Clinical Note

Can worms cause chest pain?

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Pleural effusion is commonly seen in patients with heart failure and tuberculosis (TB), and it has a different etiological factor. A rare cause of pleural effusion, eosinophilic pleural effusions (EPEs) secondary to hook worms infestation, is discussed herein.

A 35-year-old Indian male was presented to the accident and emergency with a history of right sided chest pain for one month duration with gradual onset stabbing in nature not radiated, or referred to any other site aggravated by breathing, and has no obvious relieving factors. No history of fever, cough, contact with patient with chronic cough and with active pulmonary TB, coughing with blood, sweating, and loss of weight. He came 2 months ago from India, working as a builder. He is single and sharing a room with 5 other persons, never smoked neither asthmatic nor allergic to any medicine. On examination, he seems healthy in a slim afebrile condition. Other vital signs were stable. Examination on the head eyes, ears, nose and throat revealed no abnormality. He was not pale, not cyanosed, and no lymphadenopathy. Chest examination revealed evidence of the right sided pleural effusion. Cardiovascular system (CVS), abdominal, central nervous system (CNS), and musculoskeletal examination were not remarkable. A complete blood count (CBC) showed white blood cell (WBC) of 8000 u/l [normal range (NR): 4000-11000 u/l), hemoglobin (Hb) 14.6 g/dl (NR: 13-17 g/dl), and platelets 279000/ul (NR: 140000-449000). Differential of WBC neutrophils was 45.8%, lymphocytes 20%, monocytes 10.8%, eosinophils 22%, total protein 75 g/dl, serum albumin 39 g/dl, total bilirubin 9 umol/l, serum glutamate pyruvate transaminases (SGPT) 19 u/l, serum glutamate oxaloacetate transaminases (SGOT) 20 u/l, alkaline phosphatase (ALP) 67 u/l, normal urea, and electrolytes. Chest x-ray (CXR) of the right sided pleural effusion has no apical infiltrate, hilar adenopathy or parenchymal lung disease. Pleural fluid analysis showed total a protein of 61 g/l, albumin 32 g/l, lactate dehydrogenase (LDH) 1137 u/l and glucose 5.7 mmol/l. Gram stain showed no organism, acid-fast bacilli (AFB) smear and TB culture were negative for mycobacterium TB, and cytology was negative for malignancy. Differential of WBC showed a neutrophils of 5%, lymphocytes 32%, monocytes 5%, eosinophils 52%. A stool examination for ova and parasites, showed hook worms (**Figure 1**). He was given 400 mg albendazole once, and in follow up, pleural effusion as well as peripheral eosinophilia disappeared.

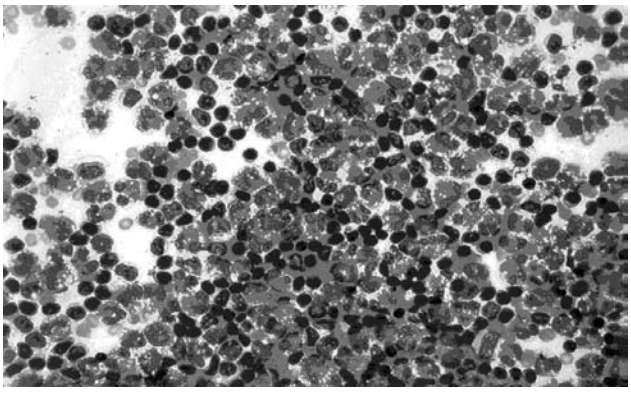

Figure 1 - Cytology smear stained with modified giemsa (diff quick) stain showing a dense inflammatory reaction of predominantly eosinophils.

Eosinophilic pleural effusions are defined as those that contain at least 10% eosinophils. The EPEs account for 5-16% of exudative pleural effusions. Eosinophilic pleural effusions are caused by the presence of air or blood or both in the pleural space, infectious or other inflammatory diseases, malignancy, pulmonary emboli, asbestos exposure, and drug reactions. Differences in the clinical features suggest that a variety of mechanisms operate to induce eosinophilic pleural inflammation and pleural fluid accumulation.

Human and animal studies indicate that interleukin (IL)-5 is an important common contributor of different pathogenetic pathways.[1] Several studies were conducted looking for the etiology of EPEs. One of the studies showed that EPEs was identified in 44 out of 476 patients (9.2%).[2] Malignancy was diagnosed as often in eosinophilic as in non eosinophilic effusions (20.5% versus 20.1%). The only diagnoses that were significantly associated with eosinophilic effusions were idiopathic (25% versus 8%; p=0.001), and post thoracic surgery (11% versus 3%; p=0.023). Median survival was 7.7 months for those with a non eosinophilic effusion compared to 16.8 months for those with eosinophilia (p=0.017). This difference in survival persisted after adjustment for age and diagnosis. In reviewing the literature, we found 3 cases of EPEs secondary to intestinal worms,[3,4] and all of them responded nicely to treatment as our patient who was given albendazole and responded well. Hookworm infections are common in the tropics and subtropics.[5,6] Infection is rare in regions with less than 40 inches of rainfall annually. The species of hookworms, which produce human disease, varies geographically.

Ancylostoma duodenale causes infection in the Mediterranean countries, Iran, India, Pakistan, and the Far East. Necator americanus infects humans in the North and South America, Central Africa, Indonesia, Islands of the South Pacific, and parts of India. Hookworm infections should be treated with mebendazole (100 mg orally BID for 3 days or 500 mg once). Alternative agents include pyrantel pamoate (11 mg/kg per day for 3 days, not to exceed 1 g/day) or albendazole (400 mg once).

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