

Letter to the Editor

Cardiac magnetic resonance imaging for the diagnosis and follow-up of Loeffler's endocarditis

To the Editor:

Loeffler's endocarditis (LE) is an acute form of primary restrictive cardiomyopathy. Patients affected often have a hypereosinophilic syndrome and cardiac involvement. Hypereosinophilia and cardiac

involvement are also seen in the allergic eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome). Warfarin is generally indicated because of association between LE and embolic events.

We report a case of a 44-year-old woman with a history of asthma and Raynaud phenomenon, treated with oral prednisone, inhaled bronchodilator, and corticosteroids. The patient reported chest pain, fever (maximum 38.5°C), arthralgia, and exertional dyspnea in the last month. The patient showed sinus tachycardia

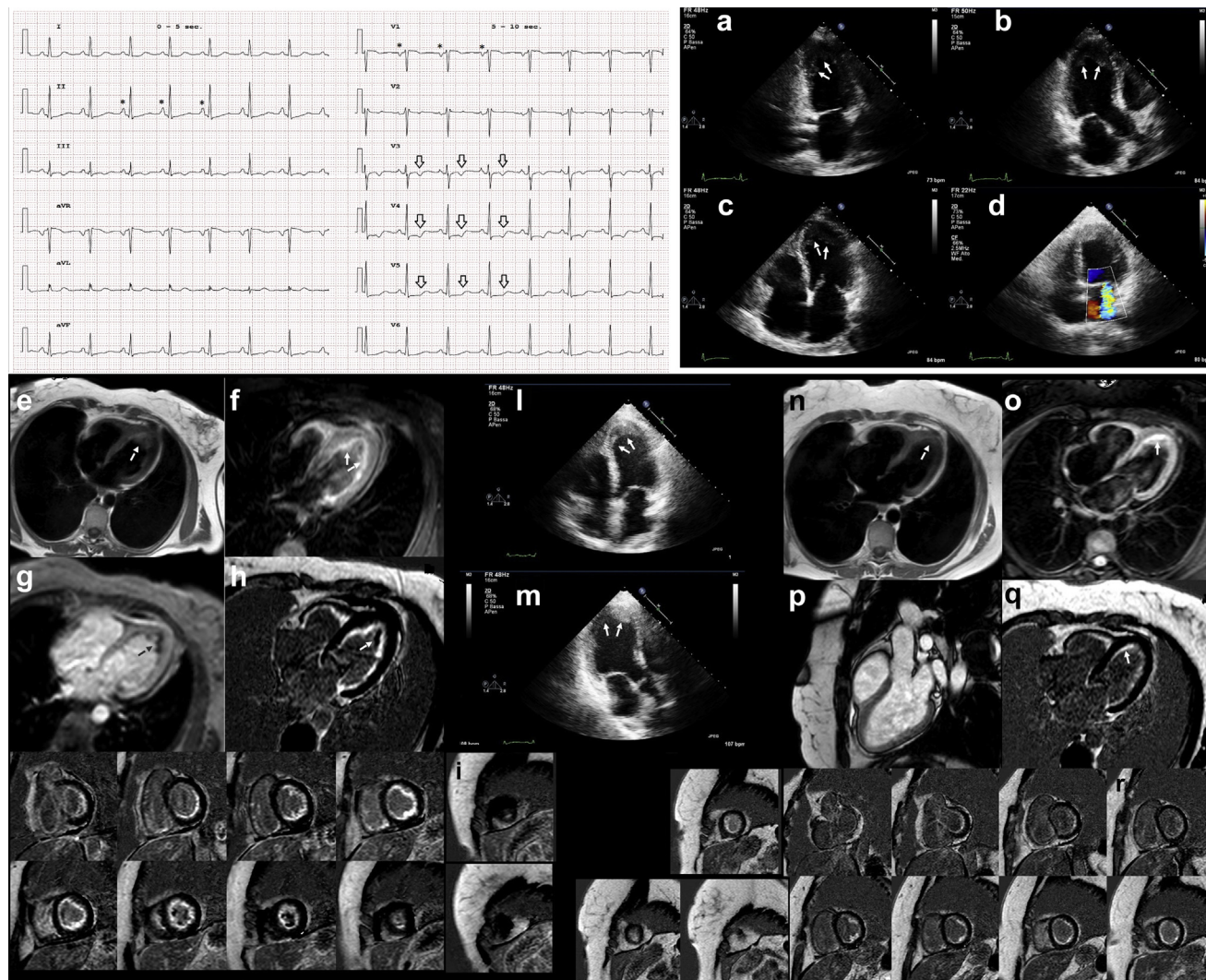


FIG 1. Electrocardiogram (aspecific ST/T anomalies [arrows], atrial enlargement [asterisks]). **a**, Echocardiography: infiltration with different echogenicity (arrows). **b**, Mass obliterating the left apex (arrows). **c**, Infiltration in left and in right apex (arrows). **d**, Moderate degree mitral regurgitation. **e**, Diffuse subendocardial thickening with obliteration of left ventricular apical lumen (arrow). **f**, Diffuse hyperintensity subendocardial signal; wall edema (arrow). **g**, First-pass images: hypointensity signal in the left and right ventricular apex (arrow). **h**, (Late enhancement study) diffuse hyperintensity subendocardial biventricular signal. Hypointense mass in left ventricular apex suitable with mural thrombus (arrow). **i**, Subendocardial hyperintense signal. **l** and **m**, Reduction of the mass in the left apex (arrows). **n**, Reduction of endocardial thickening (arrow). **o**, Subendocardial signal in midapical segments of the interventricular septum is mildly increased (arrow). **p**, Reduction of endocardial thickening. **q**, Diffuse reduction of hyperintensity left and right ventricular subendocardial signal (arrow); hypointense endoventricular mass compatible with mural thrombus is absent. **r**, Diffuse reduction of hyperintensity signal in subendocardial left ventricular wall and free right ventricular wall.

(105 bpm), and her blood pressure was 130/70 mm Hg. Laboratory tests showed the following: hemoglobin levels, 9.6 g/dL; white blood cells, 13,300/dL; eosinophils 4,750/dL (35.7%); erythrocyte sedimentation rate, 70 mm/1 h; C-reactive protein, 8 mg/dL; ANA (6.3; normal value, <3); anti-cyclic citrullinated peptide antibodies (6.1; normal value, <5), while pANCA (19.3; normal value, <2) titres were increased, cANCA mildly increased (2; normal value, <2), anti-DNA, and anti-ENA negatives. Troponin levels were 0.54 ng/mL. Neutrophils (52%), eosinophils (32%), lymphocytes (10%), and mastocytes (6%) were found at peripheral smear. Antibodies to hepatitis virus, *Treponema pallidum*, and HIV and tests for parasites were negative. Therapy with ceftriaxone 1 g/d and clarithromycin 500 mg $\times$ 2/d was started with improvement in symptoms and fever reduction 4 days later. Electrocardiogram showed diffuse ST/T anomalies, more evident in anterior-apical leads, and biatrial enlargement (Fig 1). At chest computed tomography scan, areas of ground glass in the posterior segments of the upper lobes and other small pseudonodular areas in the remaining segments, moderate bilateral pleural effusion, and minimal pericardial effusion were evident. On bronchoalveolar lavage, eosinophilia was found. Nerve conduction study suggested mononeuropathy multiplex.

A transthoracic echocardiogram revealed the presence of large areas with different echogenicity in the left and right ventricular apex, involvement of mitral and tricuspid subvalvular apparatus, and moderate secondary insufficiency, apical hypokinesis, preserved left ventricular systolic function, impaired diastolic function, right and left atrial dilation, and increased right ventricular systolic pressure (Fig 1, a-d).

Cardiac magnetic resonance imaging (cMRI) showed left and right ventricular normal size and mass, mildly reduced volume index, diffuse thickening of the left and right ventricular apex with obliteration (Fig 1, e-i), apical hypokinesis with preserved left and right ventricular systolic function (see Videos E1-E4 in this article's Online Repository at www.jacionline.org), diffuse subendocardial hyperintensity signal suggestive for wall edema (Fig 1, e-i), hypointensity signal in the left ventricular apex and minimum hypointensity in the right ventricular apex (Fig 1, e-i), large subendocardial hyperintense signal in the left ventricle (whole apical and middle segments, basal segments of the inferior wall, lateral and inferolateral), and subendocardial hyperintense signal of the right ventricle (apex, middle, and basal segments of the inferior and lateral wall). An apical left ventricular hypointense area ($1.5 \times 1.2 \times 0.5$ cm) was suitable with the presence of thrombus (Fig 1, e-i).

The diagnosis of Churg-Strauss syndrome associated with LE was therefore established and the treatment started with oral prednisolone and methotrexate, with anticoagulation with warfarin being continued. Cardiac biopsy was not performed. One month and 3 months after the initiation of therapy, follow-up echocardiography showed partial reduction of the infiltration areas within left and right ventricular apex (Fig 1, l-m). Five months later, cMRI control showed a reduction in subendocardial thickening (see Videos E5-E8 in this article's Online Repository at www.jacionline.org). Subendocardial signal within midapical segments of the interventricular septum was mildly increased (Fig 1, n-r). A diffuse reduction of hyperintensity signal in the subendocardial left ventricular wall, free wall, and right ventricular apex was found; hypointense endoventricular signal (thrombus) was absent

(Fig 1, n-r). During 6-month follow-up, no hospital admission for cardiovascular complication was observed; the patient was treated with prednisone and methotrexate. Eosinophil levels normalized after treatment (0.1%).

We therefore report a case of LE associated with Churg-Strauss syndrome; transthoracic echocardiogram, the most commonly used for clinical diagnosis of several cardiovascular illnesses, does not always correctly identify the interface between thrombus and thickened endocardium. cMRI, instead, allows a thorough assessment of the site and the extent of subendocardial lesions, by analyzing tissue characteristics.¹ The use of different sequences T1-T2 weighted as well as delayed enhanced imaging allows a precise definition of thrombotic endoluminal masses. In late images after administration of contrast medium, areas of endomyocardial fibrosis can be well identified.²

Late gadolinium enhancement cMRI offers the novel capacity to quantify the burden of myocardial fibrosis, a common end point of most cardiomyopathies and a robust marker of cardiovascular events related to the presence of advanced tissue disease.³ cMRI is the benchmark for the assessment of cardiac volumes, ejection fraction, and cardiac mass,⁴ and also allows a detailed view and a precise assessment of the right ventricle.

Despite the fact that diagnosis and follow-up of LE can be achieved noninvasively with echocardiogram, cMRI may be more sensitive and enable early diagnosis by detecting changes in the ventricular structure and function that may not be detected by echocardiogram, with an excellent capacity in detecting myocardial infiltrates (sensitivity, 80%; specificity, 94%; positive predictive power, 92%; and negative predictive power, 85%⁵). Late gadolinium enhancement correlates well with the biopsy findings of inflammatory infiltrates.⁶

Diagnosis may be confirmed by percutaneous endomyocardial biopsy, but, because endomyocardial involvement may be patchy, a false-negative biopsy result is also possible.⁷ However, cMRI may provide optimal anatomical target for biopsy.⁸

The current case demonstrates not only the diagnostic potential of cMRI to visualize cardiac involvement in hypereosinophilic syndrome but also morphologic changes after treatment with a noninvasive approach. cMRI is useful in differentiating LE from other causes of restrictive cardiomyopathy on the basis of the presence of typical cMRI findings (diffuse, global, endocardial hypoperfusion in first-pass contrast-enhanced perfusion, diffuse subendocardial delayed enhancement, and thrombus formation). Other common causes of restrictive cardiomyopathy often result in diffuse irregular hyperenhancement with noncoronary distribution (ie, cardiac amyloidosis and sarcoidosis).

Thanks to its high tissue resolution, cMRI may be therefore valuable for lesion assessment, therapeutic guidance, and prognostic stratification, over other imaging tools.⁹

In conclusion, cMRI may play a crucial role in the diagnosis of LE. The cMRI, over echocardiography, allows multiple scanning planes with excellent spatial resolution and the possibility of tissue characterization.

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Video legend.

VIDEO 1: four-chamber view, left ventricular apex hypokinesis.

VIDEO 2: two-chamber view, left ventricular apex thickening.

VIDEO 3: two-chamber view, right ventricular preserved systolic function.

VIDEO 4: short-axis view, preserved left ventricular systolic function.

VIDEO 5: two-chamber view, reduction of right ventricular thickening.

VIDEO 6: two-chamber view, reduction of apical thickening.

VIDEO 7: short-axis view, preserved biventricular systolic function.

VIDEO 8: four-chamber view, apex with preserved contractility.