VOLUME - 11, ISSUE - 09, SEPTEMBER - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Pulmonary Medicine

A RARE CASE OF TROPICAL PULMONARY EOSINOPHILIA

Dr.Hemant Sharma*	Junior Resident -3 *Corresponding Author
Dr. Bhumin Patel	Senior Resident
Dr. P.V. Potdar	Head Of Department

ABSTRACT Pulmonary diseases associated with parasitic infections of the lung are rare. Tropical pulmonary eosinophilia, caused by the filarial nematodes *Wuchereria bancrofti* and *Brugia malayi*, is endemic in the tropical and subtropical areas of Asia and Africa. The pathogenesis is due to an exaggerated immune response to the filarial antigens which includes type I, type III and type IV reactions with eosinophils playing an important role. The disease affects less than 1% of patients with lymphatic filariasis, mainly young adult males. It has an onset of several months with respiratory symptoms mainly fever, cough, dyspnea and wheeze. In India, it is mostly found around the coastal regions from Kerala and West Bengal to Tamil Nadu.

KEYWORDS : Tropical pulmonary eosinophilia, Microfilariae, Diethylcarbamazine, Microfilariae

INTRODUCTION

Lymphatic filariasis is a neglected tropical disease caused by nematodes of the Filaroidea family, the most important in humans being *Wuchereria bancrofti* (Figure 1a), *Brugia malayi* (Figure 1b) and *Brugia timori*. Tropical pulmonary eosinophilia (TPE) is the result of a hypersensitivity reaction to the microfilariae of *Wuchereria bancrofti* and *Brugia malayi* trapped in the pulmonary microcirculation. As a disease, it was first described in 1943 in India by Weingarten in patients with suspected tuberculosis who had "spasmodic bronchitis", eosinophilia and fine spotting on chest radiography. Although it primarily affects the lung, approximately 7% of the patients show extrapulmonary and extra lymphatic manifestations. In the natural history of the disease, pulmonary pathological findings include 1) acute eosinophilic infiltration in the form of eosinophilic bronchopneumonia, 2) eosinophilic abscesses or a mixed cellular infiltrate (eosinophils and histiocytes) which appear 6 months to 2 years after infection and 3) in some chronic cases, the development of interstitial, peri bronchial and perivascular fibrosis associated with the presence of histiocytes. In contrast to patients who develop TPE, most individuals who live in filarial-endemic regions and who often are subject to recurrent filarial infections usually show suppressed immune responses and minimal clinical symptoms. In conclusion, the precise role of eosinophils in the development of TPE is still largely unknown.

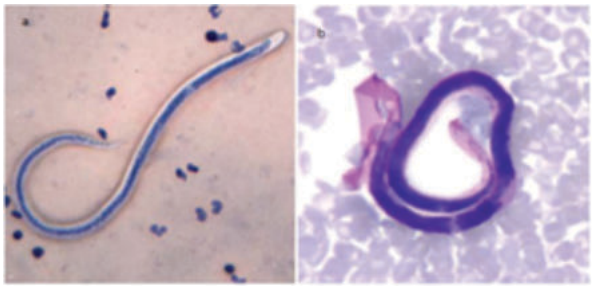

Figure 1: a) Microfilaria of *W. bancrofti* in a thick blood smear stained with Giemsa; b) Microfilaria of *B. malayi* in a thin blood smear stained with Giemsa.

Case Presentation

A 18/M patient resident of Bihar came to OPD with chief complain of malaise, anorexia, paroxysmal dry cough with dyspnea. He also reported a 2kg weight loss over the past 6 months but denied having fever or chills. The past medical history was otherwise unremarkable. Physical examination revealed bilateral basilar wheezing. On routine blood examination leukocytosis with eosinophilia (eosinophil 72%) with absolute eosinophil count 32,338 with Total Serum IgE 896IU/ml, (Figure 4) Elevated serum anti-filarial IgE, IgG (*W. bancrofti*) were found positive (Figure 5) but Tryptase were negative. Pulmonary function testing revealed mild lung restriction and decreased diffusion capacity. Microfilariae were not found in blood or sputum, and examination of stool or urine for ova and parasites is negative. Chest X-Ray ill-defined, diffuse reticulonodular infiltrates with a mottled appearance primarily affecting the mid to lower lung fields (Figure 2), HRCT show reticulonodular pulmonary opacities (Figure 3).

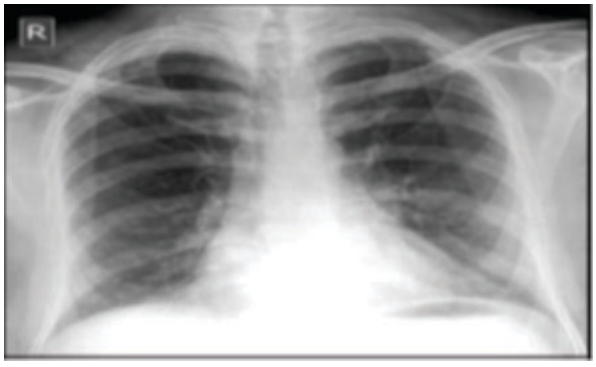

Figure 2- Chest X-ray showing diffuse reticular nodular opacities.

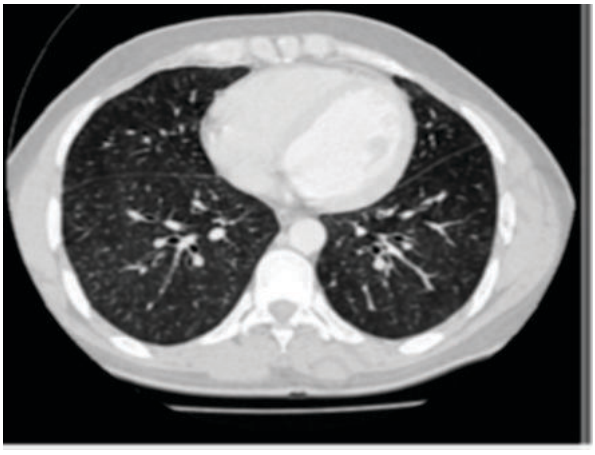

Figure 3- HRCT showing bilateral diffuse randomly distributed micronodules shadows.

LABORATORY TEST REPORT

Patient Name	:	Registered On : 21/05/2022,02.02 PM	Received On : 21/05/2022, 3.05 PM
Lab No.	: PN137R210522001	Collected On : 21/05/2022,02.00 PM	Reported On : 21/05/2022,09.21 PM
Age/Gender	: 18 Years / MALE	Normal Range For : Adult Male	Report Status : FINAL
Ref. Doctor	: MGM HOSPITAL	Barcode :	
Referred By	: Purva Diagnostic Centre	Aadhar No :	
Centre Code	: PN137R		
Contact No	:		

For Authenticity Scan QR Code

Total IgE

Parameters	Value	Unit	Biological Ref Range
Total IGE	896.0	IU/ml	IN MALE < 250 IU/ML

REFERENCE RANGE : Normal (allergy free IgE) levels IU/ml

0 - 4 days : Less than 1.5 IU/ml | **5 days - 12 months** : Less than 15 IU/ml

1 - 5 years : Less than 60 IU/ml | **6 - 9 years** : Less than 90 IU/ml

10 - 16 years : Less than 200 IU/ml

Adults : Less than 100 IU/ml

Figure 4

220011000102495

PID NO: P112200182666
Age: 18.0 Year(s) Sex: Male

Reference: Dr.MY SELF
Sample Collected At:
Health Care Laboratory
Shop No 3,maruti Nandan Society,near Mnr School,kamothe Navi Mumbai,kamothe - 410209.
Processing Location : Metropolis Healthcare Ltd, Unit No. 409- 416, 4th Floor, Commercial Building-1, Kohinoor Mall, Mumbai-70

VID: 220011000102495
Registered On: 23/05/2022 03:44 PM
Collected On: 23/05/2022 3:45PM
Reported On: 23/05/2022 09:10 PM

Investigation	Observed Value	Biological Reference Interval
Filaria Antigen detection (EDTA Whole Blood)	**Positive**	Negative

Method: Immunochromatography

Interpretation:

1. The test is structured to indicate the presence or absence of W.bancrofti antigen in the sample.
2. The absence of antigen does not exclude Filariasis caused by other nematode species.

Figure 5

Treatment

The WHO recommends a standard therapy of oral DEC at a dose of 6 mg/Kg divided TID for 21 days. The rapid response to DEC treatment is characteristic of this disease. DEC treatment has shown to rapidly reduce eosinophil lung and blood count and titers of specific anti filarial IgE. Patient completed 21 days of therapy with DEC (6 mg/kg per day) and noted marked improvement of his respiratory symptoms. The eosinophil count 1 month after the completion of treatment had decreased to 7%.

DISCUSSION

Pulmonary infiltrates with eosinophilia is a broad group of disorders infectious, inflammatory, and allergic etiologies. The differential diagnosis of eosinophilic lung disease includes Loeffler syndrome secondary to helminth infection or drug reaction; allergic bronchopulmonary aspergillosis; chronic eosinophilic pneumonia; vasculitis, such as Churg-Strauss syndrome, polyarteritis nodosa, and Wegener granulomatosis; and idiopathic hyper eosinophilic syndrome. Although all of these diseases share similar clinical and laboratory features, there are several criteria that must be met to diagnose TPE, including residence in an area in which filaria is endemic, peripheral eosinophilia, absence of microfilariae in peripheral blood, increased anti filarial antibody titer, elevated serum IgE level, and a favorable clinical response to diethylcarbamazine. A history of paroxysmal nocturnal asthma and radiographic evidence of pulmonary infiltrates are supportive but not diagnostic.

Diagnostic Criteria for TPE

Relevant exposure in endemic area
Paroxysmal nocturnal cough, dyspnea
Infiltrate on chest radiograph
Leukocytosis with eosinophilia
Elevated serum IgE
Elevated serum antifilarial IgE, IgG (*W. bancrofti*, *B. malayi*)
Clinical improvement with diethylcarbamazine treatment

Alternative anti filarial drugs (e.g., ivermectin) or a trial of corticosteroids may be useful therapies for the chronic variant of the disease, although controlled studies of these agents are lacking. A subset of patients with apparent TPE may fail to respond to diethylcarbamazine; whether these patients have diethylcarbamazine-resistant TPE or disease due to other parasites is unclear, as current serologic testing does not distinguish between human lymphatic filarial antigens and antigens on certain other parasites. Untreated disease usually persists for weeks to months. Untreated TPE may remit spontaneously,

but it commonly recurs within months to years. It is important to treat TPE early in the course of the disease, since although seldom fatal, untreated TPE often leads to the development of irreversible pulmonary fibrosis.

CONCLUSION

Tropical pulmonary eosinophilia is an occult form of filariasis which is the result of a hypersensitivity reaction to trapped microfilariae or the antigenic products of *Wuchereria bancrofti* and *Brugia malayi* present in the pulmonary microcirculation. This disease affects mainly young males from endemic countries, however, the rise in immunosuppressive conditions associated with increasing rates of traveling and migration are responsible for the emergence of cases in non-endemic countries. The disease affects initially the lung airways and is characterized by the development of eosinophilic alveolitis that may progress, in chronic cases, to interstitial, peri bronchial and perivascular fibrosis. Diethylcarbamazine is the mainstay pharmacological treatment, however, in a group of patients, a persistence of airway inflammation and significant alterations of lung function may be seen despite treatment. Better diagnostic tests and pharmacological drugs are needed to minimize morbidity and avoid the development of interstitial pulmonary fibrosis which profoundly affects the quality of life of these patients.

REFERENCES

1. O'Bryan L, Pinkston P, Kumaraswami V, et al. Localized eosinophil degranulation mediates disease in tropical pulmonary eosinophilia. *Infect Immun*. 2003;71
2. Boggild AK, Keystone JS, Kain KC. Tropical pulmonary eosinophilia: a case series in a setting of nonendemicity. *Clin Infect Dis*. 2004;39(8)
3. Weingarten RJ. Tropical eosinophilia. *Lancet*. 1943;1:103–105
4. Mullerpattan JB, Udwadia ZF, Udwadia FE. Tropical pulmonary eosinophilia—a review. *Indian J Med Res*. 2013;138(3)
5. Vijayan VK. Tropical pulmonary eosinophilia: pathogenesis, diagnosisand management. *Curr Opin Pulm Med*. 2007