Acute Life-Threatening Toxocarial Tamponade*

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An unusual case of life-threatening visceral larva migrans (toxocariasis) is reported herein. The patient was admitted with acute dyspnea and bilateral pleural effusion; rapidly pericardial tamponade developed. Blood and body fluid eosinophilia were elevated. Extensive investigations revealed no malignant process or vasculitis, but toxocara infection was confirmed by rising specific antibody titers. The high seroprevalence of Toxocara antibodies, particularly in children, suggests that a diagnosis of visceral larva migrans should be considered before a diagnosis of systemic hypereosinophilic syndrome even when clinical presentation is unusual. Prophylaxis against this widespread polymorphic zoonotic infection is desirable in view of the potentially dramatic consequences of infestation.

(CHEST 1997; 112:1692-93)

Key words: larva migrans; pleural effusion; tamponade

Abbreviation: OD=optical density

A combination of acute eosinophilic pericarditis and pleural effusion with associated blood hypereosinophilia is unusual and might suggest lymphoma or paraneoplastic syndrome, drug reactions, vasculitides such as Churg-Strauss syndrome, idiopathic hypereosinophilic syndrome, or tissue-invasive parasitic infestation. Herein is the report of a case of life-threatening tamponade with bilateral pleural effusion consequent to toxocarial infection.

CASE REPORT

A 50-year-old man was admitted to our hospital with severe dyspnea 17 days after operation for acute appendicitis. Pleural effusion of the left costophrenic angle had been observed at surgery. The blood cell count showed 600 eosinophils/mm³, and histopathologic examination of the removed appendix was unspecific. His previous medical history disclosed no abnormalities. On admission, blood pressure was 120/70 mm Hg, pulse was 125 beats per minute, and respiratory rate was 36 breaths per minute. Physical examination showed only softened heart and respiratory sounds. There was no skin rash, urticarial lesion, joint pain or inflammation, or splenic enlargement. Chest roentgenograms showed bilateral pleural effusion with normal parenchyma but an enlarged cardiac image. Blood cell counts showed 15,400 leukocytes/mm³ with 2,200 eosinophils/mm³. Hemoglobin level was 13 g/Dl, and the platelet count was 330,000/mm³. Blood chemistry findings disclosed only mildly elevated liver enzymes. Serum protein electrophoresis was normal, and the erythrocyte sedimentation rate was 42 mm at the 1st hour.

A few hours after the patient was admitted to the hospital, acute tamponade developed. On immediate subxyphoid pericardiomy, more than 800 mL of pericardial fluid with a cellular component of 20% eosinophils was evacuated, and chest tubes were inserted which drained 1,500 mL of fluid with a 7% eosinophil content from each pleural space. Both liquids contained 45 g/L of protein. Pleural and pericardial biopsies showed nonspecific subacute inflammation, with sparse eosinophil infiltration. No sign of vasculitis could be observed. Appropriate staining revealed no pathogens, and viral, bacterial, and parasitologic investigations showed no abnormalities.

Thoracoabdominal CT, esophagogastrooduodenoscopy with biopsies, bone marrow aspiration, and biopsy were uninformative. Stool examinations revealed no parasites (Bailenger and Baerman methods). Blood cultures were negative for organisms.

Viral serologic tests for HIV-1, HIV-2, human T-cell lymphotropic virus-1 and -2, and hepatitis B and C were all negative; antineutrophil cytoplasmic antiantibodies and antinuclear antibodies were absent. The IgE level was 149 kIU/L (normal, <150

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kIU/L). No antibodies were detected towards Fasciola hepatica or Trichinella spiralis, but a first serum sample tested for larva migrans (LMD Laboratories; Carlsbad, Calif) was positive with an enzyme-linked immunosorbent assay reading of 0.5 optical density (OD) (normal, <0.3 OD) at day 1 of hospitalization. Subsequent samples showed increasing titers with 0.7 OD at day 10 and 2.5 OD at day 17.

A diagnosis of acute toxocariasis was made, and the patient was treated with anthelmintic therapy at day 10 (ivermectin, 200 μg/kg/d) and day 25. His respiratory condition and pericarditis improved. His anti-Toxocara antibody titer dropped to 0.8 OD and his blood eosinophil level was 300/mm³ 15 days after treatment. One year after this acute illness, he was in good health, and no relapse occurred.

**DISCUSSION**

Toxocara canis and *T cati* are roundworms which commonly infect dogs and puppies, on one hand, and cats, on the other, respectively. Human infection occurs following ingestion of embryonated eggs from the environment. The larvae penetrate the gut wall and begin a migration through the tissues. This can continue for several years. Seroprevalence studies suggest that 2 to 3% of adults and 7 to 14% of schoolchildren in England have been exposed to *T canis*. Even in urban areas, adults and children can be infected by eating contaminated raw food.

Most disorders consequent to *Toxocara* infection are due to damage caused by an inflammatory immune response. Infected subjects often are asymptomatic, but infected children may display cough, fever, abdominal pain, hepatomegaly, and skin lesions. Severe infections are rare but may cause respiratory distress or myocarditis. Ocular infection can lead to impairment of vision.

Blood hypereosinophilia, which is a frequent sign of tissue invasion by parasites, is inconstant in toxocariasis, and the most reliable diagnostic method is an enzyme-linked immunosorbent assay test which has 98% specificity since larvae are hardly ever found upon pathologic examination. Pleural effusion is an unusual symptom of toxocariasis, and only two previous cases have been reported. Like the patient reported herein, these subjects were dyspneic with blood hypereosinophilia. Their pleural fluid contained eosinophils, but histopathologic studies failed to detect the parasite and diagnosis was based on serologic tests. A single case showed eosinophilic ascites, but as best as can be determined, the patient reported herein is the first in whom tamponade is associated with *T canis* infection. Other native parasitic diseases have not been associated with pericarditis or tamponade. The causative role of the parasite is suggested by the absence of any sign of malignancy, autoimmune disease, or vasculitides despite comprehensive investigation.

Further, there was a prolonged improvement in clinical status and regression of specific antibody titers after institution of treatment with ivermectin.

Antihelminthic drugs, such as ivermectin or albendazole, are available to treat patients, but no large-scale studies have demonstrated their effectiveness in symptomatic cases; therefore, supportive care remains essential in acute illness. In this case, ivermectin was chosen because it has fewer side effects. Reduction of the risk of environmental contamination by appropriate veterinary care of domestic dogs and cats seems desirable in view of the possible dramatic consequences of *Toxocara* infection.

**ACKNOWLEDGMENTS:** We thank J.C. Petithory for his advice and contribution in finding appropriate documents and T. Greenland for his reading of the manuscript.

**REFERENCES**


**Diverse Presentation of Aberrant Origin of the Right Subclavian Artery**

**Two Case Reports**

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Aberrant origin of the right subclavian artery occurs in up to 1% of the population and can result in a wide range of symptoms. In this report, two cases of this anomaly are presented. In the first case, a patient developed fatal group A streptococcal aortitis. In the second case, the patient complained of chronic cough and intermittent dyspnea. The embryologic genesis of this abnormality is discussed and the

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