A 71-year-old white woman was admitted for the evaluation of progressive dyspnea. Her medical history was remarkable for 3 episodes of pneumonia and asthma with peripheral eosinophilia.

As a part of her diagnostic evaluation, a contrast-enhanced computed tomographic scan of the chest was performed, which excluded pulmonary embolism and interstitial lung disease. Trans-thoracic echocardiogram revealed right-sided chamber enlargement, with a mildly hypokinetic right ventricle and an estimated right ventricular systolic peak pressure of 58 mmHg. Moreover, an area of increased echogenicity was noted at the apex of the left ventricle (LV) (Movie I in the Data Supplement). Cardiac magnetic resonance imaging (MRI) (Siemens Magnetom Avanto 1.5T VB15; Siemens Healthcare, Erlangen, Germany) was obtained to characterize the mass further. Cine MR images (Movie II in the Data Supplement) confirmed the presence of an apical LV mass with obliteration of the midventricular and apical segments and associated regional dysfunction. After the administration of 0.1 mmol/kg gadopentetate dimeglumim (Magnevist, Bayer, Germany), early postcontrast images (Figure 1A) demonstrated the avascular nature of the mass consistent with thrombus. Delayed images (Figure 1B and 1C) revealed diffuse sub-endocardial hyperenhancement, most apparent in the midventricular and apical segments. In addition, there were patchy midmyocardial areas of delayed hyperenhancement (Figure 1B), suggesting an element of myocarditis and there was hyperenhancement in the mitral leaflets (Figure 1C) indicating valvular involvement. The findings were suggestive of early necrotic stage endomyocardial fibrosis with diffuse involvement of the LV and mild to moderate regional systolic dysfunction. After this study, left and right heart catheterization with endomyocardial biopsy was performed to confirm the diagnosis of eosinophilic endomyocarditis. Pertinent laboratory findings included 24% eosinophils (reference <5%) and an absolute eosinophil count of 3300 per μL (reference <450 per μL). Bone marrow biopsy demonstrated trilineage hematopoiesis but was hypercellular with a marked increase in the number of eosinophils; neoplasm of a myeloproliferative, primary eosinophil, lymphoid, or mast cell lineage could be ruled out. Three days after admission, the patient acutely decompensated and arrested with pulseless electric activity, ultimately passing away despite maximal resuscitative efforts. Autopsy was performed, which demonstrated an extensive 4-cm thick, layered, dark red to tan mural thrombus, marked fibrous thickening and eosinophilic infiltration of the LV subendocardium, also involving the mitral valve (Figure 2A). Microscopically, the LV endocardium demonstrated marked fibrous thickening with associated areas of inflammation consisting predominantly of eosinophils within the fibrous tissue and immediate subendocardium. The thrombus within the LV was composed of fibrin, plasma, and red blood cells with marked organization and areas of neovascularization. Sections of the LV myocardium showed scattered inflammation consisting almost exclusively of eosinophils with focal areas of associated myocyte necrosis and myocardial fibrosis (Figure 2B).

Essential criteria for the diagnosis of hypereosinophilic syndrome are peripheral blood eosinophilia of >1500 eosinophils/mL for ≥6-month duration, characteristic end-organ damage, and no evidence of eosinophil clonality. MRI is an excellent modality for diagnosing ventricular thrombus and the degree of obliteration of the ventricular cavity and can thus play an important role in the prompt work-up of patients with suspected eosinophilic endocarditis. Delayed-enhancement sequences seem to be helpful in identifying and categorizing eosinophilic endomyocarditis in the early acute necrotic or late fibrotic stage. Contrast echocardiography using microbubbles of air or high molecular weight gas is an alternative method for evaluating myocardial injury and myocardial perfusion. Contrast echocardiography can also help in delineation of endocardial borders on ultrasound and identifying filling defects within the chambers. Furthermore, transesophageal echocardiography would be an appropriate tool to evaluate the cardiac chambers for thrombi better, especially in patients with poor acoustic windows. However, transesophageal echocardiography requires moderate sedation, which could further compromise the already labile cardiovascular system in severe cases.
of Loeffler syndrome. Computed tomographic angiography is an accurate technique for the imaging of cardiac structures and coronary arteries and can clearly visualize ventricular thrombi. However, the limited contrast resolution of the computed tomography does not allow the accurate evaluation of myocardial fibrosis or inflammation.

In this case, we describe the ability of cardiac MRI to diagnose healthy and pathological myocardium along with valvular involvement noninvasively, in excellent correlation with histopathologic findings, for the purpose of characterizing complex, uncommon disease, such as eosinophilic endocarditis, accurately.

Disclosures
Dr Schoepf is a consultant for and receives research support from Bayer, Bracco, GE Healthcare, Medrad, and Siemens Healthcare. The other authors report no conflicts.

References

Key Words: endocarditis ▪ heart failure ▪ magnetic resonance imaging ▪ myocarditis ▪ pathology

Figure 1. Early (A) and late (B and C) gadolinium-enhanced MRI are shown. Early postcontrast MRI (A, look-locker sequence; field of view, 350×262 mm²; slice thickness, 8 mm; acquisition matrix, 192×72; echo time/repetition time, 1.3/2.4 ms; flip angle, 50°; inversion time, 615 ms) shows extensive left ventricular (LV) thrombus as a nonenhancing mass (*) filling the entire apical and midventricular LV cavity. High-resolution, segmented, late gadolinium enhancement MRI (B, magnitude 2-chamber; C, phase-sensitive 4-chamber inversion recovery images, ultrafast gradient echo sequence, field of view, 275×340 mm²; slice thickness, 8 mm; acquisition matrix, 156×256; echo time/repetition time, 3.3/8.5 ms; flip angle, 25°; inversion time, 300 ms) also shows the thrombus (*) and reveals subendocardial enhancement along the LV consistent with endomyocardial fibrosis. Focal myocardial hyperenhancement in the posterior wall indicates myocarditis (B, arrow). Subtle delayed hyperenhancement is also seen in the mitral valve annulus, in the anterior and posterior leaflets (C, arrows), and in the chordae tendineae (B, open arrows).

Figure 2. A. Acute thrombus in the left ventricle involving the chordae tendineae of the mitral valve (white arrow). The thrombus is overlying a fibrous, thickened endocardium (black arrows). This photograph was taken after removal of the organized thrombus. B. Scattered eosinophilic infiltration (arrow) and fibrosis within the myocardium of the left ventricle (Hematoxylin & Eosin stain; original magnification, ×400).
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SUPPLEMENTAL MATERIAL

Supplemental Video Legends

Supplement Movie I

Transthoracic echocardiogram reveals right-sided chamber enlargement with mild right ventricular hypokinesis and a left ventricular apical mass.

Supplement Movie II

Two chamber (left panel) and three chamber (right panel) long axis cine magnetic resonance imaging (2D balanced Steady-State Free Precession sequence, field of view 350×350 mm², slice thickness 6 mm, acquisition matrix 192×192, echo time/repetition time 1.3/2.3 ms, flip angle 80°) show a mass in the apex extending towards the midportion of the LV.