Journal of Cardiology Cases 7 (2013) e4–e7

Contents lists available at www.sciencedirect.com

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Spontaneous multivessel coronary artery dissection associated with cannabis use

Thouraya Filali (MD)*, Dhaker Lahidheb (PhD), Mehdi Gommidh (MD), Badii Jdaida (MD), Nadhem Hajlaoui (MD), Wafa Fehri (PhD), Habib Haouala (PhD)

Cardiology Department, Military Hospital of Tunis, Montfleury 1008, Tunisia

ARTICLE INFO

Article history:
Received 7 May 2012
Received in revised form 12 July 2012
Accepted 3 August 2012

Keywords:
Spontaneous coronary artery dissection
Cannabis
Acute coronary syndrome

ABSTRACT

Cannabis is the most widely used illicit drug in the world. It is generally considered to be a drug with low toxicity. Nevertheless, there are several case reports of myocardial infarction following cannabis use in otherwise low-risk individuals. We report the first case of a cannabis user presenting with acute coronary syndrome related to multivessel coronary artery dissection.

Introduction

Cannabis is the most widely used illicit drug in the world and in Tunisia. It has several effects on multiple organ systems. Diverse cases of cannabis-related acute coronary syndrome (ACS), even in the presence of normal coronary arteries, have previously been reported in the medical literature [1]. However, the relationship is not completely understood and several mechanisms of action have been proposed [2,3]. We present here the first case of a cannabis-related ACS with multivessel coronary artery dissection. Only one case of isolated coronary artery dissection associated with both cannabis and heroin use has been reported in the literature [4].

Case report

A 40-year old man was admitted to our intensive care unit with acute retrosternal chest pain. His past medical history revealed no coronary risk factor and no previous history of chest pain. He was an occasional cannabis smoker but revealed that for the last month he had smoked cannabis daily and in large amounts especially on the previous day. He denied cocaine use. No physical exercise and no emotional stress were noted before the onset of acute chest pain. The electrocardiogram showed a transient ST-elevation in anterior leads (Fig. 1). Physical examination was unremarkable. A 2D echocardiogram did not show any structural abnormalities and the ejection fraction was normal. Blood tests showed elevated T troponin at 5.64 ng/ml (reference value <0.01 ng/ml) with mild elevation of creatine kinase at 495 UI/l (reference value <190 UI/l). Due to dynamic resting ST segment changes on the electrocardiogram, the patient was referred for urgent coronary angiography. This showed luminal narrowing of the proximal segment of the left anterior descending (LAD) artery with dissection extending to its second segment without flow limitation. There were also dissections of the proximal segment of the circumflex and the mid segment of the right coronary artery (Figs. 2 and 3). Based on the complexity of the coronary lesions with multivessel coronary artery dissection and the clinical presentation (ACS with transient ST-elevation), the patient was referred for coronary artery bypass graft. Urine toxicology testing was positive for cannabis but negative for cocaine or any other recreational drug. At operation, a hematoma was found over the proximal LAD. The clinical course after surgery was uneventful.

Discussion

Spontaneous coronary dissection is a rare cause of ACS. Risk factors for coronary dissection comprise pregnancy, the use of oral contraceptives, connective tissue disorders, Marfan's syndrome, Ehlers–Danlos disease, blunt trauma, intensive exercise, and cocaine abuse [4]. No previous case of a 3-vessel coronary artery dissection related to cannabis use has been reported in the literature.

Cannabis is derived from the plant *Cannabis sativa* [5]. There are several preparations derived from this plant widely used for their euphoric and psychoactive effects [5]: marijuana, hashish, charas, ganja, and others. Cannabis toxicity is regarded to be low and a large previous cohort study showed no association of cannabis with cardiovascular disease hospitalization or mortality [6]. However,

* Corresponding author.
E-mail address: filalithouraya@voila.fr (T. Filali).

1878-5409/$ – see front matter
http://dx.doi.org/10.1016/j.jccase.2012.08.002

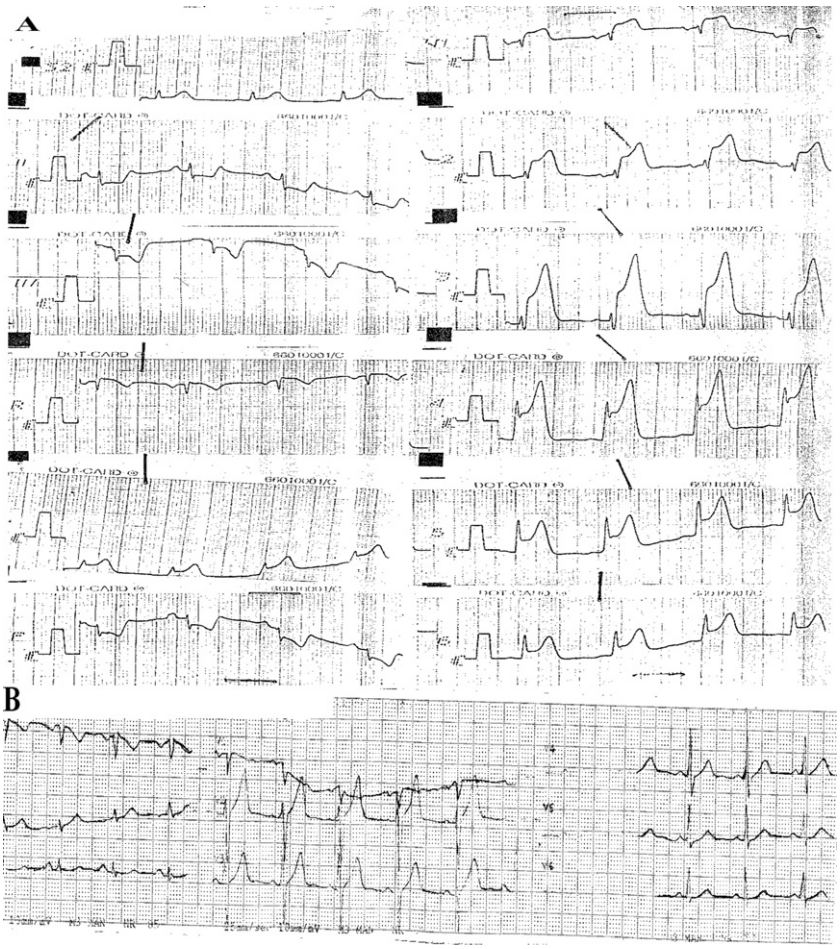

Fig. 1. Electrocardiogram (A) showing transient ST-elevation in anterior leads; electrocardiogram (B) showing regression of the ST-elevation.

evidence exists nowadays to support the cardiovascular effects of cannabis use [7]. In fact, several cases of ACS in young people without coronary risk factors and following cannabis smoking have been reported. Most of these patients had either normal coronary arteries or minimal irregularities on angiography [8]. The risk of myocardial infarction is 4.8 times higher within the hour after cannabis smoking, although this heightened risk seems to decline rapidly beyond the first hour [9]. Cannabis use can lead to frequent premature ventricular beats [8]. Even sudden death has been reported in cannabis users with or without coronary artery disease [5,10]. Adverse cardiovascular effects of cannabis are now well recognized, but actually not completely understood. The mechanisms, by which cannabis exerts its toxicity on the myocardium and the coronary vasculature, seem to be multifold. Tetrahydrocannabitol (THC) is the main active constituent reaching peak levels rapidly in the bloodstream during cannabis smoking [11]. It mediates its effect by activation of cannabinoid receptors which are found in many tissues including the heart and blood vessels [11]. THC leads to sympathetic stimulation with a rapid and substantial dose-dependent increase in heart rate and increase in cardiac output by more than 30% [8,12]. Increase in blood pressure can occur when sitting or supine and acute fall can occur when standing with orthostatic hypotension due to decrease in peripheral vascular resistance [8,12]. Cannabis smoking is also associated with rise in cardoxyhemoglobin and thus a decrease in oxygen carrying capacity [12]. All these effects lead to increased oxygen demand with reduced blood supply causing myocardial ischemia. In addition cannabis has marked vascular effects due to the vasoconstrictor effect of THC [8] and hence can worsen angina [3]. Vasospasm is consequently another patho-physiological mechanism that has been suggested, particularly to explain cases of coronary or cerebral ischemia where blood vessels have shown normal conditions in the affected areas after the ischemic event [10]. Cannabis smoking significantly lowers angina threshold for several hours following its use [2]. It also enhances oxidative stress with increased oxidized low-density lipoprotein formation, increased factor VII activity, activation and aggregation of platelets, and induction of an inflammatory response [8]. These effects of cannabis may trigger plaque disruption particularly in the presence of a vulnerable, but not necessarily stenotic, atherosclerotic plaque [9]. Plaque rupture from vasoconstriction with the procoagulation effects of cannabis on platelets will predispose to thrombosis [12,13]. In our patient who had no coronary risk factors, we believe that direct and indirect hemodynamic effects of cannabis such as vasoconstriction, sympathetic stimulation in addition to complex biochemical effects of cannabis, triggered multiple coronary dissections. As it has been proposed for cocaine, we think that the elevated arterial blood pressure associated with coronary vasospasm during cannabis smoking may generate sudden increases in the shear stress on the arterial wall, leading to an increased risk of dissection in those with normal coronary arteries like our patient and to atherosclerotic plaque rupture in those with coronary disease [14].

Although young people were most likely to report cannabis use, such use among older adults was at present higher than it had been a decade earlier [15]. Consequently the prevalence of