Figure 3. Right atrial-free wall abscess during atrial opening

ative TEE confirmed the eradication of the infective source in RA, and an arteriovenous fistula was embedded as a new HD access.

Discussion

Intravascular devices make the patients susceptible to frequent transient bacteremia, particularly by *S. aureus* as the most common causative microorganism of IE in ESRD patients (2). On the other hand, extension of device-related infections to adjacent cardiac tissues is another mechanism underlying IE and subsequent myocardial abscess in populations undergoing HD (3). However, despite the high prevalence of vascular access-related infection, right-sided valvular IE is still unusual in patients undergoing HD (2).

In the presence of valvular endocarditis, the perivalvular area is the expected site of associated abscess. The formation of a distant myocardial abscess is very rare (4). In the current case, despite TV endocarditis, two large-sized myocardial abscesses were extraordinarily located at the IVC entrance.

We believe that tricuspid IE and abscess formation in our patient was induced by direct inoculation from a septic foci established in RA by an infected intra-atrial catheter. Moreover, a possible initiating factor might be the damage of the RA wall endocardium owing to direct irritation of the catheter or jet stream caused by HD, which made the RA more vulnerable to thrombosis and infection.

Conclusion

Our case demonstrates that neither negative blood cultures nor normal TTE is sufficient to rule out IE or myocardial abscess in a highly suspected patient in the context of HD. As a matter of fact, TEE plays a crucial role in the improvement of patient outcomes by boosting early diagnosis and helping to determine an appropriate treatment plan, particularly for right-sided lesions (5, 6).

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Video 1. A large, hypermobile vegetation on the catheter tip (24×15 mm) during TEE.
Video 2. TEE view of two RA wall abscesses.

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Acute fulminant eosinophilic myocarditis due to *Giardia lamblia* infection presented with cardiogenic shock in a young patient

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Introduction

Acute eosinophilic myocarditis is a relatively rare condition that may be associated with various eosinophilic diseases, such as parasitic infection, allergies, drug hypersensitivity, granulomatous disease, connective tissue disease, vasculitis, or primary

hypereosinophilic syndrome (1). It is usually associated with peripheral eosinophilia, but it may rarely present with a normal peripheral eosinophilic count (2, 3). Herein, we describe a case of acute eosinophilic myocarditis due to *Giardia lamblia* presented with cardiogenic shock for a normal peripheral eosinophilic count upon admission, which was successfully treated with high-dose corticosteroids and metronidazole therapy.

Case Report

An 18-year-old male patient was admitted to our intensive coronary care unit with complaints of chest pain, fatigue, shortness of breath, and syncope. The patient's medical history revealed that he had been well until two days prior, which is when a fever, headache, and sore throat developed. The patient had no known allergies and he never drank alcohol, smoked, or used illicit drugs. He had not noted any insect bites and denied ingesting any raw meat, freshwater fish, or liver. On admission, the patient was tachypneic, and his body temperature was 38°C. On physical examination, the patient's blood pressure was measured to be 80/50 mm Hg; pulse rate, 103 bpm; and oxygen saturation, 87%: he was breathing ambient air, and it was increased to 95% through a nasal cannula. On auscultation, bilateral crackles were heard on the patient's lungs, and his heart was beating rapidly without murmurs. Electrocardiography revealed an ST-segment elevation in the DI and aVL leads with a reciprocal change in the DII, DIII, and aVF leads (Fig. 1). Laboratory analysis showed that the cardiac enzyme levels [creatine kinase myoglobin band and troponin, 10.5 g/mL (normal range: 0 to 2.8 ng/mL) and 2.23 ng/mL (normal range: 0 to 0.045 ng/mL), respectively], brain natriuretic peptide levels [2.824 pg/mL (normal range: 0 to 88 pg/mL)], and C-reactive protein levels [26.5 mg/dL (normal range: 0 to 8 mg/dL)] were elevated. A complete blood count revealed that the blood eosinophil count was within the normal range (480/μL) with marked leukocytosis (23.300/μL). Transthoracic echocardiography revealed a left ventricular ejection fraction of 20% with severely depressed left ventricle wall motion (Video 1a, 1b). After admission, the patient's general condition rapidly deteriorated: inotropic support with noradrenaline was started in response to the cardiogenic shock. An urgent coronary angiography was performed, providing a diffuse coronary spasm in the left anterior descending artery and circumflex artery (Video 2); however, nitroglycerin was not given because of the patient's low blood pressure. A right ventricular endomyocardial biopsy was performed in the acute phase, which showed extensive eosinophilic inflammatory cell infiltration, severe interstitial edema, and moderate myocardial necrosis. Infectious causes and giant cells were ruled out (Fig. 2a, 2b). After confirming the diagnosis for myocarditis, a high dose of corticosteroid was administered. Then, 1 g/day of IV methylprednisolone was initiated for 3 days, followed by 2 weeks of oral prednisone at 50 mg/day with a slow taper. An extensive work-up for the cause of eosinophilic myocarditis was performed, including antinucle-
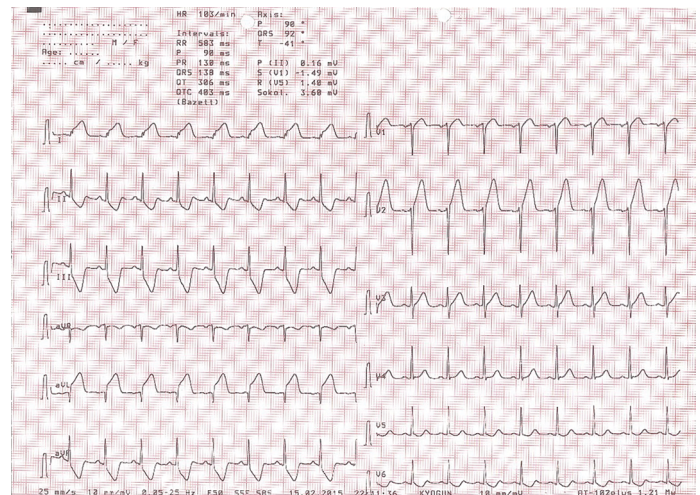

Figure 1. Electrocardiography revealed an ST elevation in the DI and aVL leads along with reciprocal changes in the DII, DIII, and aVF leads
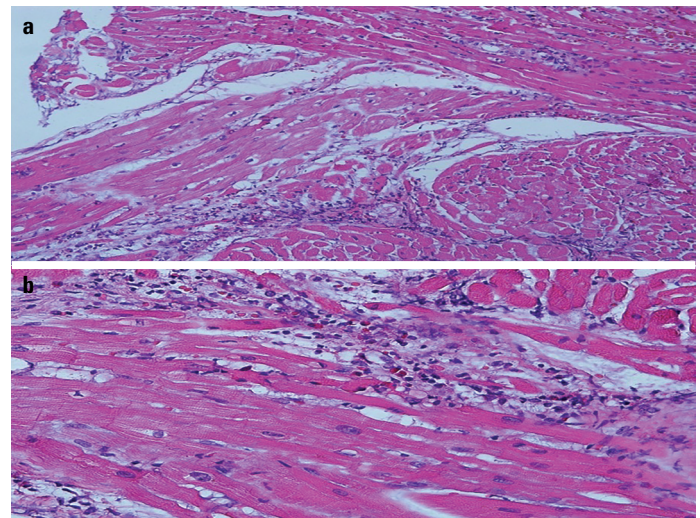
Figure 2. (a) There were signs of atrophy and nonspecific degenerative changes in the muscle fibers, interstitial inflammation, edema, and mild fibroplasia (H&E, x200). (b) Interstitial inflammation within the myocardium mainly comprised lymphocytes accompanied by eosinophilic leucocytes, as well as very few neutrophilic leukocytes (H&E, x400)

ar antibodies and anti-neutrophil cytoplasmic antibodies, all of which yielded negative results. The patient's stool studies and parasite serology for *Toxocara*, toxoplasmosis, and hydatid cysts were negative. Antigens for *Giardia lamblia* were detected in the feces using an immunoenzymatic test (ELISA *Giardia lamblia* antigen), and an examination of the stool revealed the presence of cysts and trophozoites. A diagnosis of eosinophilic myocarditis due to *Giardia lamblia* infection was then confirmed, and a 500 mg metronidazole treatment two times per day was started. After treatments with metronidazole and high-dose corticosteroid, the patient's control angiogram showed completely normal coronary arteries, and his left ventricular function dramatically improved (Video 3a, 3b, 3c). The patient was discharged with a completely normal electrocardiography result 28 days after admission (Fig. 3).
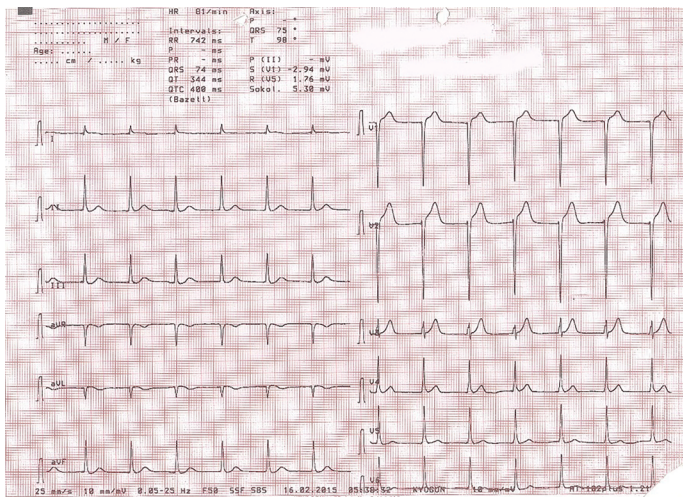
Figure 3. Completely normal electrocardiograph of the patient after treatments with metronidazole and high-dose corticosteroid

Discussion

Acute fulminant eosinophilic myocarditis is a rare disorder of unknown etiology characterized by diffuse or focal myocardial inflammation with eosinophilic infiltration (4, 5). Although several causes have been described in the literature, the exact cause of eosinophilic myocarditis is frequently unknown (6). In this case, eosinophilic myocarditis was associated with *Giardia lamblia* infection; after treatment with metronidazole therapy, the patient made a full recovery.

Peripheral blood eosinophilia is one of the diagnostic criteria and an important clue for the early diagnosis of eosinophilic myocarditis; however, it may not be present in certain patients in the early stage of the disease. Moreover, in a subset of patients, peripheral blood eosinophilia may not develop during the course of the illness (7). Our patient had a normal peripheral blood eosinophilic count upon admission. In some cases, it is difficult to differentiate between acute eosinophilic myocarditis and acute myocardial infarction due to their similar presentations and a lack of specific evidence, which may delay diagnosis. This delay may lead to the formation of mural thrombosis and scarring on the myocytes, as well as progressive eosinophilia may lead to endomyocardial fibrosis and restrictive cardiomyopathy in the advanced stages of the disease (1).

Endomyocardial biopsy remains the gold standard for the diagnosis of eosinophilic myocarditis. In addition, its role in differentiating between acute necrotizing eosinophilic myocarditis and other infectious myocarditis is critical, as corticosteroid therapy is contraindicated in patients with infectious causes (8). Endomyocardial biopsy findings are characterized by diffuse myocardial necrosis, which is associated with extensive eosinophilic infiltration of the myocardial interstitium, focal myocyte dissolution, perivascular infiltration, and myocardial interstitial fibrosis (9). However, endomyocardial biopsy is not a highly sensitive diagnostic procedure since the infiltration in myocarditis is usually focal. Therefore, in case clinicians strongly suspect this disease, a repeat biopsy should be performed (10).

Giardia lamblia is a flagellated unicellular eukaryotic microorganism that causes diarrheal illness known as giardiasis. *Giardia lamblia*'s life cycle comprises two main forms: cystic and trophozoite (11). While the infection can spread in different ways, the most common mode of transmission is drinking contaminated water with the presence of a *Giardia lamblia* cyst. The cysts transform into trophozoites in the proximal small intestine after exposure to the acidic environment of the stomach (11). The trophozoite in its vegetative form causes symptoms of diarrhea and malabsorption (12). *In vivo* and *in vitro* studies have demonstrated that neither the Giardia organism can invade the intestinal mucosa nor can it produce a toxin (13). In addition, it is well known that the surface of *Giardia lamblia* exhibits a significant antigenic variation of its cysteine-rich proteins (13). Therefore, it is considered that antigen mimicry between the myocardium and the surface proteins of the organism may be an underlying mechanism of myocarditis.

The management of acute eosinophilic myocarditis includes stopping the offending agent along with treating acute heart failure. While corticosteroids play an important role in the treatment of acute eosinophilic myocarditis, the role of immunosuppressive therapy in other causes of myocarditis remains controversial. Therefore, early diagnosis and treatment are crucial before the initiation of this therapy (14). With aggressive supportive care, complete ventricular recovery occurs in a majority of patients with acute fulminant eosinophilic myocarditis.

Conclusion

In conclusion, acute fulminant eosinophilic myocarditis due to *Giardia lamblia* may present with a cardiogenic shock and ST-elevation myocardial infarction in some patients. Hence, it should be considered during differential diagnosis. To the best of our knowledge, this is the first case report of acute fulminant eosinophilic myocarditis due to *Giardia lamblia* infection presented with cardiogenic shock.

Video 1a. Left ventricular ejection fraction of 20% along with severely depressed left ventricle wall motion in the parasternal long axis view.

Video 1b. Left ventricular ejection fraction of 20% with severely depressed left ventricle wall motion in the parasternal short axis view.

Video 2. Coronary angiography showed a diffuse coronary spasm in the left anterior descending artery and the circumflex artery in the left cranial oblique view.

Video 3. After starting high-dose corticosteroid, the patient's left ventricular function dramatically improved.

Video 3a. Left ventricular wall motion in the parasternal long axis view.

Video 3b. Left ventricular wall motion in the parasternal short axis view.

Video 3c. Left ventricular wall motion in the apical 4-chamber view.

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