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Case Report

Tropical pulmonary eosinophilia-mimicking acute severe asthma: a case report

Poulomi Roy*, Shalini Verma, Shrish Bhatnagar

Department of Pediatrics, Era's Lucknow Medical College and Hospital, Lucknow, Uttar Pradesh, India

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***Correspondence:**
Dr. Poulomi Roy,
E-mail: poulomiroy29@gmail.com

ABSTRACT

Tropical pulmonary eosinophilia (TPE) is a rare but serious infection characterized by wheezing, fever, and eosinophilia, with clinical features such as paroxysmal cough, wheezing, and dyspnoea. TPE is diagnosed by a history of filarial endemic regions and peripheral eosinophilia >3,000/mm^3. It can be easily missed but can be a differential for bronchial asthma and tuberculosis. A 15-year-old male with cough and breathlessness for three years was diagnosed with TPE after laboratory investigations showed leucocytosis with eosinophilia. A provisional diagnosis was considered, and diethylcarbamazine (DEC) was started at a dose of 6 mg/kg body weight for 21 days, with improvement in the patient's symptoms. TPE affects 51.4 million people globally and is often misdiagnosed as other conditions like bronchial asthma, military tuberculosis, and interstitial lung disease. The diagnostic criteria for TPE include a history of residence or travel to a filarial endemic region, paroxysmal and nocturnal cough with dyspnoea, leucocytosis with peripheral blood eosinophilia>3000/mm^3, elevated serum IgE and filarial antibody titres, pulmonary infiltrations in chest X-ray, and clinical improvement with DEC.

Keywords: TPE, Filariasis, DEC, Peripheral eosinophilia

INTRODUCTION

Tropical pulmonary eosinophilia (TPE) is a rare but serious manifestation of infection with lymphatic filarial parasites.[1] This condition, which is mostly observed in the Indian subcontinent and other tropical regions, is characterised by wheezing, fever, and eosinophilia. The most common etiology is *Wuchereria bancrofti* and *Brugia malayi*.[1] Common clinical features seen are paroxysmal cough, dyspnoea, wheezing and systemic manifestations like fever and weight loss. A history of residence in a filarial endemic region and a finding of peripheral eosinophilia >3,000/mm^3 should initiate consideration of this disease. Other criteria for the diagnosis of TPE include an absence of microfilariae in the blood, high titres of anti-filarial antibodies, raised serum total IgE >1,000 U/mL, and a favourable response to the anti-filarial, DEC citrate, which is the recommended treatment.[3] The radiological features include ill-defined nodules and interstitial infiltrates. Spirometry usually shows a mixed obstructive and restrictive pattern.[4] The diagnosis can be easily missed since it is rarely encountered and can mimic many other conditions. This case should be kept in mind as a differential for h/o cough with wheezing. Some of the differentials include bronchial asthma and tuberculosis. Here we present a case of a 15-year-old male who was initially misdiagnosed as a case of bronchial asthma and treated later on diagnosed as a case of TPE. Thus, it should be noted that TPE may be wrongly diagnosed. We also stress early diagnosis and treatment of this condition to avoid unfavourable outcomes.

CASE REPORT

A 15-year-old male complained of cough and breathlessness on exertion on and off for 3 years. The cough was non-productive, more at night. There was no

history of hemoptysis, chest pain, fever, night sweats, tubercular contact, or significant weight loss. There was a history of on and off bronchodilator use. There was no significant family history of allergic conditions like atopy or asthma.

On presentation to our hospital with a pulse rate of 88 beats per minute, respiratory rate of 22 per minute with SpO_2-97% at room air axillary temperature of 97.6 F, and blood pressure of 110/74 mmHg. On general examination, there was no significant finding. On auscultation bilateral wheeze was present. Rest systemic examination was within normal limits. The patient was initially managed with supportive care and bronchodilators were started but there was poor response. Initial laboratory investigations were as follows Hb 14.3 g/dl, Total leukocyte counts 30,800 cells/cumm, eosinophils 80%, absolute eosinophil count 26850 cell/cumm, platelet count 1.6 lakhs (Table 1). Peripheral blood smear showed leucocytosis with eosinophilia, no abnormal cells/ hemiparasites were seen. Chest x-ray showed reticulonodular opacities in both lungs. (Figure 1). A restrictive pattern was noted in the pulmonary function test. A revised diagnosis of TPE was considered. Serum IgE 1501 IU/ml and filaria antibody IgG and IgM detected. In stool examination, no parasites were detected (Table 1).

The patient was initially treated with bronchodilators and after the diagnosis of TPE was established DEC was started at a dose of 6 mg/kg body weight in three divided doses for 21 days. The patient was compliant. Over the course of treatment, the patient had significant improvement in symptoms, and wheeze improved. On follow-up, there was complete resolution of symptoms and investigations showed decreased peripheral eosinophilia.

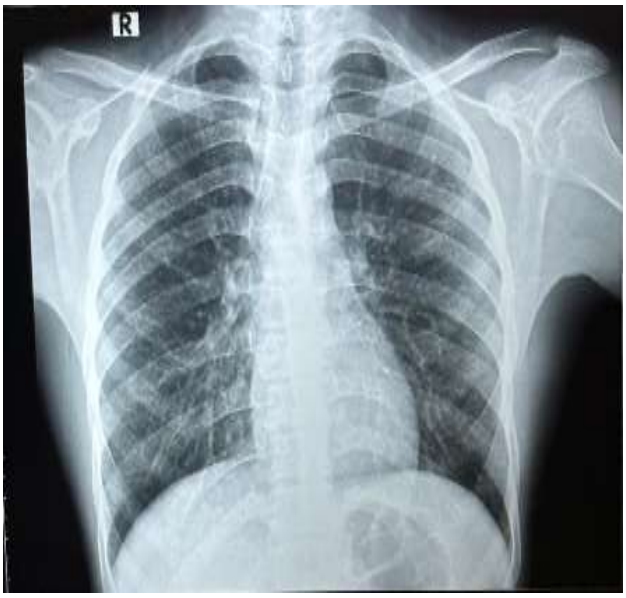

Figure 1: Chest X-ray showing bilateral reticulonodular opacities in both lung fields.

Table 1: Laboratory investigations.

Parameters	Observed value	Normal value
Hb (g/dl)	14.3	10-15.5
TLC	30,800	5000-10,000
D/L/C	07/12/80/01	
AEC (IU/ml)	24640	<170
RBC (million cells/mcl)	4.25	4.0-5.5
Platelet (lakhs/mcl)	1.6	1.5- 4.5
Serum IgE (IU/ml)	1501	0-100
Filaria antibody IgG and IgM	Detected	

DISCUSSION

TPE occurs due to a hypersensitivity reaction to the microfilariae of *Wuchereria bancrofti* and *Brugia malayi* in the pulmonary microcirculation.[5] Globally, 51.4 million people are estimated to be infected by filariasis.[6] We present a case of a 15-year-old male, resident of the northern part of India presenting to our facility with symptoms of prolonged non-productive cough and no relief from bronchodilator use, diagnosed previously as difficult to treat asthma. A revised diagnosis of TPE was established based on history, examination, and clinical findings. DEC was started to which the patient responded well.

TPE requires a high index of clinical suspicion and should be considered in patients who present with peripheral eosinophilia and respiratory tract symptoms, particularly when they come from areas that are highly endemic for filaria. Several reports have revealed TPE being misdiagnosed with other clinical conditions such as bronchial asthma, military tuberculosis, and interstitial lung disease.[7] A case report by Ray et al reported a similar TPE case misdiagnosed as military tuberculosis.[8] A study by Randev et al reported a case of a 5-year-old girl who was misdiagnosed with bronchial asthma.[9] A case report by Behera et al reported a case of TPE masquerading as interstitial lung disease.[10] The diagnostic criteria for TPE include a history of residence or travel to a filarial endemic region, paroxysmal and nocturnal cough with dyspnoea, leucocytosis with peripheral blood eosinophilia>3000/mm^3 raised serum IgE and filarial antibody titers, infiltrations in chest X-ray and, improvement on treatment with DEC.[7]

CONCLUSION

Whenever a patient from a filaria-endemic area presents with cough, breathing difficulty, or wheezing, TPE should always be taken into consideration. TPE can result in respiratory impairment, as well as mortality, in an untreated individual. Hence, early detection and DEC therapy are essential for reducing morbidity and mortality.

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