Case Report

Loeffler's endocarditis with hypereosinophilic syndrome – a rare case study report

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ABSTRACT

This case report describes the clinical presentation of Loeffler's syndrome in a 50-year-old male non-diabetic and non-hypertensive who presented with complaints of pedal edema and breathlessness, signifying New York Heart Association III cardiac failure. There was mild itching, which was intermittent and insidious in onset. On general examination, there were pallor and bilateral pedal edema, which were pitting in nature. Differential leukocyte count showed 34.0% polymorphs and eosinophilia with an eosinophil count of 51%. Electrocardiography depicted low-voltage complexes. Chest X-ray showed cardiomegaly with increased computed tomography (CT) ratio >0.6 and blunting of costophrenic angles. 2D echocardiography showed apical obliteration and biatrial enlargement with increased left atrial (LA) volume. A restrictive filling pattern was observed on color Doppler, indicating grade III diastolic dysfunction with an E/A of 4.5 and E/E' of 14. Contrast-enhanced CT was suggestive of the presence of left ventricular thrombus. The diagnosis was Loeffler's endocarditis with hypereosinophilic syndrome with congestive cardiac failure.

Key words: Eosinophilia, Hypereosinophilic syndrome, Loeffler`s syndrome

Loeffler's endocarditis is a rare form of restrictive cardiomyopathy that occurs in association with hypereosinophilic syndromes [1,2]. Varied etiologies characterize it and have different clinical outcomes [3,4]. The hallmark is inflamed endomyocardial tissues and its infiltration with eosinophils, which might be attributed to hypersensitivity, rheumatic diseases, myeloproliferative diseases, malignancies, and even idiopathic [5]. It has three clinical stages: (i) The acute or necrotic stage, which shows eosinophilic infiltration with degranulation, inflamed cardiac tissues with necrosis; (ii) thrombotic stage has a mural thrombus, and (iii) fibrotic stage chiefly exhibits fibrosis and restrictive physiology [6-8].

In Loeffler's endocarditis (also known as an eosinophilic endomyocardial disease), pedal edema is one of the signs of right-sided heart failure, which occurs due to restrictive cardiomyopathy caused by endomyocardial fibrosis and eosinophilic infiltration. The present case came to the cardiology with pedal edema and breathlessness that were evaluated and diagnosed as Loeffler's endocarditis. The rationale for reporting this rare case of Loeffler's endocarditis is to raise suspicion in cases with signs suggestive of congestive cardiac failure with eosinophilia so that they can be diagnosed earlier before their deterioration or development of intraventricular thrombotic or fibrotic changes.

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CASE STUDY

A 50-year-old male, who is non-diabetic and non-hypertensive, came with complaints of pedal edema for the past 6 months and breathlessness for 2 months, signifying New York Heart Association III cardiac failure. The pedal edema was bilateral, with pitting edema around the ankles mostly in the evenings. It was chronic and progressive followed by breathlessness on slight physical activity. There were no complaints of orthopnea, paroxysmal nocturnal dyspnea, ascites and chest pain, syncope, giddiness, or fever. The patient was addicted to consuming alcohol regularly and also chewed tobacco for the past 10 years. There was no family history suggestive of heart failure, sudden cardiac death, or other heart conditions among other immediate family members.

On general examination, the patient was of normal build, was afebrile with a heart rate of 82/min, and regular and peripheral pulses were well felt. The blood pressure was 100/60 mmHg with signs of raised jugular venous pressure. The patient exhibited mild pallor and pedal edema. There was no cyanosis, clubbing, lymphadenopathy, hepatomegaly, or ascites. On inspection, there

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was a visible apical impulse in the left fifth intercostal space on the left side. Prominent pulsations were seen in the area overlying the left ventricular (LV) region. The apex beat was displaced slightly downward and outward, suggesting ventricular hypertrophy and possibly cardiomegaly. There were no thrills or parasternal heave. There was no hepatojugular reflex. There are no signs of cardiac dullness on percussion. Heart sounds were normal. No murmurs were heard, ruling out valvular lesions.

Peripheral smear depicted microcytosis with mild hypochromia as well as polychromasia and a few teardrop cells. Differential leukocyte count showed 34.0% polymorphs and eosinophilia (eosinophil count 51%). Bone marrow aspiration showed an increased number of eosinophilic precursors and hypocellular marrow with myelofibrosis and fibrosis in the ratio of 40:60.

The 12-lead electrocardiography (ECG) showed normal sinus rhythm, normal axis, and low limb voltage with T inversion in v2–v6 as shown in Fig. 1. The chest X-ray posteroanterior view showed signs of cardiomegaly with an increased CT ratio (>0.6). The blunting of costophrenic angles indicating plural effusion is depicted in Fig. 2. Two-dimensional echocardiography (2D ECHO) was performed using the parasternal long-axis view which showed an enlarged left atrium and obliteration of the LV apex (Fig. 3a). Similarly, the apical four-chamber view in 2D ECHO showed apical obliteration and biatrial enlargement with increased LA volume of 82 mL (Fig. 3b). A normal LA volume index is generally ≤28 mL/m^2. Larger values indicate potential enlargement and may be associated with increased risk of cardiovascular events. In this case, the LA volume was raised by more than twice the normal LA volume, suggestive of LA enlargement. Color Doppler also depicted the flow through an obliterated apex as shown in Fig. 4a. When the pulse wave across mitral valve inflow was observed, it showed restrictive filling pattern indicative of "diastolic dysfunction characterized under Grade III" showing an "E/A ratio" of 4.5 and an E/E' of 14 (Fig. 4b). These are the echocardiographic parameters for assessing diastolic function through evaluation of flow of blood into the left ventricle during diastole.

E/A is the ratio of the peak early diastolic filling velocity (E) to the peak atrial filling velocity (A). An E/A ratio >1.0, and especially >2, suggests possible diastolic dysfunction, where the heart does not fully relax and fill properly, whereas "E/e" is the ratio of the peak early diastolic filling velocity (E) to the mitral annulus early diastolic velocity (e'). An E/e' value of 14 indicates significant diastolic dysfunction and elevated LV filling pressures, which can be a sign of heart failure or other heart conditions. When the tissue Doppler across medial septal mitral annulus was performed, it was observed that the value for the flow across mitral valve was 6.19 cm/s and that through the lateral annulus was 8.8 cm/s as shown in Fig. 5. The normal velocity typically ranges from 70 to 130 cm/s. A lower velocity, especially in combination with other findings, could suggest impaired LV relaxation or diastolic dysfunction. The color Doppler flow

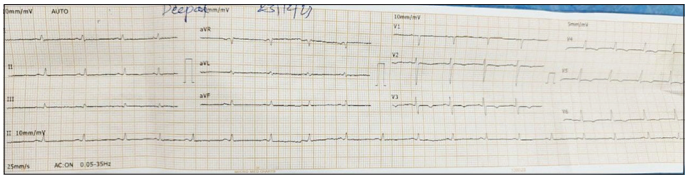

Figure 1: 12-lead electrocardiography showing low voltage complexes

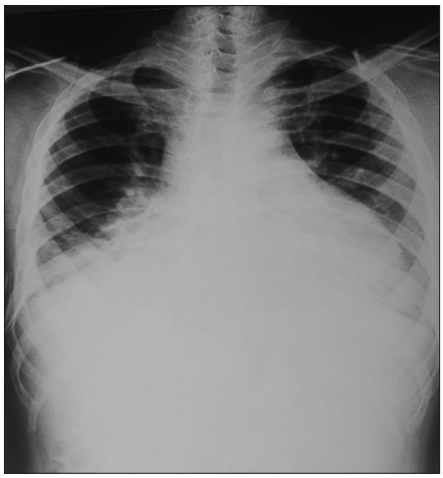

Figure 2: Chest X-ray posteroanterior view

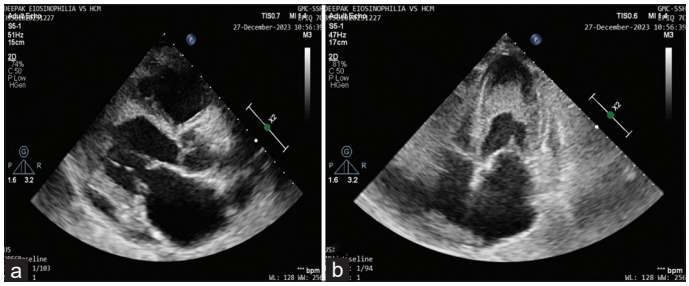

Figure 3: (a) Parasternal view in 2D echocardiography (ECHO) showing enlargement of left atrium and obliteration of left ventricular apex, (b) Apical four chamber view in 2D ECHO showing obliteration of apex and biatrial enlargement with an increased left atrial volume

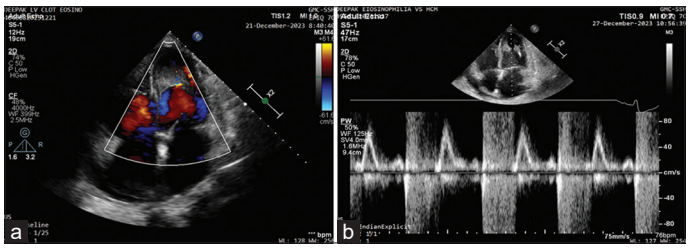

Figure 4: (a) Color Doppler showing flow through the obliterated apex, (b) Pulse waveforms across the mitral valve

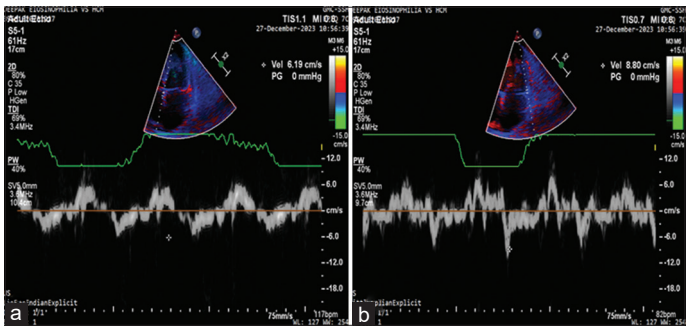

Figure 5: (a and b) Color Doppler flow velocity across the mitral valve

velocity across the tricuspid valve was 3 m/s as shown in Fig. 5. Peak tricuspid regurgitation velocity of ≤2.8 m/s is normal. This confirms tricuspid regurgitation, suggesting a high-pressure gradient. The inferior vena cava (IVC) was congested and showed non-collapsing with respiration. During respiration, a congested, non-collapsing IVC suggests elevated right atrial pressure or fluid overload. Contrast-enhanced computed tomography (CECT) was done to differentiate hypertrophied myocardium from an intraventricular thrombus. A hypodense area was observed within the LV apex, suggestive of the presence of thrombus (Fig. 6). Thrombus indicates a severe complication of the disease, characterized by the formation of a blood clot within the left ventricle. This can lead to serious complications such as embolism and heart failure.

After diagnosis as myelofibrosis, the patient was started on prednisolone 60 mg daily after which the eosinophil count decreased. For etiological diagnosis, molecular markers such as platelet-derived growth factor receptor (PDGFR)-alpha, PDGFR-beta, fibroblast growth factor receptor, and bone marrow biopsy with immunohistochemistry were required. However, these investigations were not available at our institute. Therefore, the patient was referred to the National Cancer Institute for further management. Later, he was lost to follow-up as he did not return for the next scheduled follow-up visit.

DISCUSSION

Loeffler's endocarditis is a rare condition with a range of clinical manifestations. In this case study, the patient presented with pedal edema and breathlessness. The differential diagnoses include an apical variant of hypertrophic cardiomyopathy (Yamaguchi disease) and endomyocardial fibrosis. On conducting laboratory and imaging tests, Loeffler's endocarditis was the presumptive diagnosis. Despite the non-prominent ECG changes, the diagnosis was confirmed by imaging modalities and blood investigations showing massive eosinophilia. Immunosuppressive therapy was given using corticosteroids and also mycophenolate and was started to reduce the eosinophil count. The diagnosis being made earlier in the thrombotic stage, the prognosis was good with proper treatment, and the condition was salvageable.

The underlying mechanism for the development of Loeffler's endocarditis, which leads to congestive cardiac failure, is the presence of endocardial fibrosis, which limits the diastolic filling [9,10]. Eosinophilic infiltration damages the myocardium, leading to both left and right ventricular dysfunction, elevated filling pressures, and further development of congestive symptoms [11].

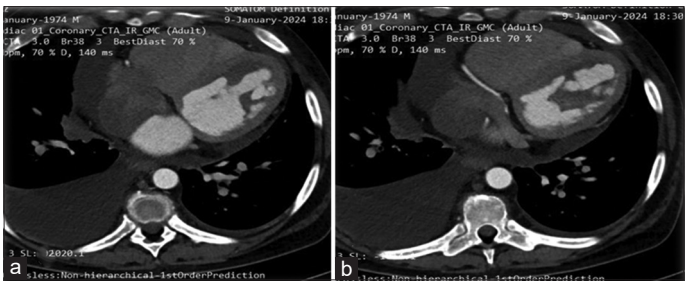

Figure 6: (a and b) Contrast-enhanced computed tomography showing an intraventricular thrombus

Loeffler's endocarditis poses diagnostic difficulties due to non-specific clinical features, mimicking other cardiac as well as non-cardiac conditions. Therefore, the utilization of imaging modalities such as CECT, chest magnetic resonance imaging, and transtracheal endoscopy (TTE), along with histological verification by an endomyocardial biopsy (EMB) aids the classification and diagnosis of Loeffler's endocarditis [12]. In addition, strain analysis also showed a reduction in apical values, pointing toward "inverse apical sparing" [13]. This is commonly seen in cases of apical hypertrophic cardiomyopathy. Myocardial strain patterns along with TTE, particularly contrast-enhanced TTE, are effective in detecting intracavitary thrombi [14] and differentiating Loeffler's endocarditis from other forms of apical hypertrophic cardiomyopathies and LV non-compaction [15].

Advanced disease stages demonstrate cardiac physiology akin to restrictive cardiomyopathy [11]. Pericardial involvement causes complex interplay ranging from restrictive to constrictive physiology. Therefore, parameters such as pulmonary, hepatic, mitral venous flow, Doppler indices, chest MRI, and sometimes minimally invasive procedures like right heart catheterization can delineate predominant physiological patterns. EMB showing eosinophilic infiltration and necrosis becomes the gold standard for diagnosis. Eosinophilia should raise clinical suspicion when cardiovascular symptoms are present [12]. Loeffler's endocarditis may however be seen independent of eosinophilia [15]. Thus, comprehensive assessments using a multimodal approach are effective when Loeffler's endocarditis is suspected.

Loeffler's endocarditis is managed according to the underlying etiology, stage, and severity. Immunosuppressive therapy is useful in idiopathic hypereosinophilic syndromes. Corticosteroids and immunosuppressants decrease eosinophil counts and also the inflammation. Thrombotic disease needs anticoagulation, while heart failure is to be treated in fibrotic Loeffler's endocarditis. Treatment response is determined by disease stage, with the acute necrotic stage having a better prognosis as compared to the thrombotic and fibrotic stages. Therefore, the essence of effective management is timely diagnosis using multimodal imaging.

CONCLUSION

Loeffler's endocarditis, a rare disease, has several etiologies as well as clinical presentations, characterized by myocardial infiltration of eosinophils with subsequent tissue damage. Multimodal imaging techniques and EMB for histological verification establish the diagnosis and stage of disease. Treatment depends on the underlying etiology, stage, and severity. If identified early, treatment with steroids can be initiated early. Once fibrosis occurs, management becomes more difficult; refractory cases – surgical intervention may be required. Patients with hypereosinophilia should be closely monitored for the development of thrombosis, fibrosis, and restrictive cardiomyopathy.

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