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CASE REPORT

A Rare Case of a Primary Cardiac Tumor Presenting as Fatal Ventricular Tachycardia

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Abstract

Primary cardiac tumors are extremely uncommon. Here, we report the case of a patient with a primary left ventricular interstitial tumor presenting with hemodynamically unstable ventricular tachycardia. In response to hemodynamically unstable ventricular tachycardia, an implantable cardioverter-defibrillator was inserted. One month after defibrillator implantation, the patient developed episodes of high ventricular tachycardia that could not be effectively terminated by catheter radiofrequency ablation, thus further confirming that the ventricular tachycardia was induced by the left ventricular interstitial tumor. The patient is doing well on medical therapy to date.

Keywords: Arrhythmia; Primary cardiac tumor; Ventricular tachycardia

Introduction

Primary cardiac tumors are rare, with an autopsy frequency of 0.001%–0.28% [1]. Primary cardiac tumors are mostly benign and account for 90% of all primary cardiac tumors. Patients with primary cardiac tumors can be asymptomatic, or can develop symptoms such as arrhythmias and obstruction, depending primarily on the size and location of the mass [2]. Here, we report a primary interstitial tumor at the left ventricle (LV) in a symptomatic patient with fatal ventricular arrhythmia.

Case Report

A 50-year-old man who experienced syncope was referred to our hospital in March 2021 with a chief complaint of palpitation for 3 months. He had a 10-year history of hypertension without any prior structural heart disease or dysrhythmia history. The patient's medical history indicated that, when he had a palpitation attack 10 days prior, he was aware of the palpitations being extremely rapid and the rhythm being regular, with symptoms of chest tightness, dizziness, nausea, vomiting, blurred vision, and confusion. The patient was sent to the local hospital immediately. At that time, his blood pressure could not be measured, and he was diagnosed with cardiogenic shock. At the local hospital, he was immediately treated with electrical defibrillation. Soon, he regained consciousness and was referred to our hospital for further treatment. Physical examination showed no significant abnormalities, and his chest X-ray and abdominal ultrasonography findings were unremarkable. Subsequently, the patient underwent coronary angiography; the results indicated 40% narrowing in the 1st diagonal branch opening of the left anterior descending artery. A 24-hour ambulatory ECG (Holter) showed 2411

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premature ventricular contractions. Transthoracic echocardiography indicated a slightly thickened left ventricular wall (Figure 1A and B), but no evidence of left ventricular outflow, inflow tract obstruction, or wall motion abnormality of the LV. The following quantitative parameters were determined: left ventricular ejection fraction, 65%; left ventricular fractional shortening, 32%; left ventricular end-diastolic diameter, 43 mm; and left ventricular end-systolic diameter, 29 mm. Cardiac magnetic resonance (CMR) showed a cardiac interstitial tumor located in the near epicardium of the anterior wall at the base of the LV, measuring 17 mm in diastole. The mass was hypointense on the T1 and T2 weighted sequences (Figure 2A and B). Postgadolinium sequences demonstrated nodular enhancement of the lesion (Figure 2C and D). The clinical characteristics of the patient are shown in Table 1. According to the imaging findings, we excluded the possibility of cardiac metastasis and suspected that ventricular arrhythmia was induced by the primary left ventricular interstitial tumor. The location of the tumor, the characteristics of T1 and T2 weighted hypointensity, and the delayed gadolinium enhancement on CMR strongly suggested a benign cardiac fibroma [3]. However, because of the risks and cost of the procedure, the patient refused to undergo biopsy, mass resection, or heart transplantation. The patient underwent implantation of a dual-chamber implantable cardioverter-defibrillator (ICD) because of hemodynamically unstable ventricular arrhythmia. In addition, a decision was made to treat the patient with bisoprolol fumarate 10 mg/day and antihypertension therapy. On the 10th day, he was discharged in stable condition with the ICD.

One month later, the patient was readmitted to our hospital with symptoms of worsening palpitation and was diagnosed with ventricular tachycardia. The 12-lead ECG (Figure 3) showed multiple wide malformed QRS with positive homotopic changes in leads V1–V6 and two premature narrower QRS, which were ventricular fusion waves indicated by arrows in the long V1 lead, thus demonstrating a sustained monomorphic ventricular tachycardia originating from the left ventricle. The 24 h Holter monitoring results revealed an average heart rate of 76 bpm, 21,824 premature ventricular contractions, and an episode of sustained monomorphic VT lasting approximately 3 hours at a heart rate of 123 bpm (Figure 4). According to the 24 h Holter monitoring, the patient's premature ventricular contractions and ventricular tachycardia stemmed from the same site, and the reason for the absence of ventricular tachycardia termination by the ICD might have been related to the slow frequency of ventricular tachycardia. After exclusion of surgical contraindications, the patient received a cardiac electrophysiological study and a catheter radiofrequency ablation. Ablation with 3D mapping in the cardiac summit and LV did not eliminate ventricular premature contraction and VT, thus further confirming that the patient's ventricular arrhythmia was associated with the cardiac interstitial tumor located in the near epicardium of the anterior wall at the base of the left ventricle. Later, the patient was administered antiarrhythmic therapy with bisoprolol 10 mg/day and amiodarone (first-week dose 60 mg/day, second-week dose adjusted to 40 mg/day, third-week dose adjusted to 20 mg/day, and discontinuation after 3 weeks). Subsequently, he was

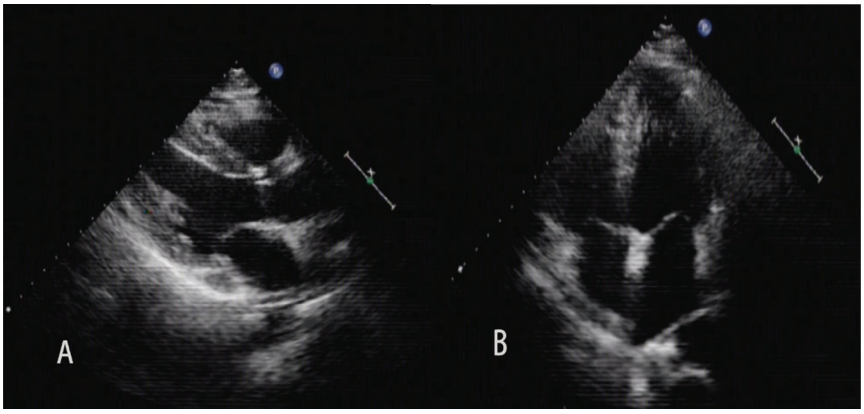

Figure 1 Transthoracic Echocardiography Showing the Slightly Thickened Left Ventricular Wall. (A) Long axis view of the left heart. (B) Apical four-chamber view.

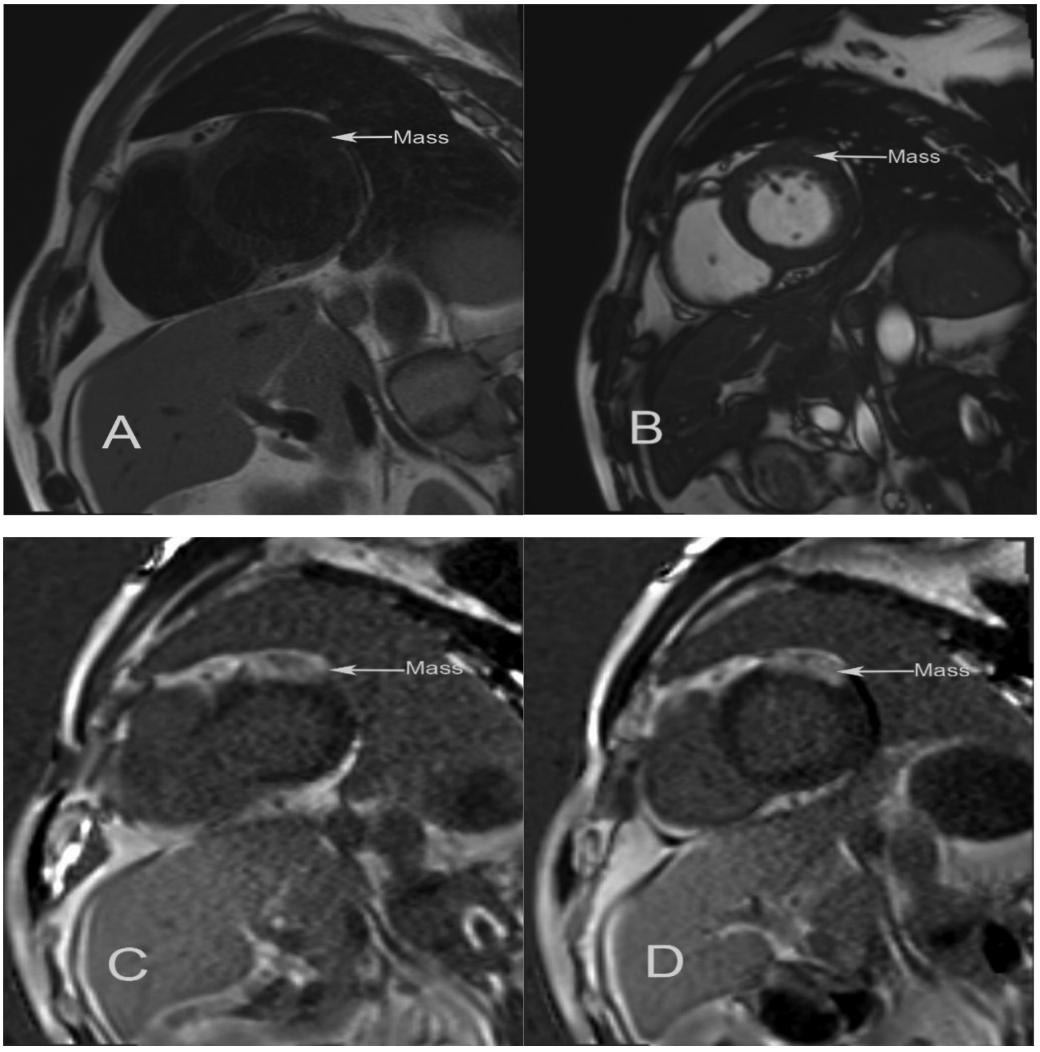

Figure 2 (A) Two-chamber T1-weighted image showing a hypointense mass (arrow). (B) Two-chamber T2-weighted image showing low signal in the mass (arrow). (C) and (D) Two-chamber delayed enhanced images showing nodular enhancement of the mass (arrows).

Table 1 Clinical Characteristics of the Study Participant.

Characteristic	Details
Sex	Male
Age (year)	50
Tumor location	Left ventricle
Maximum diameter (mm)	17
Invasion of surrounding tissues	None
Pericardial effusion (if any)	None
Morphology	Irregular shape
Boundary	Clear
Density	Heterogeneous
Mild to moderate enhancement	Yes

discharged with close follow-up. He has remained symptom-free over 12 months of follow-up.

Discussion

Primary cardiac tumors are more rare than secondary cardiac tumors and are mostly benign; papillary fibroelastoma is the most common type [4]. Primary cardiac tumors can occur anywhere in the heart, such as in the endocardium, myocardium, and epicardium. Diagnosis is usually established by echocardiography, computed tomography, magnetic

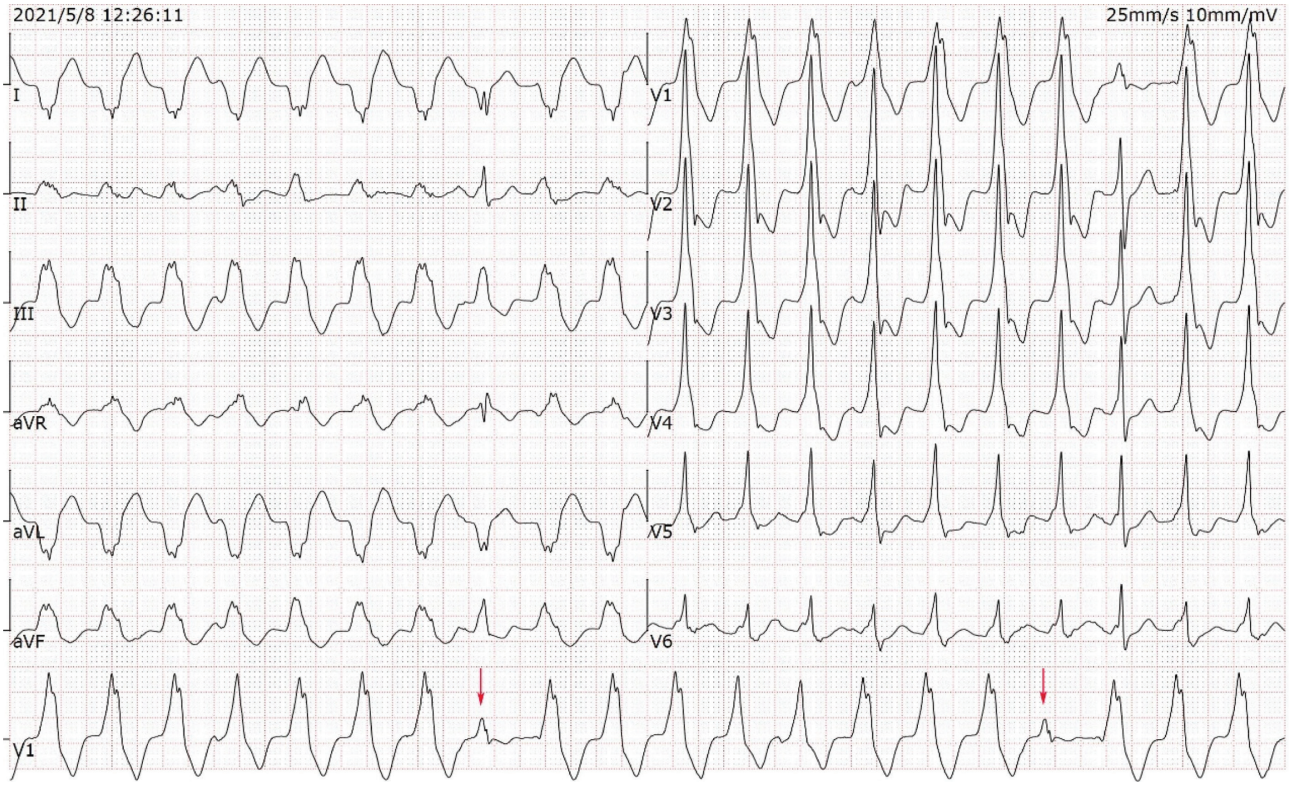

Figure 3 Twelve-Lead ECG Showing a Wide-QRS-Complex Tachycardia and Two Ventricular Fusion Waves Indicated by Arrows in the Long V1 Lead.

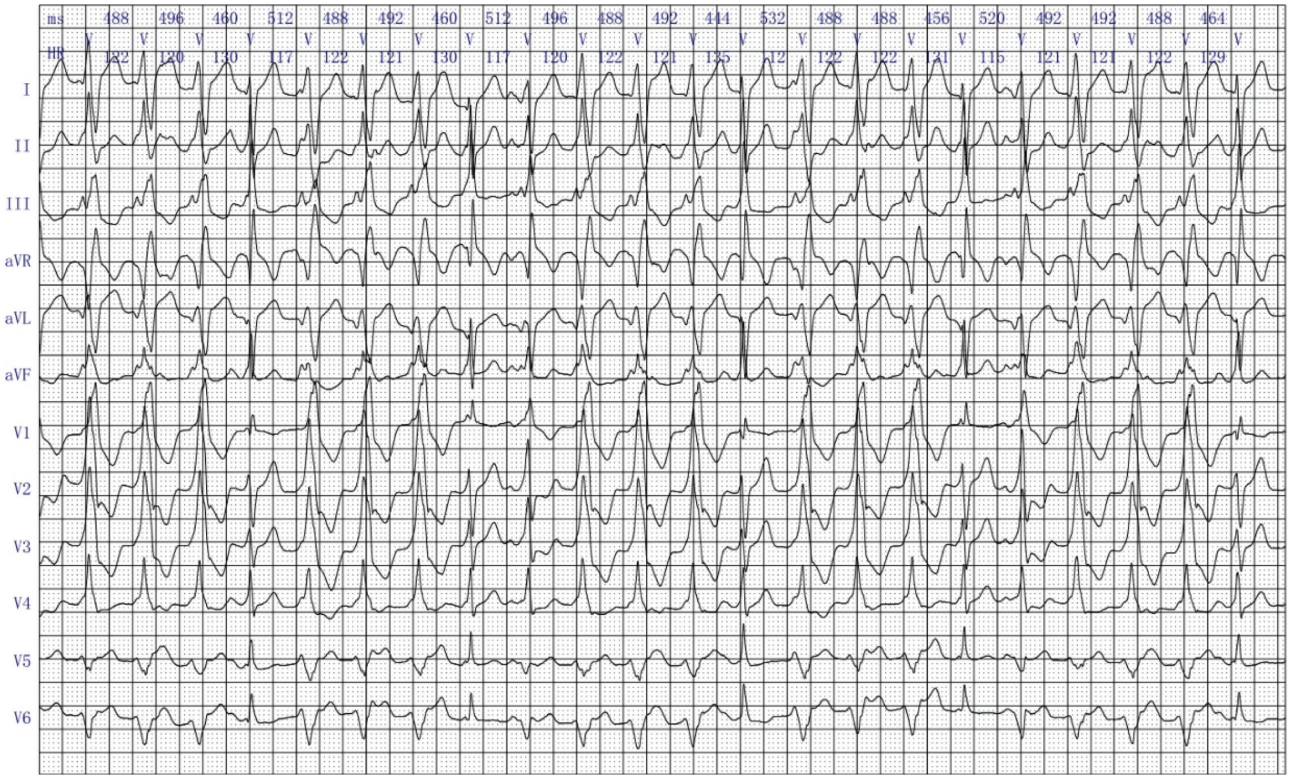

Figure 4 Twenty-Four Hour Holter Monitoring Revealing an Episode of Sustained Monomorphic VT.

resonance imaging, and tissue biopsy. Owing to the typically asymptomatic nature of primary cardiac tumors, these tumors are often diagnosed incidentally on imaging performed for unrelated issues or postmortem [5]. The primary cardiac tumor rarely manifests as hemodynamically unstable ventricular tachycardia, as in our case. The results of transthoracic echocardiography and CMR were suggestive of an interstitial tumor of the left ventricle, but the patient did not undergo a tissue biopsy because of the associated risk. Coronary angiography and echocardiography excluded common causes of ventricular tachycardia such as ischemic or non-ischemic cardiomyopathy and hypertrophic cardiomyopathy, thus confirming the underlying structural cause of the ventricular tachycardia. Cardiac fibromas, most originating in the left ventricular free wall [6], typically appear isointense relative to the myocardium, or hypointense on T1-weighted images and hypointense on T2-weighted images in MR imaging [3, 7, 8]. T2 hypointensity strongly suggests the presence of fibromas and is uncommon in any other type of cardiac tumor [3]. In terms of presentation, the association of cardiac tumors with ventricular arrhythmias, particularly ventricular tachycardia, has been described in previous literature. These ventricular arrhythmias may arise from macroreentry circuits around or within tumors, potentially involving different refractory periods within the tumor mass, and compression of the His bundle or bundle branches [5, 9].

Treatment of primary cardiac tumors includes antiarrhythmic medications, ICD placement, surgical excision, and even heart transplantation. Surgical resection is therapeutically recommended for primary cardiac tumors causing arrhythmias, obstruction, or valvular dysfunction [2]. A retrospective single-center study has found that 24% of pediatric patients with a primary cardiac tumor develop clinically significant arrhythmias, particularly VT; moreover, surgical resection, including total and subtotal resection, is an important and effective treatment strategy for tumor-associated ventricular tachycardia [10]. In the present case, the patient did not choose to undergo biopsy, resection of the cardiac mass, or heart transplantation. Instead, a dual-chamber ICD was implanted to relieve symptoms and avoid the risk of sudden death. However, the patient was readmitted 1 month later with an episode of ventricular tachycardia, and the ICD did not play a role in terminating episodes of ventricular arrhythmias in our patient, possibly because of the slow frequency of ventricular tachycardia. Subsequently, to further terminate the episodes of high ventricular tachycardia, the patient underwent catheter radiofrequency ablation, but the ablation in the cardiac summit and LV did not work. Therefore, we believed that the ventricular arrhythmia in our patient might have resulted from the primary interstitial tumor of the left ventricle. The patient was started on amiodarone and bisoprolol. Fortunately, on conservative management with amiodarone and bisoprolol, ventricular tachycardia had not recurred during 12 months of follow-up. In conclusion, we report a case of a primary cardiac tumor presenting as hemodynamically unstable ventricular tachycardia. The possibility of a cardiac tumor should be considered in patients with ventricular arrhythmias, although cardiac tumors as an etiology of ventricular arrhythmias are extremely rare.

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Ethics

The study was performed in accordance with the Declaration of Helsinki, and written informed consent for publication of the patient's details was obtained from the patient's next of kin.

Conflict of Interests

The authors report no financial relationships or conflicts of interest regarding the contents of the manuscript.

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