A PRESUMPTIVE CASE OF TOXOCARIASIS ASSOCIATED WITH EOSINOPHILIC PLEURAL EFFUSION: CASE REPORT AND LITERATURE REVIEW

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Abstract. Human toxocariasis is a helminthozoanosis caused by Toxocara sp. Larval migration of the organism through the tissues can result in eosinophilia associated with a broad spectrum of clinical manifestations. We report a case of eosinophilic pleural effusion and CD8 cell deficiency associated with Toxocara sp. The symptoms of this patient responded promptly to a nonsteroidal anti-inflammatory medication (naproxen). This is only the fourth reported case of a pleural effusion associated with Toxocara.

Human toxocariasis is a helminthozoanosis due to migration of Toxocara species larvae through the human body causing visceral larva migrans.1,2 We report a patient with an eosinophilic pleural effusion and a CD8 cell deficiency associated with Toxocara infection whose symptoms responded to naproxen.

A 54-year-old man with a history of night sweats for one year presented with complaints of left-sided pleuritic chest pain, worsening shortness of breath, orthostatic symptoms, sleepiness, and fatigue for five weeks. He had 31 pit bulls that he used as hunting dogs and reported a history of not washing his hands after handling the dogs. On examination, he had decreased breath sounds in the posterior left base. Roentgenographic studies showed a left pleural effusion. Thoracentesis showed 900 mL of yellow cloudy fluid with 29,000 white blood cells (WBCs)/mm³, 36% neutrophils, 43% eosinophils, a lactate dehydrogenase level of 617 units/liter, and a protein level of 4.1 g/dL. The results of bacterial, fungal, and acid-fast bacilli stains, cultures, and cytologic analysis were negative. Over the next two days, the patient experienced fever (103°F [39.4°C]), chills, worsening of chest pain, and shortness of breath. Re-evaluation showed a large and loculated pleural effusion. He was then admitted to the Medical Center of Central Georgia Hospital in Macon, Georgia.

Laboratory evaluation showed a WBC count of 8,550/µm³ with 21% eosinophils. The level of C-reactive protein was increased (34.5 mg/dL) (normal range = 0.020–0.722 mg/dL), as was the erythrocyte sedimentation rate (119 mm/hour) (normal range = 0–20 mm/hour), and a cellular immunodeficiency test showed an absolute CD8 cell count of 163/µL (normal range = 315–788/µL). The effusion was drained with chest tubes. It contained 18,360 RBCs/µm³ and 15,720 WBCs/µm³ with 82% neutrophils, 8% eosinophils, 7% lymphocytes, and 3% monocytes with negative stain and culture results. He continued to have a fever after treatment with ticarcillin clavulnate (3.1 grams every six hours) and became very lethargic, confused, and forgetful over the next day. Magnetic resonance imaging of the brain showed normal findings. Cerebrospinal fluid was found to be clear and had a glucose level of 48 mg/dL (normal range = 40–70 mg/dL), a protein level of 35 g/dL (normal range = 15–45 mg/dL), 0 WBCs/µm³, and 42 RBCs/µm³. He was given naproxen (375 mg orally every 12 hours) and showed an immediate decrease in his fever. Over the next two days he had resolution of his pleuritic pain and mental status. The level of IgG antibody to Toxocara was elevated (titer = 1.4) (< 0.9 = negative, 0.9–1.1 = equivocal, > 1.1 = positive). Results of serologic tests for cytomegalovirus, Trichinella, Blastomyces, and Histoplasma were negative. The level of antibodies to Coccidioides was at the upper limit of normal. On follow-up, the patient has remained asymptomatic with no further accumulation of fluid or fever.

Human infection with Toxocara usually presents as visceral larva migrans.1,2 Symptoms are nonspecific and are due to the inflammatory response to Toxocara. Pulmonary manifestations are usually a transient form of Loeffler’s syndrome or simple eosinophilic pneumonia.3 Eosinophilic pleural effusion is a rare manifestation of this infection, with, to the best of our knowledge, only three previous case reports.3–5 As in our case, this was presumptively proven serologically in all three case reports. One of these cases was treated with mebendazole, another with albendazole and diethylcarbamazine, and the third with steroids and thiabendazole.5 An enzyme-linked immunosorbent assay for Toxocara has shown a sensitivity of 86% and a specificity of 91%.6 To our knowledge, this is the first case report of a Toxocara infection clinically responding to a nonsteroidal anti-inflammatory drug. Also of note was the CD8 cell deficiency in our patient. At this time, we cannot state if he had an underlying CD8 cell deficiency or if infection with Toxocara contributed to this finding.

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