Malignant Mimicry

An Unusual Cause of VT and AV Conduction Disease

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Abstract

We present a case of a patient with unexplained asymmetric hypertrophy who presented with ventricular tachycardia and atrioventricular block and was diagnosed with lung disease metastatic to the myocardium.

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Key Words

Ventricular tachycardia • Heart block • Imaging

Case Presentation

A 56-year-old female with no known cardiac history and a history of T3N0 NSCLC status post right upper lobectomy and chemoradiation therapy with cisplatin and etoposide in 2010 and a negative screening positron emission tomography (PET) scan 2 months prior presented in 2013 with the acute onset of chest discomfort. Baseline 12-lead ECG was normal (Figure 1 A). Upon presentation to an outside hospital, she had a wide complex tachycardia most consistent with left bundle branch-type VT with a right superior axis and negative concordance in the precordial leads at a cycle length of 250ms (Figure 1 B). Due to hemodynamic instability, she was cardioverted with no recurrence of VT thereafter. Transthoracic echocardiogram (TTE) revealed preserved biventricular systolic function with asymmetric left ventricular septal hypertro-

phy (Figure 2: PSLA TTE view), and a presumptive diagnosis of hypertrophic cardiomyopathy (HCM) was made. Of note, there was no known family history of SCD, VT, or HCM, and had not had previous ECGs or TTEs. She underwent single-chamber secondary prevention ICD implantation. Two months later, she presented with worsening dyspnea on exertion and was referred for stress testing, during which time she developed AV block with peak exertion (Figure 3: ETT ECG). She was therefore referred to our institution for both endomyocardial biopsy and ICD upgrade to a dual-chamber device, which she underwent without complication. Histopathologic evaluation of the right ventricular endomyocardial biopsy from the intraventricular septum revealed metastatic lung adenocarcinoma (Figure 4 A and B). Subsequent surveillance imaging revealed new lung nodules, and chemotherapy was initiated. To our knowledge, this is the first reported case of metastatic lung adenocarcinoma to the heart presenting with VT and/or AV block that masqueraded as a variant of HCM.

Discussion

Metastatic disease in the setting of breast and lung cancer, lymphoma, leukemia, melanoma, and various sarcomas affect the heart and/or pericardium in approximately 10% of cases [1]. Interestingly,

Figure 1. (A) Baseline 12-lead electrocardiogram obtained at a routine appointment approximately 6 months prior to the presentation demonstrates normal sinus rhythm with no conduction disease and no electrocardiographic evidence of structural or ischemic heart disease. (B) A 12-lead electrocardiogram upon presentation to the emergency department demonstrates ventricular tachycardia of a predominantly left bundle branch right superior axis with negative concordance in the precordial leads at a cycle length of approximately 240 bpm. The VT morphology suggested an origin in the apical septum.

this incidence is higher now due to improvements in chemotherapy and radiotherapy, likely because of enhanced disease monitoring and longer life expectancy [2]. Adenocarcinoma of the lung is the most common malignancy resulting in metastasis to cardiac structures and is likely due to both the prevalence of the disease and the lungs' proximity to the heart and pericardium [3].

The clinical profile of myocardial involvement by metastatic disease remains poorly defined and, like

our case, may mimic more common pathology upon clinical presentation. Badri et al. recently described a similar patient with a history of lung cancer who presented with MMVT and was noted to have biopsy-proven squamous cell carcinoma of an unknown primary metastatic to the RV [4]. In our case, we felt that a presentation of sustained VT and asymmetric LVH in a middle-aged patient with no personal or family history of cardiac disease and no previous cardiac symptoms whatsoever warranted further evalu-

Figure 2. Transthoracic echocardiogram parasternal long axis demonstrating asymmetric hypertrophy of the interventricular septum causing it to be almost as twice as thick as the inferolateral wall (2.1 vs. 1.1 cm). Normal left ventricular cavity size and regional/global systolic function. No evidence of systolic anterior motion of the mitral valve nor left ventricular outflow tract obstruction at rest.

Figure 3. A 12-lead electrocardiogram after peak exercise testing demonstrates sinus rhythm at approximately 95 bpm with long-cycle 6:5 Mobitz I (Wenckebach) AV block.

ation prior to presumptively diagnosing the patient with HCM. Because the patient's ICD implantation preempted cardiac MRI testing, we felt that the next step would be endomyocardial biopsy, which ultimately revealed the diagnosis.

In patients with metastatic disease with cardiac involvement, palliative chemotherapy should be targeted at the underlying malignancy [3]. After her diagnosis was made, our patient was referred for chemotherapy (pemetrexed and carboplatin) and subsequent radiation. She developed a malignant pericardial effusion that necessitated surgical treatment with a pericardial window. The patient remains in remission 8 months after her initial diagnosis.

This case illustrates that unexplained asymmetric hypertrophy presenting with VT or AV block in a patient with no history of HCM should prompt consideration of infiltrative cardiomyopathies, including the rare possibility of metastatic disease in patients with a history of malignancy.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

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Figure 4. Figure 4. (A) Right ventricular septal endomyocardial biopsy demonstrates a population of poorly-differentiated malignant epithelial cells growing in nests and cords, infiltrating the native myocytes (Hematoxylin and Eosin, 40x original magnification). (B) Immunohistochemical staining demonstrates strong nuclear positivity for the lung adenocarcinoma marker TTF-1 in these cells (40x original magnification). These malignant cells were also positive for a cytokeratin cocktail (AE1/AE3 and Cam5.2), but were negative for mesothelial markers WT-1 and Calretinin (not shown).

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