

Received: 2020.12.18

Accepted: 2021.03.25

Available online: 2021.04.26

Published: 2021.05.27

# Toxocariasis-Associated Acute Perimyocarditis with Cardiogenic Shock: A Case Report

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Statistical Analysis C  
Data Interpretation D  
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Literature Search F  
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**Conflict of interest:** None declared

**Patient:** Female, 21-year-old  
**Final Diagnosis:** Toxocariasis-associated acute perimyocarditis with cardiogenic shock  
**Symptoms:** Dizziness • epigastric pain • headache • vomiting  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** Cardiology

**Objective:** Rare disease  
**Background:** Toxocariasis is an infection due to ingestion of the helminth parasite larvae found in dogs (*Toxocara canis*) or cats (*Toxocara cati*). Symptoms vary from being asymptomatic to shock, depending on the organ invaded by the parasite. However, cardiac involvement with shock in toxocariasis is very rare.  
**Case Report:** A 21-year-old woman without any history of underlying conditions visited the Emergency Department because of epigastric pain, vomiting, headache, and dizziness. Her blood pressure was 80/60 mmHg. Computed tomography (CT) of the brain showed no abnormal lesions. The abdominal-pelvic CT with contrast showed right pleural effusion, pericardial effusion, and focal ascites in the pelvic cavity. Laboratory tests revealed an elevation of eosinophils (40%) and cardiac enzymes (creatinine kinase-MB 27.6 ng/mL, high-sensitive cardiac troponin T 1.21 ng/mL). The transthoracic echocardiogram showed left ventricular systolic dysfunction (ejection fraction 44%) and moderate pericardial effusion. She was presumptively diagnosed with hypereosinophilic perimyocarditis and admitted to the Intensive Care Unit for shock. The pericardial effusion increased during treatment; therefore, pericardiocentesis was performed. Analysis of the pericardial effusion showed eosinophilia (eosinophils 90%) and the serologic test for parasites was positive for *Toxocara* and *Sparganum*. A combination therapy of albendazole, praziquantel, and corticosteroid resolved the pericardial effusion and the peripheral blood eosinophil count normalized. She was discharged without any other complications. At Outpatient Clinic follow-ups and observations over the next 2 years there were no abnormal findings, including pericardial effusion or eosinophilia.  
**Conclusions:** Toxocariasis rarely causes perimyocarditis with cardiogenic shock. Patients who present with pericardial effusion and eosinophilia need to be evaluated for parasitic infection.


**Keywords:** Myocarditis • Pericarditis • Shock, Cardiogenic • Toxocariasis

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## Background

Toxocariasis is a human illness caused by the larvae of *Toxocara canis* and *Toxocara cati*, the most common intestinal parasites in dogs and cats, respectively [1]. Humans are generally infected with *Toxocara* by the ingestion of soil, water, and foods contaminated with *Toxocara* eggs or larvae [2]. Humans can be infected by eating undercooked food contaminated with embryonated eggs, especially in Asian countries [3-5]. With the exception of mechanical injury in the eyes or brain caused by migrating larvae, clinical manifestations of toxocariasis are mainly caused by an immunopathogenic mechanism [6]. There are 2 types of human toxocariasis: visceral larva migrans (VLM), which usually invades major organs, including the liver, lungs, and central nervous system; and ocular larva migrans (OLM), which mainly affects the eyes and optic nerve [6-8].

Classic VLM is a systemic form of toxocariasis, which invades the liver and less frequently, the other organs (lungs, kidneys, skin, brain, and muscle) are involved [7,8]; cardiac involvement is rare [9]. Here, we report a case of toxocariasis-associated perimyocarditis with shock in a 21-year-old woman.

## Case Report

A 21-year-old woman visited the Emergency Department with epigastric pain, nausea, vomiting, headache, and dizziness for the past 3 days. She had taken medications including nonsteroidal anti-inflammatory drugs for an upper respiratory infection at an Outpatient Clinic. There were no underlying medical conditions or remarkable family history, including any history of ingestion of raw cow meat and liver. She had no pets (no dog or cat). She lived in an urban area and had not made any recent overseas trips. She was single and did not have a baby. She denied any history of playing in the sand box as a child.

On examination, she appeared to be acutely ill. The examination showed blood pressure of 80/60 mmHg, pulse rate of 100/min, respiratory rate was 20/min, and body temperature was 36.6°C. Heart auscultation revealed normal S1 and S2 without murmurs, rubs, or gallops. An electrocardiogram on arrival showed normal sinus rhythm with ST-segment elevation in the anterior precordial leads. Laboratory tests revealed a white blood cell count of 24 500/uL with eosinophilia (neutrophils 46%, lymphocytes 11%, monocytes 2%, and eosinophils 40%). The erythrocyte sedimentation rate was 16 mm/h (normal, <20 mm/h) and C-reactive protein was 0.5 mg/dL (normal, <0.5 mg/dL). The creatine kinase-MB (CK-MB) was 27.6 ng/mL (normal, <6.3 ng/mL), and high-sensitive cardiac troponin T was 1.21 ng/mL (normal, <0.014 ng/mL) (Table 1). A computed tomography (CT) of the brain showed no abnormal lesions. The abdominal-pelvic CT with contrast showed right pleural

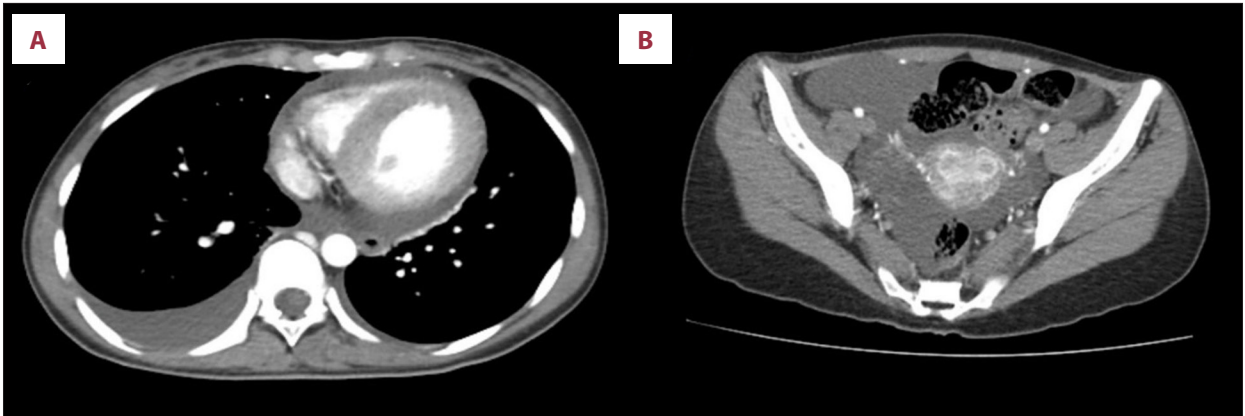
**Table 1.** Results of initial laboratory tests at admission.

Complete blood count		
White blood cells (per $\mu$ L)	24 500	(3500~10 500)
Segment neutrophils (%)	46	(40~60)
Lymphocytes (%)	11	(20~40)
Monocytes (%)	2	(2~8)
Eosinophils (%)	40	(1~4)
Hemoglobin (%)	14.6	(12.1~15.1)
Hematocrit (%)	45.5	(36.1~44.3)
Platelets (per $\mu$ L)	294 000	(150 000~450 000)
Blood chemistry		
BUN (mg/dL)	21.8	(6~20)
Creatinine (mg/dL)	0.6	(0.6~1.3)
AST (IU/L)	35.0	(10~40)
ALT (IU/L)	20.0	(10~40)
Total bilirubin (mg/dL)	0.7	(<1.0)
Sodium (mmol/L)	133	(135~147)
Potassium (mmol/L)	4.9	(3.5~5.5)
Chloride (mmol/L)	99.5	(98~106)
ESR (mm/h)	16.0	(0~20)
CRP (mg/dL)	0.5	(<3.0)
CK-MB (ng/mL)	27.6	(<5.0)
hs cardiac troponin T (ng/mL)	1.2	(<0.04)

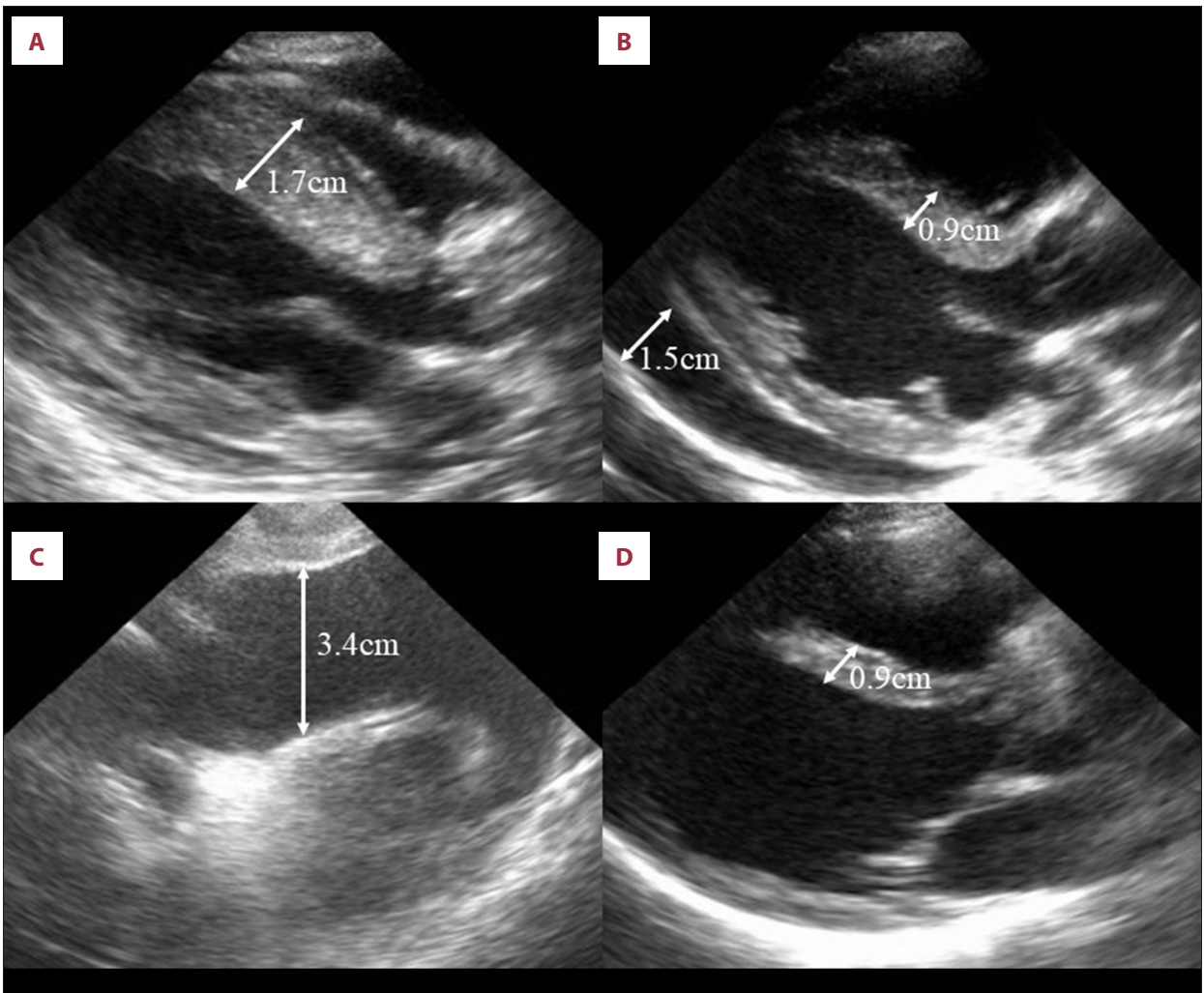
ALT – alanine aminotransferase; AST – aspartate aminotransferase; BUN – blood urea nitrogen; CK-MB – creatinine kinase-MB; CRP – C-reactive protein; ESR – erythrocyte sedimentation rate; hs – high-sensitive.

effusion, pericardial effusion, and focal ascites in the pelvic cavity (Figure 1A, 1B); however, there was no hepatomegaly. The transthoracic echocardiogram revealed left ventricular (LV) systolic dysfunction (ejection fraction 44%), with moderate pericardial effusion (fluid thickness, 0.9~1.3 cm). She was presumptively diagnosed with hypereosinophilic perimyocarditis and admitted to the Intensive Care Unit (ICU).

After admission to the ICU, corticosteroid (methylprednisolone 60 mg/day), ceftriaxone (2.0 g/day), and catecholamine (dopamine 20 mcg/kg/minute and dobutamine 10 mcg/kg/minute) were administered intravenously. Another transthoracic echocardiogram was performed 3 days later as a follow-up. Her LV systolic dysfunction had recovered (ejection fraction 73%); however, the pericardial effusion had increased (Figure 2A-2C). She underwent pericardiocentesis with pericardial fluid analysis. The pericardial effusion was exudative and differential counts of white blood cells revealed eosinophilia (eosinophils 90%, monocytes 8%, and lymphocytes 2%) (Table 2).



**Figure 1.** Computed tomography of the abdomen and pelvis at admission. (A) Right pleural effusion, pericardial effusion. (B) Focal pelvic ascites.



**Figure 2.** Changes in the pericardial effusion and interventricular septal swelling. (A) Initial transthoracic echocardiogram showed interventricular septal swelling (1.7 cm) and mild pericardial effusion. (B, C) Follow-up echocardiogram after admission showed increased pericardial effusion (1.5 cm in the parasternal view and 3.4 cm in the subcostal view) and improved interventricular septal swelling (0.9 cm). (D) Transthoracic echocardiogram after treatment showed no pericardial effusion and improved interventricular septal swelling.

**Table 2.** Results of the serologic parasite tests.

	Results	Normal range
<i>Toxocara canis</i>	Positive 2.37	0~1.00
<i>Sparganum</i>	Positive 1.28	0~1.00
<i>Clonorchis sinensis</i>	Negative 0.29	0~1.00
<i>Paragonium swetermani</i>	Negative 0.25	0~1.00
<i>Cysticercus cellulosae</i>	Negative 0.22	0~1.00

Serologic tests for parasites were performed (Table 3). The enzyme-linked immunosorbent assay to detect immunoglobulin G antibodies against the *Toxocara* excretory and secretory antigens of *Toxocara canis* was positive. The serologic test for *Sparganum* was positive, while the other serologic tests for *Clonorchis sinensis*, *Paragonimus westermani*, and *Cysticercus cellulosae* were negative. Based on the results of these serologic tests, she was administered albendazole 400 mg 2 times daily for 2 weeks and praziquantel 600 mg 3 times a day for 1 day. Praziquantel was added due to the possibility of a superinfection by *Sparganum*. Intravenous methylprednisolone (60 mg/day) was administered for 6 days, then changed to oral prednisolone and tapered over 1 month. After the combination therapy with albendazole, praziquantel, and corticosteroid, the pericardial effusion resolved as seen on the following transthoracic echocardiogram (Figure 2D). The relatively enlarged heart and peripheral blood eosinophil count normalized during hospitalization (Figures 3A-3C, and 4). The patient was discharged without any other complications. In the following 2 years of Outpatient Clinic follow-up observations, there were no further abnormal findings, including pericardial effusion and eosinophilia.

**Table 3.** Pericardial effusion study.

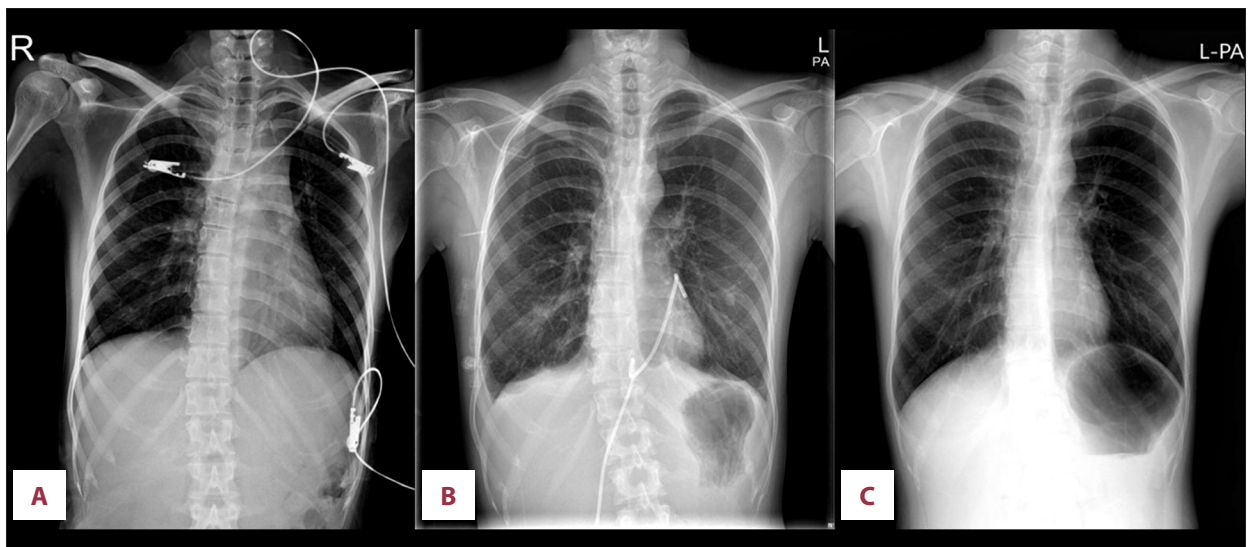
	Results	Normal range
pH	8.0	6.82~7.59
Red blood cells (per $\mu$ L)	11 408	None
White blood cells (per $\mu$ L)	7358	<100
Mononuclear cells (%)	8	
Lymphocytes (%)	2	
Eosinophils (%)	90	
Protein (mg/dL)	5294.2	<3000
Glucose (mg/dL)	106.0	
Amylase (IU/L)	26.3	
LDH (IU/L)	1169.0	<300

LDH – lactate dehydrogenase.

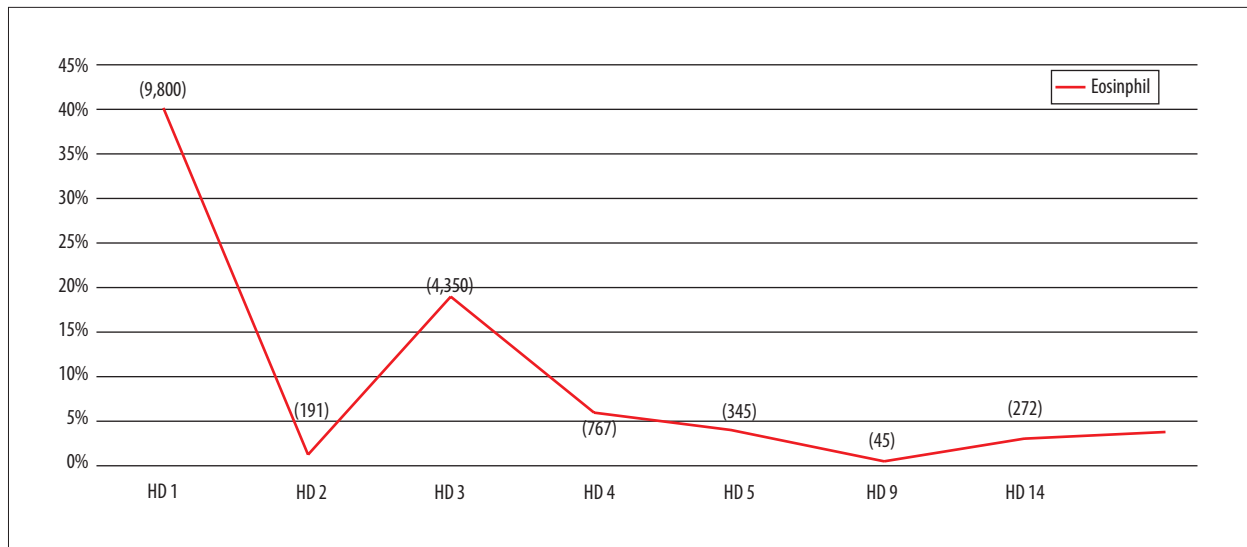
This case occurred several years ago and the authors have not obtained informed consent from the patient. However, the patient has been sufficiently anonymized and could not be identified from the information in this case report.

## Discussion

It is rare for toxocariasis to invade the heart, and it is especially unusual for it to manifest as shock. Extracorporeal membrane oxygenation treatment has been required to treat toxocariasis with cardiogenic shock [10]. In the present case, the parasitic infection was identified in a perimyocarditis patient with shock accompanied by eosinophilia, which improved with medication.



**Figure 3.** Serial changes in the chest X-ray. (A) Initial chest X-ray at admission. (B) X-ray after pericardiocentesis. (C) X-ray before hospital discharge.



**Figure 4.** Changes in the serum eosinophil count during treatment. HD – hospitalization day.

In most cases, *Toxocara* infections are not serious, and many adults infected by a small number of larvae do not notice any symptoms. The characteristics of classic VLM are usually fever, eosinophilia, hepatosplenomegaly, and pulmonary fibrosis. However, if toxocariasis invades the whole body, it can cause severe symptoms [6,8]. Cardiac involvement in *Toxocara* infections, although rare, can cause potentially life-threatening myocarditis/pericarditis with heart failure or cardiac tamponade [11,12]. Thus, globally it has become relevant for clinicians in developed settings to consider parasitic infections, including *Toxocara*, in the differential diagnosis of myocardial and pericardial disease.

A confirmed diagnosis of toxocariasis is a tissue biopsy to prove the existence of the *Toxocara* larvae; however, this method is invasive, less sensitive, and time-consuming. Western blotting is useful in the immunodiagnosis of toxocariasis for species identification and genetic analysis of the *Toxocara* species, although it is not widely used for the diagnosis of toxocariasis [13,14]. Therefore, toxocariasis is diagnosed indirectly based on the history of raw or undercooked meat consumption, clinical symptoms and signs, eosinophilia, increased immunoglobulin E levels, and positive serology. There are various serological tests to diagnose toxocariasis. Using the enzyme-linked immunosorbent assay based on the third stage of the *Toxocara canis* larvae is common [1,6]. The *Toxocara* infection does not always cause symptoms and is self-limited. Treatment is not necessary before symptoms occur. Severe toxocariasis is treated with antiparasitic drugs, including albendazole or mebendazole, in combination with anti-inflammatory drugs (non-steroidal anti-inflammatory drugs and corticosteroids) [6,12].

The patient in the present case report was Korean, and the prevalence of toxocariasis accompanied by eosinophilia in Koreans

is 20% to 40% [5,13]. Unlike other countries, there is a high prevalence of toxocariasis infection in Korean adults due to the consumption of raw meat and liver [4,5]. Therefore, it is possible that the patient in this case was infected by *Toxocara* ingestion without her knowledge.

In this case, the *Sparganum* antigen was also detected through the parasitic tests and was treated with an antiparasitic drug. However, when the tests are positive for 2 types of parasitic antibodies, it is reasonable to accept it as a cross reaction between the 2 antigens rather than an overlapping infection. Therefore, the positive result for *Sparganum* was likely to be a cross reaction between the 2 antigens, *Toxocara canis* and *Sparganum*, respectively.

Perimyocarditis is defined as myocarditis with some degree of concurrent pericardial inflammation (ie, pericarditis). Acute pericarditis and myocarditis often occur together, although they are not typically equivalent. Myopericarditis and perimyocarditis are similar in that they are accompanied by pericardial effusion and cardiac enzyme elevation (ie, troponin I or T, and CK-MB fraction). However, the regional wall abnormality and LV systolic dysfunction are only accompanied by perimyocarditis [15]. In the present case, the patient showed LV systolic dysfunction with a reduced ejection fraction, suggesting a myocarditis-dominant syndrome (ie, perimyocarditis).

Establishing the underlying cause of perimyocarditis/myopericarditis is of the utmost importance as it can alter the treatment and outcome. Perimyocarditis and myopericarditis share common infectious or noninfectious etiologic agents (including systemic inflammatory diseases, drug- or vaccine-related, rarely iatrogenic) [16]. Among infectious pathogens, viral infections, including the *Coxsackie B* virus, are the most

common causes. A large group of bacterial pathogens, including *Mycobacterium tuberculosis*, and fungal pathogens have been reported as possible etiologic agents. In addition, there are reports of rare cases in which myopericarditis/perimyocarditis is associated with parasitic pathogens, including *Toxocara*, *Trypanosomes*, and *Toxoplasma* [15,17].

The present case report has several limitations. First, we did not conduct a western blot procedure for the immunodiagnosis of toxocariasis. Therefore, only the possibility of a cross reaction between toxocariasis and sparganosis was known. The level of brain natriuretic peptide was not checked and virus tests had not been conducted before steroid therapy because we focused on the eosinophilia and did not consider that she could have a viral disease. We should have considered disseminated strongyloidiasis in the differential diagnosis because strongyloidiasis could be a cause of pericardial effusion with eosinophilia, which could be resolved with albendazole [18].

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## Conclusions

We report a case of perimyocarditis caused by *Toxocara canis*, which presented with hypotension, LV dysfunction, and pericardial effusion. Cardiac involvement in a *Toxocara* species infection is a rare and potentially life-threatening complication. A high index of suspicion is necessary to establish an early diagnosis and initiate the appropriate treatment. Patients who present with pericardial effusion and eosinophilia need to be evaluated for parasitic infection.

## Department and Institution Where Work Was Done

Heart Center, Konyang University Hospital, Daejeon, South Korea.

## Conflicts of Interest

None.