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Atrioventricular Nodal Reentrant Tachycardia Triggered by Marijuana Use: A Case Report and Review of the Literature

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Abstract

Marijuana is the most commonly abused recreational substance. With the increasing legalization of marijuana, its use is expected to rise. Delta-9-tetrahydrocannabinol (THC) is the psychotropic component of marijuana, acting via CB1 and CB2 G-protein coupled cannabinoid receptors. Marijuana has serious cardiovascular effects including tachycardia, orthostatic hypotension, angina and myocardial infarction to name a few. Previous reports by our group and others documented various arrhythmias other than atrioventricular nodal reentrant tachycardia (AVNRT) that are associated with marijuana use. In this report, we present a case of AVNRT associated with marijuana use. Marijuana in high doses stimulates parasympathetic nerves. While parasympathetic stimulation can increase the refractory period of the fast conduction pathway, it has no effect on the slow and retrograde pathways, therefore its use creates an ideal milieu for AVNRT initiation and maintenance. Our case report highlights the importance of including marijuana use in the differential diagnosis, as a possible trigger, for patients presenting with AVNRT that is otherwise unexplainable.

Keywords

marijuana; arrhythmia; avnrt

1. Introduction

Marijuana is the most common drug of abuse in the United States, with use expected to rise due to legalization for medical and recreational purposes [1]. Delta-9-tetrahydrocannabinol

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(THC) is the psychotropic component of marijuana, acting via CB1 and CB2 G-protein coupled cannabinoid receptors, which are also present in the heart [2,3]. Marijuana with increased potency has become available in recent years [4]. Tachycardia, hypotension, myocardial infarction and decreased time to angina are among the cardiovascular effects reported with marijuana use [5]. To our knowledge, we present the first reported case of atrioventricular nodal reentrant tachycardia (AVNRT) associated with marijuana use, and discuss the possible mechanism of marijuana initiated AVNRT.

2. Case Report

A 40 year-old male with no known past medical history presented with palpitations that started within one hour after smoking marijuana. The palpitations were associated with substernal chest pain, which was non-radiating, not pleuritic, and not related to change in position. The patient denied dizziness or syncope. Exercise tolerance at baseline was more than 1 mile. At time of presentation, his heart rate was 190 beats per minute, blood pressure was 117/57 mmHg, respiratory rate was 18 per minute, and he was afebrile. Electrocardiography revealed the rhythm to be AVNRT (Figure 1). The patient spontaneously converted to normal sinus rhythm with premature ventricular complexes (Figure 2). Bloodwork revealed no electrolyte abnormalities and the patient was euthyroid. Urine toxicology was positive for marijuana use and negative for other illicit drugs. Mild troponin elevation was noted, with values of 0.018, 0.213, and 0.138 ng/mL (normal value < 0.010 ng/mL). The levels were attributed to demand ischemia. The patient underwent AVNRT ablation without complications.

3. Discussion

Two distinct atrial impulses approach the atrioventricular node (AV) node—one from the fast pathway located in the anterior portion of the triangle of Koch, and the other via the posterior pathway located in the posterior portion of the triangle of Koch. As the name suggests, the anterior pathway has faster conduction but a longer refractory period; and the posterior pathway conducts more slowly but has a shorter refractory period. This difference in the refractoriness of the two pathways is key in the pathophysiology of AVNRT. In normal sinus rhythm the conduction occurs via the fast pathway. In susceptible individuals, a premature atrial beat may find the fast pathway refractory; however, the slow pathway may be available for conduction. If the impulse reaches the common end when the fast pathway has repolarized there may be retrograde conduction via the fast pathway back to the atrium. Thus, a re-entrant pathway of the “slow to fast” variety of AVNRT may be established. This type of AVNRT is referred to as typical AVNRT [5,6]. In typical AVNRT the P-waves may be buried in QRS complex or appear at the end of the QRS complex [7]. Nicotine, alcohol, exercise, stimulants, and a surge in vagal tone are reported triggers of AVNRT [8].

The effect of marijuana on the conduction system of the heart are not fully understood. Various arrhythmias reported to date are summarized in Table 1. A change in P-wave morphology may be noted following marijuana use suggesting an effect on the atrium [33]. Decreased sinoatrial (SA) conduction, delayed atrium to bundle of His (A to H interval) conduction, and a decreased AV node refractory period are reported effects of THC [34].

Autonomic nervous system mediated increases in SA and AV node conduction have been reported [35]. There are differences in the regional sympathetic and parasympathetic neuronal distributions in the heart. Vagal influence is predominant in the SA node and sympathetic nerves predominate in the atrium [36]. Marijuana's effect on sympathetic and parasympathetic nerves appear to be dose dependent, with sympathetic nerves stimulated at lower doses and parasympathetic at higher doses [37]. Vagal tone has different effects on the fast and slow pathways. Vagal tone increases the refractory period of the fast pathway but does not have this effect on the slow pathway and retrograde fast pathway [38]. This may explain the occurrence of AVNRT during periods of heightened parasympathetic tone [38]. Marijuana induced parasympathetic stimulation may cause inhibition of the fast pathway, and a premature atrial complex generated at this time may be conducted antegrade via the slow pathway with subsequent retrograde conduction, thus initiating AVNRT.

In conclusion, marijuana may be a trigger for AVNRT. Health care providers should be aware of this and consider marijuana use as a potential trigger of AVNRT. A focused history and urine toxicology screen may aid in the diagnosis.

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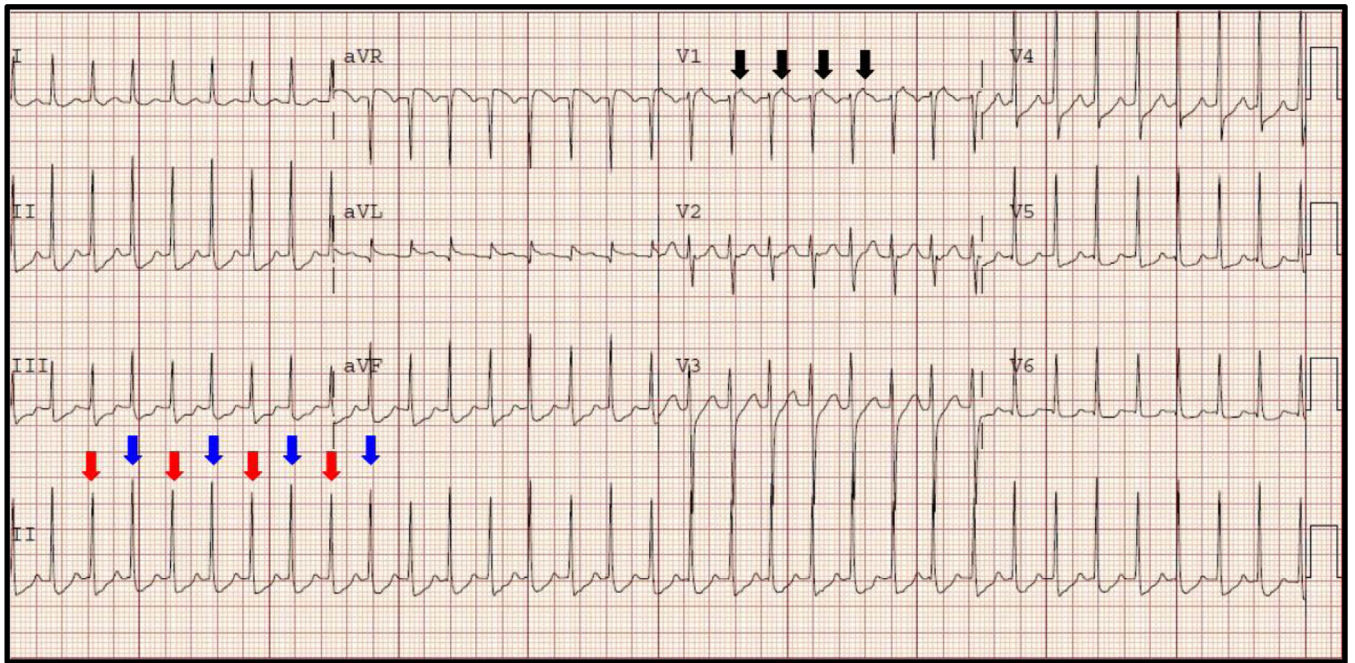


Figure 1. ECG showing AVNRT at the time of presentation. Note electrical alternans indicated by red and blue arrows. Retrograde P waves are marked with black arrows

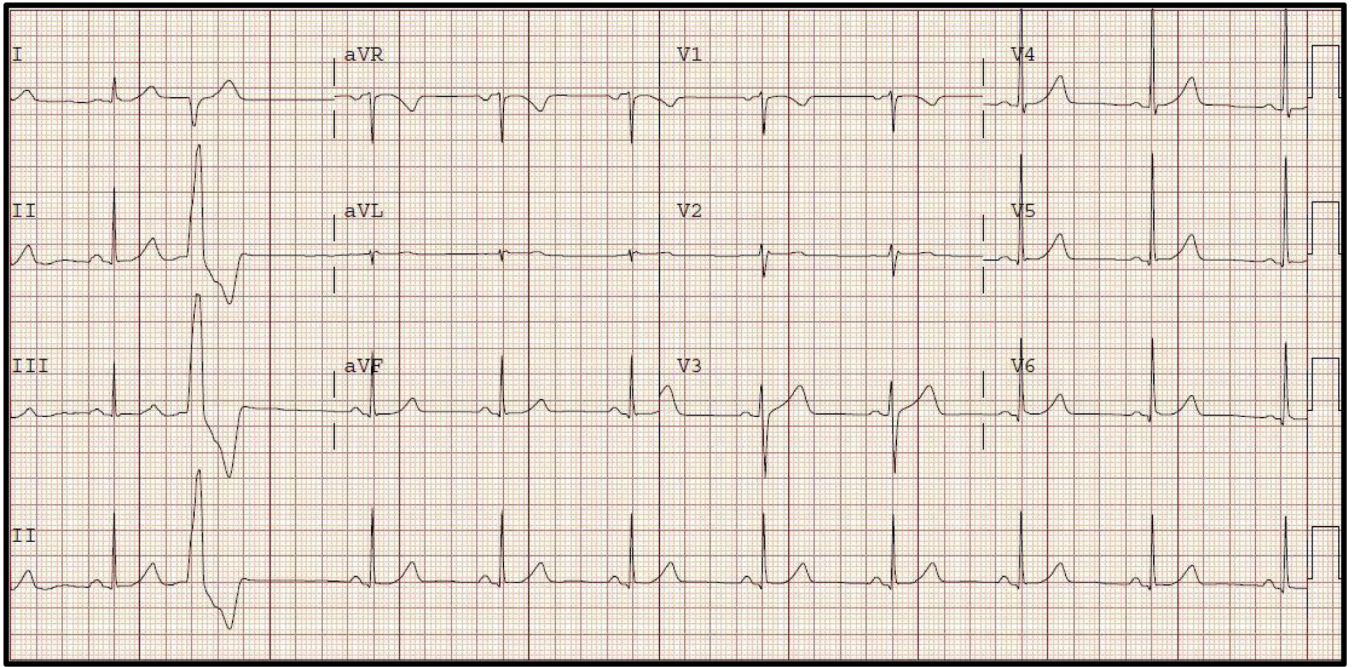


Figure 2.
ECG showing normal sinus rhythm and premature ventricular complex after spontaneous conversion from AVNRT

Table 1.

Arrhythmias Associated with Marijuana Use

Case	Year and author	Reported arrhythmia
1	1981, Akins [9]	Sinus bradycardia, first degree atrioventricular block, second degree atrioventricular block
2	2000, Kosior [10]	Atrial fibrillation
3	2000, Singh [11]	Atrial fibrillation
4	2001, Kosior [12]	Atrial fibrillation, supraventricular tachycardia unknown type
5	2001, Kosior [12]	Atrial fibrillation
6	2003, Rezkella [13]	Ventricular tachycardia
7	2005, Fischer [14]	Atrial flutter converted to atrial fibrillation after adenosine
8	2005, Charbonney [15]	Atrial fibrillation
9	2007, Dacarett [16]	Brugada pattern
10	2008, Baranchuk [17]	Ventricular fibrillation
11	2009, Sanchez-Lazaro [18]	Ventricular tachycardia
12	2009, Sattout [19]	Asystole, Ventricular tachycardia
13	2011, Fernandez-Fernandez [20]	Asystole, ventricular fibrillation
14	2012, Ramero-Punche [21]	Brugada pattern, frequent ventricular premature complexes
15	2012, Duffley [22]	Ventricular tachycardia
16	2013, Menahem [23]	Incomplete right bundle branch block, asystole, ectopic atrial tachycardia
17	2013, Kouzam [24]	Non-sustained ventricular tachycardia
18	2014, Hartung [25]	Ventricular fibrillation
19	2014, Hartung [25]	Sudden cardiac death
20	2014, Singh [26]	Atrial fibrillation
21	2016, Brancheau [27]	Asystole / sinus arrest
22	2016, Valle-Alonzo [28]	Brugada ECG pattern
23	2016, Orsini [29]	Ventricular fibrillation
24	2017, Yalsin [30]	J waves (type III pattern)
25	2017, Yalsin [30]	J waves (type II pattern)
26	2017, Doctorian [31]	Ventricular fibrillation, Brugada ECG pattern
27	2018, Theetha Kariyanna [32]	Brugada ECG pattern