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Clinical Case Report

Unresectable cardiac pseudoneoplasms causing ventricular tachycardia

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ABSTRACT

Cardiac pseudoneoplasms are rare and benign. According to World Health Organization, they are classified as tumor-like lesions. We report two patients with recurrent ventricular tachycardia (VT) in whom magnetic resonance imaging revealed a pathological mass occupying a large part of the left ventricle. The localization of both tumors precluded the possibility of resection; thus, only surgical biopsy was performed. After deducting the prospect of malignancy of the tumors, we treated both patients with amiodarone and implantation of a cardioverter–defibrillator [implanted cardioverter/defibrillator (ICD)]. VT is one of many probable symptoms indicating a tumor within the heart; therefore, treatment with an ICD should only be considered after a more thorough diagnosis.

1. Introduction

Ventricular tachycardia (VT) has different origins including a structural heart disease (e.g., ischemic heart disease, cardiomyopathies and sarcoidosis), antiarrhythmic drugs or even a structurally normal heart. In our two patients who were admitted to the Department of Cardiology, an intramyocardial mass was responsible for this type of arrhythmia.

2. Case report (1)

A 47-year-old male patient with a recent onset of angina, dyspnea and palpitations was referred to the cardiology department. A maternal family history of myocardial infarction and hypertension was reported. The patient had not previously taken any medications. During electrocardiographic monitoring, sustained monomorphic VT 214 bpm (Fig. 1a) was present and an urgent direct current cardioversion was performed. Transthoracic echocardiography revealed massive obturation of the intraventricular septum (IVS) and the apex, increased end-diastolic diameter of left ventricle (LV) (60 mm), mild systolic dysfunction (ejection fraction was measured to be 45%) and some fluid in the pericardium. No atherosclerotic coronary disease was found on coronary angiography.

Cardiac magnetic resonance was performed on a 1.5-T scanner (Siemens Avanto, Erlangen, Germany) with the use of 0.1 mmol/kg gadolinium DTPA (Fig. 1b). The study revealed pericardial fluid surrounding LV and the right ventricle (31 mm) and a pathological mass (37×67 mm) near the apical and middle segments of IVS and the apex. It was hypointense on T_1-weighted images and showed late enhancement 10 min after the administration of gadolinium. Moreover, it was hypointense on T_1-weighted images with fat suppression (STIR, *s*hort *t*au *i*nversion *r*ecovery). Further imaging studies did not reveal any metastases.

Our initial treatment consisted of the evacuating the blood-tinged fluid (about 400 ml). A total of 60 ml of the obtained fluid was sent to the pathology department. Results concluded that the fluid contained a few atypical cells, which corresponds to reactive mesothelial cells and clusters of small mesothelial cells. Thus, surgical biopsy was performed by means of a median sternotomy and a sample of the tumor was analyzed histopathologically (Fig. 1c and d). The specimen analyzed at the pathology department consisted of four fragments from the core and from the periphery of the tumor. On a cross-section, it was observed to be a solid, yellow tissue. Microscopically, each of the collected fragments was composed of fat tissue with some areas of fat necrosis. The adipose tissue was mature and had a few fibrous septae. There were no atypical adipocytes or lipoblasts, no brown fat was found and the tumor did not present any features of malignant lesion. One of the fragments contained a bundle of cardiomyocytes. The immunohistochemistry results [S100, Des, Ki67, CD68 and CD34 (negative) and SMA (positive); Fig. 1c] together with the histopathological image suggest that the lesion could represent lipomatous hypertrophy.

Surgical treatment was not a valid option because there was no possibility to resect the tumor without damage to the left ventricular systolic function, since the tumor occupied a significant portion of muscle in the LV. Administration of amiodarone after admission to the hospital successfully protected the patient against the recurrence of

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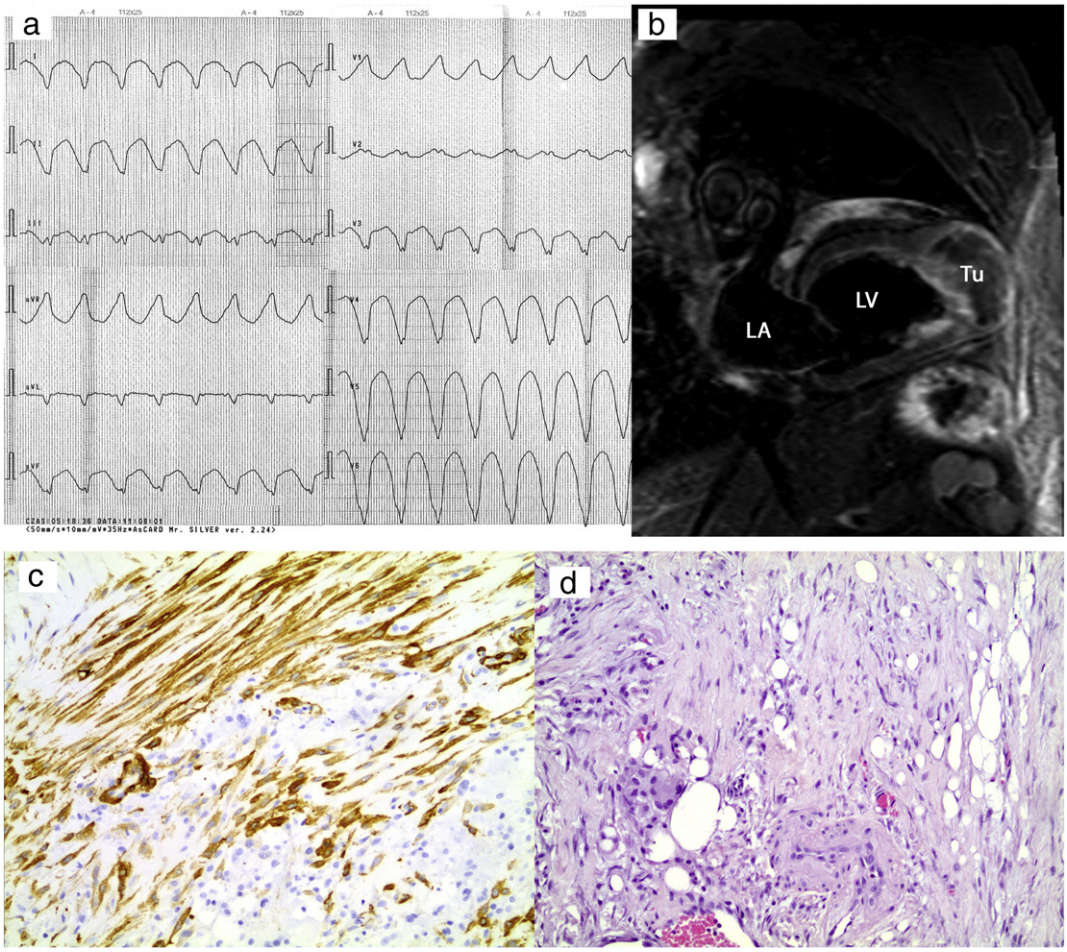

Fig. 1. (a) Electrocardiogram (50 mm/s) — monomorphic sustained VT 214 bpm caused by cardiac tumor. (b) Tumor involving apex of the LV and IVS. Fat-suppressed T_2-weighted image (STIR), two-chamber view shows heterogeneous hypointense mass (LA, left atrium; Tu, tumor). (c) Immunohistochemical stain shows expression of smooth muscle actin protein by bundles of cardiomyocytes, SMA (original magnification 200×). (d) Fat tissue with the areas of fat necrosis, giant cells and myofibroblasts, hematoxylin and eosin (original magnification 200×).

VT. Moreover, a cardioverter–defibrillator was implanted as a secondary prevention of arrhythmia.

The patient is closely followed up. During 1 year, there was no evidence of arrhythmia recurrence in the device's memory. The patient is in a clinically good condition — no symptoms of heart failure (New York Heart Association I) and the oxygen consumption in the cardiopulmonary exercise test and the level of N-terminal pro-brain natriuretic peptide is normal. In the control computed tomography, the lesion size has not changed.

3. Case report (2)

A 60-year-old female patient was admitted to the cardiology department with a recently worsening state of exercise tolerance and an increasing exercise dyspnea. Her previous cardiac history includes hypertension and hyperlipidemia. She stopped smoking 20 months prior to admission. Paternal history of myocardial infarction was reported. Medication taken was as follows: aspirin, bisoprolol, ramipril and atorvastatin. The electrocardiogram monitoring revealed asymptomatic sustained monomorphic VT 162 bpm (Fig. 2a). During transthoracic echocardiography, a pathological thickening of the inferior-inferolateral walls of the LV and the inferior part of IVS was found. The mass resulted in a significant mitral annular distortion and consequently caused the functional enlargement of the left atrium (56 mm).

Cardiac magnetic resonance (Fig. 2b) showed the presence of a pathological mass (68×33 mm) in the inferior-inferolateral walls of the LV and the inferior part of the IVS. In the area of the apex, it also involved the pericardium. It was hypointense on T_2-weighted images. T_1-weighted inversion recovery gradient echo showed late enhancement. Pleural fluid was also observed. The ejection fraction was 25% with global hypokinesia of the LV walls. Electrophysiological study confirmed monomorphic VT 157–147 bpm. A whole-body positron emission tomography revealed no outbreaks suspected of metastasis.

Pharmacological treatment with amiodarone and typical heart failure therapy was provided, which resulted in no incidence of VT afterwards. The surgical biopsy was performed by videothoracoscopy. Five pieces of tissue from the core of the tumor and a few pieces from the periphery of the tumor were sent for histopathological examination. Microscopically, the specimen was composed mainly of hyalinized connective tissue with a few vessels of a small diameter. One fragment of the examined tissue contained mature adipose tissue, without any observed atypia, intermingled with hyalinized connective tissue. There were areas of dense calcification and foci of chronic inflammation in three of the fragments; however, none of them contained "myxomatous" tissue or amorphous, fibrinous material (Fig. 2c and d). The immunohistochemical stains for S100, SMA and CD34 were negative (Fig. 2e and f) and that of Ki67 was less than 1%.

Considering the specimen, the diagnosis of a pseudoneoplasm was rendered. As seen in the first case, resection of the tumor was impossible due to its localization; therefore, the patient had only a cardioverter–defibrillator implanted.

After a few months of no follow-up (due to lack of patient's compliance), she was admitted again to another hospital with multiple organ failure due to long-lasting VT of a rate below the