Unusual Case of an Intramyocardial Tumor Presenting With a Ventricular Tachycardia

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A 64-year-old man with history of hyperlipidemia and type 2 diabetes mellitus presented with sudden onset of atypical chest pain and dyspnea at rest. He denied palpitations or dizziness. Examination revealed blood pressure of 132/81 mm Hg and a heart rate of 153 bpm. The ECG showed a wide-QRS-complex tachycardia compatible with a sustained monomorphic ventricular tachycardia (Figure 1). An amiodarone drip was started, and the tachycardia abruptly terminated during the infusion. In the ECG performed shortly after (Figure 2), normal sinus rhythm with T-wave inversion (transient) in the lateral leads (I, aVL) was evident, probably related to cardiac memory phenomenon. There was no evidence of further arrhythmias while the patient was maintained on oral amiodarone treatment. A chest x-ray showed moderate cardiomegaly and an abnormal left mediastinal contour owing to a bulge in the surface of the left ventricle (Figure 3A and 3B). A transthoracic echocardiogram was performed, demonstrating a normal-size left ventricle, with mild septal hypertrophy, normal systolic function of both ventricles, and no pericardial effusion. A coronary angiography excluded any occlusive disease but revealed a cardiac mass with multiple “feeding” collaterals from the left anterior

Figure 1. Twelve-lead ECG showing a wide-QRS-complex tachycardia compatible with a sustained monomorphic ventricular tachycardia (atrioventricular dissociation is evident in lead II).
descending coronary artery (Figure 4). A cardiac magnetic resonance imaging confirmed the presence of an intramyocardial tumor (≈34×35 mm) in the basal and midcavity anterior segments of the left ventricle. In terms of tissular characterization, it demonstrated an isointense signal on T1-weighted images and a hyperintense signal on T2-weighted images with fat suppression indicating high water content (Figure 5A) and a ring-enhancing pattern on late gadolinium images (Figure 5B and 5C), and it was unremarked on steady-state free precession images (Figure 5D). Although this pattern is common for a number of cardiac masses and therefore nondiagnostic for a specific tumor, its size, myocardial infiltration, and heterogeneous ring-enhancing pattern on delayed enhancement (indicating tumor vascularization or necrosis) suggest a malignant nature of the lesion.

His detailed history revealed an antecedent of a local renal cell carcinoma (RCC) treated by left radical nephrectomy 6 years earlier. The retrieved materials were sent to histological examination confirming a T1N0 tubulopapillary RCC (hypernefroma), limited to the renal parenchyma. The surgical borders and the perirenal fat, as well, were tumor free.

The cardiac mass was considered an unresectable tumor, and it was not feasible to undergo a biopsy because of the huge surgical risk owing to the great vascularization of the mass.

Given the patient’s past history, the most probable explanation for the cardiac tumor was a metastasis of a RCC, without being able to eliminate the possibility of a primary malignancy (such as a sarcoma) based just on the magnetic resonance imaging images.

The overall frequency of cardiac tumors is quite low, with an estimated cumulative prevalence of 0.002% to 0.3% at autopsy and 0.15% in echocardiographic series. Metastatic involvement of the heart is ≈40 times more prevalent than primary cardiac tumors and is generally associated with a poor prognosis. Tumors that are most likely to involve the heart and pericardium include lung and breast cancers, melanoma, leukemia, and lymphoma.

![Figure 2. Twelve-lead ECG performed after ventricular tachycardia termination.](image)

![Figure 3. Chest x-ray of the patient. A, Posteranterior view. B, Lateral view.](image)
Primary cardiac malignancies are very rare, the majority being sarcomatous in origin. Angiosarcoma is the most common primary malignancy of adulthood, whereas rhabdomyosarcoma is more prevalent in children. Isolated metastasis of RCC to the left ventricle of the heart, which it could be in our patient’s case, is a rare entity that has rarely been reported in the medical literature (<5 cases). There have been rare reports of solitary metastasis of RCC to the right ventricle, demonstrating the most common way of spread of this kind of tumors: the transvenous spread through the great veins (up to 10% of patients with RCC have tumor thrombus involving the renal vein and inferior cava and 1% have tumor thrombus extending into the right atrium allowing the tumor to reach the surface of the right side of the heart). Cardiac metastases from RCC in the absence of vena cava extension are extremely rare, although metastasis to the left ventricle, as to any other organ, is possibly due to hematogenous spread. The risk of metastatic disease after radical nephrectomy for local cell carcinoma depends on the stage of the primary tumor. Metastases are very unusual in the pathological stage T1 N0, as it was the case of our patient; moreover, if this happens, RCC has a clear predilection to metastasize to the lungs, bone, lymph nodes, and brain.

In terms of presentation, cardiac tumors have a known association with ventricular dysrhythmias, especially ventricular tachycardia. These arrhythmias can be secondary to tumor involving autonomic fibers or coronary arteries. Patients with arrhythmias due to cardiac tumors should initially receive medical treatment such as antiarrhythmic agents. Surgical resection, if possible, is indicated when there is no response to medical treatment or when the patient’s condition worsens. In cases of antiarrhythmic-resistant ventricular tachycardia, electric cardioversion should be performed. Survival is poor in an inoperable tumor as in the case of our patient. Even so, after almost 1 year of follow-up, the patient is still alive, and the mass remains with the same shape and size in the last magnetic resonance imaging that was performed.

Disclosures
None.

Figure 4. Coronary angiography revealing a cardiac mass with multiple feeding collaterals from the left anterior descending coronary artery.

Figure 5. A, Two-chamber T2-weighted image shows high-signal in mass (arrow). B and C, Two-chamber and short-axis delayed enhanced images show ring enhancement of the mass (arrows). D, Two-chamber steady-state free precession image shows nodule thickening of the anterior wall at midcavity level (arrow).
References


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