



Filarial Tropical Pulmonary Eosinophilia: a condition masquerading Asthma, a series of 12 cases.

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Devki Nandan : Dr.Nandan supervised in guiding the diagnosis and management of the cases, critically reviewed and revised the manuscript, and approved the final manuscript as submitted.

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Filarial Tropical Pulmonary Eosinophilia: a condition masquerading Asthma, a series of 12 cases.

ABSTRACT:

INTRODUCTION: Tropical Pulmonary Eosinophilia (TPE) is a form of occult filariasis, clinically characterized by paroxysmal cough, wheezing and dyspnea which is often misdiagnosed and treated as asthma. These manifestations result from a host immune response to trapped antigens of the Microfilarial parasites *Wuchereria bancrofti* or *Brugia malayi* in the pulmonary microcirculation.

CASE STUDY: We describe 3 rare presentations of TPE (cor pulmonale, cystic lung disease, and respiratory distress mimicking acute severe asthma) in our series of 12 cases. All cases were from filaria endemic areas and presented with cough, wheezing and dyspnea, either alone or in combination. Subsequent work-up revealed peripheral eosinophilia, raised serum IgE levels and positive serum filarial antibody and/or antigen in all the cases.

RESULTS: All patients were treated with diethylcarbamazine (DEC), while few required inhaled /systemic corticosteroid. Prompt improvement in clinical symptoms with a decrease in eosinophil count was seen in all. Two cases relapsed requiring a second course of DEC. Long-term outcome was good, however, there was a persistence of restrictive lung function and echocardiographic feature of pulmonary hypertension in the patients with cystic lung disease and cor pulmonale, respectively.

CONCLUSION: TPE should always be considered in patients from filaria endemic areas presenting with cough, dyspnea or wheezing. High eosinophil count ($>3 \times 10^9$ cells) with raised IgE level (>1000 IU/ml) in such cases should alert the physician to look for TPE. Early diagnosis and treatment can prevent disease progression and complications.

Key words: Tropical Pulmonary Eosinophilia, Asthma, Cor Pulmonale, Cystic lung disease, Reticulonodular shadows.

Abbreviations: TPE: Tropical Pulmonary Eosinophilia; DEC: Diethylcarbamazine; PFT: Pulmonary Function Test; ECHO: Echocardiography; FVC: Forced Vital Capacity; FEV1: Forced Expiratory Volume in one second; HIV: Human Immunodeficiency Virus; ECG: Electrocardiogram; CT: Computed Tomography; HRCT: High Resolution Computed Tomography.

INTRODUCTION:

Filarial tropical pulmonary eosinophilia is a form of occult filariasis resulting from host immune response to antigens derived from the microfilarial parasites, *Wuchereria bancrofti* and *Brugia malayi* [1]. It was first described in 1940 by Frimodt-Moller as a syndrome of wheezing, fever, eosinophilia and bilateral mottling of the lungs [2]. Lymphatic filariasis is endemic in tropical and subtropical areas of South America, Asia, Oceania and Africa. It is estimated that more than 120 million people worldwide are infected with lymphatic filariasis, more than 90 percent of which are due to *W. bancrofti*, while the remainder is due largely to *B. malayi* [3]. Filariasis is a major public health problem in the Indian subcontinent with heavily endemic states being Bihar, Andhra Pradesh, Orissa, Tamil Nadu, Gujarat and Kerala. Filarial TPE is found in <0.5 % of filarial infections [1]. The antigenic constituents of the degenerating microfilariae trapped in the pulmonary microcirculation trigger an immune response which include type I, II and IV hypersensitivity reactions [4, 5, 6]. Eosinophils play a central role in the pathogenesis causing severe eosinophilic inflammation of the lower airways [7]. Clinical manifestations are non-specific, but respiratory symptoms are predominant [4]. The respiratory symptoms chiefly include cough, breathlessness, wheezing

and chest pain due to which TPE is often misdiagnosed as asthma especially in endemic areas [8]. Clinical suspicion and screening are important tools in early diagnosis and treatment of TPE. Presence of peripheral eosinophilia and raised serum IgE in such clinical settings should highlight the need for specific laboratory tests to detect TPE. Both antigen and antibody detection tests are available. Antibody-based diagnostic assays use four recombinant antigens: Bm14, WbSXP, BmSXP and BmR1 [9,10,11,12,13]. These are based on the detection of anti-filarial IgG4 antibodies. The BmR1 ELISA is highly sensitive (~100%) for the detection of *Brugia malayi*, while Bm14 ELISA detects both *W. bancrofti* and *B. malayi* (sensitivity~91%-96%). Both WbSXP and BmSXP are highly sensitive (91% and 95% respectively) and specific (96-99%) for *W. bancrofti*. Filarial antigen tests are also available and are considered more specific because some antibody tests may exhibit cross-reactivity with other helminthes causing TPE like syndromes [14]. Detection of the Og4C3 antigen by ELISA is sensitive and specific for *W. bancrofti* [15,16,17], however, it fails to detect *B.malay*i infection. Hallmark of filarial TPE is a prompt response to diethylcarbamazine (DEC), however untreated or partially treated cases may progress to interstitial lung disease [4].

Case Study:

We present a series of 12 patients with TPE diagnosed in the Pediatric Pulmonology clinic and Pediatric Pulmonology ward of PGIMER, Dr.RML Hospital, a tertiary care teaching hospital situated in New Delhi. The diagnosis of TPE was based on the following criteria: (1) residence in an endemic area, with a paroxysmal cough, dyspnea and wheezing, (2) presence of peripheral eosinophilia (eosinophil count $>3 \times 10^9/L$; normal range : $0.1 - 0.6 \times 10^9 /L$ [18]) (3) raised serum total IgE >1000 U/ml, (4) elevated titer of anti-filarial antibody or detection

of anti-filarial antigen in blood, (5) favourable response to DEC and; (6) absence of microfilaria in both day and night peripheral smear samples [19].

Description of cases

Case 1. A 14-year-old boy presented with one-year history of cough, shortness of breath and left-sided chest pain. The cough was dry and persistent with no diurnal variation or specific aggravating factors. The dyspnea was initially brought on by activities such as running upstairs and while playing but for the last two months, he was experiencing it on walking short distances, associated with palpitation. He had no history of orthopnoea or paroxysmal nocturnal dyspnea. He was receiving nebulizations occasionally for his symptoms. His past history was insignificant.

On examination, his vitals were stable with SpO₂ of 96% at room air. Cardiovascular examination revealed grade 2 left parasternal heave, with palpable P₂, single loud S₂ and pan-systolic murmur at the left lower sternal edge. Spirometry showed FVC of 85% and FEV₁ of 81%, with no significant bronchodilator reversibility (only 7% improvement in FEV₁ from baseline 15 minutes after nebulized salbutamol). Chest x-ray revealed prominent pulmonary conus, and ECG showed right axis deviation, P pulmonale, and right ventricular strain pattern. On 2D-echocardiography dilated right chambers, severe tricuspid regurgitation (PG=68mmHg) and moderate pulmonary regurgitation were observed (fig 1). Blood investigations showed eosinophilia (3.876×10^9 cells/L), serum IgE level of 3760 IU/ml and positive serum filarial antigen. Stool examination was negative for ova and cysts. So a possibility of TPE was considered. Meanwhile, contrast enhanced CT chest (fig 2a and 2b) revealed centrilobular nodules in bilateral postero-basal segments, mild cardiomegaly with mild dilatation of right ventricle and an enlarged pulmonary artery at its bifurcation. These features were also suggestive of pulmonary embolism but on CT angiography there was no

such evidence. Work up for rheumatic heart disease, connective tissue disorders, vasculitis, allergic bronchopulmonary aspergillosis, and HIV infection were negative. So he was treated with DEC (300mg), inhaled bronchodilator and low dose furosemide.

At 21 days follow up cough had resolved however he was still dyspneic on exertion. The eosinophil count had decreased to 0.3×10^9 cells/L, while 2D ECHO revealed moderate tricuspid regurgitation with a gradient of 55 mmHg. At this point, prednisolone was added (0.5 mg/kg/day) and continued for 6 weeks, along with furosemide. At 6 months follow up he was asymptomatic, with normal eosinophil count and 2D ECHO showing trivial TR (gradient of 45mmHg). He was continued on low dose furosemide and kept on regular follow up.

Case 2: A 14-year-old boy presented with two-year history of cough, exertional dyspnea, and occasional left-sided chest pain. He had no orthopnea or paroxysmal nocturnal dyspnea. His past and family history were unremarkable. He was taking regular anti-asthma medications without any significant improvement until he was admitted to our hospital. Physical examination revealed decreased air entry over the left infraclavicular, axillary and suprascapular areas. There were no added sounds. Rest of the examination findings were within normal limits. Laboratory investigation revealed an absolute eosinophil count of 2.350×10^9 cells/L, with a serum IgE of 6400 IU/ml. Stool examination did not reveal ova or cysts. Chest x-ray showed a large thin-walled cavity in the left upper zone with a fluid level. HRCT chest (fig 3) showed a thin-walled cavity with a dependent fluid level in the left upper lobe with mild perifocal fibrotic opacities and traction bronchiectasis with air trapping in the adjoining parenchyma. Test for filarial antigen and antibody in serum were positive. Pulmonary function test showed both restrictive and obstructive pattern with FVC of 68% and FEV1 of 61% with only 5% improvement in FEV1, 15 minutes after nebulized salbutamol. He was treated with 300mg of DEC daily for three weeks along with inhaled steroid and bronchodilator. After three weeks of treatment, his symptoms resolved and the

absolute eosinophil count decreased to 0.2×10^9 cells/L /mm³, however he continued to have restrictive change on spirometry (FVC=72%).

Case 3: A 7-year-old boy presented to the emergency room with complaints of cough for 7 days, and fast breathing for 2 days. He had no fever or preceding upper respiratory infection, neither did he have similar episodes in the past. Clinical examination revealed a tachypneic child with suprasternal, intercostal and subcostal retractions, with SpO₂ of 89% at room air and bilateral wheeze on auscultation. He was initially treated with nebulized salbutamol and budesonide. Chest x-ray showed bilateral reticulonodular shadows (figure 4), while blood investigations revealed an eosinophilia of 25.8×10^9 cells/L, and serum IgE level of 21000 IU/ml. Test for serum filarial antigen was positive. He was treated with DEC (6mg/kg/day) and a short course of oral prednisolone (5 days). At 21 days follow up he was asymptomatic, with an eosinophil count of 0.4×10^9 cells/L.

Results:

Demographic profiles of the cases are shown in table 1. The median age was 9.5 years, with a male to female ratio of 3:1. Eleven patients were from Bihar which is a state in North India, known to be endemic for filariasis, (figure 5) [20], which has an average temperature of 22°C in winters and 39° C in summers. One patient was from a city in Eastern Uttar Pradesh bordering Bihar. The median duration of illness was 15 months, with the shortest being 7 days (patient 8) and longest being 72 months. Table 2 shows the clinical features at presentation. Cough (91.6%), breathlessness (75%) and wheezing (75%) were the most common presenting symptoms. Three (25%) of them presented in the emergency with severe respiratory distress mimicking acute severe asthma. None of the patients had fever, organomegaly, lymphadenopathy or lymphedema.

One patient had a previously documented diagnosis of asthma (patient 6) and was on high dose inhaled budesonide for one year, without any significant improvement. Nine (75%) patients had received only symptomatic treatment for their symptoms with nebulized/ inhaled bronchodilators and steroids before being referred to us.

Chest radiography was done on all the patients. Reticulonodular infiltrates were seen in 6 patients (50%). One patient had a large cyst on the left upper zone, while another one had prominent pulmonary conus. Rest of the patients (33%) had normal chest x-ray findings. Spirometry was done in 3 patients (patient 6, 10 and 11), which revealed a combined obstructive and restrictive pattern in two patients (6 and 11), and a normal pattern in one patient (patient 10). Fraction of exhaled nitric oxide (FENO) was not measured in any of the cases.

The laboratory parameters are shown in table 3. The eosinophil count was $> 3 \times 10^9$ cells/L in 10 patients (83%), with a median count of 7.024×10^9 cells/L. Serum IgE level was elevated in all the cases, with a median level of 5046 IU/ml. Ten patients tested positive for filarial antigen, while three patients tested positive for filarial antibody (IgG by ELISA). Stool examination for ova and cysts; and peripheral smear for microfilaria were negative in all cases. All of them were treated with DEC for 21 days and followed up regularly. Two (16.6%) of the cases relapsed requiring a second course of DEC.

Discussion:

Pulmonary infiltrates with eosinophilia include a group of disorders that are characterized by pulmonary symptoms, blood/sputum eosinophilia and/or abnormal chest radiograph, caused by a variety of infectious, inflammatory, and allergic conditions. The differential diagnosis includes Loeffler syndrome secondary to helminth infection or drug reaction; Vasculitis such as Granulomatosis with Polyangitis, Churg-Strauss syndrome, Polyarteritis nodosa; Allergic

bronchopulmonary aspergillosis and Chronic eosinophilic pneumonia [21]. TPE is one such condition which is unique and often goes undiagnosed. This series aims to describe its clinical presentation and how it can be identified, or differentiated from other conditions, specially asthma, by some of its characteristics such as prevalence in filaria endemic areas, elevated peripheral eosinophils and serum IgE levels; and prompt response to treatment with Diethylcarbamazine. Diagnosis of TPE can further be confirmed by detection of filarial antigen and/or anti filarial antibody in the serum [19].

TPE is most commonly seen in the age group between 15-40 years, with a male to female ratio of 4:1[22]. In our series, the median age at presentation was 9.5 years with a male to female ratio of 3:1. Filarial TPE predominantly presents with cough, breathlessness, chest pain and wheezing [4]. In our series, the most common presenting symptom was cough, followed by breathlessness and wheeze. Symptoms are usually nocturnal however we did not observe any symptoms with diurnal variation in our cases. Extra-pulmonary manifestations were not seen in our series.

Our series highlight the importance of awareness about TPE among physicians, as seen in one case in which the patient had been diagnosed and treated as asthma with high dose inhaled corticosteroid for one year without significant improvement, while another nine (75%) gave a history of receiving nebulized bronchodilators and steroids each time for their recurrent symptoms while remaining undiagnosed. Three of our cases presented in the emergency room with respiratory distress mimicking acute severe asthma. The symptoms of TPE mimic that of asthma, however, failure to respond to asthma medications, with the presence of peripheral eosinophilia and history of travel to or being from an endemic area should raise the suspicion of TPE.

Cor pulmonale is not a usual presentation; however, one case presented with pulmonary artery hypertension and dilated right heart chambers secondary to TPE. Association between pulmonary hypertension and filariasis has been reported in a study conducted in Sri Lanka by Obeyesekere I and De Soysa N [23].

Spirometry in TPE usually reveals mixed obstructive and restrictive pattern [24,25]. Two out of three patients in our series in whom spirometry was performed had mixed obstructive and restrictive pattern. None of the patients showed bronchodilator reversibility, that is, an improvement in FEV1 of more than 12% from baseline, 15 minutes after inhaled or nebulized bronchodilator (salbutamol)[26]. Radiography findings in TPE vary, the most common presentation being reticulonodular shadows. Other findings include miliary mottling, bronchiectatic changes, lymphadenopathy, cavitation, consolidation or pleural effusion, and interstitial shadows. Literature shows that up to 20% patients may have normal chest x-ray, although we observed a higher percentage of patients (33%) with normal chest radiography [4]. Six (50%) cases had reticulonodular shadows, while one had a cystic lesion and one showed prominent pulmonary conus. However, chest x-ray findings are not specific and should not be used as criteria for ruling out TPE. We also observed a rare radiological finding of a large cyst in the left upper lobe in one of our cases. Various atypical presentations of TPE such as lung mass, consolidation, and pleural effusion have been reported in adults. Viswanathan R [27] reported an atypical radiological finding of a cavity in the center of a densely mottled area, observed during his study on 205 adult patients with TPE.

In TPE, peripheral eosinophilia is striking, that may range from 3 to 80×10^9 cells/L [21,24]. All patients in our series had peripheral eosinophilia (median 7.024×10^9 cells/L), however, in two of our cases, eosinophil count was less than 3×10^9 cells L. Markedly elevated levels of IgE was also seen in all the cases (median 5046 IU/ml) [28].

Treatment of TPE is with oral DEC, given at a dose of 6 mg/kg/day in three divided doses for 21 days [29]. Steroids have been reported to have beneficial effects [4], however, the exact dose and duration in combination with DEC is not determined. All patients in our series were treated with 21 days course of DEC, and all of them responded to treatment. Three cases with respiratory distress required a short course of systemic steroid (prednisolone 2mg/kg/day for 5 days), while one patient with pulmonary hypertension was given a 6 weeks course of low dose prednisolone. Two (16.6%) of the cases relapsed and required a second course of DEC.

This study highlights the need for physicians to have awareness about screening and diagnosis of TPE in patients from filaria endemic areas. Because of similarity in clinical presentation, it is often misdiagnosed and incorrectly treated as asthma, which also contributes further to delay in diagnosis and progression of the disease. However it is also noted that despite having symptoms for prolonged duration, some of the patients did not have any complications, highlighting the need to further understand the natural course of this condition. Further it is difficult to make any statistical conclusions from this study because of the small sample size and the retrospective design.

Conclusion: TPE is a chronic progressive illness which may lead to irreversible pulmonary damage and permanent deficits in pulmonary function when left untreated [4]. It is also a potential mimicker and often misdiagnosed as asthma. It is therefore vital for physicians to keep a high level of suspicion of TPE in patients from filaria endemic areas presenting with cough, dyspnea or wheezing. High eosinophil count ($>3 \times 10^9$ cells/L) with raised IgE level (>1000 IU/ml) in such cases should alert the physician to look for TPE. The outcome is linked to chronicity of disease and advanced disease may show a partial response to treatment probably due to already established pulmonary fibrosis.

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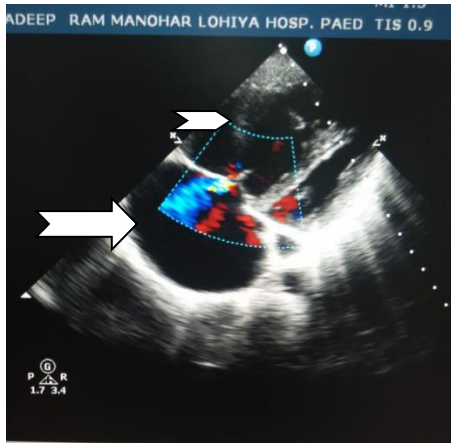


Fig. 1 (case 1): 2D ECHO showing dilated RA (arrow) and RV (arrowhead), with flattened intraventricular septum and severe TR (gradient of 68 mmHg)

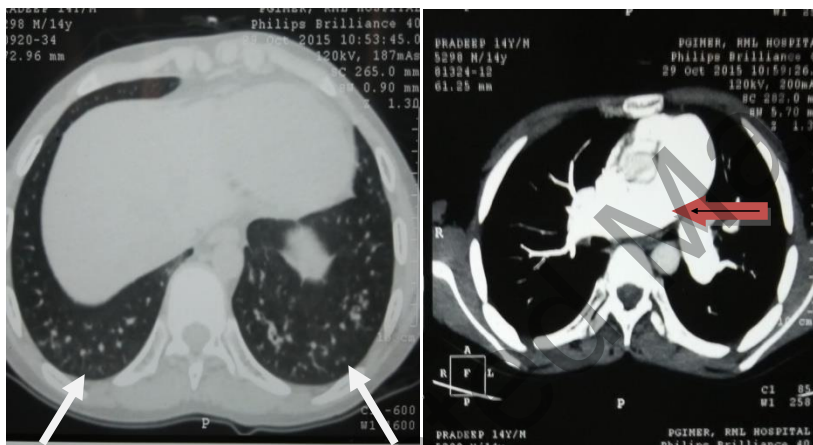


Fig.2 (case 1): CT chest showing bilateral centrilobular nodules in the postero-basal segments (white arrows), and CT angiogram film showing dilated pulmonary artery (red arrow).

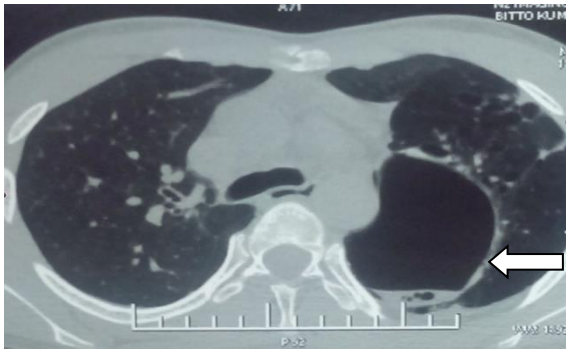


Fig. 3(case 2): CT chest showing a thin walled cavity with a dependent fluid level (arrow) in the left upper lobe with mild perifocal fibrotic opacities with air trapping in the adjoining parenchyma.



Fig. 4 (case 3) : Chest x ray showing bilateral diffuse reticulonodular infiltrates.

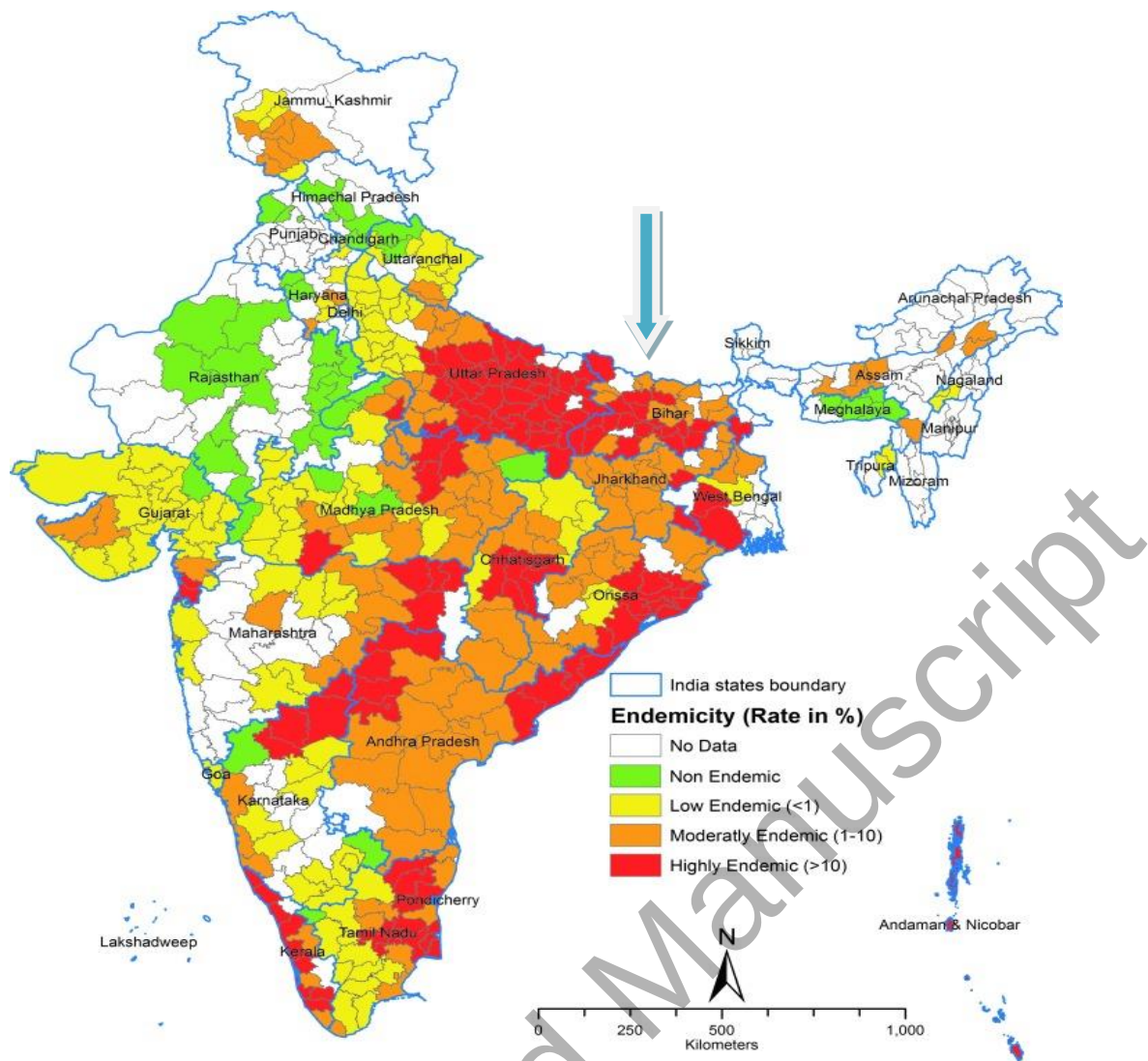


Fig 5: District-level endemicity map of Lymphatic Filariasis in India (Sabesan et al. 2000); arrow indicates the State where majority of the cases in the series were from.

Table 1: Characteristics of patients with tropical pulmonary eosinophilia

S. no.	Age (years)	Sex (M/F)	Place of origin	Duration of symptoms (months)	Treatment / Medication history
1	4	F	Bihar	18	Bronchodilator on an as-needed basis Short term courses of nebulized steroids
2	14	M	Bihar	24	Bronchodilator on an as-needed basis
3	8	F	Bihar	2	Bronchodilator on an as-needed basis
4	6	M	Bihar	1	Bronchodilator on an as-needed basis
5	12	M	Bihar	24	Bronchodilator on an as-needed basis
6	14	M	Bihar	24	MDI Budesonide for 12 months
7	7	M	Bihar	0.29	Not known
8	12	M	Bihar	0.23	Not known
9	7	M	Bihar	48	Bronchodilator on an as-needed basis
10	14	M	Uttar Pradesh	12	Bronchodilator on an as-needed basis
11	11	F	Bihar	72	Bronchodilator on an as-needed basis
12	5	M	Bihar	8	Bronchodilator on an as-needed basis

Table 2 : Clinical features of patients with tropical pulmonary eosinophilia at presentation

Presenting clinical feature	n (%) of patients
Cough	11/12 (91.6%)
Breathlessness	9/12 (75%)
Wheeze	9/12 (75%)
Fever	0/12 (0%)
Chest pain	1/12 (8%)
Respiratory distress	3/12 (25%)

Table 3: Laboratory parameters and chest radiography findings of cases

Patient	Absolute eosinophil count ($\times 10^9$ cells/L)**	Serum IgE level (IU/ml)	Filarial antigen	Filarial antibody	Chest x-ray findings	FEV1 (%) (FEV1/FVC)
1	15.250	9560	Positive	ND	Reticulonodular infiltrates	Not done
2	3.2	>400	Positive		Normal	Not done
3	5.588	1200	Not done	Positive	Normal	Not done
4	3.584	28821	Not done	Positive	Normal	Not done
5	28.800	1814	Positive	ND	Reticulonodular infiltrates	Not done
6	2.350	6400	Positive	positive	Left upper zone cyst	61% (0.89)
7	16.648	3400	Positive	ND	Reticulonodular infiltrates	Not done
8	25.850	21000	Positive	ND	Normal	Not done
9	0.800	3000	Positive	ND	Reticulonodular infiltrates	Not done
10	3.876	3760	Positive	Positive	prominent pulmonary conus	81% (0.94)
11	14.880	3346	Positive	Positive	Reticulonodular infiltrates	70%

						(0.93)
12	4.230	1844	Positive	ND	Reticulonodula infiltrates	Not done

** Normal eosinophil count: $0.1-0.6 \times 10^9$ cells L

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