Letter to the Editor

A case description of chronic eosinophilic leukemia with loeffler endocarditis initially characterized by cerebral infarction: the role of 2-dimensional speckle tracking echocardiography in diagnosis

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Introduction

Eosinophilia is a clinically rare condition. It can be primary, including idiopathic eosinophilia syndrome and chronic eosinophilic leukemia (CEL), or secondary, most often caused by parasitic infections (1). Loeffler endocarditis is a disease in which an abnormal increase in the number of eosinophils causes damage to the heart, which in turn leads to a series of clinical symptoms (2). This case reported an interesting case of a middle-aged male admitted with cerebral infarction as the first symptom, laboratory tests suggesting abnormal eosinophilia, echocardiography showing thrombosis in the left ventricle, and a final diagnosis of CEL with Loeffler endocarditis. With the treatment of corticosteroids, imatinib, and anticoagulants, the patient's hematology and myocardial injury improved significantly, and this was assessed using 2-dimensional (2D) speckle tracking echocardiography. Endomyocardial biopsy (EMB) is often considered essential for a definitive diagnosis of Loeffler endocarditis, whereas cardiac magnetic resonance imaging (MRI) provides valuable non-invasive assessment. However, in the absence of EMB, and in urgent situations where diagnosis and treatment must be made expeditiously, echocardiography and its newer techniques are essential for the early diagnosis of Loeffler endocarditis to ensure timely and effective treatment (3).

Case presentation

A 56-year-old man with no past medical history was admitted to the Second Affiliated Hospital, Jiangxi Medical College, Nanchang University due to headache and dizziness with visual field defects for 3 days in another hospital on 31 July 2023. Cranial computed tomography (CT) and cranial MRI showed cerebral infarction in the left temporal lobe and occipital lobe. He had been treated with vasodilation and lipid-lowering therapy. Since the symptoms did not improve and he experienced decreased muscular strength in his right limb as well as numbness. He was referred to our hospital for additional evaluation and treatment. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was provided by the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Initial laboratory examinations revealed marked

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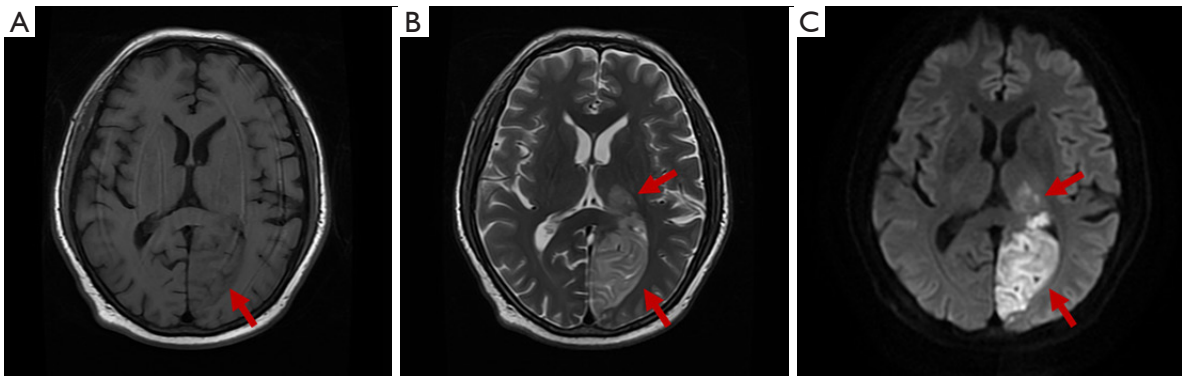

Figure 1 Cranial MRI. In the left occipital temporal lobe and thalamus, there are large patches of slightly longer T1 and slightly longer T2 signals (A), and T2 FLAIR (B) and DWI (C) are hyperintense (arrows). MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery; DWI, diffusion-weighted imaging.

eosinophilia (eosinophils 43.4%, normal range, 0–8%; absolute eosinophil count of 3.05×10^9/L, normal range, 0–0.5×10^9/L), mild anemia [hemoglobin (Hb) of 98 g/L, hematocrit 31.9%], thrombocytopenia, as well as abnormal liver and kidney function. The levels of α-hydroxybutyrate dehydrogenase (α-HBDH, 281.33 U/L, normal range, 72–182 U/L), lactate dehydrogenase (LDH, 334.11 U/L, normal range, 120–250 U/L), and brain natriuretic peptide (BNP, 281.00 pg/mL, normal range, 0–100 pg/mL) were elevated. Troponin (Tn) was 0.011 ng/mL (chemiluminescence assay: normal range, 0–0.05 ng/mL). D-dimer (DD) value was 3.85 mg/L fibrinogen equivalent unit (FEU) (cut-off value <0.5 mg/L FEU). Vitamin B12 was >2,000 pg/mL, folic acid (FA) was 1.95 ng/mL, ferritin (FER) was 390.0 ng/mL, and homocysteine (Hcy) was 16.18 μmol/L. Glycated hemoglobin (HbA1c) was 5.6%, and serum glucose was 6.12 mmol/L 2 hours after meals. Erythrocyte sedimentation rate (ESR) was 10.74 mm/h, and fibrinogen (Fg) is 3.15 g/L. Auto-immunity tests yielded negative results and thyroid function and hormones were within limits.

Cranial MRI showed acute cerebral infarction in the left occipital temporal lobe and thalamus, scattered ischemic foci in the brain, and mild cerebral atherosclerosis (*Figure 1*), and CT angiography of brain vessels was not performed. A chest CT showed no significant abnormalities.

To clarify the cause of cerebral infarction, the patient underwent 2-dimensional (2D) transthoracic echocardiography (TTE). TTE revealed a left ventricular ejection fraction (LVEF) of 71% (normal range, 50–70%), tricuspid annular plane systolic excursion (TAPSE) of 17 mm (normal range, ≥16 mm), normal internal diameters of each atrium and ventricles, no valvular involvement, and no pericardial effusion. However, a honeycomb-shaped slightly echogenic area was visible from the level of the left ventricular papillary muscle to the level of the apex, measuring approximately 31 mm × 32 mm × 33 mm (*Figure 2A-2C*), with a loose internal structure, an uneven edge of the left ventricular surface, and poor range of motion. In conjunction with laboratory tests, ultrasound diagnosis considered Loeffler endocarditis and left ventricular thrombus. To further clarify the extent of the thrombus, the patient underwent contrast-enhanced ultrasound, which showed a left ventricular thrombus of approximately 32 mm × 33 mm × 30 mm (*Figure 2D*).

The patient reported no history of allergies. Pulmonary function tests suggested moderately restrictive ventilatory dysfunction. In addition, the patient was positive for *Treponema pallidum* antibody (anti-TP), herpes simplex virus type I IgG antibody (HSVI-IgG), herpes simplex virus type II IgG antibody (HSVII-IgG), cytomegalovirus IgG antibody (CMV-IgG), and rubella virus IgG antibody (RV-IgG). Parasitic and oval diseases were excluded based on fecal examination results. Fungal infection was ruled out.

Eosinophilia persisted upon repeated blood tests before admission (eosinophils 40.8%, normal range, 0–8%; absolute eosinophil count of 2.49×10^9/L, normal range, 0–0.5×10^9/L). Bone marrow smear cytology showed myeloproliferative activity, a normal granulocyte/erythrocyte ratio (G/E =2.2), significant eosinophilia (40%), and hyperproliferative megakaryocytes with impaired maturation (*Figure 3*). Most importantly, due to the

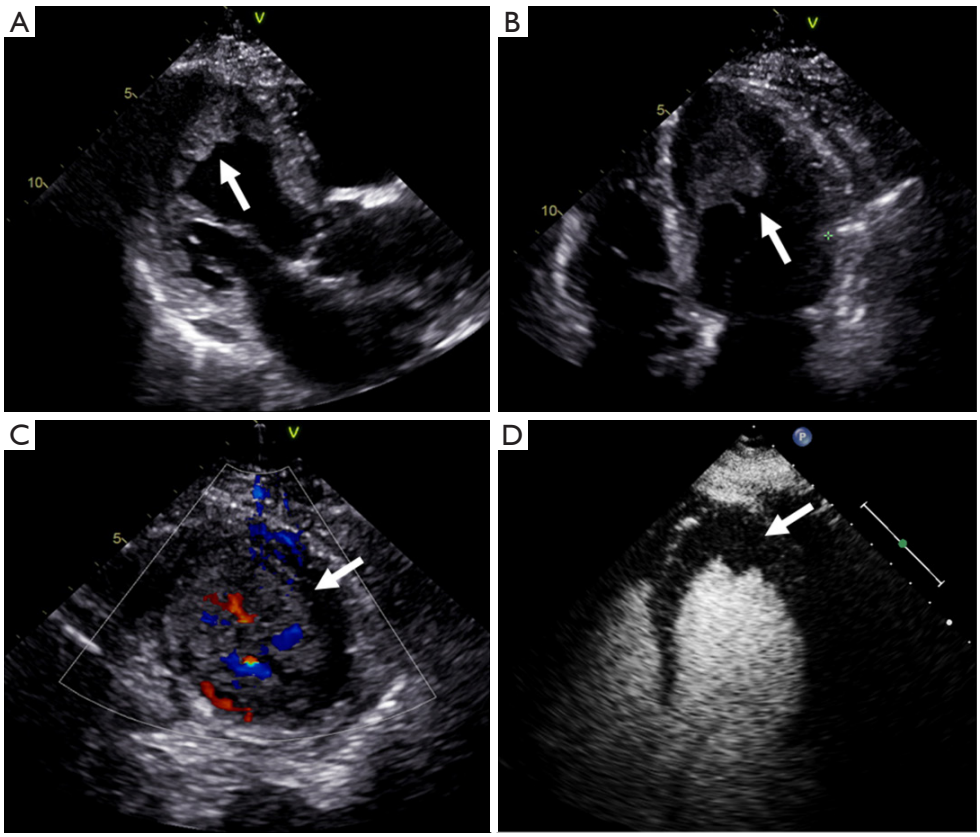

Figure 2 Two-dimensional echocardiography images. (A) Apical 3-chamber and (B) 4-chamber views show a thrombosis (arrows) extending from the left ventricular papillary muscle to the apex. Horizontal section of the short axis apex of the parasternal left ventricle (C) shows the left ventricle filled with thrombus, and color Doppler shows a small flow signal (arrow). (D) Contrast-enhanced ultrasound shows left ventricular thrombus without contrast microbubble development (arrow).

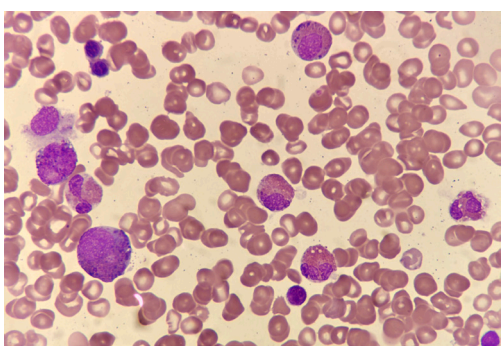

Figure 3 Bone marrow aspirate smear (Wright's staining, 1,000× magnification), showing significant proliferation of abnormal immature eosinophils.

suspicion of eosinophilic myeloid leukemia, polymerase chain reaction/fluorescence in situ hybridization (PCR/FISH) was performed and identified the gene fusion, and the leukemia-associated fusion gene test (56 screenings) returned positive for the FIP1-like 1/platelet-derived growth factor receptor alpha (*FIP1L1-PDGFRA*) fusion gene.

Conditions associated with intracardiac thrombosis such as ischemic cardiomyopathy, valvular heart disease, dilated cardiomyopathy, and chronic heart failure were excluded. The diagnosis was consequently CEL with Loeffler endocarditis and cerebral artery embolic infarction. The patient was treated with prednisone acetate (40 mg/day), imatinib (100 mg/day), and anticoagulation (warfarin, 3 mg/day, international normalized ratio (INR): 2–3, continued until the thrombus disappeared). The number of peripheral blood eosinophils decreased significantly after imatinib treatment.

The patient was re-examined in the Second Affiliated Hospital, Jiangxi Medical College, Nanchang University after a month; the number of peripheral blood eosinophils

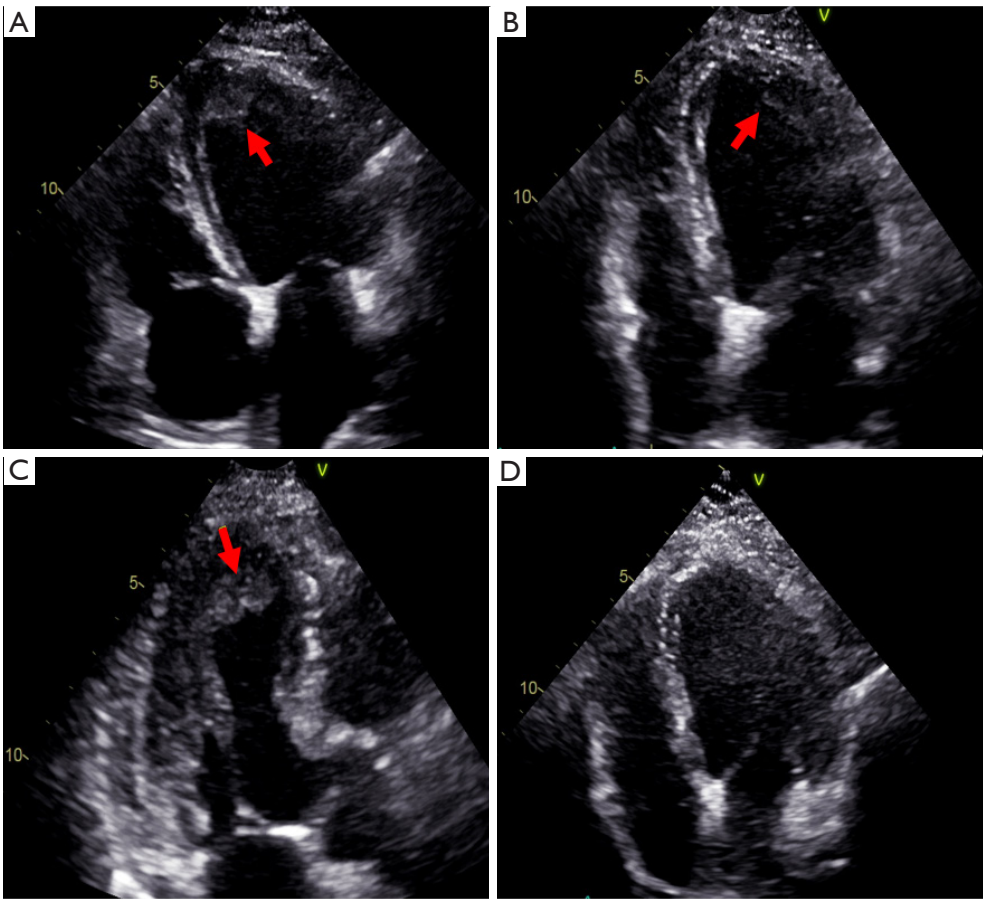

Figure 4 Echocardiography images after imatinib treatment. (A) Apical 4-chamber view after 1 month of treatment, showed that the left ventricular thrombus (arrow) was slightly smaller than before. (B) Apical 4-chamber view after 6 months of treatment, showed a thrombus (arrow) at the apex of the left ventricular lateral wall, significantly smaller than previous one. (C) 3-chamber cardiac view after 6 months of treatment, showed local thickening of the endocardium (arrow) at the apex and significant reduction of thrombosis. (D) Apical 4-chamber view after 1 year of treatment, showing that the thrombus at the apex has completely disappeared.

was roughly normalized [eosinophils 3.40%, normal range, 0–8%; absolute eosinophil count of 0.10×10^9/L, normal range, $(0–0.5)\times10^9$/L], and the echocardiography showed that LVEF was 69% and the left ventricular thrombus was smaller than before, with a range of 20 mm × 15 mm × 12 mm (*Figure 4A*). The patient was re-examined 6 months later; the peripheral blood eosinophils were still normal [eosinophils 1.50%, normal range, 0–8%; absolute eosinophil count of 0.09×10^9/L, normal range, $(0–0.5)\times10^9$/L], and the echocardiography revealed that LVEF was 70% and the thrombus was significantly reduced compared with the previous one, with a size of approximately 15 mm × 5 mm × 5 mm, and the patient's condition was stable (*Figure 4B,4C*). The patient was re-examined 1 year later: echocardiography showed complete resolution of the thrombus (*Figure 4D*) and eosinophils were within normal limits [eosinophils 2.60%, normal range, 0–8%; absolute eosinophil count of 0.15×10^9/L, normal range, $(0–0.5)\times10^9$/L]. At the same time, the patient discontinued anticoagulation. The patient underwent CT re-examination after 1 month of treatment, but there was no obvious improvement in the cerebral infarction. The patient's cerebral infarction was deemed an irreversible injury.

To evaluate the myocardial injury of the patient, the results of 3 hospital admission echocardiography examinations were imported into the GE Echo PAC 204 workstation (GE Healthcare, Chicago, IL, USA) for offline analysis. The stratified strain technique was used to evaluate

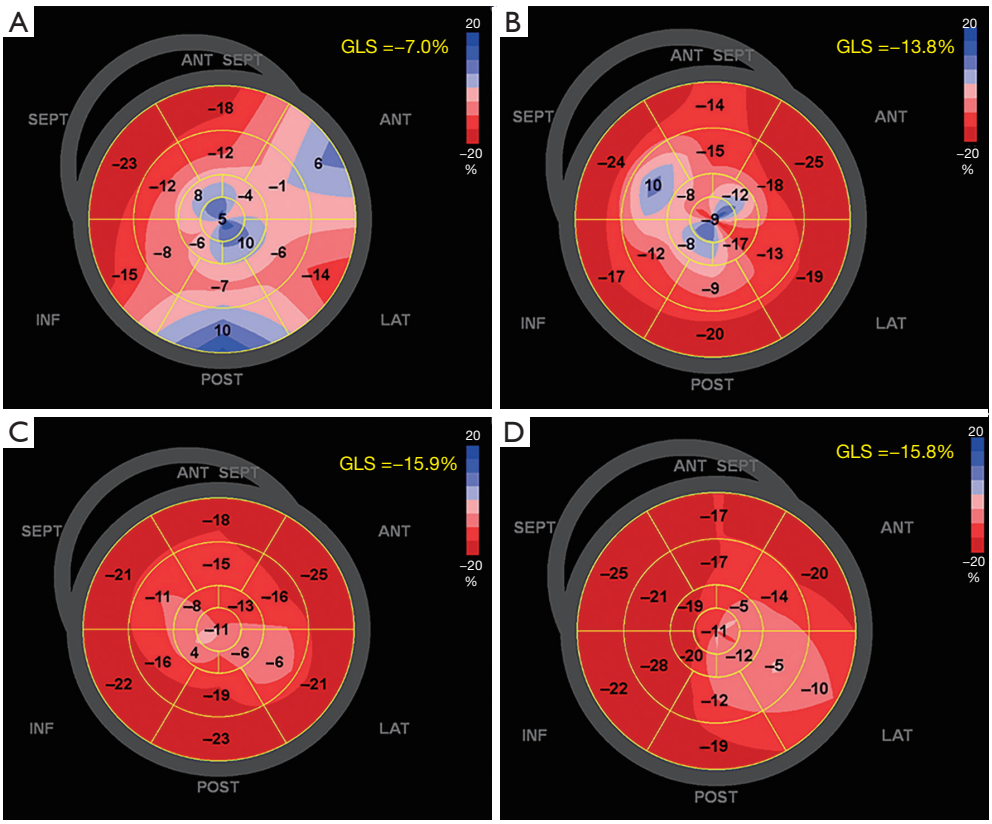

Figure 5 Bull's-eye diagram of stratified strain technique. (A) The GLS of the subendocardial myocardium at first admission was −7.0%. (B) The GLS of the subendocardial myocardium was −13.8% after 1 month of imatinib treatment. (C) The GLS of the subendocardial myocardium was −15.9% after 6 months of imatinib treatment. (D) The GLS of the subendocardial myocardium was −15.8% after 1 year of imatinib treatment. GLS, global longitudinal strain; ANT_SEPT, anteroseptal; INF, inferior; LAT, lateral; POST, posterior.

Table 1 The average longitudinal strain of subendocardial, middle, and subepicardial myocardium before and after treatment

Time	GLS-endo	GLS-mid	GLS-epi
First admission	7.7%	6.6%	5.8%
1 month after treatment	13.2%	13.0%	11.2%
6 months after treatment	17.7%	15.1%	13.0%
1 year after treatment	19.9%	16.9%	14.6%

GLS-epi, global longitudinal strain of epi-myocardium; GLS-endo, global longitudinal strain of endo-myocardium; GLS-mid, global longitudinal strain of mid-myocardium.

the endocardial strain, and the myocardial damage before and after treatment was evaluated. The global longitudinal strain of the subendocardial myocardium before treatment, 1 month after treatment, 6 months after treatment, and 1 year after treatment were −7.0%, −13.8%, −15.9%, and −15.8% respectively (*Figure 5*). The results of patient stratified strain analysis are shown in *Table 1* and *Figure 6*.

Discussion

Eosinophilia is a rare hematologic disease characterized by an eosinophil count >1.5×10^9/L in peripheral blood and bone marrow, and multi-systemic eosinophilic infiltration, with a prevalence of about 0.035/100,000 (4). Eosinophilia is often accompanied by damage to multiple organs such as the heart, lungs, skin, gastrointestinal tract, and nervous system, among which the heart is the most common organ involved (about 40%), and is the main cause of death in patients (5,6). Varga *et al.* (2) reported a case of positive *FIP1L1-PDGFRA* myeloid leukemia with Loeffler endocarditis who was admitted to the hospital for congestive heart failure (CHF), where, despite significant improvement in hematological indices after treatment,

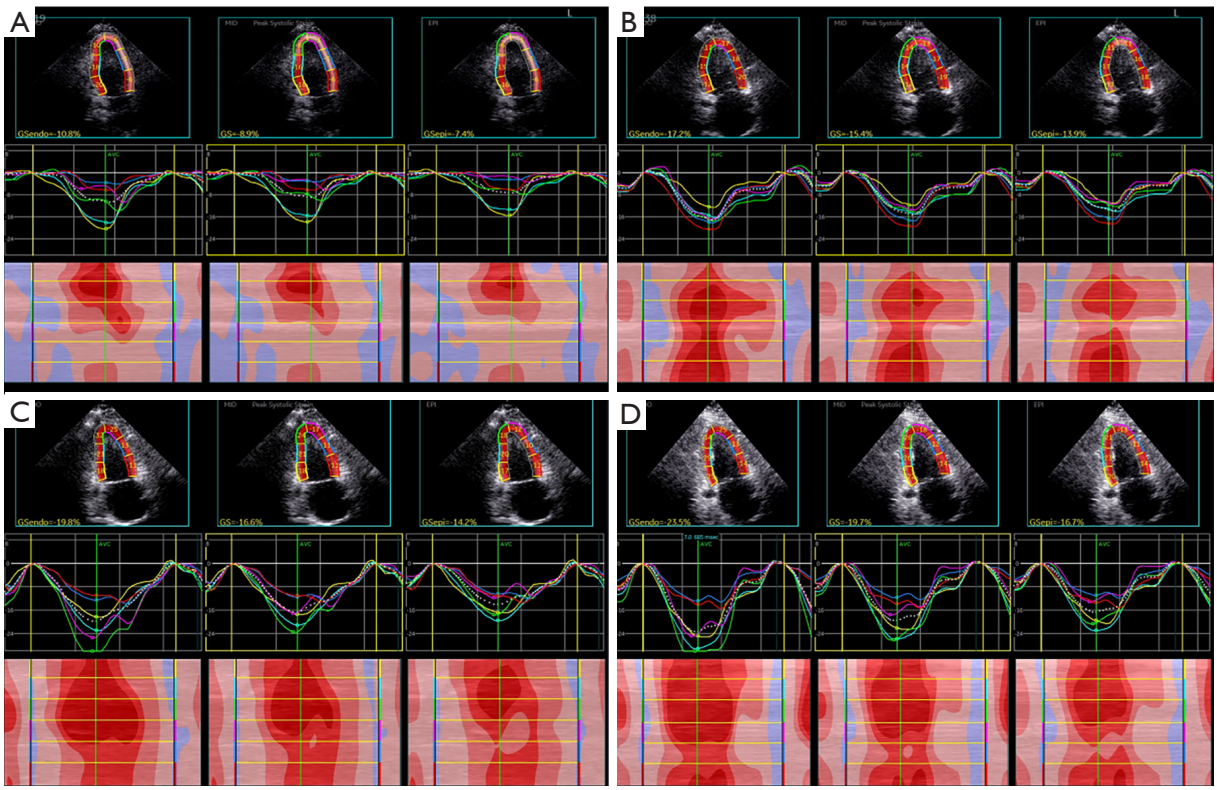

Figure 6 Longitudinal peak strain values and curves of subendocardial, middle, and subepicardial myocardium in the 2-chamber cardiac sections before and after treatment. (A) First admission; (B) 1 month after treatment; (C) 6 months after treatment; (D) 1 year after treatment.

cardiac impairment continued to progressively worsen and multiple complications (including embolization) eventually led to the patient's death. CHF is a serious complication in advanced Loeffler endocarditis. Therefore, early and accurate identification of the cause of heart failure is particularly important to ensure effective treatment.

Eosinophilia can be classified into 4 categories: hereditary, secondary, primary, and undetermined significance. Among them, secondary eosinophilia is a benign process, mainly caused by parasitic infection (7); primary eosinophilia is mostly a malignant process and has been redefined as CEL (8). CEL is a rare chronic myeloproliferative disorder caused by the clonal proliferation of eosinophil precursor cells. In patients with primary eosinophilia, there is a high probability of positivity for the *FIP1L1-PDGFRA* fusion gene (9). Mapelli *et al.* (10) reported a case of Loeffler endocarditis following coronavirus disease 2019 (COVID-19) infection, suggesting that COVID-19 infection, although rarely clinically symptomatic, may be a trigger for eosinophilic syndromes that were previously completely asymptomatic. Amelotti *et al.* (11) considered asthma a major clinical feature of eosinophilic granulomatosis with polyangiitis (EGPA), with more than 90% of patients reporting asthma. However, in the present case, the patient did not have significant asthma symptoms. Padhiyar *et al.* (12) reported a case of CEL characterized by oral and genital ulcers combined with *FIP1L1-PDGFRA* fusion, which also suggests that CEL could involve multiple systems, leading to a diversity and complexity of clinical presentations.

Loeffler endocarditis is a common cardiac complication of eosinophilia, which is usually accompanied by ventricular thrombosis. In addition, embolism can occur anywhere in the heart, chest, or lower extremities (13). Loeffler endocarditis is very rare in clinical practice, caused by eosinophilia involving the heart. First reported by Wilhelm Loeffler in 1936, it is more common in males. The pathogenesis is still unclear and may be related to direct damage of the endocardium by eosinophilic infiltration and degranulation of toxic substances, resulting in an

imbalance of coagulation–anticoagulation regulation (14). The pathological process can be divided into 3 stages: acute inflammatory necrosis, thrombotic, fibrotic, and eventually restrictive cardiomyopathy. Acute inflammatory necrosis usually occurs within 5 weeks and generally has no specific clinical manifestations due to lymphocyte and eosinophil infiltration of endocardial tissue, eosinophil release mediators, and cytotoxic molecules (e.g., basic protein, eotaxin, and peroxidase), resulting in endocardial and cardiomyocyte necrosis. The thrombotic phase lasts an average of 10 months and is caused by eosinophils releasing cytokines, increased thrombin production, and platelet-activating factor, leading to local arteriolar necrosis and mural thrombosis. Echocardiography is specific at this stage; when ventricular thrombosis is found without endocardial thickening, it needs to be differentiated from ventricular thrombosis due to ventricular aneurysm, myocardial insufficiency, and local myocardial damage. In additional, atrial fibrillation, valvular disease, dilated cardiomyopathy, and chronic heart failure may also be associated with intracardiac thrombus formation. When these conditions are excluded, the possibility of Loeffler endocarditis should be considered. Endocardial fibrosis is equivalent to the last stage and manifests as a classic restrictive cardiomyopathy. Echocardiography shows endocardial thickening, increased echogenicity, enlargement of both atrium, decreased ventricular diastolic function, limited valvular motion, and a restrictive filling pattern in the mitral spectrum doppler or, more rarely, dilated cardiomyopathy (15).

The clinical manifestations of Loeffler endocarditis are non-specific, and acute coronary syndromes such as chest tightness and chest pain may be present, making it highly prone to misdiagnosis of acute heart disease such as myocardial infarction, infective endocarditis, and acute heart failure (16,17).

The diagnosis of Loeffler endocarditis requires meet the following 2 conditions (4): (I) peripheral blood eosinophils $>1.5\times10^9$/L, lasting for more than 6 months; (II) signs of cardiac involvement, such as chest tightness, dyspnea, and other clinical manifestations, as well as echocardiograms, cardiac MRIs, and other imaging tests suggesting associated cardiac lesions, and so on. Loeffler endocarditis is difficult to diagnose in the early stages, and once diagnosed, it is usually accompanied by obvious cardiac involvement, and the condition is more severe.

This case is extremely rare in clinical practice. The patient had no specific clinical symptoms at the beginning of the disease, and the patient's first admission presented with typical symptoms of cerebral infarction. To determine the cause, a 2-dimensional (2D) echocardiogram was performed, which showed left ventricular thrombosis. In addition, combined with the patient's laboratory tests, it was found that the patient had a significant increase in eosinophils. Therefore, Loeffler endocarditis is initially suspected for an ultrasound investigation. In order to clarify the classification of eosinophilia, the patient actively underwent bone marrow biopsy, leukemia-related fusion gene testing, parasitic infection screening, and other examinations. It was finally found that the patient was positive for the *FIP1L1-PDGFRA* fusion gene, and combined with the patient's medical history and other relevant auxiliary examinations, the diagnosis of CEL with Loeffler endocarditis could be confirmed.

At present, the gold standard for the clinical diagnosis of Loeffler endocarditis is EMB (18). However, EMB examination is traumatic in nature, the patient had a history of cerebral infarction, and 2D echocardiography and contrast-enhanced ultrasound examination had found left ventricular thrombosis, indicating that the patient was in the thrombotic stage. EMB may cause thrombus detachment and cause multi-organ embolism, which is extremely risky and has great limitations in clinical application, so the patient did not undergo an EMB. When EMB cannot be performed, a multimodality approach (clinical and imaging) could guide diagnosis and therapy (19). Fortunately, echocardiography plays a very important role in diagnosing Loeffler endocarditis (20). The 2D echocardiography can intuitively and clearly show the size of the thrombus in the heart chamber, dynamically observe the changes of the thrombus, determine whether the valve is involved, whether it causes cardiac obstruction, and so on, and also evaluate cardiac function and hemodynamic changes. Long-term follow-up should be performed on such patients to monitor changes in blood clots in the heart chambers (3). Contrast-enhanced ultrasound can further define the boundaries of the thrombus and assess perfusion (21). In this case, the sonographic findings were typical, with significant thrombotic echogenicity; importantly, because rapid diagnosis and treatment are crucial to prevent potentially fatal progression or irreversible myocardial dysfunction, the utility of 2D echocardiography as a diagnostic tool should be framed in a more limited manner. In the absence of an EMB, 2D echocardiography is valuable in urgent cases where diagnosis and treatment must proceed swiftly.

It is well acknowledged that cardiac MRI is the gold standard for non-invasive assessment of heart disease (22).

Cardiac MRI is useful for diagnosing, stratifying, and monitoring Loeffler endocarditis without invasive procedures. Cardiac MRI can be used for morphological assessment using delayed enhancement sequences to comprehensively assess systolic and diastolic function, as well as tissue characterization, particularly myocardial fibrosis. As a result, it can identify specific stages of the disease and easily distinguish between inflammation and fibrosis. Caruso (23) reported a case of Loeffler endocarditis with fibrosis detected by cardiac MRI. Fibrosis with delayed enhancement on cardiac MRI typically presents with linear or patchy hyperintensity of the endocardium, frequently accompanied by an adjacent thrombus at the apex. Nevertheless, the patient in this case was in the thrombosis stage and had not progressed to the fibrosis stage, and the diagnosis was largely clear based on echocardiography and genetic testing for leukemia. In addition, since the clinical symptoms improved dramatically after imatinib treatment, the physician did not believe an extra cardiac MRI was necessary to save treatment costs.

The stratified strain technique can assess the myocardial strain of the epicardium, media, and endocardium and analyze the endocardial damage in patients with Loeffler endocarditis so as to determine the depth of myocardial injury (24). It is very valuable for follow-up of patients with myocardial injuries before and after treatment. The normal ventricular wall is composed of 3 layers of myocardial fibers with different structures and orientations. The endocardial and epicardial myocardium are longitudinal myocardium, which are mainly responsible for the long-axis function of the left ventricle; the middle myocardium is the annular myocardium, which is responsible for radial and circumferential functions. Different diseases can cause damage to different layers of the myocardium, resulting in different stratified strain situations (25). The results of stratified strain in this case showed that the degree of endocardial myocardial injury changed significantly before and after treatment, and the endocardial myocardial injury was severely damaged when the patient was first admitted to the hospital. The myocardial injury gradually improved after treatment and progressively returned to normal. This indicates that the degree of myocardial damage in Loeffler endocarditis is reversible. Therefore, echocardiography is essential for the early diagnosis of Loeffler endocarditis, which can help clinicians intervene early and prevent irreversible myocardial damage to a large extent. Furthermore, ischemic heart disease, valvular disease, and cardiomyopathy can also lead to impaired endocardial longitudinal strain, and echocardiography shows decreased LVEF and endocardial thickening. Once myocardial damage is found, it is often irreversible.

Currently, CEL is treated mostly depending on the existence of *FIP1L1-PDGFRA* fusion gene mutations, with imatinib being the most effective tyrosine kinase inhibitor (26). Imatinib has a high selectivity for oncogene kinase, can block its adenosine triphosphate (ATP) binding site, reduce its activity and proliferation, and has no effect on normal cell growth while exerting its efficacy and safety (27).

Conclusions

Loeffler endocarditis is currently difficult to diagnose early, and once detected, it is generally associated with severe cardiac involvement, a poor prognosis, and a high death rate; therefore, early detection and treatment are critical. Echocardiography and 2D speckle tracking echocardiography provide substantial advantages in the early detection and assessment of myocardial injury in Loeffler endocarditis.

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Footnote

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-24-1383/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was provided by the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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