

Persisting "asthma" in tropical pulmonary eosinophilia

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We report a case of tropical pulmonary eosinophilia which masqueraded as asthma for four years.

Case report

A 37 year old Indian man, resident in England for 11 years, gave a four year history of intermittent cough, wheeze, and breathlessness. At the onset of his illness asthma had been diagnosed and bronchodilators prescribed. He had never smoked. Referral to a chest clinic was prompted by worsening symptoms for four months, accompanied by sweating and weight loss of 6.5 kg. On examination he looked well but he had a persistent dry cough. There was diminished chest expansion, and auscultation revealed generalised expiratory wheeze as well as many late inspiratory bilateral basal and mid zone crackles. Detailed questioning established that his symptoms had originally started one month after a holiday in north west India.

The chest radiograph showed 2-4 mm nodules in the mid and lower zones superimposed on a background of hazy shadowing (fig 1). Simple spirometry indicated a restrictive ventilatory defect: FEV₁ was 1.1 litres (37% of predicted), forced vital capacity (FVC) 1.35 l (34% of pre-

dicted) and FEV₁/FVC ratio 81%. Total lung capacity (TLC) was reduced at 2.3 l (41% of predicted) and gas transfer was impaired (TLCO = 16.7 ml min⁻¹ mm Hg⁻¹ (5.6 mmol min⁻¹ kPa⁻¹); 56% of predicted). The total peripheral white blood count was 11.8 × 10⁹ l, with 66% eosinophils. The filarial fluorescent antibody titre was very high at 1/512. Total serum immunoglobulin E (IgE) was considerably raised at 5700 U/ml (mean normal 122 U/ml). Stool examinations for parasites and blood film examinations for filariae (including nocturnal samples) gave negative results. Skinprick tests for common allergens, including *Aspergillus fumigatus*, all gave negative results; and precipitins against *Aspergillus* were not detected in the serum.

After these investigations tropical pulmonary eosinophilia was diagnosed and diethylcarbamazine (total dose 11.4 g over 25 days) was added to the patient's pre-existing regimen of salbutamol and beclomethasone inhaled from pressurised aerosols. Within a week of starting this treatment his cough and breathlessness had improved and his peak expiratory flow rate (PEFR) rose simultaneously (fig 2). Oral aminophylline (225 mg twice daily) was added to his treatment in an attempt to minimise early morning dips in PEFR.

The chest radiograph had cleared by eight weeks. Repeat lung function tests at this time showed appreciable improvement: FEV₁ was 2.0 l (67% of predicted), FVC 2.4 l (60% of predicted), TLC 3.3 l (59% of predicted), and TLCO 23.2 ml min⁻¹ mmHg⁻¹ (7.8 mmol min⁻¹ kPa⁻¹; 78% of predicted). The absolute eosinophil count fell to

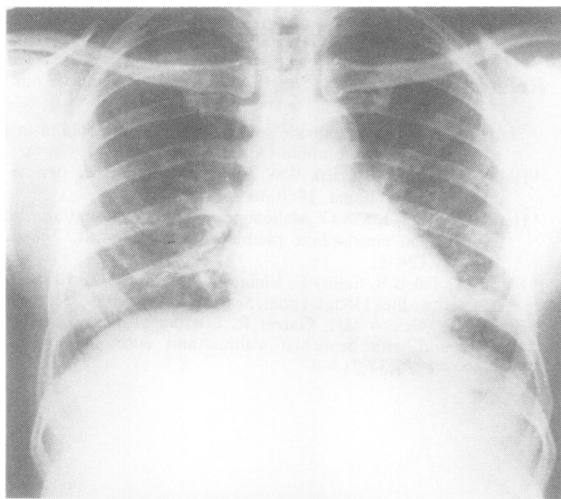


Fig 1 Chest radiograph at presentation.

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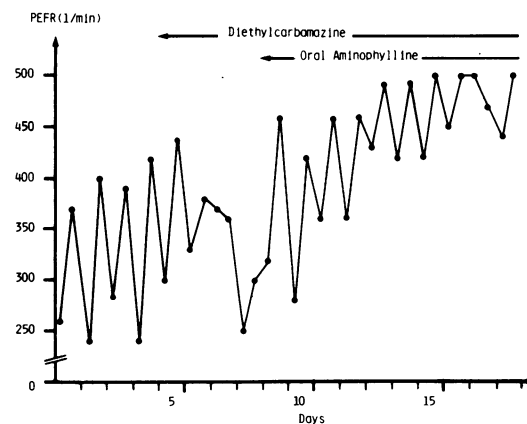


Fig 2 Peak expiratory flow rate (PEFR) before and after the start of treatment, showing maximum and minimum PEFR values within each 24-hour period.

0.6×10^9 l although the fluorescent filarial antibody titre remained 1/256. The total serum IgE fell to 840 U/ml. The patient had been able to discontinue all bronchodilator treatment on his own initiative and he has remained well for six months without any treatment.

Discussion

Tropical pulmonary eosinophilia is endemic in filarial zones, most commonly in India, South East Asia, Africa, and South America. There are occasional reports of the disease among recent immigrants to the United States,¹ but we have failed to find any documented cases diagnosed in Britain in the last 30 years.

The illness usually presents with respiratory symptoms, most commonly paroxysmal cough, breathlessness, and if symptoms are recent wheezing.² The disease is nevertheless a systemic one, and features such as fever, lassitude, and weight loss are frequent.² Current diagnostic criteria include considerable blood eosinophilia ($> 3.0 \times 10^9$ /l), a high filarial antibody titre, chest radiographic changes in the form of lung infiltrations with or without small nodules (a normal film does not exclude the diagnosis), and resolution of the illness after diethylcarbamazine.^{2,3} The filarial aetiology of tropical pulmonary eosinophilia is beyond doubt, although the precise immunopathological mechanisms remain uncertain. Microfilariae are not seen in blood, but their presence in lung biopsy specimens suggests that the illness is an immunological response to microfilariae trapped in pulmonary capillaries.^{4,5} The filarial antibody titre is certainly much higher than in other forms of filariasis,⁶ and serum IgE levels are very high.⁷ Lung function tests show different patterns at various stages of the disease. Within the first month the defect is one of airflow obstruction, an accompanying restrictive defect occurring between one and four months.⁸ In chronic cases with established fibrosis a purely restrictive lung defect predominates.⁹

Our patient fulfilled all the criteria for the diagnosis of tropical pulmonary eosinophilia. There was an appreciable clinical response to diethylcarbamazine, as well as objective improvements in the eosinophil count, total serum IgE concentration, and results of lung function tests, although the latter did not return to predicted values. It is worth emphasising that cough and intermittent wheeze were prominent symptoms, and that the clinical diagnosis of asthma had prevailed for four years. Serial PEFR recordings before treatment supported the history of intermittent wheeze, showing the circadian variability of airflow obs-

truction described in asthma¹⁰ (fig 2). This is not a recognised feature of chronic tropical pulmonary eosinophilia, where lung fibrosis and restriction are reported.^{2,9}

We report this case for three reasons. Firstly, increased ease of international travel makes tropical pulmonary eosinophilia a "tropical" disease which may be encountered in Britain. Secondly, variable airflow obstruction may persist in chronic tropical pulmonary eosinophilia, making the diagnosis a possibility even in "asthmatics" who left an endemic area several years before. Finally, uncritical acceptance of PEFR measurements may lead to a diagnosis of asthma,¹⁰ hindering further consideration of the underlying pathology in this disease.

References

- McKeehan FR. Tropical eosinophilia in an America college: a report of two cases. *J Am Coll Health Ass* 1975;**24**:92-4.
- Udwadia FE. Pulmonary eosinophilia. In: Hertzog H, ed. *Progress in respiration research*. 7. Basel: Karger, 1975:35.
- Danaraj TJ. Eosinophilic lung (tropical eosinophilia). In: Maegraith BG, Gilles HM, eds. *Management and treatment of tropical diseases*. Oxford: Blackwell Scientific Publications, 1971:151.
- Joshi W, Udwadia FE, Gadgil RK. Etiology of tropical eosinophilia. A study of lung biopsies and review of published reports. *Am J Trop Med Hyg* 1969;**18**: 231-9.
- Webb JKG, Jöb CK, Gault EW. Tropical eosinophilia: demonstration of microfilariae in lung, liver and lymph nodes. *Lancet* 1960;**i**:835-42.
- Danaraj TJ, Da Silva LS, Schacher JF. The serological diagnosis of eosinophilic lung (tropical eosinophilia) and its etiological implications. *Am J Trop Med Hyg* 1959;**8**:151-9.
- Neva FA, Kaplan AP, Pacheco G, Gray L, Danaraj TJ. Tropical eosinophilia. A human model of parasitic immunopathology, with observations on serum IgE levels before and after treatment. *J Allergy Clin Immunol* 1975;**55**:422-9.
- Nesarajah MS. Pulmonary function in tropical eosinophilia. *Thorax* 1972;**27**:185-7.
- Udwadia FE. Tropical eosinophilia: a correlation of clinical histopathologic and lung function studies. *Dis Chest* 1967;**52**:531-8.
- Hetzel MR. The pulmonary clock. *Thorax* 1981;**36**: 481-6.