. Despite these recent reports the effect of marijuana and its active components on ion-channels in general, cardiac ion channels in particular is largely lacking. Previous investigators have reported a case of acute marijuana intoxication that presented with Brugada like-ST segment elevation [15,16,17,18]. We here present a case of type I Brugada pattern associated with marijuana use.

## 2. Case Presentation

A 36-year-old, previously healthy, African-American male who presented to our institution with precordial chest pain of one-hour duration. The pain was characterized as sharp, severe, aggravated by deep inspiration. The patient also complained of “fever, fatigue and nausea”, but was afebrile at presentation. The patient admitted to smoking “four joints” over the previous 30 hours. Family history was significant for “premature cardiac death” of two maternal cousins in their second decade of life. 12 lead electrocardiogram (ECG) revealed sinus rhythm at a rate of 91 bpm, with an incomplete right bundle branch block pattern, coved ST elevation pattern in lead V1, V2 consistent with a Brugada Type I pattern (Figure 1). The patient was found to have no evidence of coronary artery disease by coronary angiography while a 2 D echocardiogram showed no evidence of structural heart disease and a preserved left ventricular function. Serial troponin levels were not elevated; urine toxicology confirmed marijuana use, and he remained afebrile. No supraventricular or ventricular arrhythmias were noted during the hospital stay. Procainamide challenge did not reproduce brugada pattern after normalization of EKG. No ventricular arrhythmias were noted. Patient was subsequently discharged and was asked to return to hospital he developed fever.

## 3. Discussion

Marijuana is the most common drug of abuse and has more than 460 active chemicals including  $\delta$ -9- tetrahydrocannabinol (THC) [19]. THC mainly acts via CB1 and CB2 receptors, the activation of the former is atherogenic while the activation of the latter is anti-atherogenic [20]. Multiple isolated case reports of arrhythmias including Brugada pattern have been reported in the past [4–18]. Despite multiple case reports of arrhythmias associated with marijuana use, research exploring the effect of THC/marijuana on cardiomyocytes and ion channels are largely lacking.

Anandamide, an endogenous cannabinoid is known to inhibit the ventricular voltage-dependent sodium and calcium ion channel independent of CB1 and CB2 receptor activation [21]. Anandamide inhibits INA current in neurons [22] and *Xenopus* oocytes [23], and no evidence exist suggesting involvement of cardiac sodium channels. A greater expression of the outward potassium channels (Ito) in epicardial as compared to, endocardial cells results in the characteristic spike-and-dome action potential (AP), highlighted by the typical “saddleback” electrocardiogram pattern. The negative T-wave in the Type I Brugada pattern may be attributed to prolongation of repolarization in the epicardial (Ito rich) myocytes, creating heterogeneity of repolarization between the epicardial and endocardial cells and

such heterogeneity provides the substrate for phase two reentry in Brugada [24]. Interestingly, in our patient, infusion of procainamide after normalization of the ECG did not reproduce the Brugada pattern arguing against the involvement of sodium channels. Since intravenous flecainide, which was reported to reproduce marijuana induced Brugada pattern [25] is not available in the US, we could not decisively exclude the involvement of sodium channels. Although high vagal tone has potential for induction of the Brugada pattern [26], we doubt vagotonic properties of marijuana were involved as the resting HR was not slow. This patient was advised to cease use of marijuana, and to seek medical attention for a fever that persists despite antipyretic therapy and to inform anesthesia of the possibility of Brugada in the event he requires surgery. This patient, as well as his first degree relatives, would benefit from genetic testing and counseling.

In summary, we present an interesting case of marijuana induced Type I Brugada pattern in a patient who admittedly consumed a large quantity of the inhaled agent. Further studies to understand the effect of marijuana on cardiac ion channels are needed.

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**Figure 1.**  
EKG of the patient showing Coved ST-segment elevation in lead V1, V2 consistent with type 1 Brugada pattern