Eosinophilic Myocarditis—An Unusual Cause of Left Ventricular Hypertrophy

Vanderbilt Heart

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Abstract: Eosinophilic myocarditis is a rare condition in which inflammation of the heart results in an infiltrative cardiomyopathy that is often difficult to diagnose in the acute setting. It sometimes presents as left ventricular hypertrophy. The authors present a case of a 79-year-old woman with a history of Non-Hodgkin's lymphoma who presented with acute heart failure with marked left ventricular hypertrophy. Echocardiography demonstrated abnormalities consistent with an infiltrative cardiomyopathy, and endomyocardial biopsy showed findings consistent with eosinophilic myocarditis. The patient was managed with diuresis and glucocorticoid therapy, and within 4 weeks of her admission, her clinical status had improved and her echocardiogram normalized. The prompt diagnosis and treatment of this patient's myocarditis likely resulted in her favorable outcome. This illustrates the need for a broad consideration of all the potential causes of hypertrophy and the necessary diagnostic strategies and therapeutic options.

Key Indexing Terms: Left ventricular hypertrophy; Eosinophilia; Myocarditis; Glucocorticoids. **[Am J Med Sci 2015;349(4):358–362.]**

Left ventricular hypertrophy (LVH) denotes an increase in ventricular wall mass and is commonly described as concentric or eccentric thickening of the myocardium. Concentric hypertrophy typically occurs in the setting of chronic pressure overload. The left ventricular wall thickness is increased; however, the diameter of the ventricle is commonly unchanged or even reduced. Histologically, newly laid sarcomeres are added in parallel to the existing functional units.[1] Eccentric hypertrophy, however, occurs in the setting of chronic volume overload of the ventricle. The ventricular wall thickness is increased, as is the diameter of the ventricle. Histologically, sarcomeres are added in series.[1]

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LVH is typically noted on echocardiogram by the 2-dimensional (2-D) measurement of the septal and posterior walls of the left ventricle. There are multiple etiologies of LVH, and the causative factor is not always readily apparent. Concentric hypertrophy is commonly associated with long-standing hypertension or aortic stenosis, whereas eccentric hypertrophy is often the result of aortic or mitral insufficiency and ischemic or idiopathic cardiomyopathies. Infiltrative cardiomyopathies, such as amyloidosis, result from the abnormal deposition of substances within the ventricular walls and may cause both concentric and eccentric thickening patterns on echocardiography. Although rare, hypertrophic cardiomyopathy, mucopolysaccharidoses, cardiac oxalosis, Friedreich's ataxia, Danon's disease, Fabry's disease and inflammatory cardiomyopathies including eosinophilic myocarditis (EM) often result in increased left ventricular wall mass and wall thickening.[2] Alternatively, cardiac sarcoidosis, hemochromatosis and Wegener's disease are infiltrative processes that typically result in a dilated left ventricular cavity with eccentric ventricular hypertrophy.[2] Myocardial biopsy is often required to establish a diagnosis in these patients.

We present a case of a 79-year-old woman with a history of Non-Hodgkin's lymphoma who presented with significant congestive heart failure and marked LVH. The case outlined illustrates the need for a broad consideration of all the potential causes of LVH and the necessary diagnostic strategies and therapeutic options.

CASE PRESENTATION

A 79-year-old woman with a history of non-Hodgkin's lymphoma and hypertension presented to our institution with progressive shortness of breath, orthopnea and chest pain. She had recently completed 3 cycles of bendamustine and rituximab approximately 2 months before her presentation. Her initial physical examination revealed a blood pressure of 91/59 mm Hg, a pulse of 106 bpm, a respiratory rate of 24 per minute and an oxygen saturation of 96% on room air. The patient had jugular venous distention of 12 cm of water, a S3 gallop and a holosystolic murmur auscultated at the left lower sternal border. Inspiratory crackles were present at the lung bases. The

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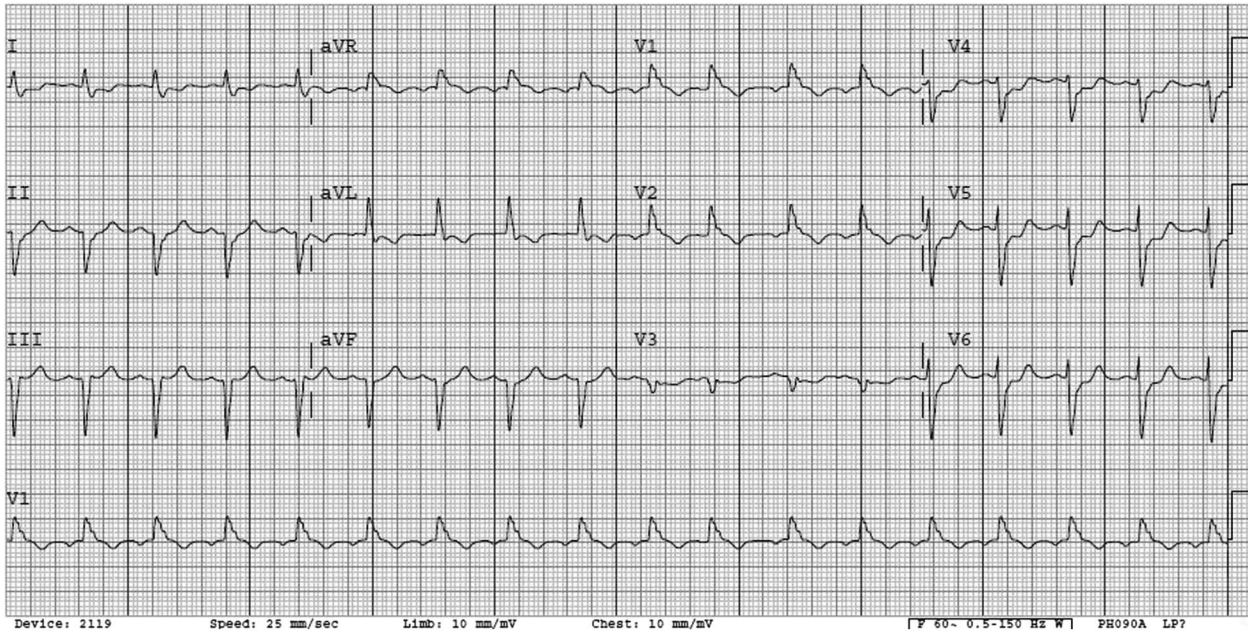

FIGURE 1. Electrocardiogram at presentation shows sinus tachycardia, right bundle branch block, left anterior fascicular block and nonspecific repolarization abnormalities.

initial laboratory evaluation was remarkable for white blood cells count: 16,000 cells per microliter (reference: 3.9–10.7), absolute eosinophil count: 480 cells per microliter (reference: 30–510), creatine phosphokinase (CK): 123 U/L (reference: 30–300), CK-MB: 16.72 ng/mL (reference: <6), troponin I: 2.94 ng/mL (reference: <0.05) and B-type natriuretic peptide: 9,046 pg/mL (reference: <100), all of which were significantly abnormal. Additionally, her creatinine was 1.53 mg/dL (reference: 0.7–1.5), and her liver transaminases were mildly elevated. Her calcium was 10.4 mg/dL, the upper limit of normal. Blood and urine bacterial cultures were negative throughout the hospital course.

The patient's baseline electrocardiogram (ECG) was notable for sinus tachycardia (100 beats per minute), right bundle branch block, left anterior fascicular block, nonspecific repolarization abnormalities and low voltage across the precordial leads (Figure 1). There were no prior ECG tracings available for comparison. Chest radiography revealed bibasilar opacities consistent with prior scarring without other acute abnormalities. A transthoracic echocardiogram showed severe global left ventricular systolic dysfunction with a left ventricular ejection fraction estimated at 20% to 30%. There was moderate concentric LVH with speckling of the myocardium consistent with an infiltrative process (Figure 2). Stage 1 diastolic dysfunction was present, and a small pericardial effusion and dilated inferior vena cava were evident, suggesting markedly elevated right-sided filling pressures (Figures 3 and 4). The valves were structurally and functionally normal.

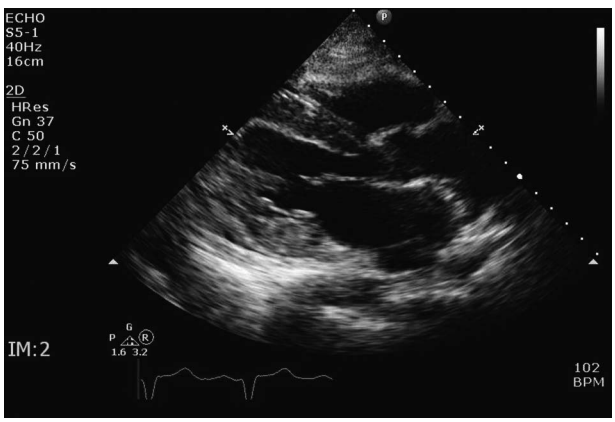

FIGURE 2. A surface echocardiogram at presentation shows moderate concentric LVH and speckling of the myocardium concerning for an infiltrative process. LVH, left ventricular hypertrophy.

An infiltrative process was suspected given the clinical picture, and a right ventricular endomyocardial biopsy was performed. Histologic findings included focal myocyte necrosis, diffuse inflammation with lymphocytes, eosinophils and neutrophils and interstitial edema (Figure 5) consistent with a diagnosis of EM. Repeat measurements of peripheral eosinophil count remained within the normal range.

Her initial treatment included diuresis with intravenous loop diuretics and a transient milrinone infusion due to evidence of cardiogenic shock. Ultimately, she was initiated on an angiotensin-converting enzyme inhibitor and a beta-blocker when hemodynamic stability was achieved. After tissue diagnosis, the patient was started on intravenous methylprednisolone 250 mg intravenously every 12 hours for a total of 48 hours. Her regimen was then decreased to prednisone 30 mg orally twice daily for 2 days followed by a gradual taper over the following 3 weeks.

Twenty days after hospital discharge, she was symptom free and without signs of volume overload. A transthoracic echocardiogram at that time demonstrated normal left ventricular ejection fraction of 65%, normal wall thickness, no pericardial effusion and normal inferior vena cava diameter (Figure 6). An additional echocardiogram 5 months later confirmed these findings. She is now asymptomatic after over 1-year follow-up.

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