

## Early Stage Loeffler's Endocarditis Detected by Transthoracic Echocardiography

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Loeffler's endocarditis involves progressive eosinophilic infiltration of the endocardium, which leads to apical thrombotic obliteration of the ventricle and endomyocardial fibrosis, that may finally represent a characteristic feature of restrictive cardiomyopathy. This paper presents a case of a 44-year-old male with symptoms of dyspnea and peripheral hypereosinophilia, who was diagnosed with early stage Loeffler's endocarditis via multicardiac imaging modalities.

**Key Words:** Loeffler's endocarditis, Transthoracic echocardiography, Thrombus

### INTRODUCTION

Hypereosinophilic syndrome (HES) is rare disease characterized by 1) persistent eosinophilia ( $>1,500$  eosinophils/ $\mu\text{L}$ ) without obvious etiology of hypereosinophilia and 2) eosinophil-mediated multiple organ dysfunction.<sup>1</sup> Heart is one of the frequently involved organ (40-50% of HES) and characteristic features of fibrous thickening of the endocardium leading to apical obliteration and thrombus formation and finally resulting in restrictive cardiomyopathy with heart failure is well known as Loeffler's endocarditis.<sup>1-4</sup>

We present a case of a 44-year-old male patient with typical morphologic feature of early phase of Loeffler's endocarditis in transthoracic echocardiography (TTE) and cardiac magnetic resonance imaging (CMR).

### CASE

A 44-year-old man without significant past medical history

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was admitted our hospital due to aggravated dyspnea for one month. His blood pressure was 110/70 mmHg, heart rate was 88/min, respiratory rate was 20/min and body temperature was 37.1°C. His breathing sound was decreased with mild rale in both lower lung fields. Chest X-ray revealed loculated right pleural effusion and electrocardiography demonstrated normal sinus rhythm with high voltage in lateral precordial lead (V4, 5, 6). Laboratory findings showed marked eosinophilia (11,155/ $\mu\text{L}$ ), elevated serum total immunoglobulin E (5,000 KU/L), mildly anemia (10.7 g/dL), mildly elevated troponin I (0.12 ng/mL) and elevated erythrocyte sedimentation rate (39 mm/hr). Anti-neutrophil cytoplasmic antibodies, fluorescent antinuclear antibody test and stool parasite test were all negative. Chest computerized tomography revealed no pulmonary thromboembolism, abnormal endobronchial lesion or enlarged lymphadenopathy but multiple nodules with surrounding halo at both lungs. CMR imaging revealed mild hypokinesia of left ventricular apex at dynamic study, diffusely thickened and enhanced endocardium with large hypointense semilunar thrombus at viability study (Fig. 1). Bone marrow biopsy revealed normocellular marrow with marked increase of normally matured eosinophils and FIP1L1-PDGFR tyrosine kinase was shown that there is no evidence of abnormal myeloid proliferative disease. TTE revealed different echogenic layering in the apical segment of left ventricle, mild

tricuspid valve regurgitation and borderline values of diastolic dysfunction (Fig. 2).

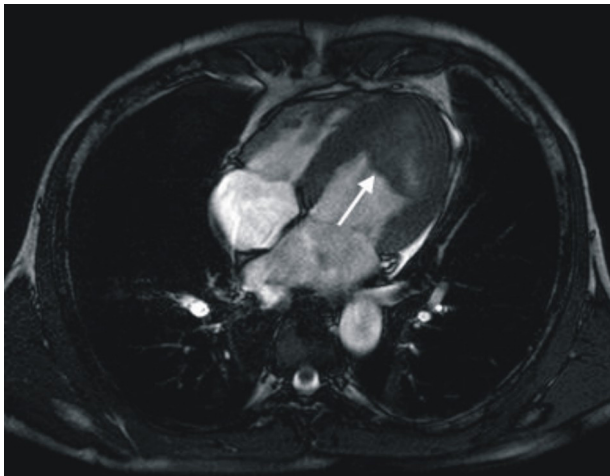
A clinical and echocardiographic diagnosis of Loeffler's endocarditis was thus considered, oral corticosteroid (prednisolone 60 mg), oral furosemide (40 mg/day for 3 days) and anticoagulation by systemic heparinization, which is changed into warfarin were planned. After 4 days, the symptom of dyspnea was disappeared, there was more decreased pleural effusion at follow-up chest X-ray and absolute eosinophil count was decreased from 11,155/ $\mu$ L to 1,065/ $\mu$ L, an indication of a good prognosis. Oral corticosteroid was tapered gradually by 30 mg at follow-up 1 month visit. But after 2 months, he suffered from severe right calf pain suddenly, right popliteal artery total occlusion was diagnosed at computerized tomography. We executed the peripheral artery thrombectomy by Fogarty catheter. Thrombus was confirmed by biopsy. Follow-up TTE at the same time showed more significantly decreased amounts of the thrombus and no definite diastolic dysfunction (Fig. 2). He was treated on the tapered

oral corticosteroid (prednisolone 20 mg) and anticoagulation (warfarin 5 mg) consistently.

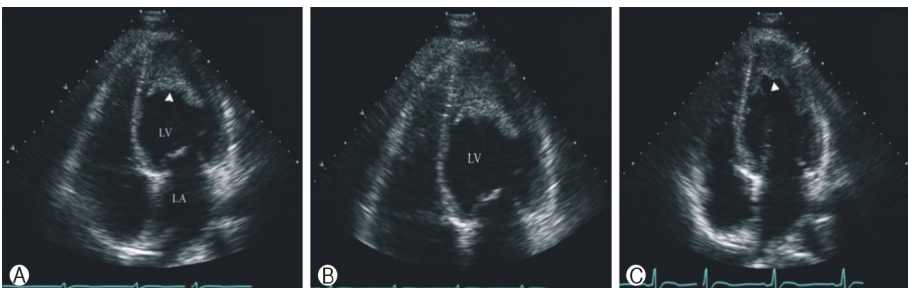
## DISCUSSION

We reviewed the early stage of Loeffler's endocarditis with borderline diastolic dysfunction by follow-up TTE and embolic event as peripheral artery total occlusion due to thrombus. Loeffler's endocarditis is considered as the late cardiac feature characterized by progressive eosinophilic infiltration of endocardium, leading to endomyocardial fibrosis, apical thrombotic obliteration of ventricle, which may finally represent characteristic feature of restrictive cardiomyopathy.<sup>5-9</sup> Eosinophils-mediated heart injury classifies through three stage.<sup>1</sup> First, acute necrotic stage as clinically asymptomatic phase means aggressive infiltration of eosinophils and lymphocytes on endomyocardium, leading to inflammatory reaction by eosinophil degranulation and its cytokine initial activation.<sup>1,10</sup> At the second stage, intracardiac mural thrombus is formed by thrombus outbreak along the damaged endocardium and it can induce embolic events.<sup>5</sup> In this stage, patients are able to suffer from cardiopulmonary symptoms. At the last stage, endomyocardial fibrosis is major component of pathologic feature of HES, leading to restrictive cardiomyopathy, aggressive valve regurgitation due to invasion of the chordae tendineae, finally Loeffler's endocarditis.<sup>1,7,11</sup>

Corticosteroids have been used in first line treatment of HES, initial corticosteroids dose is over 40 mg daily (or prednisolone equivalent) and then by improvement of patient's symptom, slow tapering to lowest effective dose of corticosteroid is warranted.<sup>3,12</sup> By using corticosteroid, nearly 70% of HES patients treated a rapid reduction in absolute eosinophil count.<sup>2</sup> Oral anticoagulation must be considered to prevent the spread of thromboembolic event, ventricular obliteration and remodeling.<sup>5,6</sup>



**Fig. 1.** Cardiac magnetic resonance imaging of the heart shows large hypointense apical thrombus (arrow) with diffusely thickened and non-enhanced endocardium of left ventricle.



**Fig. 2.** At the apical 4 chamber view, initial transthoracic echocardiography reveals different echogenic layering—considered as apical thrombus (arrow head) of left ventricle (A, B). Initial E/E' is 11.4 and E/A ratio is 0.8. Follow-up echocardiography (2 months later) shows significant regression of apical thrombus of left ventricle (C). Follow-up E/E' is 8.8 and E/A ratio is 0.64.

At cardiac involvement of HES, left ventricular thrombus due to endomyocardial damage by profound eosinophil, apical obliteration, the last finding into Loeffler's endocarditis can evolve gradually. By echocardiography follow up, we observed marked resolution of left ventricular thrombus and no significant diastolic dysfunction. Even if there are no critical evidences of restrictive cardiomyopathy suggestive Loeffler's endocarditis by echocardiographic imaging, we may consider the early detection and high possibility of changing to third stage as Loeffler's endocarditis from second stage. We report that TTE will be a valuable diagnostic tool of early stage of Loeffler's endocarditis.

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