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Eosinophilic Pulmonary Fibrosis

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ABSTRACT

Pulmonary fibrosis is commonly characterized by inflammation of the alveolar wall, leading to derangement of normal alveolar architecture, and interstitial as well as intra-alveolar fibrosis. The eosinophil is a major source for several of these key pro-fibro genic cytokines during the early stages of fibrosis. The percentages of eosinophil in peripheral blood and Bronchoalveolar lavage fluid are essential parts of the evaluation. This review will focus on the recent findings, which suggest novel potential roles for the eosinophil in the pathogenesis of pulmonary fibrosis.

Keywords: Eosinophilia, Cytokines, Interleukin, TPE

INTRODUCTION

Pulmonary fibrosis, meaning scarring in the lung tissue, is an umbrella term for more than 200 different lung diseases that all look very much alike. In pulmonary fibrosis, the scar tissue builds up in the walls of the air sacs of the lungs, and eventually the scar tissue makes it hard for oxygen to get into the blood. Low oxygen levels (and the stiff scar tissue itself) can cause to feel short of breath, particularly when walking and exercising. The most common symptoms of pulmonary fibrosis are cough and shortness of breath. Symptoms may be mild or even absent early in the disease process. As the lungs develop more scar tissue, symptoms worsen. One third of patients suffering from tropical pulmonary eosinophilia develop fibrosis of lungs.

DESCRIPTION OF CASE

A male aged about 65 years presented to the Sanaria Lymphology Clinic and Research Center with complaints of dry, hacking, nonproductive cough, which was frequently paroxysmal and nocturnal. Asthma-like attacks were associated with breathlessness and wheezing. Other symptoms included weight loss, fatigue, and malaise. Dyspnea on exertion was uncommon. Chest findings were minimal. Rhonchi, crepitation (especially over the mid zones and bases) and wheezing could be auscultated. The absolute eosinophil count was 4,500/µL (reference value<400/µL). The serum immunoglobulin E level was 1400 units/mL (reference value for adults<158 IU/ml). The serum OG_4C_3 test for detection of circulating adult filarial antigen was moderately positive, i.e., 512 A.U (antigen units). The negative value is less than 128 A.U. The biochemical parameters of blood were within normal limits and fecal examination for intestinal parasites found negative.

Photomicrograph shows a combination of air space filling and interstitial thickening in a patient with chronic eosinophilic pneumonia. The air space exudate is a combination of histiocytes and eosinophils.

Images were obtained from the patient with progressive dyspnea over one month; acute eosinophilic pneumonia was diagnosed based on the clinical presentation and Bronchoalveolar lavage showing 51% eosinophils and response to therapy.

The chest radiograph finding includes increased Broncho-alveolar markings, diffuse interstitial lesions and mottled opacities in the lower lung fields. Bronchoalveolar lavage showed presence of eosinophils and response to therapy **(Figures 1 and 2)**.

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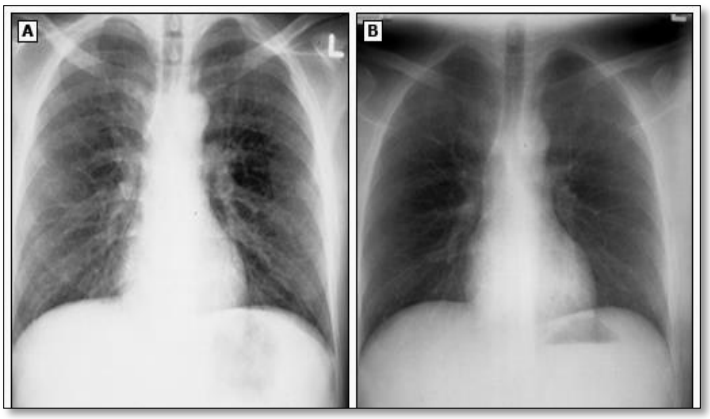

Figure 1. Tropical pulmonary eosinophilia. (A) Chest radiograph of the patient with symptoms suggestive of asthma. (B) Follow-up chest radiograph two weeks after diethylcarbazine therapy.

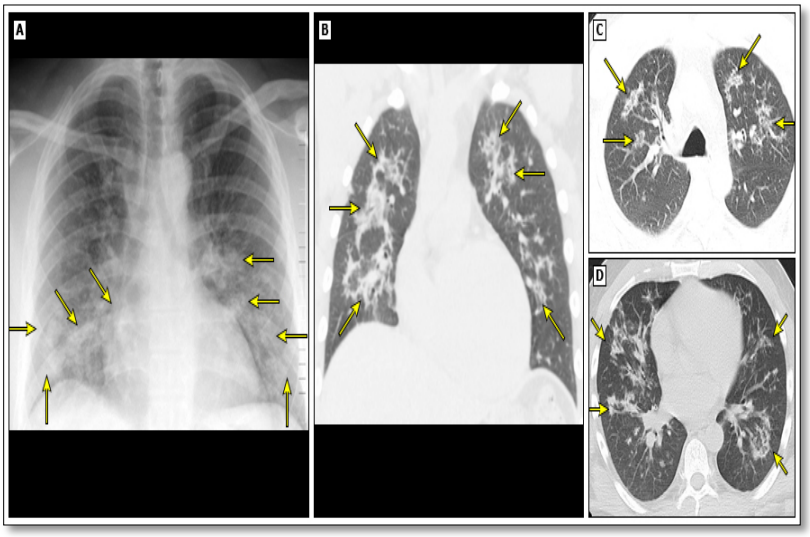

Figure 2. Acute eosinophilic pneumonia on radiography and CT.

Pulmonary function tests (PFTs) typically demonstrated a predominantly restrictive pattern together with mild to moderate airway obstruction.

A Broncho alveolar lavage (BAL) eosinophil count was done. It was found to be 48% (normal<30%).

The photomicrograph of lungs tissue of the patient revealed acute eosinophilic pneumonia with air space exudate and alveolar septal thickening **(Figure 3)**.

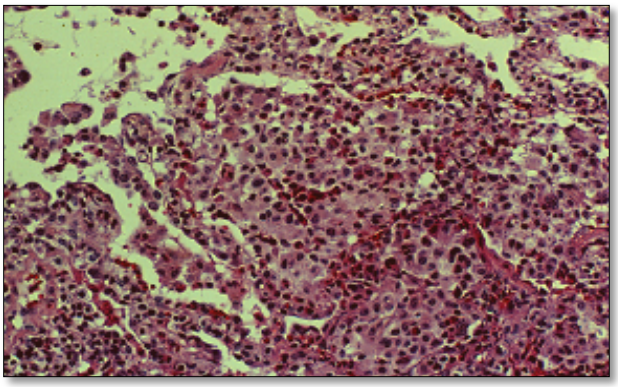

Figure 3. Chronic eosinophilic pneumonia histopathology.

DISCUSSION

Tropical pulmonary eosinophilia (TPE) is a clinical manifestation of lymphatic filariasis, a parasitic infection caused by filarial parasite *Wuchereria bancrofti* [1]. It is caused by an immune hyper-responsiveness to microfilariae trapped in the lungs. TPE is more common in individuals from the Indian subcontinent and occurs four to seven times more frequently in males than in females. There were an ongoing eosinophil interstitial infiltration and increasing pulmonary fibrosis. No microfilaria fragments identified in biopsied lung. The cardinal laboratory finding in TPE is blood eosinophilia, usually above 3000/μL [2]. The patient's absolute eosinophil count estimated every week was between 3000 and 4500/μL. An elevation in serum immunoglobulin E level is frequently observed; often above 1000 units/mL [3]. The diagnosis can be confirmed by marked elevations in filarial circulating antigen titers (OG_4C_3) [4,5]. Microfilariae are generally not detectable in peripheral blood. The chest radiograph findings included increased bronchoalveolar markings, diffuse interstitial lesions and mottled opacities (usually most prominent in the lower lung fields. Pulmonary symptoms (cough and breathlessness) are present in 25% of patients suffering from eosinophilia.

Pulmonary function tests (PFTs) typically demonstrate a predominantly restrictive pattern together with mild to moderate airway obstruction [6]. Pulmonary involvements are common in tropical eosinophilia and may result from eosinophilic infiltration of the lung with subsequent fibrosis, although the imaging studies in TPE can mimic miliary tuberculosis [7]. The presence of marked eosinophilia in TPE is an important distinguishing feature. Standard treatment consists of diethyl carbamazine (DEC) given at 6 mg/kg/day in three doses for 12 to 21 days [8]. DEC is active against both the microfilariae and adult worms, and DEC therapy is associated with a dramatic and rapid improvement in signs and symptoms in most cases. The restrictive and obstructive defects typically return toward normal if DEC is administered in the first few years of disease, although a low-grade eosinophilic alveolitis may persist. If DEC therapy is delayed, progressive interstitial fibrosis and irreversible impairment in pulmonary function can occur. Corticosteroid therapy has been used as adjunctive therapy to reduce inflammation in the acute setting but is not definitive therapy for TPE. Relapses occur in up to 20% of patients within the first five years of DEC therapy and are treated with a repeat course of the same treatment regimen.

Other agents used in the treatment of lymphatic filariasis, included oxycycline (which has efficacy against adult filarial worms via its action on their Wolbachia endosymbionts. Ivermectin (a rapid microfilaricide without efficacy on adult worms) and albendazole (which affects only adult worms) are also used [9]. Bronchospasm can generally be managed with bronchodilators, although short-term corticosteroids may be necessary in severe cases. Interleukin 5 (IL5), known as eosinophil differentiation factor (EDF), is a lineage-specific cytokine for eosinophilpoiesis [10].

CONCLUSION

Early recognition of an eosinophilic lung disease is crucial because initiation of proper treatment can often substantively alter the disease course and diminish morbidity and mortality. Ultimately, it is the combination of radiologic findings and both clinical and pathologic information that leads to the most accurate diagnosis.

LEARNING POINTS

- Patients belonging to tropical zone and suffering from eosinophilia are prone to pulmonary fibrosis.
- Early diagnosis and treatment of TPE prevent future disability and fibrosis of lungs.

CONTRIBUTOR

The author prepared the manuscript, provided the clinical data and images, conceived the case study, reviewed the literature and drafted the manuscript.

COMPETING INTERESTS

None declared.

PATIENT CONSENT

Obtained.

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