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CASE REPORT
Diseases of the heart

A case of Loeffler endocarditis with extensive right ventricular thrombosis (RCD code: III-3F.1)

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Abstract

Loeffler endocarditis is a rare cardiac disorder characterised by endomyocardial involvement of heart ventricles in patients with persistent eosinophilia The condition leads to restrictive cardiomyopathy and treatment-resistant heart failure and generally has a poor prognosis. Cardiac imaging and eosinophil count monitoring provide valuable information toward establishing the diagnosis. We present the case of a 32-year-old female patient with an abrupt onset of right-sided heart failure. The patient was diagnosed with myocardial infarction without coronary artery obstruction 2 years before the admission and was in stable condition with mid-range ejection fraction during follow-up. Her status deteriorated several days before admission with increasing dyspnoea and peripheral oedema. Echocardiography revealed severe left ventricular systolic dysfunction and normal right ventricular systolic function with a restrictive filling pattern due to massive thrombosis of its cavity, which was confirmed by cardiac computed tomography. Eosinophil count was elevated and the diagnosis of Loeffler endocarditis was established. Corticosteroids were administered together with conventional therapy for heart failure and anticoagulation. The right ventricular mass resolved gradually during 3–5 months and right ventricular filling normalised with concomitant improvement of the patient's clinical status The dosage of prednisolone was tapered over a period of 6 months, while the maintenance dose was continued for another 2 years. The patient died suddenly 2.5 years after the diagnosis of Loeffler endocarditis, with signs of acute dyspnoea prior to death. The autopsy showed mild right-sided endocardial fibrosis, extensive interstitial left ventricular fibrosis consistent with a diagnosis of chronic myocarditis of unspecified aetiology, and bronchospasm with eosinophilic material inside the bronchi. JRCD 2021; 4 (5): 115–121.

Key words: rare disease, hypereosinophilia, echocardiography, cardiac computed tomography

Introduction

Loeffier endocarditis (LE) can develop in the case of persistent eosinophilia of any cause. The endocardium of both ventricles is usually involved with the most frequent locations being the apical and inflow regions. Endocardial thickening and thrombosis are seen in the affected regions, with the late fibrotic stage of the disease leading to restrictive ventricular filling and refractory heart failure. Various cardiac imaging modalities are used in the diagnosis of LE in addition to laboratory examinations. Transthoracic echocardiography (TTE) reveals characteristic lesions in the heart ventricles, while cardiac magnetic resonance imaging (MRI) and computed tomography (CT) are used to confirm the diagnosis. Treatment with corticosteroids, which is recommended in early stages of the disease, is not effective in the late fibrotic stage, and 5-year mortality is high, with numbers reaching 30%.

Case report

A 32-year-old female was admitted to the hospital with symptoms of heart failure New York Heart Association (NYHA) class IV and signs of peripheral congestion. The patient reported that symptoms of congestion suddenly appeared several days prior to admission. Furthermore, 2 years earlier (at age 30 years) she was diagnosed with anterior ST-elevation myocardial infarction. The patient did not have any other risk factors for ischaemic heart disease such as hypertension, diabetes, or hyperlipidaemia. At the time, coronary angiography did not show any coronary stenosis or occlusion; however, a myocardial bridge of the left anterior descending artery (LAD) was revealed. The diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA) was established and the patient was treated with aspirin, clopidogrel, ramipril, and eplerenone. Left ventricular (LV)

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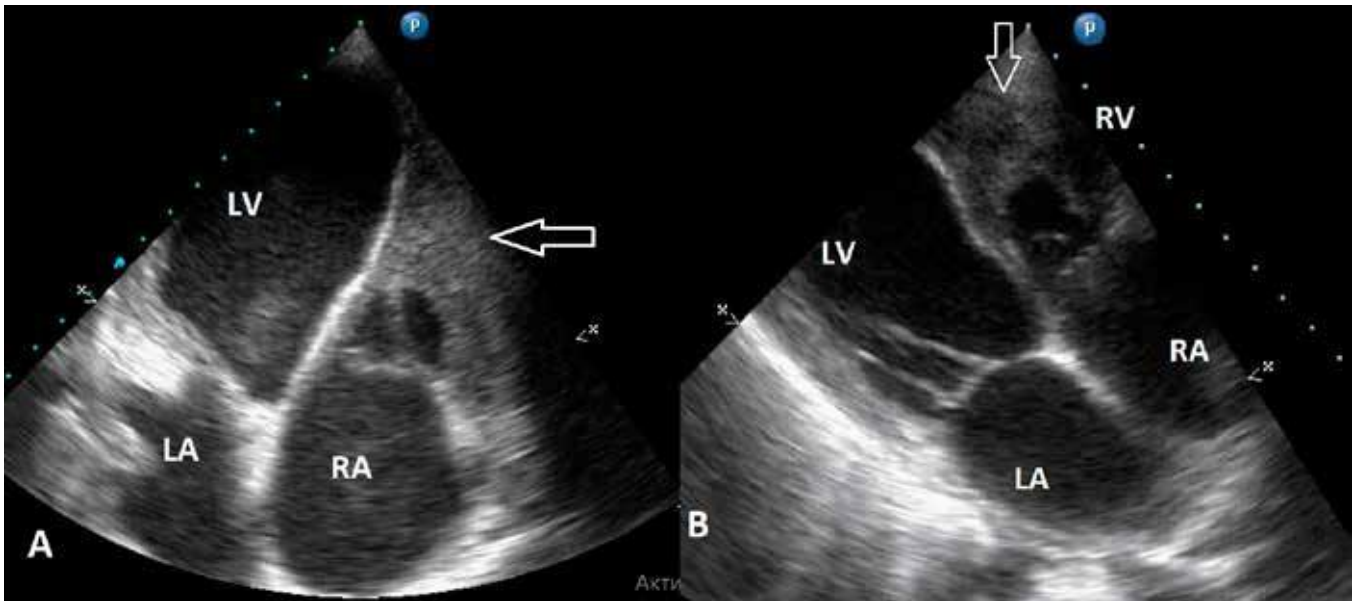

Figure 1. Echocardiographic four-chamber view (Panel A) and parasternal right ventricular inflow view (Panel B) demonstrate echogenic mass (suggestive of a thrombus) in the right ventricle obliterating its cavity (arrows). LV – left ventricle, LA – left atrium

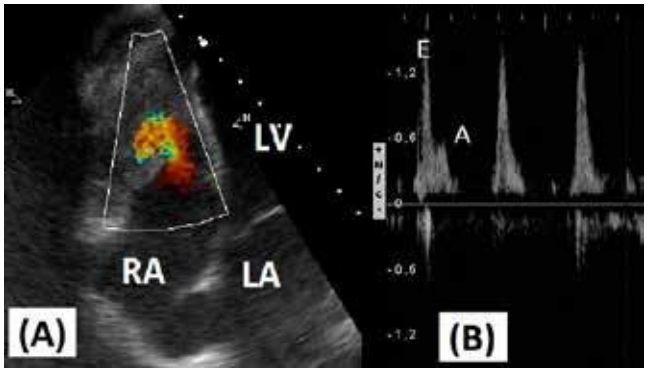

Figure 2. Panel A – Color Doppler shows turbulent flow in the right ventricle caused by the obliteration of its cavity and by high RV filling pressure. Panel B – Pulsed Doppler shows restrictive right ventricular filling pattern

systolic function was severely reduced during admission, but after treatment, ejection fraction (EF) increased to 45%. During the 2 years prior to the current admission, the patient had no symptoms of heart failure or angina pectoris, and had an EF of 45% during follow-up echocardiographic examinations.

Two months prior to the current admission, the patient complained of fatigue and cough and was treated with a short course of antibiotics and diuretics with rapid improvement of her condition. However, medical documentation regarding this episode was absent.

The abrupt onset of congestive heart failure occurred with no symptoms of angina pectoris. The patient was afebrile at the time of admission; however, her clinical condition was poor. Physical examination revealed signs of dyspnoea, orthopnoea, peripheral oedema, apical systolic murmur, tachycardia, and hepatomegaly. Chest X-ray revealed bilateral pleural effusions. Ultrasonography showed hepatic enlargement with ascites. Electrocardiography (ECG) showed QS waves in leads V_1-V_4 and signs of left bundle branch block without changes when compared to the previous ECG. Laboratory analysis: Haemoglobin was 120 g/l, white blood cell (WBC) count was 9 900/mm^3 with 11% eosinophils, 53% neutrophils, 30% lymphocytes, 6% monocytes, and sedimentation rate 8 mm/h. Results from biochemical analysis were within normal range. B-type Natriuretic Peptide level was not available. Transthoracic echocardiography showed anteroseptal LV akinesia and a diffuse decrease in LV contractility with EF of 25%. Moderate functional mitral regurgitation with an effective regurgitant orifice area (EROA) of 16 mm^2 was observed. The right ventricle (RV) was mildly dilated and contractility was normal (rather hyperkinetic). However, most of the RV cavity was occupied by an echogenic mass (Figure 1). Diastolic filling of the RV was severely diminished and pulsed Doppler showed a restrictive filling pattern with increased velocity of the peak E-wave (Figure 2, Panel A). Colour Doppler showed turbulent flow in the RV due to restricted filling (Figure 2, Panel B). Tricuspid regurgitation was moderate without signs of pulmonary hypertension and the right atrium was dilated. Additionally, the inferior vena cava was dilated with no inspiratory collapse. Thrombus or neoplasm of the RV was suspected, and chest CT was performed to specify the nature of the RV mass.

Cardiac CT with contrast revealed massive thrombosis of the RV and allowed determination of clot locations. Thrombi occupied more than two-thirds of the RV cavity, with the majority of deposits located near the apex and close to the tricuspid annulus (Figure 3).

Thrombus formation occurred in the setting of a well-contracting heart ventricle with typical localisation near the apex and valve annulus. Furthermore, eosinophil count at the time of admission was elevated. Taken together, Loeffier endocarditis was suspected.

Eosinophil count was monitored daily and comprised 17–19% of the WBC count during the following days. Maximal absolute eosinophil count was 1 632/mm^3.

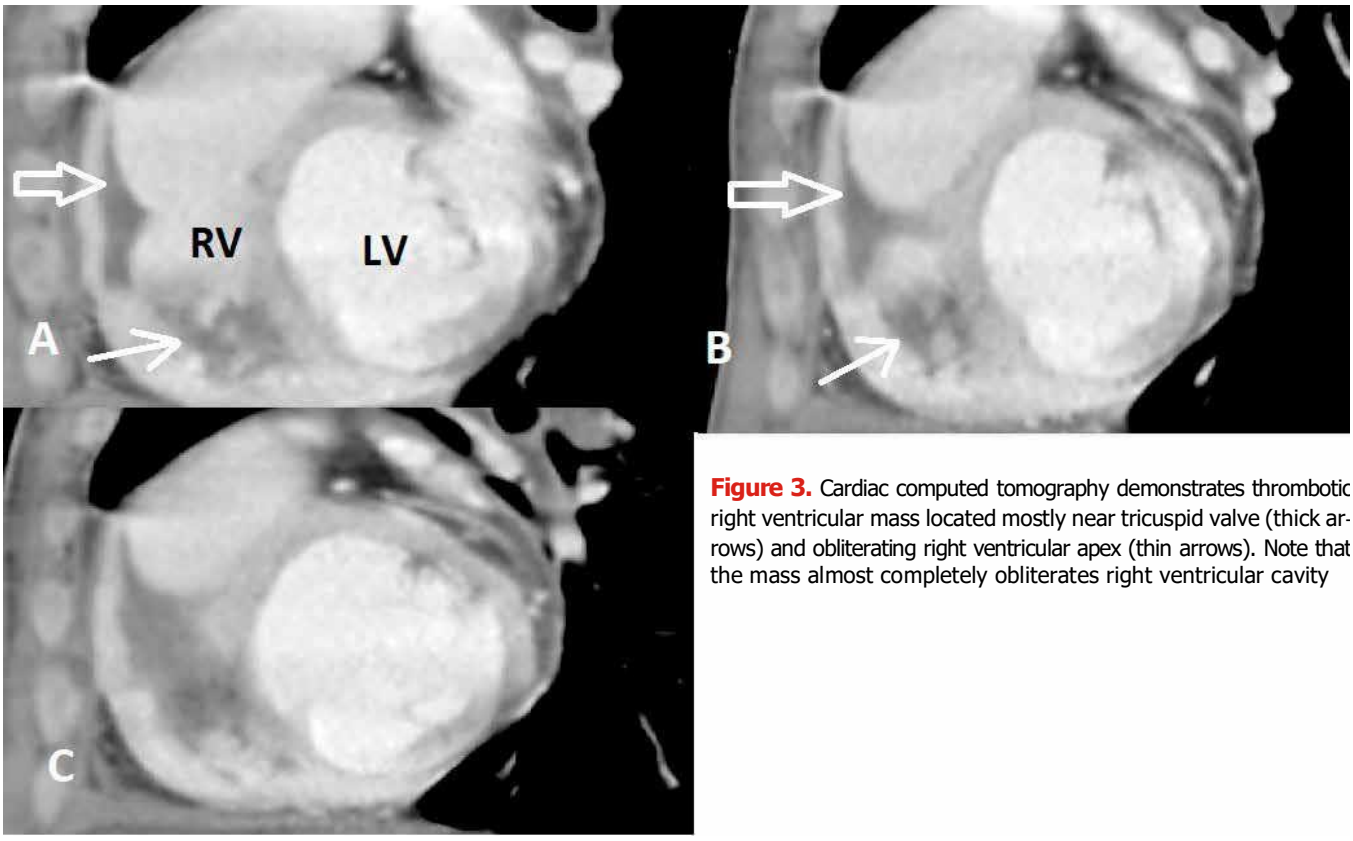

Figure 3. Cardiac computed tomography demonstrates thrombotic right ventricular mass located mostly near tricuspid valve (thick arrows) and obliterating right ventricular apex (thin arrows). Note that the mass almost completely obliterates right ventricular cavity

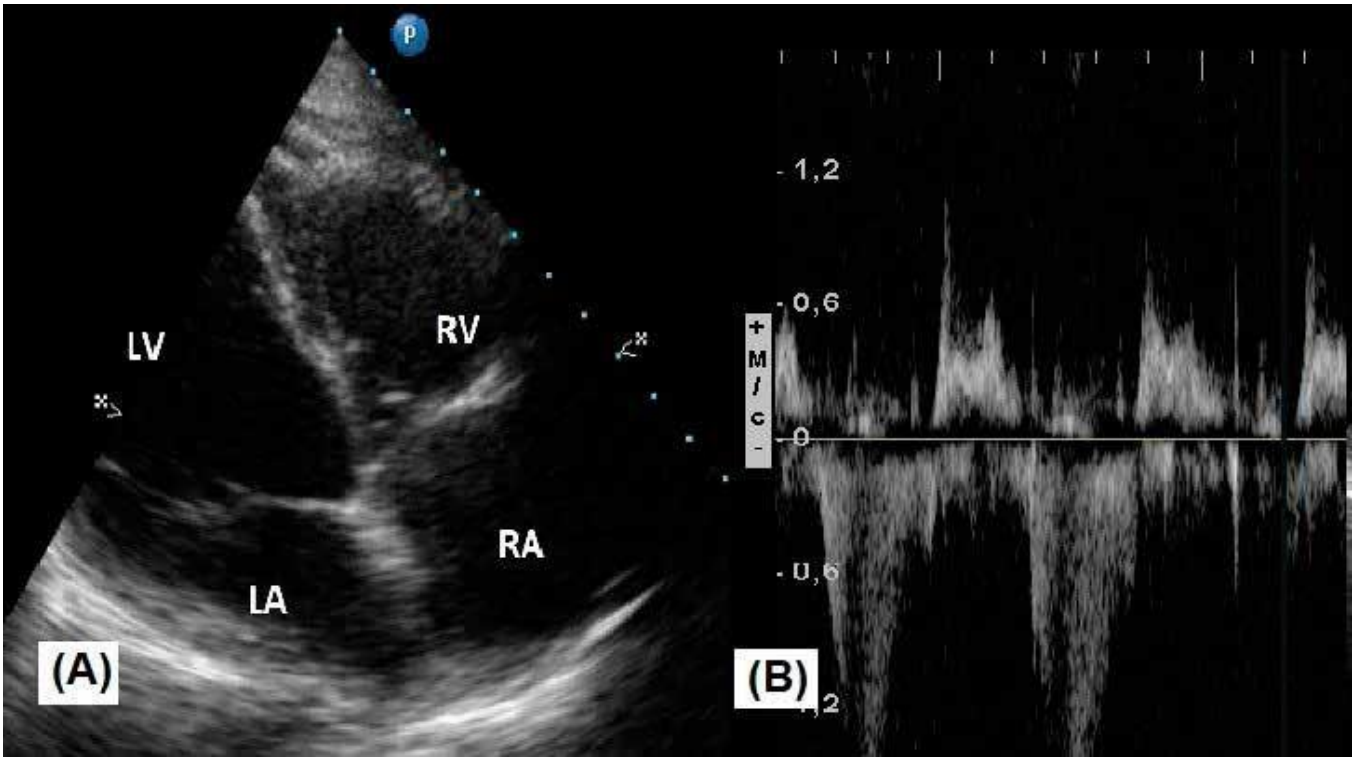

Figure 4. Echocardiographic four-chamber view after 3 month of treatment with corticosteroid. Panel A. Almost complete resolve of the thrombotic mass within the RV cavity. Panel B. Doppler-echocardiography shows the improvement of the right ventricular filling

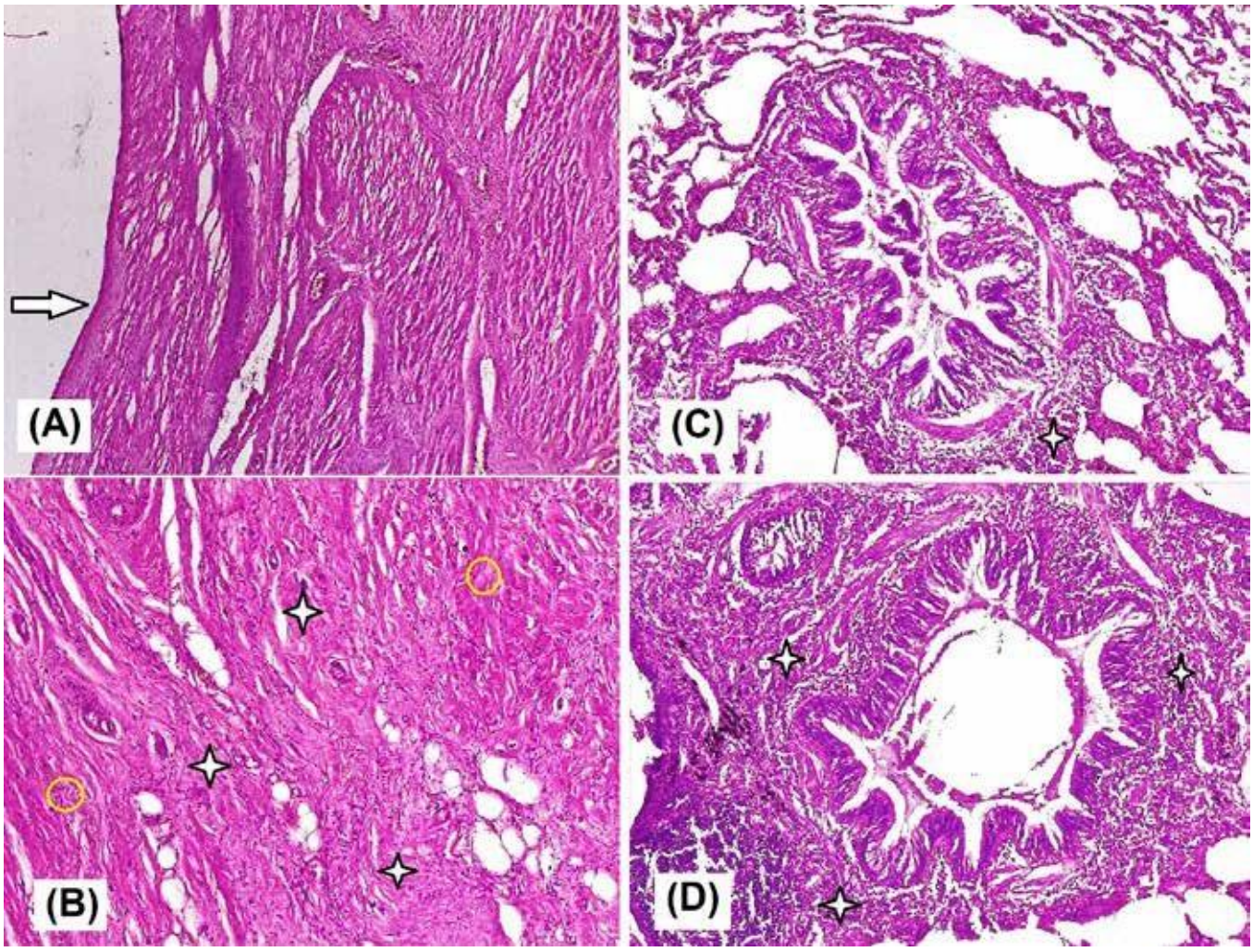

Figure 5. Pathologic examination of the obtained specimens. Panel A. Thin strip of endomyocardial fibrosis of the right ventricle (arrow), confirming the diagnosis of Loeffler endocarditis. Panel B. Extensive interstitial fibrosis of the LV myocardium. Astrerisks – interstitial fibrosis, yellow circles – cardiomyocytes. Panel C, D Specimens of lungs showed bronchial spasm with eozynophilic masses inside them. Asterisk – inflammatory perybronchial infiltrates

A diagnosis of Loeffier endocarditis was established based on imaging, laboratory, and clinical findings. Stool examination for parasites and serologic examination for Echinococcus and Toxocara were performed. Results of these tests were negative. Bone marrow biopsy revealed no signs of myeloproliferative disease. Thus, hypereosinophilic syndrome (HES) of unknown aetiology was suspected. Endomyocardial biopsy (EMB) was not available to confirm the diagnosis. On the fifth day of pharmacotherapy for heart failure (carvedilol, ramipril, eplerenone, torasemide, aspirin) and anticoagulation with heparin, the patient's clinical condition was still poor without any signs of improvement. As the patient's condition deteriorated, specific treatment could no longer be delayed. Moreover, despite receiving anticoagulation treatment, the right ventricular thrombus showed no regression on TTE.

Treatment with prednisolone 1 mg/kg (48 mg of methylprednisolone) was initiated. Anticoagulation with enoxaparin was discontinued and treatment with warfarin was initiated (titrated until INR of 3.00 was achieved). Eosinophil count decreased promptly after initiation of corticosteroid treatment. The clinical condition of the patient improved gradually, with regression of signs of congestion. After 14 days of corticosteroid therapy, follow-up TTE revealed a moderate reduction of the thrombotic masses in the RV chamber, with mild improvement in RV filling. The LV systolic function remained diminished with EF 30%. The pleural effusion was reduced. The patient was discharged from the hospital after 1 month of treatment on a tapering regimen of prednisolone with weekly monitoring of eosinophil count. Therapy with carvedilol, torasemide, eplerenone, ramipril, and warfarin with INR monitoring was continued.

The patient's status improved, her classification remained as NYHA II, and follow-up TTE showed a gradual reduction of RV thrombus size (significant regression 3 months after discharge and almost complete resolution 5 months after discharge) (Figure 4, Panel A). Diastolic RV filling normalised and turbulent flow in the RV disappeared when assessed via Colour Doppler (Figure 4, Panel B). Two years after the first manifestation of LE, the patient was receiving a maintenance dose of methylprednisolone 4 mg.

Any attempt to reduce the dose of corticosteroid was followed by an increase in eosinophil count.

Two and a half years after the initial diagnosis of LE, the patient was admitted to a local hospital for bronchitis and was reported to be in good condition after the course of treatment. However, one month later, she died suddenly with symptoms of acute dyspnoea. At autopsy, morphological examination of the obtained specimens showed a thin strip of RV endomyocardial fibrosis, confirming the diagnosis of Loeffier endocarditis (Figure 5, Panel A). No signs of post-infarction scarring were detected; however, extensive interstitial fibrosis of the LV myocardium with scant inflammatory infiltrates was revealed, consistent with the diagnosis of chronic myocarditis (Figure 5, Panel B). Examination of the lungs showed signs of bronchial spasm with eosinophilic masses inside them (Figure 5, Panel C, D). No signs of acute pulmonary oedema were revealed.

Discussion

Parietal thromboplastic Loeffier endocarditis is a rare cardiac disease with the hallmarks of hypereosinophilia, an absolute number of eosinophils above 1 500/ml, and cardiac manifestation with evidence of endocardial involvement [1, 2]. Usually, persistent eosinophilia with a duration of more than 6 months is considered to be a cause of LE [2]; however, its duration is often unknown. Cases of LE have been reported when the number of eosinophils in the blood varied significantly [3] or was within normal limits at the time of clinical manifestation [4, 5]. Loeffier Endocarditis leads to restrictive cardiomyopathy and heart failure due to endomyocardial fibrosis of one or both ventricles [2, 3, 4, 5, 13]. The disease carries with it a poor prognosis and can be fatal due to progressive and refractory heart failure or embolic complications [6]. A 5-year mortality of 30% has been reported in LE [7].

There are 3 stages of LE [2]. The first stage is necrotic, when inflammation and necrosis of the endocardium and myocardium of one or both ventricles occurs, especially in the apical regions and near the atrioventricular valves. The second stage is thrombotic, with thrombi formation on the surface of the affected endocardium. The thrombi can occupy almost the entire cavity of a heart chamber, leading to its obliteration and can lead to embolic complications [8]. The third stage of LE is fibrotic, and during this stage, endomyocardial fibrosis replaces the necrotic tissue and leads to the restrictive physiology of the affected ventricle [2, 3, 8]. There have been reports of the development of restrictive physiology in earlier stages without fibrosis as a result of subtotal ventricular obliteration by thrombi, as it was in our case [9, 10].

The endomyocardium of both ventricles is usually affected [2, 4, 5, 11], although isolated LV involvement is frequent [3, 8, 12, 13]. The mitral valve is affected infrequently [8, 9]. Isolated RV involvement in LE, as it was in our case, is extremely rare [10, 14, 15].

Diagnosis of LE remains a clinical challenge which demands a high level of awareness and knowledge of clinical signs and characteristic echocardiographic findings. Endomyocardial biopsy remains the gold standard for establishment of the final diagnosis; however, it is not available in all hospitals. Furthermore, some authors have reported cases in which EMB was uninformative or pseudo-negative [11]. A full clinical picture including laboratory analysis, echocardiographic examination, and confirmation via other imaging modalities should be taken together in the diagnostic process for LE.

Echocardiography is valuable in the thrombotic and fibrotic stages of LE with excellent sensitivity; however, it is less informative in the first stage (necrotic) of LE [2]. Apical thrombus in a ventricle with good contractility should raise suspicion for LE [2, 4, 5, 12]. The restrictive filling pattern is highly specific for the fibrotic stage, but it is seen in the early thrombotic stage as well due to obliteration of the affected chamber by thrombi [10]. Our case demonstrated a pattern of restrictive RV filling in the thrombotic LE stage followed by complete resolution of the thrombus with improvement of RV filling.

More sophisticated methods of cardiac imaging (contrast CT and MRI with gadolinium) are needed to confirm the diagnosis and evaluate the extent of endomyocardial damage and ventricular thrombosis. Cardiac CT enables differentiation of apical or valvular thrombi from tumour or vegetations [15]. In our case, cardiac CT revealed the presence of thrombus and enabled us to specify its location as apical and adjacent to the tricuspid valve, a finding which is highly specific for LE.

Cardiac MRI has an important role in LE diagnostics, as it enables identification of endocardial and subendocardial damage of the myocardium. Magnetic resonance imaging shows subendocardial late gadolinium enhancement zones due to inflammation (in early stages) and fibrosis (in late stages) [4, 10, 11, 13, 18, 19]. However, in critically ill patients, its usage could be limited due to the inability of the patient to hold their breath. In our case, cardiac MRI was unavailable.

Eosinophil count should be monitored in every case of suspected LE. A high eosinophil count has been reported in the majority of LE cases, ranging from 1 948/mm^3 [3] to 17 000/ mm^3[16], while proportions have been reported as approximately 10% [3,11] to 63% of leukocytes [15]. There have also been reports of eosinophilic myocarditis, confirmed by morphological examination, with a normal eosinophil count in peripheral blood [4, 5, 17]. The authors suggested that eosinophil count may change within a short period of time. In our patient, eosinophilia was moderate and promptly decreased after initiation of corticosteroid therapy. However, during methylprednisolone tapering, each attempt to decrease the dose below 4 mg was unsuccessful due to an increase in eosinophil count. Laboratory investigations for the causative agent should be considered in every case of hypereosinophilia, including tests for parasites, infectious and toxic agents, bone marrow biopsy for clonal eosinophilia, and allergic or autoimmune diseases.

Corticosteroids are the first-line of treatment for LE, usually at a dose of 1 mg/kg for prednisolone [2, 20]. Complete resolution of intracardiac thrombi after treatment with corticosteroids was previously reported in early-stage LE patients [9, 15]. Other recommended drugs include hydroxyurea, azathioprine, imatinib, and interferon-alpha [2]. Congestive heart failure should be treated with angiotensin-converting enzyme inhibitors, beta-blockers, and diuretics in accordance with current guidelines. Anticoagulation is needed when intracardiac thrombi are present. A delay in corticosteroid initiation in LE patients while awaiting results of examinations (ie. EMB) could lead to irreversible changes via endomyocar-

dial fibrosis formation [4, 5]. Additionally, this delay can be fatal due to the progression of heart failure [14]. Development of a rigid fibrotic endocardium is the cause of refractory heart failure due to a restrictive type of diastolic dysfunction [2, 5].

If the diagnosis of LE is established during the fibrotic stage, when massive endocardial fibrosis is already present, therapeutic options are limited. In these situations, treatment is via surgical endocardectomy (decortication of the fibrosed endocardium) [4, 5]. Therefore, imaging studies are crucial in the diagnostic process for LE, especially if EMB is unavailable.

The patient in our presented case had LE with isolated RV involvement, which is extremely rare. According to autopsy results, the patient was misdiagnosed with myocardial infarction (MI), as morphologic examination showed diffuse interstitial fibrosis consistent with chronic myocarditis. Aetiology of the chronic myocarditis could not be identified.

Cases of LE or myocarditis mimicking MI have previously been reported by other authors [16, 20–25]. In our patient, the significant decrease in LV systolic function during the second admission could have been due to LV involvement of the eosinophilic myocarditis process [25].

Our presented case demonstrates difficulties in the differential diagnosis for MI in a young patient without evidence of obstructive coronary artery disease. Myocarditis should be considered in every case, regardless of absent fever or flu symptoms prior to admission. The main limitation of our case was the absence of EMB and cardiac MRI, which were not available during the patient's hospitalisation. However, the diagnosis of LE in our patient was based on specific imaging findings (TTE and CT) together with moderate hypereosinophilia. Furthermore, the diagnosis of LE was evidenced by the effectiveness of steroid therapy and ultimately by autopsy findings.

Another limitation was the lack of consultation with a pulmonologist, who should be involved in the management of a patient with HES. The patient was consulted with a haematologist and an immunologist with an extensive search for the cause of eosinophilia. In addition, the significance of pulmonary symptoms (cough, dyspnoea, bronchitis) in the patient 2 months prior to LE manifestation was underestimated. Previous studies have reported lung involvement in 40–67% of HES cases, with the most common symptom being a persistent cough [10, 24, 26–29]. Patients with HES are often treated in pulmonology departments and bilateral infiltrates are a typical finding [24]. Pulmonary involvement could lead to bronchial hyperreactivity, and a high frequency of asthma (27%) is reported in HES patients [29]. Additional assessment of lung functions in a patient with HES should be repeatedly performed during follow-up, taking into consideration the risk of disease exacerbation.

Despite significant LV dysfunction with reduced ejection fraction in our patient, the main cause of death was not acute heart failure with pulmonary oedema, but instead, severe bronchospasm probably due to complications of HES. Extensive workup of the patient with pulmonologist and allergologist co-operation could have changed the patient's prognosis.

Conclusion

Loeffier endocarditis is a potentially fatal disease, which leads to refractory heart failure due to endomyocardial fibrosis and restrictive ventricular filling. Characteristic echocardiographic findings are valuable in the diagnostic process. Cardiac CT and MRI with contrast are imaging modalities which confirm the diagnosis, together with evidence of elevated eosinophil count. Prompt initiation of corticosteroid treatment could completely resolve the process. Furthermore, myocarditis should be considered in every case of suspected myocardial infarction without coronary artery obstruction in young patients. A multidisciplinary team of physicians should be involved in the management of a patient with hypereosinophilic syndrome.

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