

Ventricular Tachycardia in Chronic Myocardial Contusion Interest of Multimodal Imaging

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ABSTRACT

Ventricular tachycardia (VT) is a rhythmic emergency due to the poor hemodynamic tolerance, the possibility of transformation into ventricular fibrillation, and the occurrence of sudden death. It is a late complication after thoracic trauma due to ventricular remodeling and scar tissue fibrosis, the main arrhythmogenic substrate. The case we report is that of an 80-year-old patient admitted to our unit for lipothymic discomfort that has been evolving for several months. In this antecedent, we find a violent thoracic traumatism 23 years ago by accident of the public way. On admission, it has a stable hemodynamics; the surface electrocardiogram inscribes a sinus rhythm with diffuse negative T waves and reassuring biology. A few hours after his hospitalization, the discomfort will reappear with unsupported TV. Coronary angiography eliminates an ischemic cause with non-significant atheroma of the bisector. Echocardiography demonstrates a particular aspect of hypertrophy of the left ventricular apex with normal contractile function. Cardiac magnetic resonance imaging shows myocardial fibrosis in this area of hypertrophy and the cardiac computed tomography with three-dimensional reconstruction allows to visualize partial apical inferior disinsertion with an interventricular septum with a thin wall on the right ventricular slope calcified in places with an inlet opening closing in systole. The mechanism of TV in our patient is related to myocardial fibrosis and ventricular remodeling secondary to myocardial contusion 23 years ago. In this context, an implantable automatic defibrillator has been set up with half-yearly monitoring.

Key words: Chronic myocardial contusion, multimodal imaging, ventricular tachycardia

INTRODUCTION

Ventricular tachycardia (VT) is essentially a rhythmic urgency due to the poor hemodynamic tolerance, the possibility of transformation into ventricular fibrillation (VF), and the occurrence of sudden death.

It is a late complication after thoracic trauma due to ventricular remodeling and scar fibrosis, the main arrhythmogenic substrate.^[1] The multimodal cardiovascular imaging is great importance in the diagnosis and monitoring of remodeling phenomena and tissue fibrosis.

CASE REPORT

It was an 80-year-old patient who was received in our unit for faintness episodes that had been evolving for several months.

She had in her antecedents, a violent thoracic traumatism 23 years ago by accident of the public way. Two years ago, she had benefited from electrophysiological exploration for unsupported VT episodes with negative ventricular pacing and the appearance of ventricular extrasystoles. At admission, she had stable hemodynamics. The surface electrocardiogram recorded sinus rhythm with diffuse negative T waves and a reassuring biology with a serum electrolyte and a cardiac enzyme.

A few hours after his hospitalization, the discomfort had reappeared with unsupported VT [Figure 1].

Echocardiography found a particular aspect of hypertrophy of the apical wall of the left ventricle, without abnormalities of segmental and global kinetics and the left ventricular (LV) systolic function was preserved [Figure 2].

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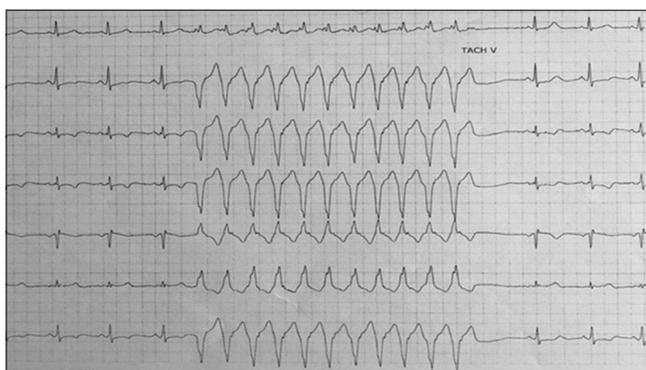


Figure 1: Electrocardiogram showing ventricular tachycardia

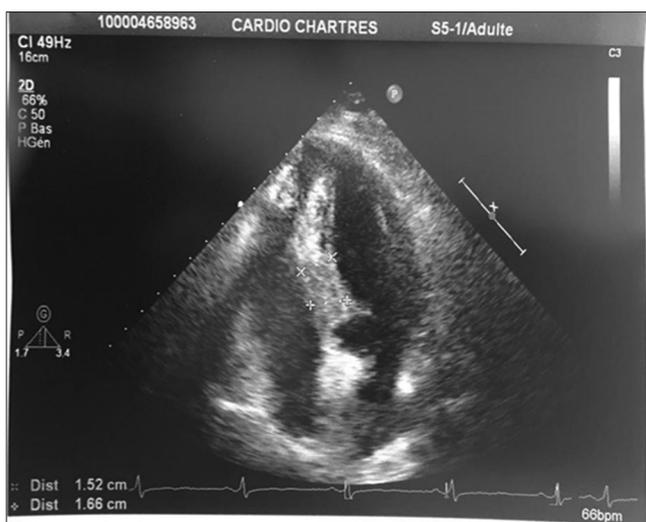


Figure 2: Transthoracic echocardiography showing apical and septal hypertrophy

Coronary angiography was normal except for non-significant stenosis of the bisecting artery.

Cardiac magnetic resonance imaging (MRI) revealed the LV hypertrophy with infiltration of the apical, septal, and inferior wall with late epicardial enhancement within this hypertrophy and an aneurysmal aspect of the LV apex [Figure 3a and b].

The computed tomography (CT) scan demonstrated partial inferior and apical disinsertion with an interventricular septum with a thin septum on the right ventricular slope calcified in places [Figure 4a and b] with an inlet opening closing in systole without expansion of the septum, Figure 4c and d.

DISCUSSION

The ventricular rhythm disorders after cardiac trauma can, under certain conditions, develop immediately or late. The occurrence of ventricular arrhythmia in this setting is a rare,

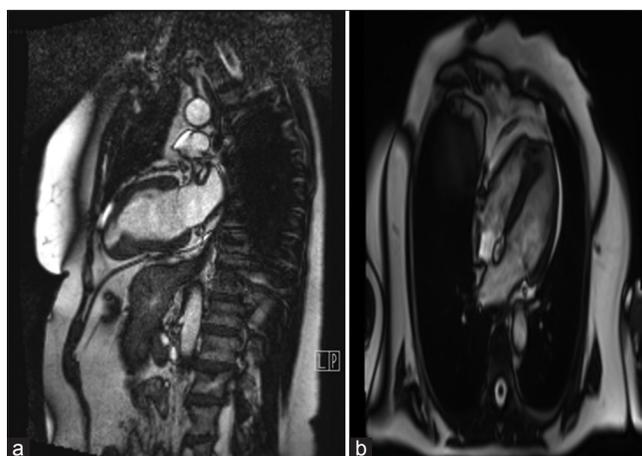


Figure 3: Cardiac magnetic resonance imaging: (a) Post-gadolinium early, (b) post-gadolinium with apical aneurysm, and epicardial enhancement

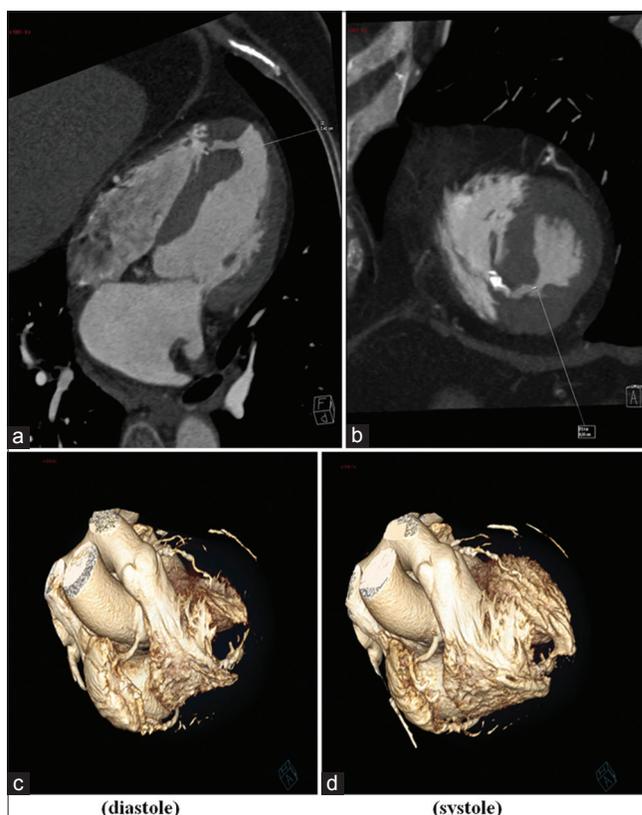


Figure 4: Computed tomography scan with three-dimensional reconstruction: Evidence of lateral apical and septal disinsertion

but potentially life-threatening complication including a trigger, automatic activity, or reentry. The acute manifestation is VF in the context of chest contusion and even in the absence of cardiac structural injury.^[2,3]

In a second time, the occurrence of ventricular arrhythmias in the long run especially TV is mainly due to ventricular

aneurysm or related to scar tissue fibrosis. Ventricular remodeling associated with tissue fibrosis is the main arrhythmogenic substrate.^[1,4]

The coronary angiography has allowed us to eliminate an ischemic cause on this VT. Thus, the mechanism of VT in our patient is related to myocardial fibrosis and ventricular remodeling secondary to myocardial contusion 23 years ago.

The visualization of this myocardial fibrosis has been possible thanks to cardiac MRI which is the non-invasive reference technique in the detection of myocardial fibrosis by the study of the late enhancement of gadolinium.^[5,6] The myocardial fibrosis is associated in our patient with localized hypertrophy at the apex with partial disunion of the apical septum and areas of calcification, visualized on the chest CT scan. The most frequent site of this disunity in the context of myocardial contusion is the right ventricle and septum than the left ventricle, and preferentially the apical portion of the myocardium. The MRI and thoracic CT were used to diagnose lesions and topography, to understand the mechanism of symptomatic VT in this octogenarian patient.

Our patient benefited as part of the primary prevention of sudden death from the implantation of an implantable automatic defibrillator with a semi-annual follow-up.

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