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Ventricular Tachycardia Revealing a Coronary Artery Spasm in a Young Cannabis Smoker in the Absence of Coronary Artery Disease: A Case Report

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Abstract **Case Report**

Vasospastic angina (VA), is characterized by symptoms of coronary angina caused by coronary vasospasm, usually in the absence of atherosclerotic changes. It typically presents with chest pain, which can be accompanied by transient electrocardiographic changes, if visualized during the attack. It can also rarely present with severe manifestations of acute myocardial angina, ventricular tachycardia (VT), ventricular fibrillation or cardiac arrest. We report the case of a 25-year-old male patient, with cannabis smoking habit, admitted for palpitations which had revealed a VT in the electrocardiogram (ECG), and for whom a coronary angiography was realised showing a coronary artery spasm (CAS) with no sign of atherosclerotic occlusion.

Keywords: Ventricular tachycardia, arythmia, coronary artery spasm, vasospastic angina, Prinzmetal's angina, acute coronary syndrome, myocardial infarction, cardiac catheterization, coronary angiography, cannabis smoking.

INTRODUCTION

VA, also known as Prinzmetal's angina, or variant angina, is a clinical entity first described by Prinzmetal *et al*., in 1959 as 'angina at rest due to CAS with transient ischemic ECG changes that resolve with short–acting nitrates' [1]. CAS are generally characterized by events that occur at rest and are associated with transient ST-segment elevation on ECG. Although uncommon, these episodes can lead to syncope, arrhythmia, or sudden cardiac arrest [2]. VA can occur in patients with or without atherosclerotic vasculature. It can have focal or diffuse involvement of coronary arteries or epicardial microvasculature [3]. We present an unusual case in which CAS resulted in VT, and cardiovascular collapse.

CASE PRESENTATION

Herein, we present a case of a 25-year-old male patient with history of cannabis smoking, with no family history of coronary artery disease (CAD) or cardiomyopathies, who initially presented to the emergency department with palpitations. The patient was hemodynamically unstable with a low blood pressure of 82/67 mmHg at his arrival. He was, therefore, immediately admitted to the intensive care unit. The ECG monitoring showed a heart rate of 198 beats per minute. A 12 lead-ECG was realised showing a VT (Figure 1). The patient received an electrical cardioversion with an electric choc of 150 Joules delivered with a biphasic defibrillator, with analgesia and short sedation, and a sinus rhythm was restored immediately afterwards as well as a stable hemodynamic state. His labs showed an elevated troponin level of 311 ng/ml (normal <0.03). A complete blood count, basic metabolic panel, serum electrolytes, and lipid panel were all within normal limits. He underwent emergency cardiac catheterization, with concerns for acute coronary syndrome, which revealed a CAS of the right coronary artery (RCA) (Figure 2). The CAS was relieved after intracoronary administration of isosorbide dinitrate (Figure 3). His transthoracic echocardiogram showed a left ventricular ejection fraction of 50 - 55 % and mild hypokinesia in the inferior wall. Our patient was managed on calcium channel blockers (CCBs) and antiarythmics. Symptoms and arrhythmia improved after optimizing doses of medical treatment. He was then discharge with optimal doses of CCBs and changing in life style with vital necessity to quitting cannabis smoking.

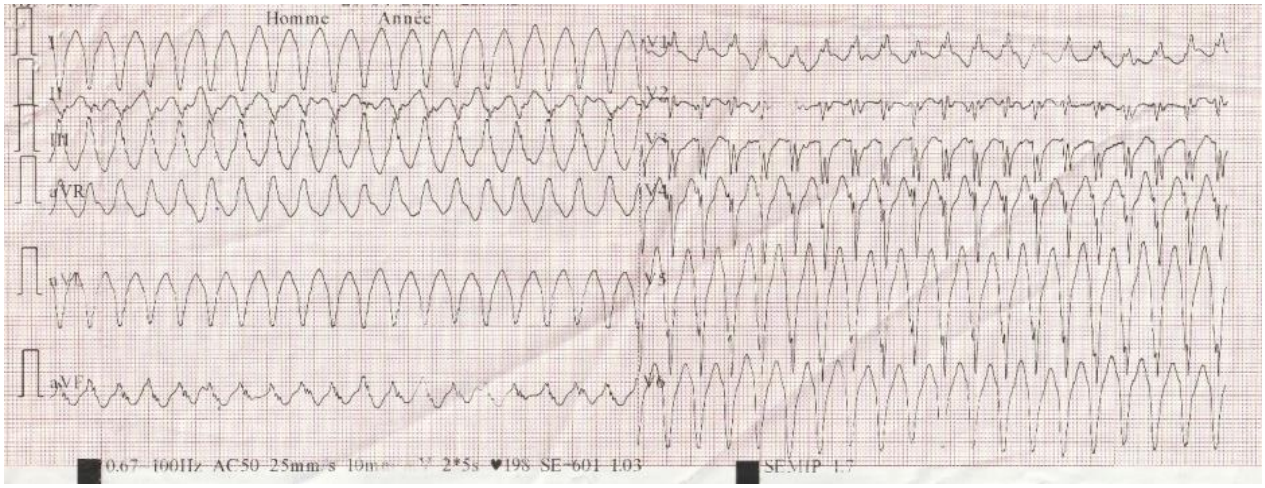

Figure 1: 12 lead-ECG showing a ventricular tachycardia at 198 beats per minute; VT score: 4

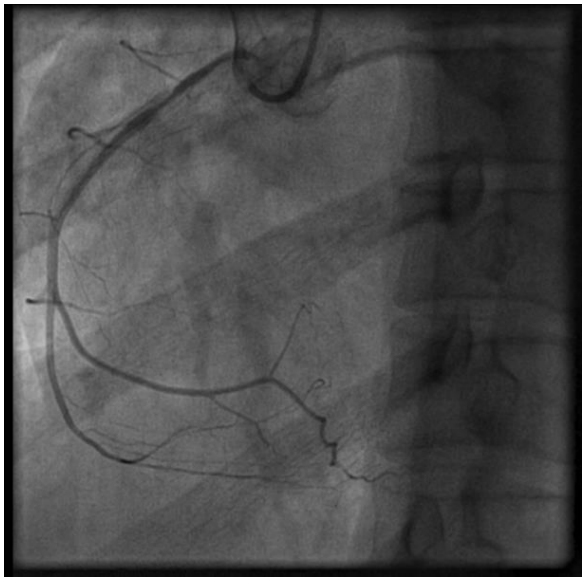

Figure 2: Coronary angiography revealing a vasospasm of the RCA

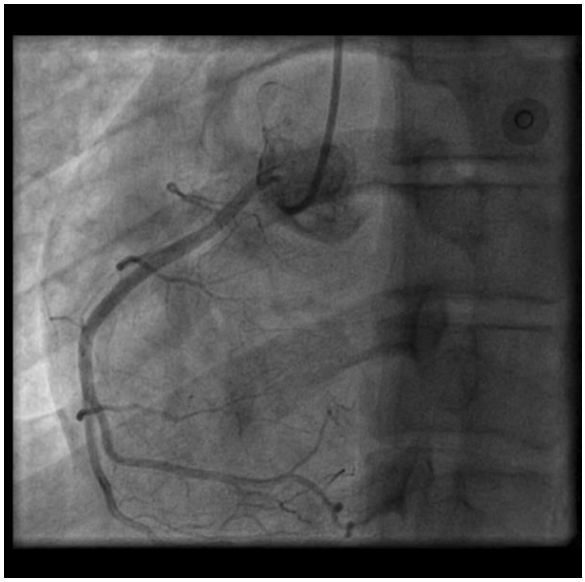

Figure 3: Coronary angiography showing RCA after coronary spasm was relieved thanks to intracoronary administration of isosorbide dinitrate

DISCUSSION

With the widespread use of coronary angiography in the early clinical management of myocardial infarction (MI), multicenter registries have been created and have reported that as many as 10% of MI patients have no evidence of obstructive CAD [4]. Focal or diffuse spasm of a major coronary artery could result in a high-grade obstruction. Transient myocardial ischemia causes angina in many patients; MI may develop in some [5].

Various forms of arrhythmia often appear during attacks of CAS associated with ST segment elevation. These include ventricular arrhythmias such as ventricular premature contractions or VT, bradyarrhythmias, atrioventricular block, and supraventricular arrhythmias. Ventricular fibrillation may also appear rarely [6, 7]. Severe MI caused by CAS can lead to life threatening ventricular arrhythmias. In addition, patients with CAS have increased ventricular irritability and prolonged QT dispersion, compared with controls, particularly during symptom-free periods [8, 9].

Classically, the clinical presentations of VA are usually thought to be short, but sometimes may be associated with serious arrhythmias [10] as in our case. It is, therefore, important to recognize its presence so that it can be managed with an aim to prevent such life-threatening arrhythmias.

It is difficult to attribute anginal presentations to VA without ruling out CAD. There are triggers that are reported to lead to VA such as substance use (cocaine, marijuana, alcohol, ephedrine–based products, amphetamines) and magnesium deficiency [11-13]. Our patient had history of cannabis (marijuana) smoking.

Takagi *et al*., reported that RCA spasm was significantly associated with ventricular arrhythmias compared with other vessels [14]. Our patient had a RCA spasm.

Pharmacological interventions have the potential to reduce or prevent spasm attacks, symptoms and life-treating arrhythmias. Aside from compliance with treatment, identification and avoidance of precipitants are an essential component of any management plan. In this regard it is essential to advise patients to quit smoking [15]. CCBs (both dihydropyridine and nondihydropyridine) and nitrates represent the mainstay of therapy because they reduce, prevent and resolve spasm attacks and thus angina and arrhythmias. These beneficial effects are mediated suppressing Ca 2+ inflow into the vascular smooth muscle and through the nitrate metabolization to nitric oxide (NO), both resulting in relaxation of vascular smooth muscle and vasodilation. Prospective double-blind studies with CCBs compared with nitrates have reported efficacy for both agents to a similar degree, in reducing spasm occurrence [16]. Importantly, the drugs can be used safely, without adverse reactions, and the dosage should be increased up to the maximal tolerated dose [15]. Notably, especially when symptoms are under control, medications should not be discontinued. As reported by Takagi *et al*., discontinuing or reducing medication is associated with symptom recurrence, higher incidence of lethal arrhythmic events and non-fatal MI [17, 18]. Drugs should preferably be taken nocturnally because of the reported circadian pattern and risk of events in the early morning [15]. Several studies have evaluated alternative therapies for CAS including nicorandil, statins, aspirin, magnesium, vitamins C and E, Iloprost, alpha receptor blockade, selective serotonin receptor inhibitors, and selective thromboxane A2 synthetase inhibition. Although considered effective in certain patients, in specific clinical settings, there is a lack of evidence and consensus on their use and further studies are needed to ascertain their effectiveness in daily clinical practice. All of these drugs can be considered if a patient is still symptomatic despite optimal therapy [15]. In our case our patient was optimally managed on maximal doses of CCBs.

CONCLUSION

VT can be an uncommon but severe manifestation during VA crises. In cases with normal coronary vasculature, it is important to recognize VA as a cause of VT in order to optimize medical management for prevention of fatal arrhythmias. CCBs and nitrates represent the mainstay of therapy. There dosage should be increased up to the maximal tolerated dose. Avoiding triggers that can lead to VA such as cannabis smoking is also primordial.

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