Ventricular Tachycardia Due to Marijuana Use in a Heart Transplant Patient

To the Editor:

Cannabis is the most consumed drug in Spain, and contrary to other drugs, its consumption is perceived as innocuous for the body.

Ever since describing a non Q wave acute myocardial infarction in 1979 after cannabis consumption, atrial fibrillation episodes, ventricular tachycardia (VT), ventricular fibrillation, angina pectoris, and acute myocardial infarction have been reported and are directly related to consumption of this substance.

Among the cannabinoids which constitute marijuana, tetrahydrocannabinol is the main one. Although its harmful effects are demonstrated, its strong association with tobacco does not allow effects to be clearly distinguished between one or another substance, or even its potentiation.

We present the case of a 29-year-old male, with a heart transplant for 5 years, due to a developed idiopathic dilated myocardiopathy.

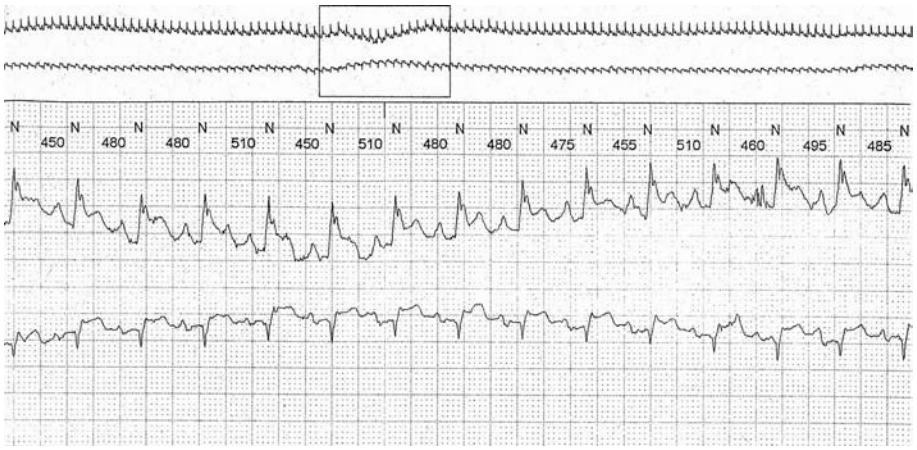

Figure 1. Episode of sinus tachycardia, subepicardial lesion.

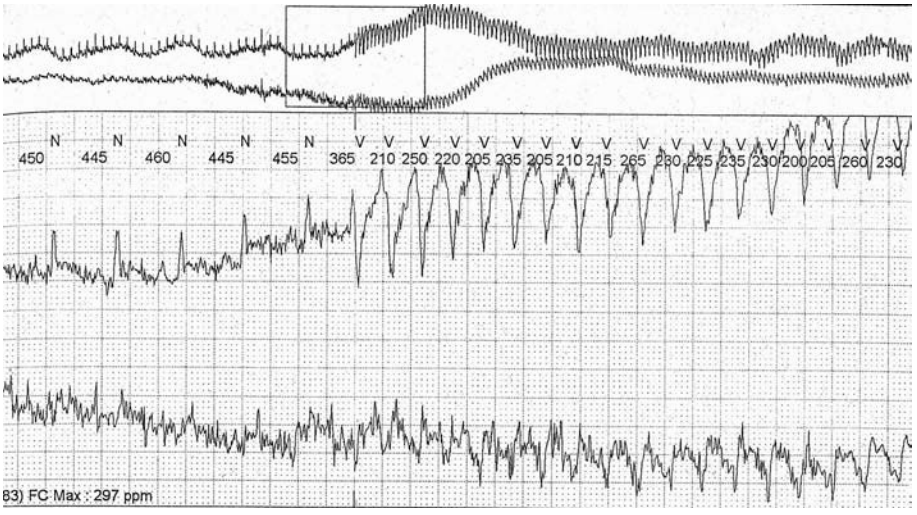

Figure 2. Ventricular tachycardia.

Early problems after the cardiac transplant were a bacterial infection and a haemorrhagic ictus without secondary effects. He later had arterial hypertension and dyslipidemia as a consequence of immunosuppressive medication.

The patient was admitted for an endomyocardial biopsy to be taken after changing immuno-suppressives (cyclosporin for everolimus due to cutaneous warts). The physical examination was normal; the echocardiography showed good ventricular function and moderate pericardial effusion with no haemodynamic compromise. The cytomegalovirus viral load was negative, and the endomyocardial biopsy did not show signs of rejection (ISHLT level 1A). A 24 h Holter was requested according to our protocol, which revealed a sinus tachycardia with an image of a subepicardial lesion and a subsequent period of VT for 45 s (Figures 1 and 2). Likewise, we observed another 3 episodes of transient subepicardial lesion with no associated arrhythmias. All of this occurred in a period of 1 h. The patient remained asymptomatic during the recording.

Following these observations, we carried out a coronariography with IVUS which showed: significant neointimal proliferation (0.8 mm) in the LAD, 2 lesions (40% and 100%) at medial and distal points from the same artery, and a lesion of 40% in the CD-2. The determination of narcotics in urine resulted positive for tetrahydrocannabinol, after which the patient confirmed his consumption of marijuana on the day of Holter and within the time frame of events.

In this instance, the VT could be as much a consequence of myocardial ischaemia due to graft vascular disease (GVD) as from an ischaemic effect from cannabis, independent or added to the GVD.

There are various mechanisms by which cannabis produces myocardial ischaemia. The increase of

sympathetic activity and parasympathetic block which it produces can increase cardiac frequency up to 100% and cardiac output up to 30%. Other mechanisms would be its direct vasospastic effect, interference in the peripheral vascular reflexes, and increase of carboxyhaemoglobin. The final result is an increase of oxygen consumption and a decrease of its contribution, mainly in the hour following cannabis consumption.[1]

In this case, the sinus tachycardia previous to the vasospastic angina episode and the close cause-effect relationship declared by the patient himself indicate cannabis as the cause of the coronary spasm, myocardial ischaemia, and the later appearance of VT. With respect to localization of the ischaemia, the absence of atrioventricular block in the Prinzmetal episode and with the limitations of Holter, it would be oriented toward a left coronary spasm.

In regard to coronary spasm, among the causes which can produce a VT are an increase of automatism, focal discharges, and unidirectional obstruction which generates re-entry, but nor can it be ruled out that it is produced after vasospasm with reperfusion.[2]

Although the short durations of subepicardial ischaemia and VT could have occurred with no symptoms in any patient, in our case, cardiac denervation is added to explain the absence of symptoms.

Bachs et al[3] carried out a post mortem analysis on 6 patients with sudden death after consuming cannabis, and they did not find any other cause or drug except cannabis. Consequently, they indicate that the cause of death for the 6 cases is of cardiac, ischaemic, and/or arrhythmic origin.

We found it interesting in this case that, even though the heart and coronary vessels were found denervated, they responded to the stimulus of marijuana, first with sinus tachycardia and later with a vasospasm.

Furthermore, we should remember the importance of determining narcotics in urine of young transplant patients, even with the slightest suspicion, given the high rate of cannabis consumption among the young population.

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