CASE REPORT

Successful early diagnosis and treatment in a case of *Toxocara canis*-induced eosinophilic myocarditis with eosinophil-rich pericardial effusion

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Accepted 18 August 2015

SUMMARY

Fulminant myocarditis can become fatal if left untreated. Treatments for most types of myocarditis, including mechanical support, are limited. However, immediate systemic corticosteroids are known to be effective against eosinophilic myocarditis; therefore, prompt diagnosis of this disease is crucial. Unfortunately, the standard diagnostic tool for myocarditis, endomyocardial biopsy, does not provide immediate histopathological findings. Thus, a rapid diagnostic tool for identifying types of myocarditis is urgently required. We report here the first case of *Toxocara canis*-induced eosinophilic fulminant myocarditis which was diagnosed based on eosinophil-rich pericardial effusion where the patient recovered with early corticosteroid therapy.

BACKGROUND

Without early diagnosis and prompt treatments, acute eosinophilic myocarditis can cause fatalities.¹ Immediate systemic corticosteroid administration is crucial for successfully treating the disease.² However, initiation of this treatment is often delayed because of late diagnosis and rapid disease progression.³

Endomyocardial biopsy (EMB) is considered the gold standard for a diagnosis and aetiology identification of myocarditis. Unfortunately, even when performed urgently, EMB requires several days to make a final diagnosis with histopathological findings, which greatly diminishes the treatment opportunities of the patients. A rapid diagnostic tool that can provide same day identification of myocarditis is therefore required.

We describe here a case of *Toxocara canis*-induced eosinophilic fulminant myocarditis that was diagnosed early based on eosinophil-rich pericardial effusion.

CASE PRESENTATION

A 27-year-old man presented to a local hospital with chest pain and cold symptoms in March of 2012. ECG revealed ST segment elevation in the inferior and precordial leads V3 through V6. On day 2, the patient's blood pressure decreased (systolic blood pressure, 60 mm Hg), and echocardiography showed a sudden depression in cardiac function with pericardial effusion of haemodynamic relevance. The patient was therefore transferred to our hospital. On admission, his blood pressure was 70/52 mm Hg and respiratory rate was 25 breaths/min. Coronary angiography, EMB and

pericardiocentesis were performed immediately after admission for the differential diagnosis of acute coronary syndrome and myocarditis. No occlusive lesion in the coronary arteries was revealed. Since cardiogenic shock persisted after pericardiocentesis, administration of catecholaminergic agents (dopamine 20 µg/kg/min and dobutamine 10 µg/kg/min) was conducted. However, the blood pressure did not recover despite administering the maximum dose of catecholaminergic agents. Therefore, the patient was placed on an extracorporeal membrane oxygenator (ECMO), and intra-aortic balloon pumping (IABP) was initiated.

Echocardiography at the time of admission showed severe hypokinesis of the left ventricular wall motion (left ventricular ejection fraction (LVEF): 13%), with a thickened and oedematous left ventricle. (figure 1). Laboratory data revealed leucocytosis (total leucocyte count, 13 800/µL; eosinophil count, 1104/µL), with elevated levels of C reactive protein (0.97 mg/dL), troponin T (1.5 ng/mL), serum eosinophilic cationic protein (114 µg/L) and serum IgE (662 IU/mL). Immunological examination revealed no changes in antibody titres for myocarditis-causing viruses (influenza virus, Coxsackievirus, echovirus, cytomegalovirus, parvovirus, mumps virus). Molecular analysis with PCR using an EMB specimen, which is thought to be the best method to detect a viral infection of myocarditis, was not performed due to a technical issue in our hospital and the limitation of public health insurance treatment in our country (Japan). Collagen disease, and drug-induced and radiation-induced myocarditis were ruled out. Pericardial effusion was of the exudative type; further, as compared with peripheral eosinophil

Figure 1 Transthoracic echocardiographic image.

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To cite: Sangen H, Tanabe J, Takano H, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-208506

Figure 2 The shift of the serum eosinophilic count, eosinophilic count of pericardial effusion and serum eosinophilic cationic protein.

counts, the pericardial effusion sample showed abundant eosinophils, with a leucocyte count of 6400/ μ L and eosinophil count of 3264/ μ L (51%). Pericardial drainage continued for 6 days after admission; the eosinophil count decreased with disease severity (figure 2).

On the basis of the eosinophil-rich pericardial effusion, we diagnosed acute eosinophilic myocarditis of unknown aetiology. Corticosteroid therapy (0.5 mg/kg/day, 30 mg daily) was initiated on the day the patient was transferred to our hospital, and continued for 3 days. We did not add other immunosuppressive therapy; subsequently, cardiac function improved dramatically (LVEF: 40%), and the ECMO and IABP were removed on the fifth and sixth day after admission.

On the sixth day after admission, EMB histopathological findings revealed massive eosinophilic infiltration with some lymphocyte and focal myocyte necrosis, but no granulomas or giant cells, suggesting acute eosinophilic myocarditis (figure 3). On the 20th day after admission, the anti-IgG *T. canis* antibody was serologically detected. On further examination at the Department of Pathology, Miyazaki Medical University, Miyazaki, Japan, immunohistochemical analysis was performed and direct binding of antibodies to the *T. canis* antigen was demonstrated. The fact that the patient routinely ate raw deer meat supported *T. canis* infection. Our final diagnosis was *T. canis*-induced acute eosinophilic myocarditis.

Figure 3 Histological pictures.

TREATMENT

- ▶ Corticosteroid therapy
- ▶ Intra-aortic balloon pumping
- ▶ ECMO

OUTCOME AND FOLLOW-UP

The patient recovered and left the hospital after 40 days of treatment, with ongoing corticosteroid therapy (15 mg) and β -blocker (carvedilol 10 mg/day). At first, the corticosteroid was reduced 5 mg every week. After the first week and then, when the dose had reached 10 mg, the corticosteroid was reduced 2.5 mg every month.

DISCUSSION

This report is the first to present the diagnostic value of eosinophil-rich pericardial effusion in early diagnosis and treatment of eosinophilic myocarditis. Although eosinophil-rich pericardial effusion is commonly encountered in eosinophilic myocarditis, the clinical relevance of eosinophilic counts in pericardial effusion fluid remains unknown.⁴ In general, peripheral blood eosinophil counts exceeding 500/ μ L are considered abnormal. However, some reports consider only values exceeding 1500/ μ L to be abnormal.⁵ In the present case, the eosinophil counts in the serum were only mildly elevated.⁶ Eosinophilic myocarditis without peripheral eosinophilia has been previously reported,⁷ wherein EMB revealed marked eosinophil infiltration and myocytosis, EMB is therefore the gold standard for diagnosing myocarditis.⁸

In the present case, early myocardial biopsy was performed on the day the patient transferred to our hospital. As the patient was in a state of cardiogenic shock, ACE inhibitor and β -blocker could not be initiated. We decided that we should initiate corticosteroid therapy only after the EMB results were received, because early immunosuppressive therapy may cause virus persistence and therefore have fatal consequences. However, in this case, deterioration of cardiac function could have led to a fatal outcome. Therefore, as early immunosuppressive therapy may be life-saving in eosinophilic myocarditis, early corticosteroid therapy was initiated based on eosinophil-rich pericardial effusion.

Since EMB does not provide rapid results, our experience suggests that eosinophil-rich pericardial effusion can be used as a diagnostic tool for identifying eosinophilic myocarditis in the absence of peripheral eosinophilia. While little has been reported regarding pericardial effusion in other types of

myocarditis, it is known that peripheral eosinophils migrate into cardiac tissues in the acute phase of eosinophilic myocarditis. Therefore, it is possible that eosinophilic counts in pericardial effusion fluid increase before peripheral counts. Further analysis of pericardial effusion fluid from myocarditis associated with other aetiologies will aid in clarifying the utility of this novel diagnostic criterion.

In this case, the patient was treated with an ECMO and IABP. IABP counterpulsation has been the most widely used mechanical haemodynamic support device for nearly five decades. It improves diastolic blood pressure, thereby improving coronary perfusion, and by its afterload reduction properties, myocardial oxygen consumption is reduced leading to an increase in cardiac output.⁹ However, on the basis of insufficient and conflicting evidence from registry data, American and European guidelines recently downgraded IABP use for cardiogenic shock from a class I to a class IIa or IIb.^{10 11} According to an IABP-Shock II trial in patients with cardiogenic shock in myocardial infarction,

usage of IABP may not be beneficial.¹² Long-term benefit of IABP in fulminant myocarditis is not established yet. However, some reports have shown positive effects of IABP in myocarditis with cardiogenic shock. We believed that mechanical support therapy with ECMO and IABP was considered effective in this case.

Early diagnosis is thus of great importance in the optimal treatment of acute eosinophilic myocarditis. Our experience suggests that early diagnosis and treatment for eosinophilic myocarditis can be successfully based on eosinophil-rich pericardial effusion.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

Learning points

- Early diagnosis is of great importance in the optimal treatment of acute eosinophilic myocarditis.
- Eosinophilic myocarditis without peripheral eosinophilia has been previously reported, where endomyocardial biopsy revealed marked eosinophil infiltration and myocytosis.
- Our experience suggests that early diagnosis and treatment for eosinophilic myocarditis can be successfully based on eosinophil-rich pericardial effusion in the absence of peripheral eosinophilia.
- In this case, the patient was treated with an extracorporeal membrane oxygenator and intra-aortic balloon pumping (IABP). American and European guidelines recently downgrade IABP use for cardiogenic shock from a class I to a class IIa or IIb. Long-term benefits of IABP in fulminant myocarditis is not established yet, however, some reports have shown a positive effect of IABP in myocarditis with cardiogenic shock. We believed that mechanical support therapy with ECMO and IABP was effective in this case.

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