CASE REPORT

Myocarditis Associated with Visceral Larva Migrants due to Toxocara Canis

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Abstract

A 26-year-old man who was diagnosed with myocarditis presented eosinophilia after having eaten raw meat several times before the admission. Since the antibody titer against Toxocara canis was high, we diagnosed that he had visceral larva migrants due to Toxocara canis associated with myocarditis and eosinophilia. He was then treated with oral albendazole and prednisolone for 4 weeks and eosinophil count and hepatic enzymes were normalized along with the decrease in the antibody titer. We consider that his myocarditis was probably caused by direct larval migration and/or by hypersensitivity reaction, for which combined therapy with albendazole and prednisolone was effective.

Introduction

Toxocara canis (T. canis), a common dog roundworm, is one of the causative agents of visceral larval migrants (VLM). When infective eggs of T. canis from contaminated meat reach the human gastrointestinal tract, they enter the portal system and reach the liver. Some larvae then migrate from the liver to the lung and the heart through the systemic circulation (1, 2). Indeed, cases of myocarditis associated with VLM have previously been reported (3). Myocarditis may occur in 10–15% of cases of VLM, and in those cases, myocarditis is accompanied by an increased level of circulating eosinophils (4). The diagnosis of VLM can be made by the antibody test against T. canis antigen. Here, we report a case of myocarditis associated with eosinophilia caused by T. canis VLM in which combination therapy with albendazole and prednisolone was effective.

Case Report

A 26-year-old man was admitted to our hospital due to dyspnea, palpitation, chest pain and general fatigue. The initial examination showed the following findings; body temperature 36.7°C, blood pressure 108/80 mmHg with paradoxical pulse, pulse rate 104/min with a regular rhythm, and a cardiac gallop rhythm. ECG showed ST-segment elevation in all leads except for the leads of aVR, V, and V (Fig. 1A). The echocardiogram showed diffuse hypokinesis of the left ventricular (LV) wall, increased LV wall thickness (14–16 mm), and pericardial effusion (18 mm) (Fig. 1B). Laboratory data on admission revealed leukocytosis (total count 9,250/mm³ and eosinophil count 1,387/mm³), elevated C-reactive protein (1.4 mg/dl) and enzymes [creatine phosphokinase (CPK) 679 IU/l, aspartate aminotransferase (AST) 124 IU/l, alanine aminotransferase (ALT) 182 IU/l and lactic dehydrogenase (LDH) 733 IU/l]. Immunological test demonstrated that antibody titers against viruses that could cause myocarditis were all negative, including Coxsackie viruses A and B, adenovirus, cytomegalovirus, echovirus, EB virus and hepatitis viruses. We diagnosed that he had acute myocarditis of unknown etiology. In 2 weeks after admission, his symptoms gradually subsided and cardiac function improved with rest alone with concomitant normalization of ST-segment on ECG and of hemodynamic variables (blood pressure 124/66 mmHg and pulse rate 66/min). However, the laboratory studies revealed that the eosinophil count had increased to 26,000/mm³ (85% of his total white blood cell count), and the serum LDH level also increased to 1,069 IU/l (Fig. 2). The antibody test against parasitic infection (ELISA method) demonstrated that T. canis antibody was positive. The patient reported that he ate raw meat several times before admission. Taken together, we diagnosed that he had myocarditis caused by T. canis VLM. Although the patient was almost free from cardiac symptoms, we treated him with a combination therapy of albendazole (1,000 mg/day, PO) and prednisolone (35 mg/day, PO), because of the obvious presence of active VML. Albendazole was continued at the initial dose and prednisolone was tapered for 4 weeks (Fig. 3). Eosinophil...
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Figure 1. A) ECG showed ST-segment elevations in leads of I, II, III, aVL, aVF and V6. B) The echocardiogram showed diffuse hypokinesis of left ventricular wall, increased wall thickness (14-16 mm) and pericardial effusion (18 mm).

Figure 2. Clinical course after admission before the initiation of the treatment. With rest alone, pericardial effusion was diminished and left ventricular wall motion was normalized, whereas the eosinophil count and serum LDH level were relatively increased.

Figure 3. Clinical course after the initiation of the therapy. The combination therapy with albendazole (1,000 mg/day, PO) and prednisolone (35 mg for initial dose, PO) effectively decreased the eosinophil count and serum LDH level. With this therapy, the *T. canis* antibody titer was once elevated, but thereafter decreased.
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count gradually decreased to 500/mm³ (13% of total WBC count) and LDH level was normalized. The antibody titer against *T. canis* became elevated after the initiation of the treatment probably due to the antigen release from the dead parasite bodies, however, the titer was thereafter decreased (Fig. 3). For 3 months after the completion of the treatment, physical examination, laboratory tests, ECG and echocardiogram showed no abnormal findings and he was able to return to work.

**Discussion**

VLM caused by *T. canis*, a syndrome occurring mainly in young children, is characterized not only by chronic eosinophilia and hyperglobulinemia, but also by the involvement of lungs, liver, eyes, the central nervous system, skin and the heart as a consequence of local larval migrations. Indeed, cases of myocarditis associated with *T. canis* have previously been reported in which granulomatous lesions or parasites were not detectable and most cases showed extensive myocardial degeneration with gross eosinophilic cell infiltration (3). Myocarditis in VLM may result from direct larval invasion to the myocardium and/or hypersensitivity reactions to the parasites. In our case, symptoms of the patient appeared after he ate raw meat several times. Cookson et al reported that in mice infected with *T. canis*, myocardial lesions were developed associated with focal infiltration of eosinopils and histiocytes and that the cardiac lesions thereafter progressed to form granulomas with necrotic debris (4). It is possible that our patient had already been sensitized to *T. canis* and could thus easily develop myocarditis.

In the present case, the combination therapy with albendazole and prednisolone was effective. Albendazole is a broad-spectrum anthelmintic that has a low toxicity for humans. However, it often causes hypersensitivity reactions by dead larvae (5), which can be effectively avoided by the additional administration of prednisolone as proven in the present case. In addition, the present patient had marked eosinophilia. Eosinophil granule proteins are highly toxic to various tissues including myocardium. We assume that we were able to avoid such toxic effects by the administration of prednisolone.

**References**