

Eosinophilic pleural effusion and giardiasis: A causal or a casual relationship?

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ABSTRACT

A case of bilateral eosinophilic pleural effusion with coincidental intestinal infestation of giardia lamblia is being reported. After reviewing the possible causes of this type of pleural effusion, no clinical or laboratory data were obtained which could explain this condition except giardiasis. Moreover the clearance of pleural effusion with the treatment of giardia with metronidazole suggests giardia as the probable cause of bilateral eosinophilic pleural effusion.

KEY WORDS: Eosinophilia, giardiasis, pleural effusion

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INTRODUCTION

Eosinophilic pleural effusion (EPE) is defined as presence of at least 10% eosinophils of the total white blood cells in pleural fluid.^[1,2] The relative incidence of EPE has been estimated at between 5% and 16% of all pleural effusions (PEs).^[1-3] The most common conditions associated with eosinophilic pleural effusions include previous thoracentesis, air or blood in the pleural cavity, asbestosis, collagen vascular disease, drug-induced pleuritis, paragonimiasis and malignancy.^[4] Despite thorough investigations, 14-20% of the EPEs remain undiagnosed and are labeled as idiopathic.^[5,6] The clinical significance of pleural fluid eosinophilia remains unclear. We report a case of bilateral eosinophilic pleural effusion in which cause could not be ascertained despite extensive investigations and was presumed to be due to giardia infection.

CASE REPORT

A 32-year-old male, barber by profession presented with complaints of right sided chest pain for 1 month, cough for 20 days and left lower chest pain for 15 days. Patient

had breathlessness on exertion for past one month which was gradual in onset. He gave history of loose motions and diffuse pain abdomen off and on for the past three months. He smoked 10-15 bidis per day since last 10 years and took 60-90 ml of alcohol per day for past 2 years. There was no history of any drug abuse. There was no history of any trauma, prolonged bed rest, any invasive procedure involving the chest or any respiratory infection. There was no history of any drug intake or exposure to pets. There was no history of any intake of over the counter medications or any other medication for any ailment. Complete blood count was within normal limits with a Hb of 10.2 gm%, total leucocyte count of 10800/mm³ with a DLC of 61% neutrophils, 30% lymphocytes, 9% eosinophils and an absolute eosinophil count of 1008/mm³. Liver and renal function tests were within normal limits. Electrocardiogram [ECG] did not show any abnormality. X-ray chest [Figure 1] showed a right sided pleural effusion. Contrast-enhanced computerized tomography (CT) scan [CECT] of chest also revealed bilateral pleural effusion (right > left) without any parenchymal abnormality [Figure 2]. Pleural fluid was aspirated from both the sides.

The pleural fluid analysis of both sides showed an exudative pleural effusion with a very high percentage of eosinophils on cytology suggestive of eosinophilic pleural effusion. Right sided PE showed a TLC of 1850/cmm with 96% eosinophils and 4% lymphocytes. Adenosine deaminase [ADA] level was 32 IU/L (N ~ 30 IU/L). Left sided pleural fluid examination showed a TLC of 6400/cmm with lymphocytes 20%, neutrophils 6% and eosinophils 74%. ADA level was 30 IU/L. Pleural fluid pH and glucose was

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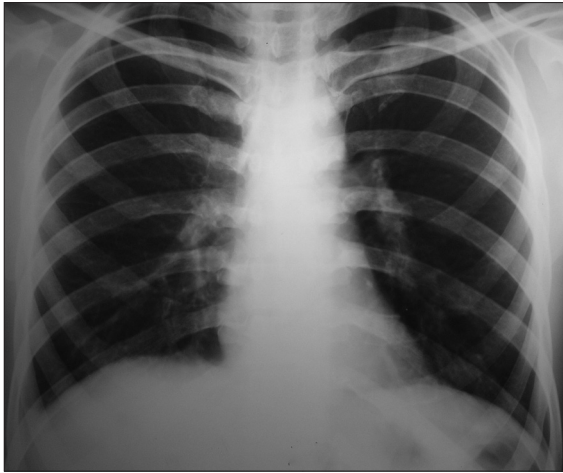


Figure 1: X-ray chest showing right sided pleural effusion

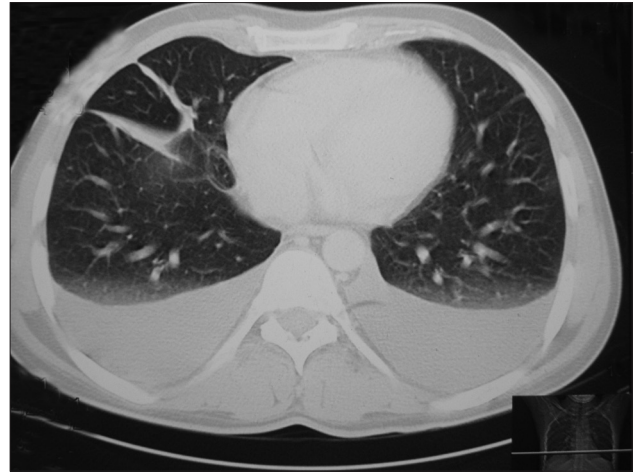


Figure 2: CT scan chest showing bilateral pleural effusion

within normal limits. Pleural fluid from both the sides was negative for malignant cells. The results of bacterial, fungal, and acid-fast bacilli stains, and cultures were negative. The only positive, probably an incidental, finding in our case was that the patient's stool for ova was positive for *Giardia lamblia*. But the pleural fluid examination was negative for giardia and any other parasite. Ultrasonography of the abdomen also revealed no abnormality.

Patient was put on metronidazole (400 mg BD) for 4 weeks for giardiasis. His abdominal symptoms showed improvement. X-ray chest after one month showed clearing of bilateral PE. After 2 years follow-up the patient is healthy and has no complaints and the pleural effusion has not recurred.

DISCUSSION

Eosinophilic pleural effusion (EPE) was first described by Harmsen in 1894 and is defined as a pleural effusion that contains at least 10% eosinophils.^[1] EPEs account for 5 to 16% of all pleural effusions^[1-3] and can be a manifestation of a great variety of diseases. It may be caused by almost every condition that causes pleural effusion. The most common conditions associated with EPE are malignancy, infections, post-traumatic, miscellaneous and idiopathic.^[5]

Parasitic infections are a sufficiently common cause of pleural disease that parasitosis should be considered in any effusion of unclear cause.^[7] They can involve the pleura with or without involvement of the adjacent lung. Parasitosis may be capable of determining immune reactions with release of eosinophilotactic substances and these chemotactic factors may be held responsible for the eosinophilia and EPE.^[8]

Among parasites that cause EPE the most frequent is *paragonimus* species^[9] which is endemic in eastern and south eastern Asia but can also be found in Africa, South America and even North America. Other

parasitic diseases reported to be associated with EPE are sparganosis,^[10] toxocariasis,^[11] cutaneous myiasis,^[12] loiasis,^[13] echinococcosis,^[14] ascariasis, amebiasis^[12] and giardiasis.^[15] Thirty nine cases of pulmonary paragonimiasis have been reported in literature from India out of which four patients presented with pleural effusion, the pleural effusion responded to the treatment of paragonimiasis.^[16]

The association of giardiasis with pulmonary eosinophilia has not commonly been reported. A single case of eosinophilic pleural effusion with coincidental intestinal infestation by *giardia lamblia* has been reported.^[17] Another report has attributed persistent bronchitis and nocturnal cough to giardiasis.^[8] But the association of bilateral pleural effusion with giardiasis has not been reported in literature. In our case, no causative etiology could be identified for the bilateral pleural effusion other than the findings of ova of giardia in the stools of the patients, a causal or a casual finding.

Considering the finding of ova for giardia as a casual finding, other causes of EPE were also kept as an etiologic possibility. Absence of Br asthma, sinusitis, and peripheral eosinophilia which was not markedly raised ruled out conditions such as Churg-Strauss syndrome, tropical pulmonary eosinophilia (TPE) and hypereosinophilic syndrome (HIES). Pleural biopsy was planned to rule out malignancy to which the patient did not give consent. Furthermore the patient showed marked symptomatic improvement on the first follow-up visit. Kravetz *et al.*^[18] have suggested that if improvement is noted, the patient can forego an extensive evaluation for other causes.

Our patient could have had bilateral pleural effusion of an unrelated etiology but its complete resolution concurrently with treatment of giardiasis with metronidazole makes this unlikely. Moreover, no recurrence of pleural effusion or the detection of any ova of giardia in the stool of the patient in a 2 years follow-up of the patient suggests giardia as the most probable cause

of bilateral pleural effusion.

Parasitic disease should be an important consideration in the differential diagnosis of undiagnosed EPE and investigations in this regard should be carried out in patients from endemic areas like India in otherwise labeled as idiopathic EPEs.

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