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Case Report

Marijuana, bigeminal premature ventricular contractions and sluggish coronary flow: Are they related?

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ABSTRACT

Premature ventricular contractions (PVCs) and slow coronary flow phenomenon (SCFP) are primarily separate entities. Each one of them has different characteristics and a diverse spectrum of presentation. However, and despite many suggested theories, a comprehensive understanding of the etiology of both of them is still a matter of debate. PVCs, which can be triggered by consuming cannabis (marijuana), and through decreasing the diastolic time (DT), can affect the slow blood flow found in SCFP even more and worsen the clinical picture in patients who have PVCs and SCFP. In this paper, we present a patient who uses marijuana and has PVCs and SCFP, try to address different aspects of PVCs and SCFP, pinpoint any suspected interaction between both of them and the role of marijuana in this context.

<**Learning objective:** (i) PVCs are extra abnormal heartbeats arising in one of the ventricles and disrupting the normal rhythm of the heart. (ii) PVCs are very common and occur in a broad spectrum of the population including those with and without underlying heart disease. (iii) SCFP is an angiographic finding characterized by delayed progression of the contrast injected inside large coronary arteries without any significant CAD. (iv) It has an incidence of 1% among patients who undergo coronary angiography, especially those presenting with acute coronary syndrome. (v) PVCs, which can be triggered by consuming cannabis (marijuana), and throughout decreasing the DT, can affect the slow blood flow found in SCFP even more and worsen the clinical picture in patients who have PVCs and SCFP.>

Introduction

Premature ventricular contractions (PVCs) are extra abnormal heartbeats arising in one of the ventricles and disrupting the normal rhythm of the heart. PVCs are very common and occur in a broad spectrum of the population including those with and without underlying heart disease. They are more common in men than women, in African Americans than whites, and in those with organic heart disease. The ability to detect recorded PVCs is mainly depending on the period of monitoring, especially in asymptomatic individuals.

Slow coronary flow phenomenon (SCFP) is an angiographic finding characterized by delayed progression of the contrast injected inside large coronary arteries without any significant coronary artery disease (CAD). It was first recognized in 1972. It is more common in male smokers [1], with an overall incidence of 1% among patients who undergo coronary angiography, especially those presenting with acute coronary syndrome [2].

Case report

A 37-year-old female with a history of mitral regurgitation (MR), hypertension, and syncope, presented to the emergency department with chest pain, dizziness, headache, and shortness of breath for the past two weeks. She also reported a history of palpitations for many years, which have increased over the past two weeks. Laboratory evaluation showed potassium of 3.6 mEq/L (N = 3.5–5 mEq/L), magnesium of 1.9 mg/dL (N = 1.5–2.3 mg/dL), and negative troponin times two. Urine was found to be positive for opiates and marijuana. The patient admitted to using marijuana for 10 years. Electrocardiography (ECG) at rest revealed normal sinus rhythm with PVCs in a bigeminal pattern (Fig. 1). On exercise stress test, the patient developed non-sustained run of ventricular tachycardia at almost 300 beats per minute. Chest X-ray and computed tomography of the chest came back normal. Nuclear medicine stress test was also normal with no evidence of reversible ischemia or scar. Transthoracic echocardiogram showed abnormal

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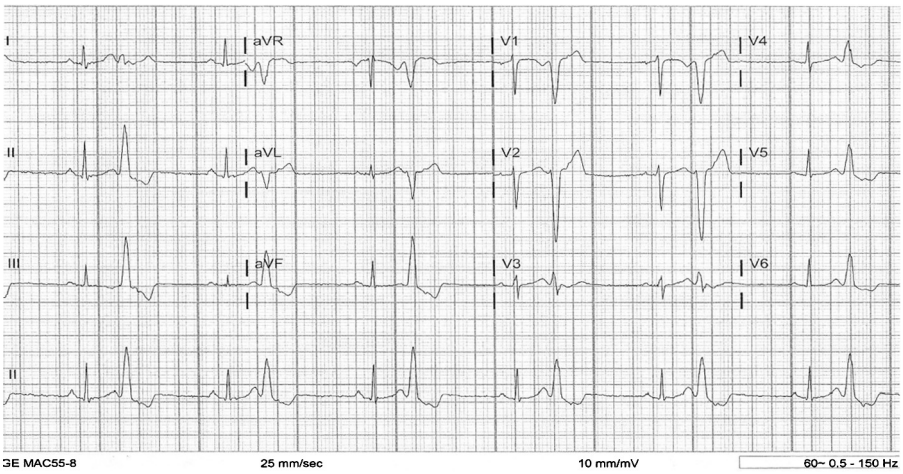

Fig. 1. Electrocardiogram of the patient showing normal sinus rhythm associated with a bigeminal pattern of premature ventricular contractions.

left ventricle diastolic filling pattern, PVCs, trace MR, and an ejection fraction of 50–55%. To rule out any significant CAD, cardiac catheterization was done and revealed slow coronary blood flow with a thrombolysis in myocardial infarction (TIMI) score of II in left anterior descending (LAD) artery (Video 1) and mildly slow coronary blood flow with a TIMI score of II–III in right coronary artery (RCA) without any overt CAD. Occasional couplets without ectopy and mild MR were also noted. The patient was put on the maximal dose of metoprolol and counseled to abstain from marijuana with a possibility to consider catheter ablation therapy in case she continues to be symptomatic with documented PVCs and ventricular tachycardia after that.

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jccase.2013.06.006.

Discussion

The prevelance of PVCs is increased in patients diagnosed with hypertension (HTN) [3] especially when it is associated with left ventricular hypertrophy, dilated cardiomyopathy and heart failure (HF), post acute myocardial infarction (MI), and congenital heart disease.

PVCs often cause no symptoms. In many patients, the presence of PVCs could result in the sensation of fluttering, pounding, skipped beats, palpitations, dizziness, and/or near syncope.

The etiology of PVCs is not well known. Many mechanisms may explain the origin of PVCs, including enhanced normal or abnormal automaticity inside the heart, triggered activity in Purkinje cells of the ventricular myocardium, or reentry. Anxiety, alcohol, caffeine, tobacco, exercise, illicit drugs, hypokalemia, HTN, ischemia, infarction, excessive calcium, drug toxicity (such as digoxin), or an underlying heart disease could result in PVCs through previously mentioned mechanisms.

ECG is the mainstay of diagnosis of PVCs. This includes standard ECG, exercise stress ECG, holter monitor, and event recorder depending primarily on the frequency of PVCs which helps to decide the best way to detect them.

Only in symptomatic patients do PVCs need to be diagnosed and treated. Beside eliminating previously mentioned possible triggers, beta blockers and calcium channel blockers are recommended as first-line therapy for symptomatic PVCs, especially with outflow tract morphology in a structurally normal heart. Antiarrhythmic medications, such as amiodarone can sometimes be tried but with caution because of its side effects.

Frequent PVCs may be associated with worsening of systolic heart failure in patients with a dilated cardiomyopathy. Small studies have suggested that in selected patients, radiofrequency ablation of ectopic ventricular foci is associated with an improvement in left ventricular function and clinical improvement in symptoms [4–7].

The 2006 American College of Cardiology/American Heart Association/European Society of Cardiology guidelines for the management of ventricular arrhythmias included suggestions regarding ablation therapy for PVCs [8]. They note that ablation therapy of PVCs may be useful if they are frequent, symptomatic, and monomorphic, if they are refractory to medical therapy, if the patient chooses to avoid long-term medical therapy, or if they consistently provoke ventricular arrhythmia storm of a similar morphology [9].

SCFP, as a separate entity, has a widely diverse presentation including chest discomfort, unstable angina, non ST elevation MI, ST elevation MI or nonsustained ventricular tachycardia. It usually presents with recurrent rest pain requiring urgent admission.

The etiology of SCFP is not completely understood. It is speculated that it is caused by acute but recurrent perturbations of microvascular function. Histopathological examination (light and electron microscope) of left and right ventricular endomyocardial biopsies taken from some patients showed fibromuscular hyperplasia, myofibrilar hypertrophy, endothelial degeneration with swollen endothelial cells encroaching on the lumen, luminal size reduction, mitochondrial abnormalities, lipofuscin deposition, and glycogen content reduction, which can cause the elevation in resting coronary artery resistances, especially toward microvasculature beds, found in SCFP. Normal and pathological zones often coexisted in the same specimen (patchy appearance). Thus, in some patients with slow coronary flow and patent coronary arteries, functional obstruction of microvessels seems to be implicated, as it is relieved by dipyridamole infusion. This shows also that small-vessel CAD can cause classic angina pectoris.

The diagnosis can be suspected when the coronary angiogram shows large patent arteries with slow flow of the angiographic contrast medium and it can be confirmed by endomyocardial biopsy.

Another study suggested that elevation in plasma homocysteine, even if it is mild, may play a role in the pathogenesis of SCFP by severely disturbing vascular endothelial function and subsequently impairing coronary blood flow [10], and showed that patients with SCFP have statistically significant raised level of plasma homocysteine compared to control subjects with normal coronary flow.