Tropical Pulmonary Eosinophilia – A Rare Presentation of a Common Disease in Tropics

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Research Article

Abstract: Tropical pulmonary eosinophilia is one of the many pulmonary infiltrates with eosinophilia. We are presenting a case with features of lung abscess in left lung field. The diagnosis was suspected and proved retrospectively after successful treatment with diethyl carbamazine (DEC). There was an elevation of gamma-glutaryl transpeptidase levels in our case, which may have a role in pulmonary infiltrates in tropical eosinophilia.

Keywords: Tropical pulmonary eosinophilia, lung abscess, DEC treatment and GGT

Introduction

Tropical pulmonary eosinophilia is one of the many pulmonary infiltrates with eosinophilia. It is caused by immunologic hyper-responsiveness to the filarial parasites Wuchereria bancrofti or Brugia malayi[1]. The intense eosinophilic alveolitis seen in acute tropical pulmonary eosinophilia is suppressed by 3 weeks of treatment with diethylcarbamazine citrate. A mild eosinophilic alveolitis along with radiological, physiological and haematological abnormalities, though with reduced intensity, persists in some patient's however[2].

Case presentation

A male patient by name Mahaveer, aged about 35years of age presented with fever, cough, wheeze, loss of weight for a period of one month. Patient lost about 5 kilograms of weight in month. He received various antibiotics including anti-tubercular treatment. The initial examination showed an ill looking pale dyspnoeic anxious patient with a pulse rate of 112/minute, respiratory rate about 30/minute, temperature of 99.80F. There was no no significant lymphadenopathy. Rhonchi were heard all over the lungs, heart did not reveal any abnormality apart from tachycardia. There was no visceromegaly. Examination of nervous system did not reveal any deficit. Haemoglobin was 7.4 Gm, total leukocyte count of 13,400/c.c. with 62 Neutrophil, 26 lymphocytes, and 12 % eosinophils. E.S.R. was 70 mm/F^{irst} hour. Blood sugar – 98mg / dL, blood urea - 32mg / dL serum Creatinine – 1.2 mg. Liver function test were within normal limits. The gamma-glutaryl transpeptidase (GGT) levels in serum were 126mg / dL (0 – 65mg for adult males). An X-ray chest (Figure 1) showed well defined cavity in left mid-zone, the same was noted in the X-ray taken 7, 20 days earlier. Old investigations revealed leukocytosis with eosinophils ranging from 02 to 14/mm^3. The patient admitted to acute medical care (AMC) unit with provisional diagnosis of un-resolving pneumonia R/O tuberculosis. Sputum examination at three occasions was negative for acid fast bacilli. Repeat blood counts on 3rd day of admission revealed an eosinophil count of 18%, while absolute eosinophil count of 3680/c.c. The diagnosis of TPE was based on the following clinical and laboratory criteria[4]: (1) clinical history supportive of exposure to lymphatic filariasis, (2) peripheral eosinophilia (eosinophil count, > 3000/c.c.), (3) elevated serum IgE levels (normal range, 0–380 IU / mL), (4) increased titres of anti-filarial antibodies, (5) peripheral blood specimen that tested negative for microfilaria, and (6) clinical response to diethylcarbamazine. Anti-filarial antibodies and IgE levels were not undertaken as there was no provision for estimating. Then a trial of diethyl carbamazine (D.E.C) 100mg thrice a day started on 4th day, apart from supportive medication. He showed slight improvement 3 days later. The temperature became normal from 4th day after revised treatment, dyspnoea and cough improved slowly, so also the general well being. The X-ray after a week showed slight improvement while the same taken after 2 weeks of D.E.C. therapy (Figure 2) revealed almost complete resolution. However D.E.C. was given for a total of 21 days, he gained a weight of 1.5 kilos after 30 days of admission. After discharge he was followed up to 2 years regularly and showed no sequel.

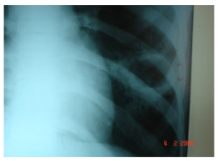

Figure 1: X-ray chest showing an abscess in left lower lobe at the time of admission

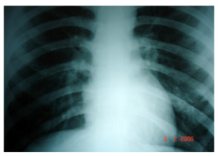

Figure 2: X-ray showing improvement in the lesion after 20 days

Discussion

Tropical pulmonary eosinophilia is one of the many pulmonary infiltrates with eosinophilia. It is caused by immunologic hyper-responsiveness to the filarial parasites Wuchereria bancrofti or Brugia malayi. Its clinical presentation includes nocturnal cough, dyspnea, wheezing, fever, weight loss, fatigue, interstitial mottling on chest radiograph, predominantly restrictive but also obstructive lung function abnormalities, and peripheral blood eosinophilia of more than 3000 per micro-liter. It can be distinguished from other PIE syndromes by the patient's history of residence in the tropics, by the presence of extraordinarily high levels of both serum IgE and anti-filarial antibodies, and by the dramatic clinical improvement after treatment with the anti-filarial drug diethylcarbamazine. Recent studies indicate that the compromised lung diffusion capacity of patients with acute tropical pulmonary eosinophilia is a function of the degree of the eosinophilic alveolitis present and that, despite a 3-week course of diethylcarbamazine, low-grade alveolitis persists in almost half of such patients; this persistent alveolitis is likely to be the cause of the progressive interstitial fibrosis seen in many untreated or inadequately treated patients with tropical pulmonary eosinophilia[1]. Tropical pulmonary eosinophilia is an occult form of human filariasis. The gamma-glutaryl transpeptidase found in the infective L3 stage larvae of Brugia malayi has been found to have similarities with the gamma-glutaryl transpeptidase present on the surface of human pulmonary epithelium. It has, therefore, been proposed that filarial gamma-glutaryl transpeptidase may play an important role in the pathogenesis of tropical eosinophilia. Airway hyper-responsiveness, manifesting as asthma-like syndrome, has been reported in tropical pulmonary eosinophilia and it has been suggested that interleukin-4 induces and interferon-gamma suppresses filarial-induced airway hyper-responsiveness. The intense eosinophilic alveolitis seen in acute tropical pulmonary eosinophilia is suppressed by 3 weeks of treatment with diethylcarbamazine citrate. A mild eosinophilic alveolitis along with radiological, physiological and haematological abnormalities, though with reduced intensity, persists in some patients[2]. TPE typically has a nonspecific presentation and may mimic a number of conditions; it is most often misdiagnosed as asthma because of pulmonary manifestations, such as paroxysmal cough and dyspnea [3,5,6,7]. Other commonly reported symptoms include malaise, fever, and weight loss [3]. Frimodt-Moller and Barton[8] in 1940 described a group of patients from a sanatorium of South India who had fever, cough, and chest pain and weight loss in association with massive blood eosinophilia. These patients had extensive bilateral miliary mottling in chest radiographs and were wrongly diagnosed as miliary tuberculosis. However, they were in good physical condition and did not have a high mortality as observed in miliary tuberculosis. They described this entity as "a pseudo-tuberculosis condition associated with eosinophilia". Rapid amelioration of signs and symptoms with diethylcarbamazine treatment is a hallmark of TPE. Although the clinical response to diethylcarbamazine is often marked, many patients are left with mild residual pulmonary disease following treatment[9].

Addendum

Author VN recollects and thanks his teachers at Kurnool Medical College, Kurnool, A. P. who taught him about TPE as a disease of symptoms of tuberculosis and signs of bronchial asthma.

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