

## A Case of Löffler Endocarditis Complicated with Listeria Sepsis

### INTRODUCTION

Löffler endocarditis (LE) is rare restrictive cardiomyopathy. *Listeria monocytogenes* is a foodborne pathogen that causes listeriosis, a relatively rare but potentially fatal gastrointestinal illness. The incidence of listeriosis in the United States is about 0.24/100 000 population (Centers for Disease Control, 2017). There is no report of Löffler endocarditis combined with Listeria infection.

We present the case of a 43-year-old woman who was admitted to another hospital because of repeated breathlessness. Her laboratory examinations demonstrated the presence of hypereosinophilia. Based on her multimodal imaging findings, the final diagnosis of Löffler endocarditis was made. During treatment, the patient developed a recurrent fever, her blood culture tests suggested listeria infection. We did the anti-infective treatment based on sensitivity tests after rehabilitation.

### CASE REPORT

The patient, a 43-year-old female, suffered from heart fatigue, shortness of breath, and frequent heart palpitations since April 2020, needing to lie elevated by high pillows and edema of both lower extremities. She was admitted to another hospital in June 2020. Physical examination: T: 36.4°C, P60 beats/min, R: 21 beats/min, BP131/71 mm Hg, clear breath sounds in both lungs, no dry and wet rales, normal heart, regular heart rhythm, no murmurs in each valve area, and mild edema of both lower extremities. Auxiliary examination included routine blood tests: eosinophil percentage 7.5%, basophil percentage 1.4%, eosinophil absolute value  $0.38 \times 10^9/L$ , basophil absolute value  $0.07 \times 10^9/L$ . Biochemical tests included total bilirubin 368  $\mu\text{mol/L}$ , direct bilirubin 13.7  $\mu\text{mol/L}$ , indirect bilirubin 23.1  $\mu\text{mol/L}$ , creatinine 82  $\mu\text{mol/L}$ , estimated glomerular filtration rate 75.75 mL/min/1.73 m<sup>2</sup>, uric acid 515  $\mu\text{mol/L}$ , potassium 4.06, trans-triiodothyronine 16 nmol/L, normal thyroid-stimulating hormone, FT3 (4.33 pmol/L) and FT4 (14.44 pmol/L). Swelling marker: serum carbohydrate antigen 125: 53.90 U/mL. Immunoglobulin and complement: immunoglobulin G 7.80 g/L, complement 30.7110 g/L, complement 40.1340 g/L. Immunological examination: no obvious abnormalities were observed in T subcells; ANA<1:80, anti-double-stranded DNA, and ENA (negative) were all negative. L; lupus anticoagulant: screening time 45.10 seconds, diagnosis time was 38.50 seconds LA1/LA21 .17; Microbial laboratory tests suggested anticardiolipin antibodies (-); ESR(-), procalcitonin 0.09 ng/mL, TBIGRA, G test, GM test, EB-DNA CMV-DNA, and TORCH- GM were negative: Schistosoma antibody (-), Plasmodium antigen (-); Serum VitB12 concentration determination (-); Cardiac color Doppler ultrasound+ right heart function measurement (2020-5-30): Whole- heart enlargement, biventricular apical endocardium thickening, calcified tricuspid regurgitation (severe), mitral regurgitation (mild), double decreased ventricular systolic function measurement values, and pericardial effusion (minor amount). The patient was initially diagnosed with eosinophilic endocarditis (Loeffler Endocarditis). Further cardiac magnetic resonance imaging (MRI) examination showed LV 54 mm, LA 40 mm, RA 52 mm, RV 28 mm, VS 9 mm, EF 46%. Myocardial perfusion ultrasound examination (June 20<sup>th</sup> 2020): We slowly

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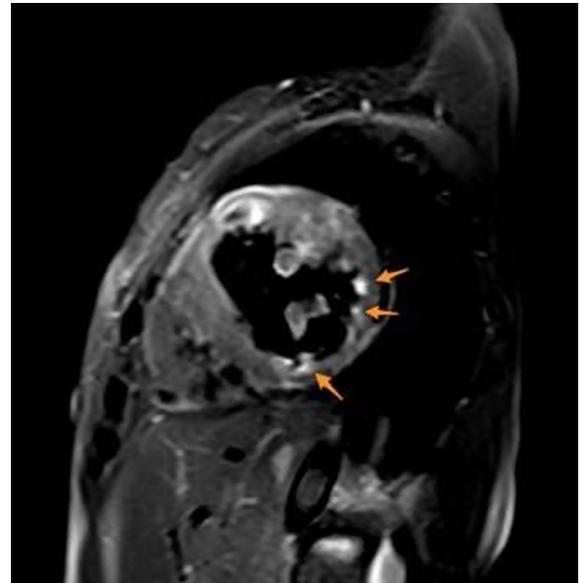
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injected Sonovitro solution 3.5 mL through the left elbow vein. Angiography showed abnormal shape of the apex of both ventricles with filling defect. There was slightly more contrast agent filling, and there were many irregular and strong echoes inside it. The maximum size of the right ventricle was 23 mm × 16 mm, and the maximum size of the left ventricle was 26 mm × 16 mm. There was an acoustic shadow behind them. There was nothing detected and no filling defect in the remaining heart cavity. Eosinophilic endocarditis with calcification is suggested. Computed tomography cardiac 3-dimensional reconstruction and enhanced scanning and chest scan showed heart enlargement, right atrium enlargement, endocardium calcification, no abnormal pulmonary veins, no signs of thrombosis in the left atrial appendage, and no phenotypic abnormalities on bone marrow aspiration and biopsy. The patient was treated with rivaroxaban for anticoagulation, furosemide diuresis, and methylprednisolone 80 mg qd intravenous infusion therapy. After the impulse treatment, the symptoms were relieved, so the dose was reduced to 20 mg qd and he was discharged. After being discharged from the hospital, the patient experienced repeated chills and high fever and was transferred to the local hospital for poor treatment results. He later came to our hospital for further treatment. Cardiac color Doppler ultrasound after admission showed double ventricular apex endocardium thickening and calcification, double atrium enlargement, severe tricuspid regurgitation, moderate pulmonary hypertension, E/A2.4, moderate mitral regurgitation, double-ventricle reduced systolic function, EF 0.48%, TAPSE 10 mm, obvious endocardial mass calcification. Computed tomography of the upper abdomen revealed hypodensity of the spleen. Enhanced blood culture results suggested *Listeria* infection. Cardiac MRI revealed double-ventricular cavity volume shrinkage as well as uneven thickening of the left ventricle, the middle segment of the ventricular septum, and the apical segment, also involving the right ventricular outflow tract, causing its stenosis. The left and right ventricle middle and apical segments of the ventricular wall, the middle segment of the ventricular septum, and the apical segment of the myocardium had middle myocardial fibrosis. For the sepsis caused by *Listeria*, cefoperazone and sulbactam were given to fight the infections. The drug sensitivity test showed that the infections were sensitive to amoxicillin, penicillin G, and compound trimethoprim, and the antibiotic was adjusted to piperacillin and tazobactam 4.5 g. After 3 days of intravenous infusion of q8h anti-infective treatment, the patient did not have a fever again. After that, sequential anti-infection therapy was continued for 1 month, and the patient's symptoms were relieved without fever (Figures 1-3).

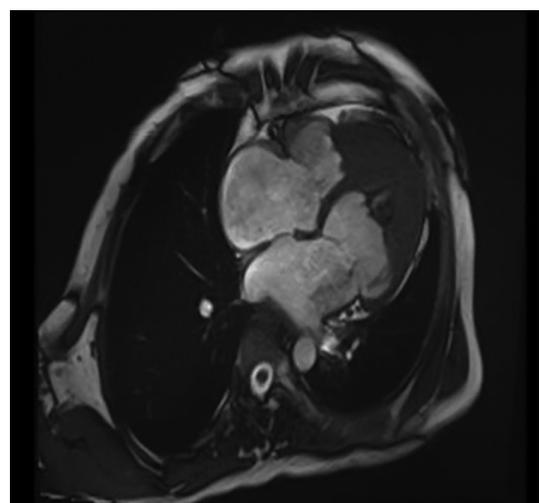
## DISCUSSION

Eosinophilic endocarditis is a restrictive cardiomyopathy, Loeffler's endocarditis is a cardiac manifestation of eosinophilia syndrome, a rare systemic disease characterized by continuous occurrence. Eosinophils cause organ damage. Non-invasive examinations, including echocardiography, cardiac MRI, and PET-CT, can provide a strong basis for evaluating the heart involvement of LE and other non-cardiac manifestations of hypereosinophilic syndrome. If



**Figure 1. Left ventricular short-axis view of cardiac MRI: endocardial thickening and calcification. MRI, magnetic resonance imaging.**

the results of non-invasive imaging are confusing or uncertain, endocardial biopsy may be considered in patients with high clinical suspicion of LE. The appropriate use of invasive and non-invasive imaging methods, combined with existing technology and the clinical characteristics of patients, is expected to lead to early diagnosis, more accurate disease staging, and timely treatment.<sup>1-4</sup> The patient's echocardiogram, contrast-enhanced ultrasound, and cardiac MRI all showed signs of eosinophil infective endocarditis, but the patient's left ventricle showed thrombosis and was prone to stroke.<sup>5</sup> Splenic embolism is contraindicated for endocardial myocardial biopsy, so myocardial biopsy was not performed. For the treatment of eosinophilic endocarditis, many cases have shown the effectiveness of glucocorticoids.<sup>6-8</sup>



**Figure 2. Heart MRI four-chamber view: the apex of the double ventricle is obviously thickened. MRI, magnetic resonance imaging.**



**Figure 3. Echocardiogram: thickening and calcification of the left ventricle endocardium.**

However, the use of glucocorticoids in patients can lead to decreased immunity and increase the risk of pathogen infection. Opportunistic infections. Possible infectious complications should be considered in patients receiving high-dose glucocorticoid therapy.<sup>9</sup> *L. monocytogenes* is widely distributed in nature and widely found in animals and plants, as well as in sludge, soil, and water environments. *Listeria monocytogenes* is inherently resistant to third-generation cephalosporins, fusidic acid, and fosfomycin. Ten strains of *L. monocytogenes* were isolated from food and food-processing environments. About 10%-80% are resistant to biocides and show high resistance to osmotic, dry, hot, cold, alkaline, and acidic stress conditions, so it can selectively survive and increase its relative total group number. Resistance of *L. monocytogenes* to ampicillin occurs very rarely, maybe in less than 0.1% of isolates. During 2012-2015, no drug resistance was detected in 2862 well-characterized isolates in China.<sup>10</sup> Although only a few patterns have been identified, the evolutionary conservation of many metabolic functions suggests that these molecules may be targets of the innate immune system.<sup>11</sup> This patient still had fever after using cefoperazone and sulbactam. According to the drug susceptibility test, after switching to piperacillin and sulbactam, the symptoms were relieved and the fever did not reappear, which once again shows the antibacterial susceptibility

properties of *Listeria*. To understand the characteristics of microbial drug susceptibility, it is important to use drugs more accurately based on the results of drug susceptibility tests.

## CONCLUSION

Clinically, eosinophilic endocarditis is rare and *Listeria sepsis* is also rare. These 2 diseases have not been reported together. This case has far-reaching significance for the diagnosis and treatment of such patients.

**Informed Consent:** Patient informed consent was waived owing to the retrospective nature of this case report.

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