Paroxysmal atrial fibrillation after recreational marijuana smoking: another “holiday heart”?

Emmanuel Charbonneya, Juan-Marc Sztajzelb, Pierre-Alexandre Polettic, Olivier Rutschmann

a Internal medicine department, University Hospital Geneva, Switzerland
b Department of cardiology, University Hospital Geneva, Switzerland
c Emergency department, University Hospital Geneva, Switzerland

Summary

Paroxysmal atrial fibrillation (AF) is a common arrhythmia that may occur after various triggers. With the exception of the well recognized “holiday heart” associated with acute alcohol consumption, the cause of AF is rarely identified in young adults. We report a case of paroxysmal AF after marijuana smoking in a 22 year old female with no other identifiable triggers and normal echocardiography. Marijuana smoking should be included in the list of possible triggers in young adults presenting with paroxysmal AF, once cardiac disease and other common causes of AF have been excluded.

Key words: marijuana (cannabis); atrial fibrillation; young patients

Introduction

Marijuana is an increasingly popular recreational drug in adolescents and young adults and is usually considered fairly safe by the users [1]. It is also used for the treatment of nausea and pain, especially in oncology and in patients suffering from AIDS [2, 3]. The most common side effects involve the central nervous system, and include dizziness, dysphoria or depression, and hallucination. Sinus tachycardia and vasovagal syncope are frequent cardiovascular effects, but only rare cases of arrhythmia have been reported [4, 5, 14].

Case report

A 22 year-old woman presented herself at the emergency department complaining of dizziness, hot flush and malaise following a syncope. During the three hours before admission, she had one single drink of alcohol and smoked more marijuana than usual (equivalent to 3–4 days smoking); she had not taken an excessive amount of other beverages or substances. Her previous medical history was irrelevant with the exception of occasional palpitations during marijuana smoking. These previous episodes were described as regular rapid heart beats resolving spontaneously. She also reported episodes of orthostatic hypotension. During the present episode, she had not felt any palpitations. Her only medication was oral contraception, but she reported daily marijuana smoking (1–2 joints) during the last two years. She did not report any cardiovascular risk factors, except the small amount of tobacco smoked with marijuana. Her physical examination was unremarkable except for irregular heartbeats at a frequency of 120–130 beats per minute.

An ECG confirmed the presence of AF (fig. 1a). A complete blood count, thyroid tests and blood electrolytes (Na, K, Mg, Ca) were normal. Blood screening for alcohol and a broad search for drugs were negative. One hour after admission, the AF spontaneously converted to sinus rhythm (fig. 1b). During continuous ECG monitoring in the emergency department, no recurrence of the arrhythmia was observed. A transthoracic echocardiography found no structural or functional anomaly. Finally, no aetiology other than the marijuana consumption could explain this AF episode. On account of her history we interpreted the initial syncope as vasovagal. Six weeks later she was in perfect condition and did not present any other arrhythmia. She seemed to have reduced marijuana consumption.
Cannabis is one of the most common recreational drugs and is consumed by all levels of the population. Most consumers are young men, who begin to smoke as teenagers. It is legally sold in some European countries, and usually considered as fairly safe by its users, in spite of the well-known negative effects of chronic abuse. Most of these effects are neuropsychological (cognitive and psychomotor impairment, anxiety, depression, psychosis), but transient spermatogenesis and female cycle disturbance have also been reported. Although hypotension and sinus tachycardia are commonly associated with marijuana smoking [9] and usually well tolerated [6, 7], more severe arrhythmias and other cardiac events are rare.

Little is known about the cardiovascular effects of 9-9-tetrahydrocanabinol (9-9-THC), [8–10] the active substance of cannabis and we do not know much about the cannabis receptors (CB1, CB2). The cardiovascular effects of cannabis have been characterized by experimental pharmacological studies on animal models [11]. In these studies, cannabinoids had an action on cardiovascular regulatory centres and on the peripheral autonomic system through the CB1 receptor. Central and peripheral CB1 receptors play a role in the regulation of heart regulatory centres and the peripheral autonomic system. The effects of cannabinoids seem to be dose-dependant. At low doses, it can inhibit the release of postganglionic catecholamines [12] but at higher doses an increase in heart rate through peripheral inhibition of car-

**Figure 1**
ECG at admission (a) and after spontaneous return to sinus rhythm (b).

**Discussion**

Cannabis is one of the most common recreational drugs and is consumed by all levels of the population. Most consumers are young men, who begin to smoke as teenagers. It is legally sold in some European countries, and usually considered as fairly safe by its users, in spite of the well-known negative effects of chronic abuse. Most of these effects are neuropsychological (cognitive and psychomotor impairment, anxiety, depression, psychosis), but transient spermatogenesis and female cycle disturbance have also been reported. Although hypotension and sinus tachycardia are commonly associated with marijuana smoking [9] and usually well tolerated [6, 7], more severe arrhythmias and other cardiac events are rare.

Little is known about the cardiovascular effects of 9-9-tetrahydrocanabinol (9-9-THC), [8–10] the active substance of cannabis and we do not know much about the cannabis receptors (CB1, CB2). The cardiovascular effects of cannabis have been characterized by experimental pharmacological studies on animal models [11]. In these studies, cannabinoids had an action on cardiovascular regulatory centres and on the peripheral autonomic system through the CB1 receptor. Central and peripheral CB1 receptors play a role in the regulation of heart regulatory centres and the peripheral autonomic system. The effects of cannabinoids seem to be dose-dependant. At low doses, it can inhibit the release of postganglionic catecholamines [12] but at higher doses an increase in heart rate through peripheral inhibition of car-

**Figure 1**
ECG at admission (a) and after spontaneous return to sinus rhythm (b).
diac parasympathetic fibres has been observed. Other mechanisms have been proposed to explain marijuana-induced arrhythmias and the role of 9,9-THC in triggering cardiac ischemia [13, 14]. Cannabis might have a direct effect on autonomic activity or on Purkinje fibres and may also have a direct effect on coronary microcirculation [14].

The timing of the acute cardiovascular effect of marijuana is described as beginning after 10 minutes and lasting for a duration of 2–3 hours [10].

This case is only the third report of marijuana-induced paroxysmal AF, but the real prevalence of these events is certainly underestimated. In this young female, none of the usual triggers for AF were found and she had consumed an unusually large amount of THC, suggesting that this episode was secondary to marijuana ingestion. This was probably caused by a conjunction of coronary hyperperfusion and autonomic disturbance.

It could be argued that the AF was a coincidental episode, although lone AF is a very rare pathology in young adults [15].

The role of simultaneous marijuana and alcohol consumption should also be discussed. Although major alcohol consumption is a well-reported trigger for AF by acting on the refraction and conduction of the atrium [16], our patient reported only a minor consumption of alcohol in conjunction with marijuana, and no alcohol was detected in her blood. Hence, we can hypothesize that the association of a small amount of alcohol with an unusual quantity of marijuana increased the potential cardiac toxicity of 9,9-THC. As both drugs are frequently taken together, this observation may be of great clinical importance in the evaluation of young patients with paroxysmal AF. When the patient denies the consumption of significant amounts of alcohol, the physician should ask about recent intake of marijuana. Unfortunately, there is no standardized method easily usable to confirm acute ingestion. Urine tests, which are commonly available, reflect chronic ingestion, due to the lipophilic characteristics of THC, its slow clearance and the presence of many metabolites [17]. Mass spectral gas chromatography, which is not easily feasible in emergency departments, is the most appropriate method available to determine the time of consumption.

**Conclusion**

In conclusion, marijuana is an unusual trigger for AF that might be underestimated in young healthy adults. Once the standard work-up has ruled out common causes for AF, the physician should look for marijuana ingestion, as 9,9-THC could precipitate arrhythmia.

**References**


