Follow-up magnetic resonance imaging of Löeffler endocarditis: a case report

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Background

Löeffler endocarditis is a condition characterized by cardiac infiltration of eosinophils. Cardiac magnetic resonance imaging (MRI) is a modality for the diagnosis of myocardial damage.

Case summary

This is the case of a 77-year-old man with acute decompensated heart failure who was admitted. Transthoracic echocardiography showed preserved left ventricular (LV) systolic function along with LV thrombi attached to the septo-apical wall and the posterior wall, consistent with Löeffler endocarditis. Cardiac MRI revealed obliteration of the LV apex and partial filling of the LV cavity, as well as near circumferential subendocardial late gadolinium enhancement (LGE) in the mid- and apical segments. T2-weighted images showed a near circumferential high-intensity area of the LV subendocardial muscle in the mid- and apical segments. High-dose corticosteroids and intravenous heparin were initiated, followed by maintenance warfarin therapy. At 18 months, follow-up cardiac MRI revealed the disappearance of the LV thrombi, and a reduction of LGE, as well as high-intensity areas in the T2-weighted images.

Discussion

The high-intensity area of T2-weighted images indicate the presence of subendocardial oedema. Eosinophil-mediated heart damage evolves through three stages: (i) acute necrotic, (ii) thrombotic, and (iii) fibrotic stages. Since the deposition of toxic eosinophil granule proteins and eosinophil infiltration injured the endocardium, the first-line treatment for Löeffler endocarditis is corticosteroid therapy. In this case, LGE in the subendocardium and the high-intensity area in the T2-weighted images were reduced at 18 months. High-intensity areas of T2-weighted images in the acute phase might indicate the possibility of therapeutic response to corticosteroid therapy.

Keywords

Löeffler endocarditis • Left ventricular thrombus • Eosinophilic endocarditis • Immunosuppressive therapy • Anticoagulation therapy • Cardiac magnetic resonance imaging • Case report

Learning points

• Transthoracic echocardiogram, enhanced computed tomography, and cardiac magnetic resonance imaging could visualize mural thrombus in the left ventricle.
• T2-weighted images in patients with Löeffler endocarditis could differentiate oedema from fibrosis (i.e. irreversible change).
• High-intensity area of T2-weighted images in patients with Löeffler endocarditis in the acute phase may indicate the possibility for therapeutic response to corticosteroid therapy.
Introduction

Löeffler endocarditis, which was first described by Löeffler in 1936, is characterized by the cardiac infiltration of eosinophils. Cardiac magnetic resonance imaging (MRI) has emerged as a new modality for the diagnosis of cardiac disease in hypereosinophilic syndrome. Late gadolinium enhancement (LGE) cardiovascular magnetic resonance was previously reported as a reliable diagnostic tool to confirm endomyocardial fibrosis. However, many studies have focused more on the LGE images than the T2-weighted images of patients with Löeffler endocarditis. This is a case of Löeffler endocarditis in which T2-weighted images showed high-intensity areas at the time of diagnosis, and this finding was reduced at 18 months.

Timeline

| 4 days prior | The patient developed shortness of breath |
| 1 day prior | Shortness of breath deteriorated  
Complete blood count (CBC) revealed elevation of eosinophils |
| Admission | The patient was emergently hospitalized for acute decompensated heart failure. The brain natriuretic protein level was 1051.6 pg/mL (≥18.4 pg/mL). Chest X-ray showed pulmonary congestion with butterfly shadow |
| First week of hospital admission | Transthoracic echocardiography (TTE) showed massive mural thrombi without regional wall motion abnormality on the affected wall. Considering the elevation of eosinophils, we suspected endocarditis and investigated to diagnose the cause of endocarditis including cardiac magnetic resonance imaging (cMRI) and enhanced multislice computed tomography, which revealed Löeffler endocarditis with left ventricular (LV) thrombosis. Immediately corticosteroid therapy and antithrombotic drugs started |
| One month after admission | CBC showed no elevation of eosinophils, and TTE showed no resolution of LV thrombus |
| Two months after admission | Follow-up TTE showed no resolution of LV thrombus |
| One and a half years after admission | Follow-up cMRI showed reduction of the LV thrombus and regression of high-intensity area of T2-weighted image and late gadolinium enhancement |

Case presentation

This is the case of a 77-year-old male who was referred to our hospital for severe shortness of breath that started 4 days before presentation. He had previous histories of bronchial asthma, sigmoid clonal cancer, small bowel obstruction, and acute duodenal ulcer. Physical examination revealed bilateral coarse crackle at the bases and absence of a heart murmur. Complete blood count showed a significant rise in the eosinophil count (2769/μL, 30.1%). The white blood cell count was 9200/μL (normal value: 3000–9000/μL), neutrophils accounted for 54.9% (30–72%) of the white blood cell count, lymphocytes 11.3% (20–53%), monocytes 3.5% (2–12%), and basophils 0.2% (0–3%). The haemoglobin level was 89 g/L (135–175 g/L) and platelet count was 183 000/μL. The brain natriuretic protein level was 1051.6 pg/mL (≥18.4 pg/mL). Both myeloperoxidase and Proteinase3 antineutrophil cytoplasmic antibodies were negative. The chest X-ray showed bilateral pulmonary opacities. Transthoracic echocardiography (TTE) revealed massive mural thrombus attached to the septo-apical wall and the posterior wall, which was consistent with Löeffler endocarditis (Figure 1A). We suspected Löeffler endocarditis to be the cause of the mural thrombi because there was no regional wall motion abnormality on the affected wall. Left ventricular (LV) systolic function was visually preserved, without significant valve regurgitation and stenosis. We did not use the modified Simpson method since it is unreliable in the setting of extensive mural thrombi. The transmigral flow pattern was pseudonormal (E/A ratio was 1.68 and the deceleration time was 161.1 ms). e’ (septal) was 5.0 cm/s and E/e’ was 12.13. Cardiac MRI revealed obliteration of the LV apex and partial filling of the LV cavity as well as near circumferential subendocardial LGE in the mid- and apical segments (Figure 2A and B). T2-weighted images showed a near circumferential high-intensity area of the LV subendocardial muscle in the mid- and apical segments (Figure 2C). We differentiated subendocardial hyperintensity from the slow flow artefact often seen on STIR/TIR sequences at the endocardial border referring to the cine images. The MRI findings of the combination of subendocardial LGE and obliteration of the LV apex enabled us to make a confident diagnosis. Although he did not have any neurological and thrombotic symptoms, we searched for signs of cerebral and other systemic embolization. Magnetic resonance imaging of the brain showed multiple small newly developed cerebral infarctions in the cortical watershed zone and the cerebellar region, while enhanced computed tomography (CT) revealed no signs of systemic embolization in the rest of his body.

High-dose corticosteroid therapy (500 mg methylprednisolone for 3 days, followed by 60 mg prednisolone daily) was initiated. The percentage of eosinophils decreased to below 0.1% one day after the initiation of the corticosteroid therapy. Intravenous heparin administration was started immediately after admission, followed by maintenance warfarin therapy. Transthoracic echocardiography at Day 26 (Figure 1B) and electrocardiogram-gated CT at Day 31 showed no significant reduction in the size of the mural thrombus in the LV cavity. The dose of warfarin was adjusted to the therapeutic PT-INR range from 2.0 to 3.0.

The patient was event-free (no cardiovascular events) at 1 year. The reduction of the thrombotic burden was confirmed using TTE (Figure 1C). Follow-up cardiac MRI at 18 months revealed the disappearance of the LV thrombus and a reduction of the endocardial high-intensity area upon gadolinium enhancement (Figure 2D and E). High-intensity areas of T2-weighted images were also reduced (Figure 2F).
Figure 1 Transthoracic echocardiographic image at 5 days showed left ventricular obliteration (A). Transthoracic echocardiographic images at 26 days revealed unchangeable thrombosis of the left ventricle by visual assessment (B). Transthoracic echocardiographic images at 1 year revealed significant reduction of thrombotic obliteration with no restrictive cardiomyopathy development (C).

Figure 2 Cardiac magnetic resonance image of the four chambers and axial view at 3 days showed obliteration of the left ventricular apex, partial filling of the left ventricular cavity, and near circumferential subendocardial late gadolinium enhancement in the mid- and apical segments (A and B; white arrows). T2-weighted images showed high-intensity area of the subendocardial muscle, which suggested oedema (C; yellow arrows). Follow-up cardiac magnetic resonance image at 18 months revealed the disappearance of the left ventricular thrombus and reduction of endocardial high-intensity area upon gadolinium enhancement (D and E). T2-weighted images at 18 months revealed the disappearance of high-intensity area (F; yellow arrows).
Discussion

Eosinophil-mediated heart damage evolves through three stages: (i) acute necrotic, (ii) thrombotic, and (iii) fibrotic stages. The early necrotic stage is usually not recognized because the clinical symptoms are not obvious in most of the cases. The second stage involves the formation of thrombi along the damaged endocardium of either one or both ventricles. In the fibrotic stage, progressive scarring may lead to endomyocardial fibrosis, restrictive cardiomyopathy, and valvular dysfunction.

A previous study showed eosinophil infiltration and deposition of toxic eosinophil granule proteins in cardiac tissue with eosinophilic endomyocardial disease. Eosinophilic granule proteins, such as major basic protein and the eosinophil peroxidase, can activate platelets and impair the anticoagulant effects of thrombomodulin. This might be one of the mechanisms of endocardial dysfunction. Therefore, corticosteroids for the reduction of eosinophilic infiltration are one of the main therapies for Löffler endocarditis.

In this case, high-intensity areas in T2-weighted images and LGE in the subendocardium were both reduced at 18 months. The regression of LGE may highlight its potential to monitor the responsiveness to treatment. This finding suggests that treatment with corticosteroids may prevent further progression to the fibrotic stage. Since T2-weighted images could differentiate oedema from fibrosis (i.e. irreversible change), high-intensity areas of T2-weighted images in the acute phase may indicate the possibility of a therapeutic response to corticosteroid therapy.

Lead author biography

Dr Shinya Ito graduated from Shiga University of Medical Science and started his career in Kobe City Medical Center General Hospital, Kobe, Japan. He is a general cardiology physician in the Department of Cardiovascular Medicine, Kokura Memorial Hospital, Kitakyushu, Japan, since 2016.

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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