A 73-year-old Japanese woman was admitted to the intensive care unit of our hospital with the diagnosis of eosinophilic pneumonia and congestive heart failure. Laboratory examination revealed a white blood cell count of 8100/mm³ with 49% eosinophils, 1568 IU/mL IgE, 467 IU/L lactate dehydrogenase with 40% lactate dehydrogenase-1, a brain natriuretic peptide level of 1223 pg/mL, and 23.5 U/mL myeloperoxidase anti-neutrophil cytoplasmic antibody. Chest x-ray demonstrated bilateral perihilar opacities. ECG showed normal sinus rhythm and QS pattern in leads V1 through V5.

A chest nodule referred to our hospital for cardiac magnetic resonance imaging. After resolution of dyspnea, we performed coronary angiography. There were no coronary arteries. Transthoracic echocardiography (TTE) demonstrated severe thickening and hyperkinesis of the posterior portion of the left ventricular (LV) wall, showing findings consistent with inflammation and fibrosis, and low signal mass, encircled by black dotted lines) and severe endocardial fibrotic thickening (Figure 1A and 1B, arrows and Movies I–III in the online-only Data Supplement), which caused the Doppler-derived systolic pressure gradient across the LV outflow tract of 74 mm Hg. Transmural flow pattern showed an E/A ratio of 2.1 with a deceleration time of 58 milliseconds, suggesting severe diastolic dysfunction. Gadolinium-enhanced cardiac magnetic resonance imaging demonstrated late gadolinium enhancement (Figure 2A, arrow) of LV endocardial segments, consistent with inflammation and fibrosis, and low signal mass, indicating subendocardial hematoma in the LV posterior wall (Figure 2A, open arrow). Significant mass was found in the posterior portion of the LV wall (Figure 2A, arrowheads). Foci consisting of acute/subacute brain embolic infarcts were found on diffusion-weighted magnetic resonance imaging. After resolution of the pulmonary edema, cardiac catheterization with coronary angiography was performed. There were no coronary arterial abnormalities. Endomyocardial biopsy specimens from the thickened portion of the LV posterior wall revealed hemorrhage in the subendocardial myocardium (Figure 3A, encircled by black dotted lines) and severe endocardial fibrotic thickening (Figure 3A, arrows). Eosinophilic cell infiltrations with necrosis were found in the myocardium (Figure 3B, asterisks), but there were no thrombus formations. Peripheral hypereosinophilia was resolved with the treatment of 1 mg/kg oral prednisolone within 10 days. After 4 weeks, eosinophil counts markedly reduced to 38/mm³ (0.5% of the white blood cell count). Prednisolone was tapered gradually. Serial TTE and cardiac magnetic resonance revealed gradual regression of myocardial thickening. Within 4 months of treatment, TTE and cardiac magnetic resonance demonstrated significant regression of myocardial thickening (Figure 2B and Movies IV–VIII in the online-only Data Supplement). There were moderate mitral regurgitation confirmed by TTE (grade 2–3/4) and the typical heart murmur at the apex on admission. Mitral regurgitation rapidly regressed 10 days after treatment with prednisolone, but mild mitral regurgitation (grade 1–2) still persisted 1 year after the treatment without any new symptoms.

Hypereosinophilia associated with Loeffler endocarditis is usually characterized by peripheral blood eosinophil counts exceeding 1500/mm³ for at least 6 months. Loeffler endocarditis includes eosinophilic infiltration and necrosis in the myocardium, resulting in the formation of mural thrombus, and fibrotic thickening. With progression, most of the patients suffer heart failure caused by the restrictive physiology or systemic emboli. In the present case, regression of the mass in the posterior LV wall may partially represent the resolution of mural thrombus and intramyocardial hemorrhage. However, there are no other findings of vasculitis typical of Churg-Strauss syndrome. The elevation of the myeloperoxidase anti-neutrophil cytoplasmic antibody, significant resolution of the mass in the posterior LV wall with steroid treatment within a few months, and TTE and cardiac magnetic resonance findings endorsed by endomyocardial biopsy specimens support the diagnosis of Loeffler endocarditis.

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Disclosures
None.
References


**Figure 1.** Transthoracic echocardiography demonstrated severe thickening and hyperkinesis of the posterior portion of the left ventricular (LV) wall during diastole (A, arrows) and systole (B, arrows), resulting in the Doppler-derived systolic pressure gradient across the LV outflow tract.

**Figure 2.** A, Gadolinium-enhanced cardiac magnetic resonance (GE-CMR) imaging reveals marked endocardial late gadolinium enhancement within the left ventricle (LV), indicating inflammation and fibrosis (arrow) and bleeding mass (open arrow) of the posterior LV subendomyocardial segment of low intensity confirmed by the biopsy specimen (Figure 3A). Significant mass was found in the posterior portion of the LV wall (arrowheads). B, Within 4 months of treatment, CMR demonstrated significant regression of myocardial thickening and improved midventricular obstruction, but the posterior portion of the LV wall was still hyperkinetic.

**Figure 3.** Histological findings of endomyocardial biopsied specimens. A, Marked endocardial fibrotic thickening (arrows) and subendocardial bleeding (Azan stain, circled by black dotted lines). B, Prominent eosinophilic cell infiltrations with necrosis in the myocardium (hematoxylin and eosin stain, asterisks) on high magnification.
Biopsy-Proven Loeffler Endocarditis Successfully Treated With Steroids

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