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LETTER TO THE EDITORS

Progression of left ventricular thrombus in Loeffler's endocarditis without eosinophilia—case report and review of the literature

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Sirs:

A previously healthy, 41-year-old man was admitted to the Department of Cardiology for the evaluation of progressive exertional dyspnea, NYHA Class III, on his return from vacation. He denied other common cardiac symptoms such as angina pectoris, nocturnal dyspnea, syncope or palpitations. He was a smoker (15 pack years) and had a history of systemic hypertension, but no known allergies. He had normal vital signs and no abnormal clinical findings.

The complete blood count showed no abnormalities, including the absence of eosinophilia. C-reactive protein, creatinine, aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, gamma-glutamyl transferase, alkaline phosphatase and bilirubin levels were within normal limits. Troponin and nt-pro-BNP were also within normal limits.

The ECG showed ST segment depression in inferiolateral leads (II, III, V4-6). Contrast enhanced computed tomography ruled out pulmonary embolism, but revealed minor enlarged mediastinal lymph nodes and small pleural effusions. Community acquired atypical pneumonia was suspected and antimicrobial treatment started (Clarithromycin, Ceftriaxone).

Endobronchial and ultrasound-guided biopsy of the lymph nodes were unremarkable, and bronchoalveolar lavage revealed a borderline CD4/CD8 quotient of 0.4 (normal 0.5–1.5% in smokers). Pulmonary function tests showed minimal restriction.

Echocardiography demonstrated normal sized heart chambers with concentric remodeling (left ventricular mass index 99 g/m^2, relative wall thickness 0.42), and normal systolic function but restrictive filling pattern (*E* wave 1.28 cm, A wave 0.37 cm; *E*:*A* 3.3, doppler-derived E deceleration time 98 ms, *E*/*e*′ 11.2), mild left atrial enlargement 37 ml/m^2, normal sized right atrium of 20 ml/m^2) and elevated pulmonary artery systolic pressure of 41 mmHg. There was no sign of hypertensive heart disease. Furthermore, contrast-enhanced echocardiography showed that the endocardial surface of the right and left ventricular apex was covered by a 5–10 mm layer of thrombotic material corresponding to the typical picture of endocarditis parietalis fibroelastica Loeffler (Fig. 1a, b).

Cardiac magnetic resonance imaging (CMR) was performed using a 3Tesla CMR system (Achieva 3.0 T TX; Philips Healthcare; Best; the Netherlands). A T_2-weighted short-tau inversion-recovery (STIR) known as „triple inversion "spin echo-sequence depicted diffuse myocardial edema – preferable in the subendomyocardial regions (Fig. 2a). Myocardial perfusion analysis was performed using T_1-weighted fast field echo imaging sequences. Subendocardial non-perfused thrombotic layers were documented Fig. 2b, c. Late gadolinium enhancement (LGE) images 10 min after injection of contrast using a T1-weighted gradient echo pulse sequence with sensitive

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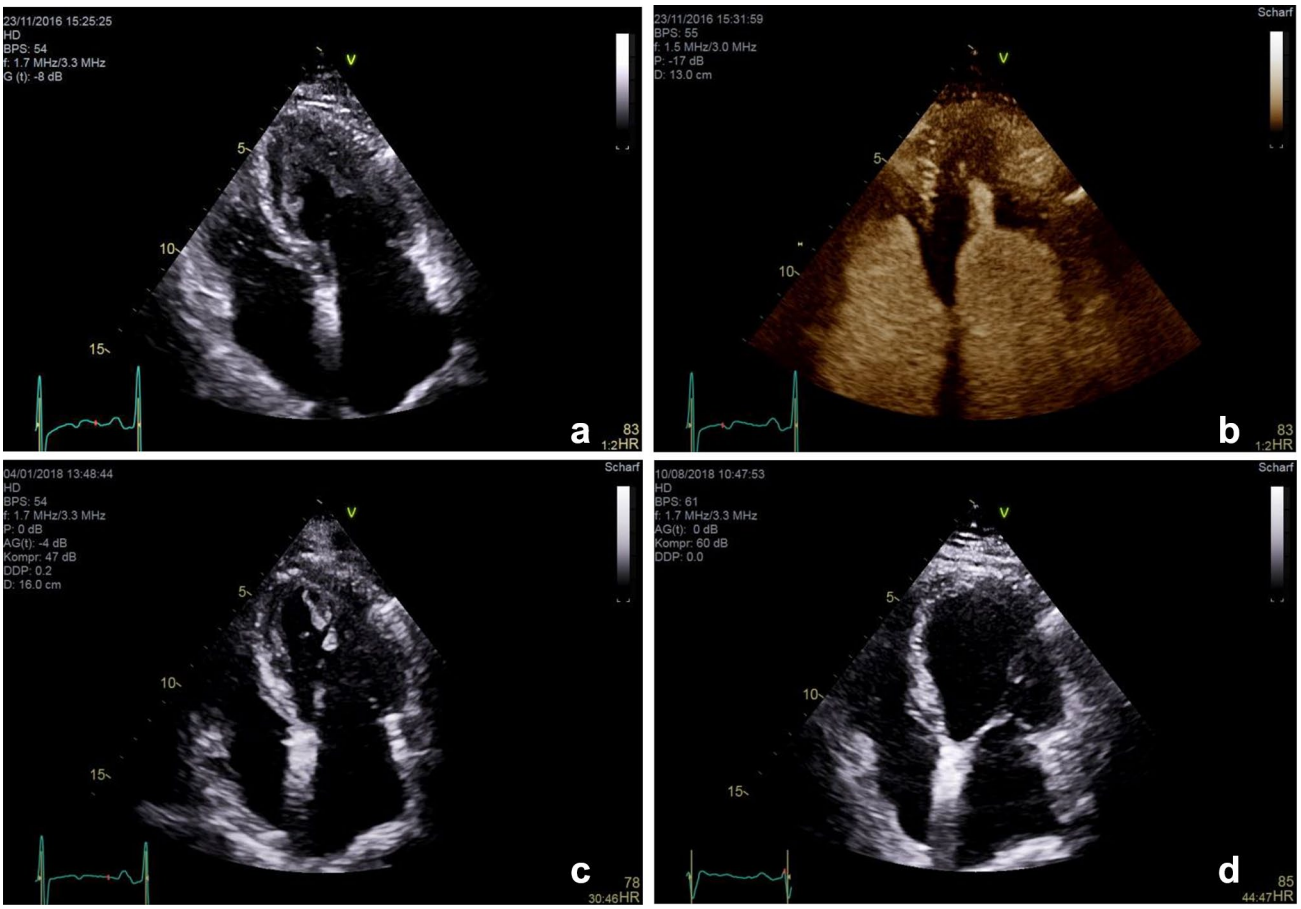


Fig. 1 Echocardiographic images. **a** Apical four-Chamber view showing biventricular apical thrombotic layer with dagger-shaped cavum (November 2016). **b** Apical four-Chamber contrast-contrast-enhanced view showing biventricular apical thrombotic layer with dagger-shaped cavum, contrast-enhanced (November 2016). **c** Apical four-Chamber view progressive thrombus growth with floating appearance (January 2018). **d** Apical four-Chamber after surgical removal of the fibrotic-thrombotic membrane (August 2018)

inversion recovery were used to document scars and fibrosis. A significant subendocardial layer of LGE could be visualized in the midapical left ventricular regions (Fig. 2d).

Right ventricular endomyocardial biopsy findings were suggestive of chronic myocardial damage with presence of adherent thrombotic material (T)—but without histological evidence of eosinophilic inflammation (Fig. 3a). The non-invasive imaging findings and histological picture were diagnostic of a late stage of Loeffler's endocarditis.

Additional investigations did not reveal any significant findings: The bone marrow smear and biopsy demonstrated normal cytology with no dysplasia or eosinophilia and a positron emission tomography displayed no abnormal findings. Furthermore, screening for antinuclear antibodies, anti-neutrophil cytoplasmic antibody, antiphospholipid syndrome, sarcoidosis, JAK2/BCR-ABL, platelet-derived growth factor receptor (PDGFRA)-mutations as well as parasitic infections was unremarkable. The above-mentioned investigations ruled out the secondary causes of hypereosinophilic syndromes (HES) [1], thereby, supporting the diagnosis of idiopathic HES with cardiac involvement.

Once a working diagnosis was established, the patient was symptomatically treated with torasemide, a beta-blocker, and an angiotensin receptor antagonist. Prednisolone (70 mg per day) was prescribed as a definitive line of therapy, in addition to Apixaban as antithrombotic therapy. The initial antithrombotic medication prescribed was Phenprocoumon (vitamine K antagonist), which was switched to Apixaban due the development of side effects (nausea, deranged liver function tests). Prednisolone was tapered to 5 mg per day within three months. The echocardiographic follow-up did not show complete resolution of the endocardial injury, but the disease remained stable. Thereafter, the patient was lost to follow-up for nearly one year and it was revealed only then at a follow-up visit that Prednisolone was stopped by his general practitioner after 10 months of treatment. Unfortunately, an echocardiography performed at this visit showed severe, progressive intraventricular growth of the thrombi Fig. 1c). The patient confirmed that he had taken Apixaban

daily. Due to the high risk of systemic embolization, the patient was scheduled for surgery a few days later. There was no evidence of peripheral eosinophilia in the complete blood count report. The patient underwent total excision of the left ventricular thrombus and fibrotic membrane/endocardium through the apex of the left ventricle without complications (Fig. 4). Histological examination of the excised specimen revealed thrombotic material (T) and fibrotic tissue (F) but no eosinophilic inflammation (Fig. 3b). Echocardiographic and CMR imaging performed during the 10-month follow-up period under treatment with Apixaban, but no prednisolone therapy, showed no signs of recurrence (Fig. 1d). The first CMR follow-up examination three months after the operation showed a mild non-significant left-ventricular myocardial edema, which was attributed to the local healing process following profound endomyocardial decortication during surgery. The second postoperative CMR examination nine months following surgery was unremarkable. All CMR studies as well as the contrast-enhanced echocardiograms did not reveal signs of thrombotic material. The restrictive left ventricular filling pattern normalized (*E* wave 1.0 cm, *A* wave 0.55 cm, *E*:*A* 1.8, *E* deceleration time 150 ms. Additionally, the indication for the use of postoperative steroid therapy was discussed. Current literature does not provide established pathways of therapy for patients with late stage Loeffler's endocarditis without evidence of local or systemic eosinophilia. Therefore, in the absence of adequate evidence in favor of postoperative steroid use and the potential for the development of side effects due to its immunosuppressive action, a decision to refrain from steroid use and to monitor the patient closely with CMR imaging, contrast-enhanced echocardiography and blood counts was taken.

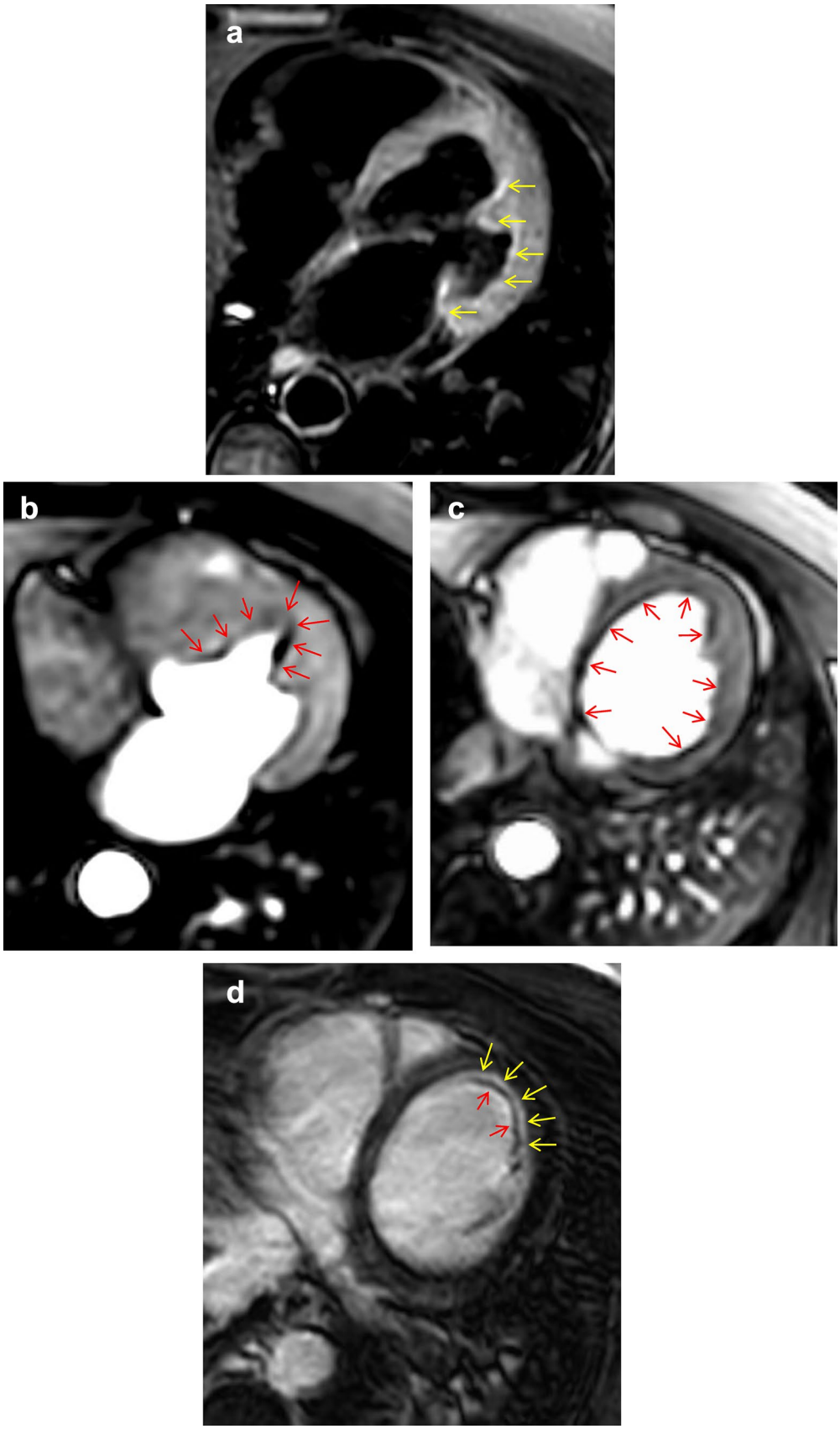
Definition Endocarditis parietalis fibroelastica Loeffler is a rare disease. Its etiology is still not completely understood. The primary hallmark is the hypereosinophilia (HE) defined as a eosinophil count $> 1.5 \times 10^9$/L on two tests ≥ 1 month apart and/or tissue HE (based on eosinophils on bone marrow biopsy or presence of eosinophilic granule proteins in tissue) [1]. HES is diagnosed by hypereosinophilia and organ damage/dysfunction directly due to HE. Eosinophils can infiltrate organs via toxic protein production. The cardiac involvement is common, occurring in up to 50% of the patients with HES [2], especially when HE lasts for months [3, 4].

History and Pathology In 1896, Reinbach described a female patient with eosinophilia and severe endocardial thickening [5], but it was Wilhelm L. Loeffler who coined the term „Endocarditis parietalis fibroplastica "in 1936 [6]. Histopathologically, three stages can be differentiated: (1) Initial stage with acute necrosis: eosinophilic endomyocarditis with eosinophilic and lymphocytic infiltration and necrosis and thrombus formation (duration approx. 5–6 weeks). Microemboli and development of a fulminant course are rare. (2) Thrombotic stage with planar endocardial thrombus formation on the base of eosinophilic infiltration and the beginning of fibrosis (duration approx. 10 months). Thrombi involve the apices of one or both ventricles, and can encroach on base of the heart into the subvalvular region. Embolic phenomena can occur. Isolated valve involvement is possible. (3) Final fibrotic stage: the repair process continues to replace inflamed myocardium with tough fibrotic tissue. The resultant scarring produces restrictive cardiomyopathy with signs and symptoms of left- and/or right-sided heart failure. Fibrosis may occur at the base of the heart and can lead to valvular regurgitation. Inflow tracts are commonly involved (duration approx. 24 months) [7, 8]. Pathophysiologically, inflammatory processes are caused by eosinophilic tissue infiltration [4, 9]. Blood eosinophilia usually corresponds to the disease's acute state, but local disease may occur without peripheral eosinophilia [10, 11]. Furthermore, the eosinophilic count can be completely normal at the time of presentation if in later stages [11], which may result in a diagnostic delay. Unfortunately, fibrosis may further progress and scarring may trigger ventricular arrhythmias [12].

Etiology HES can be either idiopathic, primary (neoplastic) or secondary (reactive) based on the etiology Table 1 [1].

Imaging and diagnosis Since cardiac involvement is common in up to 50% of patients with HE [14], echocardiographic screening for restrictive filling patterns and endocardial changes is recommended [4]. CMR is the imaging modality of choice to diagnose and follow-up Loeffler's endocarditis. CMR has higher tissue resolution and allows the assessment of subendocardial inflammation and fibrosis [12, 15, 16]. Endomyocardial biopsy (EMB) is mandatory to confirm the diagnosis and define the disease stage. The successful histological evidence of the subendocardial inflammation can be improved by CMR guidance [15]. If EMB is not available, treatment should not be delayed [4].

Treatment The treatment of Loeffler's endocarditis is mainly based on case reports and small case series. Since HES are a heterogeneous group of uncommon disorders, the treatment should be guided by the underlying etiology, the severity of the clinical manifestations and the side-effect profile of the agents used [17]. Therapeutic interventions for HES typically proceed in a stepwise fashion, beginning with immunosuppressive treatment with corticosteroids (CS) to reduce eosinophil count and counteract inflammation [18]. There is no consensus regarding the initial dose. Some reports advise initial high dose treatment (e.g. 1000 mg methylprednisolone i.v. Per day for 3–7 days) [19, 20], followed by an oral course with 0.5–1 mg/kg body weight per day. Since the underlying disease causing the eosinophilia is the primary target (e.g. anti-parasitic treatment, hematological treatment), we suggest an interdisciplinary approach to adjust the CS dose in those patients. Prednisolone 0.5–1 mg/kg body weight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◂**Fig. 2** MRI images (November 2016). **a** A T2-weighted short-tau inversion-recovery (STIR) known as „triple inversion "spin echo-sequence depicted diffuse myocardial edema (arrows)—preferable in the lateral, subendomyocardial regions. **b**, **c** Myocardial perfusion analysis using T1-weighted fast field echo imaging sequences. Subendocardial non-perfused thrombotic layers were documented (red arrows). **d** A significant subendocardial layer (yellow arrows) of LGE could be visualized in the midapical left ventricular regions with dark superficial thrombus formation (red arrows).

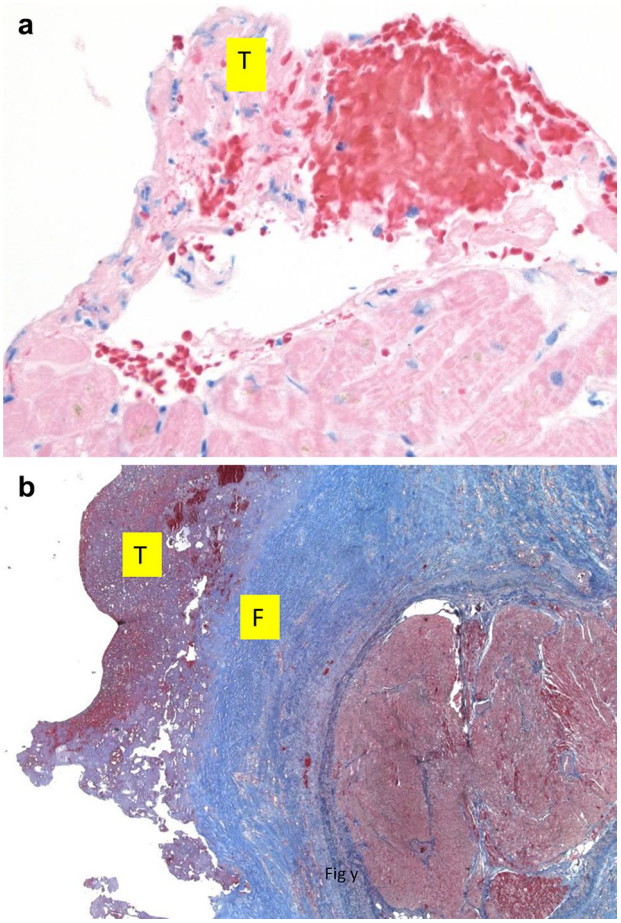


Fig. 3 Histopathological Images. **a** Histological findings in the initial endomyocardial biopsy (November 2016) with thrombotic material (T) but no eosinophilic inflammation (Giemsa stain × 400). **b** Histology of the operatively excised specimen (January 2018) with thrombotic material (T) and a thick fibrotic layer (F) (Masson Trichrome stain × 25)

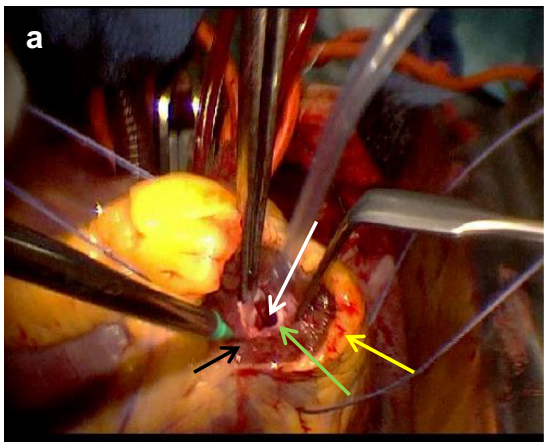
remains the standard in patients with idiopathic HES, and should be started as soon as possible [21]. The CS dose is usually tapered within three months to 5–7.5 mg per day. Treatment options of primary and secondary HES should preferably be selected by a multi-disciplinary team. Especially PDGFRA-FIP1L1-mutations may be effectively treated by target-specific drugs (e.g. tyrosine kinase inhibitor Imatinib) [3, 22, 23]. Most patients will improve symptomatically within 2–3 months. Response to treatment is best assessed by echocardiography and CMR as well as the regression of the underlying disease (e.g. eosinophil count) [21]. Treatment success and outcomes related to cardiac involvement depend on the stage of disease, as repair processes can lead to irreversible fibrosis [24, 25]. In severe cases with congestive heart failure or progressive thrombus growth, total surgical resection of the thrombus and fibrotic membrane is an option [26]. Eosinophils and the deposition of eosinophilic granule may induce a local inflammatory fibrotic process [27]. This development of a pro-inflammatory endomyocardial membrane may contribute to a chronic process of remodeling involving necrosis, fibrosis as well as the activation of coagulation due to the inflammation-coagulation-crosstalk. The surgical removal of this membrane may aid to control the local inflammation.

For CS-non-responders, additional immunosuppressive treatment with azathioprine or cyclophosphamide as well as other cytotoxic agents (e.g. hydroxyurea and vincristine) or Interferon-α are available options [28, 29]. The overall treatment duration depends on the underlying disease, but at least 1–2 years should be warranted, because of the high risk of recurrence. Cardiac recurrence cannot be reliably excluded by the measurement of eosinophils alone; cardiac imaging is mandatory [29]. After prednisolone is tapered, the patient should be under surveillance for at least 5–10 years, since cases of late recurrence are known [30].

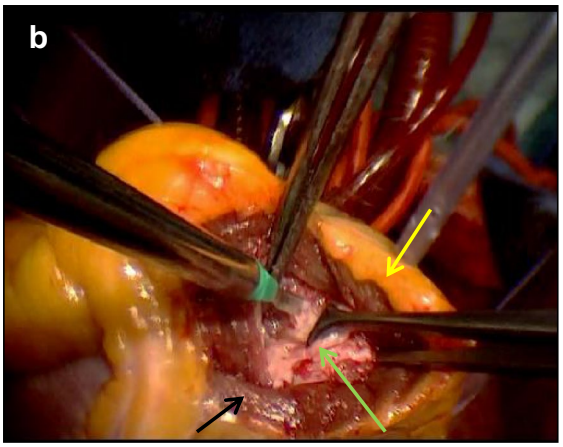
The role of antithrombotic treatment to reduce intraventricular thrombus growth remains undefined, since not all patients develop thrombotic complications. The used drugs are based on case reports. Most reports do not even state a specific antithrombotic medication. Heparin and vitamin K antagonists are the most common drugs. In our opinion, the occurrence of an embolic stroke strongly demands the use of antithrombotic therapy. Furthermore, anticoagulation may have a role in limiting thrombus development and adherence, and support the remodeling of the apical fibro-thrombotic mass [21]. The reported patient, unfortunately, developed side effects to the vitamin K antagonist, which explains the alternative treatment with Apixaban.

The overall prognosis for HES has improved, but limited data exists on the best course for cardiac disease. The two-year-mortality increases up to 50% in patients with heart failure and systemic embolization [26]. The prognosis, however, chiefly depends on the underlying disease.

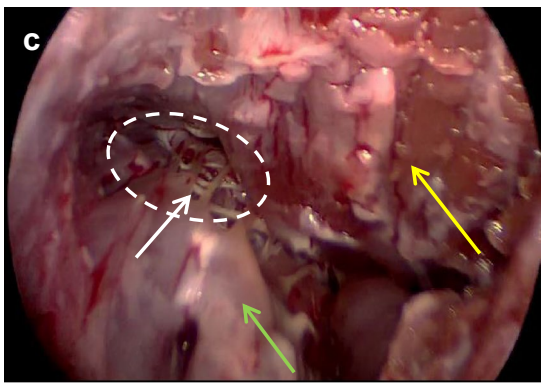
Loeffler's endocarditis is rare, but since cardiac involvement in hypereosinophilia is common, the disease is probably underdiagnosed. Besides echocardiography, CMR is the first-line imaging modality to detect endocardial damage. Biopsy remains important to define the stage of disease. As in this case presentation, eosinophilia is not



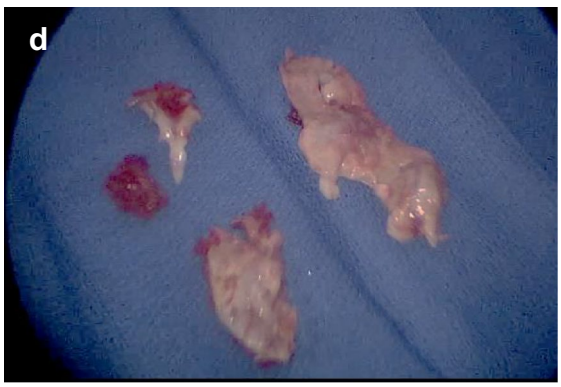
Yellow arrow: epicardial fat, Black arrow: myocardium, Green arrow: fibrotic layer covering the endocardium, White arrow: LV cavity



Yellow arrow: epicardial fat, Black arrow: myocardium, Green arrow: fibrotic layer covering the endocardium



Yellow arrow: raw endocardial surface, Green arrow: papillary muscle, White arrow: chordae tendinae, White ellipse: mitral orifice



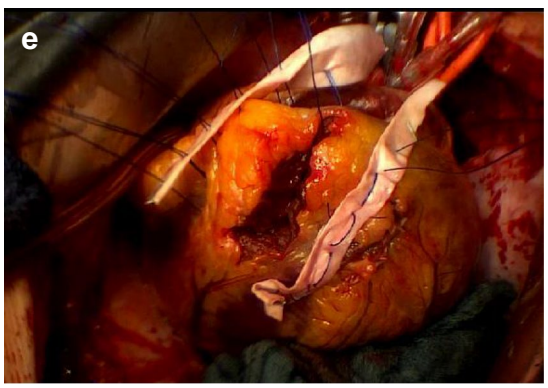


Fig. 4 Intraoperative Images (January 2018). **a** LV Apical incision. Yellow arrow: epicardial fat, Black arrow: myocardium, Green arrow: fibrotic layer covering the endocardium, White arrow: LV cavity. **b** Excision of the fibrotic layer. Yellow arrow: epicardial fat, Black arrow: myocardium, Green arrow: fibrotic layer covering the endocardium. **c** LV cavity following complete excision of the fibrotic layer. Yellow arrow: raw endocardial surface, Green arrow: papillary muscle, White arrow: chordae tendinae, White ellipse: mitral orifice. **d** Specimen of the excised fibrotic layer. **e** Closure of LV apex

Table 1 Etiologies of hypereosinophilic syndromes (mod. [1, 13])

Primary (neoplastic) HES	Secondary (reactive) HES
Stem cell, myeloid, or eosinophilic neoplasm: PDGFRA, PDGFRB, FGFR1	Production of eosinophilopoetic cytokines: vasculitis (e.g. eosinophilic granulomatosis with polyangiitis)
t-cell lymphoma	Parasitic infections (e.g. strongoloides)
t-cell lymphocytic variant	Adverse drug reactions (e.g. anticonvulsants, semi-synthetic penicillins, allopurinol)

FGFR1 fibroblast growth factor receptor 1, *HE* hypereosinophilia, *HES* hypereosinophilic syndrome, *PDGFRA* platelet-derived growth factor receptor α, *PDGFRB* platelet-derived growth factor receptor β

mandatory in late stages [11]. Treatment should not be delayed, and should be continued for at least 1–2 years. Late recurrence may occur warranting long-term follow-up.

Author contributions TK and MM wrote the first draft of the manuscript contributed equally to the paper. Prof. AH provided the echocardiographic and CMR images. PD provided the intraoperative images, made critical revisions and approved the final version. Prof. KK provided histological images. Prof. UL made suggestions for the structure and arguments for the paper, and made critical revisions. All authors reviewed and approved the final manuscript.

Compliance with ethical standards

Ethical approval As a requirement of publication, author(s) have provided to the publisher signed confirmation of compliance with legal and ethical obligations. The authors have read and confirmed their agreement according to the ICMJE authorship. The authors have confirmed that this article is not under consideration or published in any other publication.

Conflict of interest No conflict of interest of the corresponding author.

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