ISSN (print): 2589-9120
ISSN (online): 2589-9139

International Journal of Medical Science and Applied Research (IJMSAR)

Available Online at: https://www.ijmsar.com

Volume – 3, Issue –6, November – December - 2020, Page No. : 30 – 34

A Case Series On Tropical Pulmonary Eosinophilia Misdiagnosed As Miliary Tuberculosis

[1]Dr. Sanchit Mohan, 301 - High Rise Building, GT Hospital Campus, Near St. Xavier's College, Mumbai - 400001, Maharashtra, India.

[2]Dr Vishwanath Pujari, Associate Professor, Dept. of Pulmonary Medicine, Grant Medical College, Mumbai, India.

[3]Dr Priti L Meshram, Professor and HOD, Department of Pulmonary Medicine, Grant Medical College, Mumbai, India.

[4]Dr Rohit Hegde, Associate Professor, Dept of Pulmonary Medicine, Grant Medical College, Mumbai, India.

Corresponding Author: Dr. Sanchit Mohan, 301 - High Rise Building, GT Hospital Campus, Near St. Xavier's College, Mumbai – 400001, Maharashtra, India.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Tropical pulmonary eosinophilia occurs as a result of an exaggerated immune response to parasite W. Bancrofti, Brugya Malayi. It is prevalent in tropical as well as subtropical regions of the world. Tuberculosis is the most prevalent infective disease of our country and it can mimic any pulmonary disease on CXR.

Here we present a case series of 4 patients who were diagnosed to have milliary tuberculosis based on their HRCT were started on AKT based on their clinical findings, however no improvement in symptoms despite taking AKT for more than 6 months. Actually, they were suffering from tropical pulmonary eosinophilia which improved after starting corticosteroids and diethylcarbamazine.

Conclusion

Tropical pulmonary eosinophilia may be wrongly diagnosed as miliary tuberculosis if we rely solely on CXR and HRCT findings, hence a detailed history with complete blood cell count and bronchoalveolar lavage fluid analysis is mandatory in the diagnosis of tropical pulmonary eosinophilia.

Keywords: Miliary Tuberculosis, Tropical Pulmonary Eosinophilia, HRCT

Introduction

Pulmonary Eosinophilia is a group of disorders which can be idiopathic or with a cause. Out of various pulmonary eosinophilia, Tropical Pulmonary Eosinophilia is most common in tropical as well as sub-tropical regions which occurs as a result of an exaggerated immune response to the parasite W. Bancrofti and Brugia Malayi. Tuberculosis is the most prevalent infective disease of our country and it can mimic any pulmonary disease on chest X-ray.[1]

Case Series

Case-1

A 24-year-old gentleman from Bihar came to our OPD with complain of cough, dry in character, since 2 months and shortness of breath since 2 months (MMRC grade-2). No past history of Tuberculosis.

HRCT done suggestive of bilateral randomly distributed nodules, few of them forming tree in bud pattern with necrotic mediastinal lymphadenopathy (4R, 7, 10R) findings likely suggestive of Koch's. [Figure-1,2]

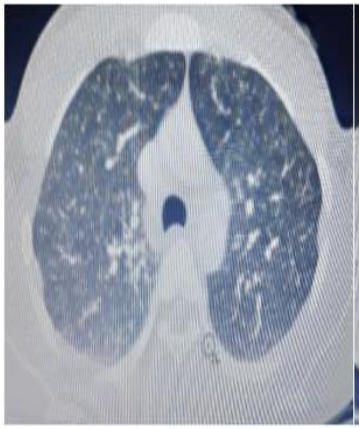
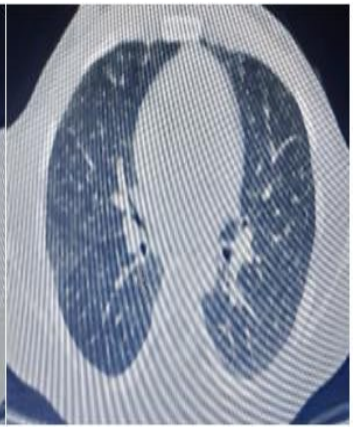

Figure - 1 **Figure – 2**

Patient was started on Anti-tubercular treatment but did not get any relief ever after 2 months of ATT. CBC showed marked leukocytosis (TLC- 46800) with marked eosinophilia (AEC-32956).Serum IgE level was 20124. Spirometry was suggestive of Mixed Ventilatory defect with good bronchodilator reversibility of obstructive component.

FEV1/FVC-65, FEV1-1.93(44), FVC-2.99(57), MMEF-2.08(47) (Post Bronchodilator).

Fiberoptic Bronchoscopy was done, BAL GeneXpert-Negative, AFB Culture- MTB not Grown, Cytology- Total counts 597 with 38% eosinophils, Aerobic culture-Negative. Filarial specific IgG raised.

Started on T. Diethlycarbamazine 6mg/kg for 21 days and T. Prednisolone 1mg/kg, ICS+LABA and ATT was withheld follow up scan showed drastic improvement.

Case-2

A 41-year-old gentleman from Gorakhpur came to OPD with complains of cough with mucoid expectoration since 1 month and fever since 1 month (low grade) with no past history of Tuberculosis.

HRCT was suggestive of multiple poorly defined Ground Glass Nodules scattered in bilateral lung fields with no apico-basal gradient, few sub centimeter sized non necrotic mediastinal lymphadenopathy in bilateral paratracheal region, findings suggestive of miliary spread of tuberculosis. [Figure-3,4]

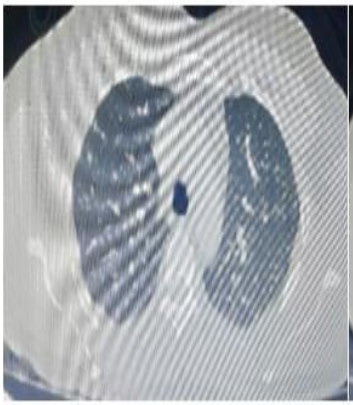
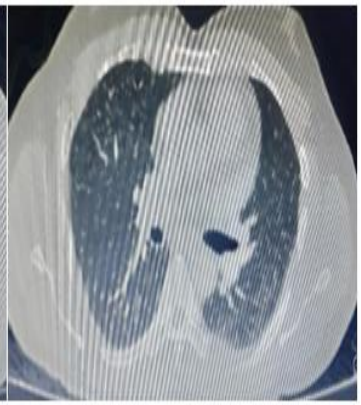

Figure - 3 **Figure – 4**

Patient was started on Anti-tubercular treatment based on clinical and radiological findings.

TLC-25000 with Eosinophils of 74%, Total IgE-7260, FISH for FIP1L1(PDGFRA)- Negative. Spirometry was done suggestive of Restrictive Ventilatory Defect FEV1/FVC-96, FEV1-2.34(68), FVC-2.45(60), MMEF-3.55 (85).

Fiberoptic Bronchoscopy was done, BAL GeneXpert-Negative, AFB Culture- MTB not Grown, Cytology- Total counts 800 with 80% eosinophils, Aerobic culture-Negative. Filarial specific IgG raised.

Started on T.Diethlycarbamazine 6mg/kg for 21 days and T.Prednisolone 1mg/kg and ATT was withheld.Follow up scan showed drastic improvement.

Case-3

A 21-year-old boy from Ranchi came with complains of cough with mucoid expectoration with shortness of breath for 20 days (MMRC grade 2) in a known case of Bronchial Asthma since childhood, no history of Tuberculosis in past. HRCT was suggestive of bilateral randomly scattered nodules, few of them forming tree in bud pattern, suggestive of tuberculosis with no mediastinal adenopathy. [Figure-5,6]

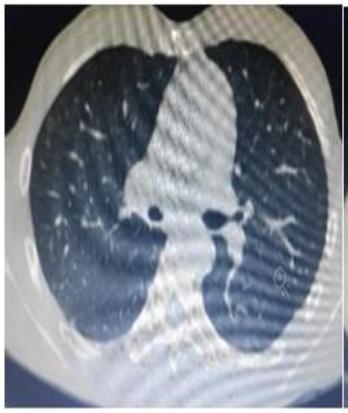

Figure - 5

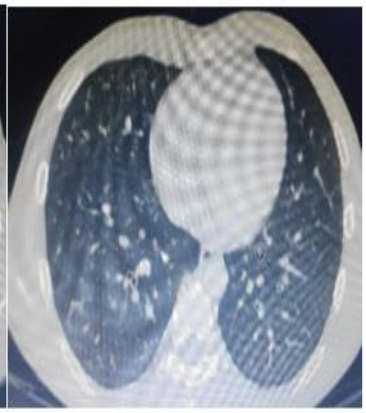

Figure – 6

Patient was started on Anti-tubercular treatment based on clinical and radiological findings. However, he developed ATT induced hepatitis and stopped medications by his own.

TLC-18900 with Eosinophils of 45%, Total IgE-12902. Spirometry was done suggestive of Moderate obstruction with good bronchodilator reversibility FEV1/FVC-62, FEV1-1.84(64), FVC-2.45(68), MMEF-1.60(54) Post bronchodilator.

Fiberoptic Bronchoscopy was done, BAL GeneXpert-Negative, AFB Culture- MTB not Grown, Cytology- Total counts 350 with 40% eosinophils, Aerobic culture-Negative. Filarial specific IgG raised.

Started on T. Diethlycarbamazine 6mg/kg for 21 days and T. Prednisolone 1mg/kg, ICS+LABA. Follow up scan showed drastic improvement.

Case-4

An 18-year-old boy came with complaint of dry cough since 3 months, shortness of breath (MMRC grade-1)since 1 month and fever since 20 days (low grade), in a known case of Bronchial Asthma since childhood but was not on any medications for the same. There was no previous history of Tuberculosis.

HRCT was suggestive of bilateral randomly scattered nodules, few of them forming tree in bud suggestive of tuberculosis with necrotic mediastinal adenopathy (4R, 7, 10L, 10R). [Figure-7]

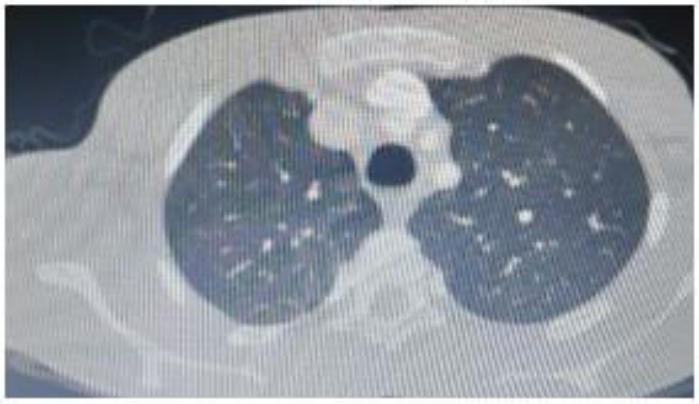

Figure – 7

Patient was started on Anti-tubercular treatment based on clinical and radiological findings.

TLC-18900 with Eosinophils of 45%, Total IgE-12902. Spirometry was done suggestive of Mixed Ventilatory defect with good bronchodilator reversibility of obstructive component FEV1/FVC-51, FEV1-0.81(22), FVC-1.60(39), MMEF-0.80(20) Post bronchodilator.

Fiberoptic Bronchoscopy was done, BAL GeneXpert-Negative, AFB Culture- MTB not Grown, Cytology- Total counts 550 with 30% eosinophils, Aerobic culture-Negative. Filarial specific IgG raised.

Started on T.Diethlycarbamazine 6mg/kg for 21 days and T.Prednisolone 1mg/kg, ICS+LABA and ATT was withheld.Follow up scan showed drastic improvement.

Discussion

Lymphatic filariasis is a tropical disease caused by W. Bancrofti, Brugia malayi and Brugia timori. One-third of the cases of *W. bancrofti* filariasis are reported from India, another third in Africa and third in South Asia.[1] Elephantiasis is the common manifestation of W.Bancrofti, which results due to obstruction of lymphatic vessels leading to lymphedema.

Tropical Pulmonary Eosinophilia occurs due to hypersensitivity reaction(type-1) to microfilariae of *Wuchereria bancrofti* and *Brugia malayi* trapped in pulmonary microcirculation. It was described by Weingarten in patients with suspected tuberculosis who

had "spasmodic bronchitis", eosinophilia and fine spotting on chest radiography in 1943, hence also known as Weingarten Pneumonia.[2]

TPE particularly occurs in young adults with male to female ratio of 4:1. It primarily affects lungs only 7% of patients shows extrapulmonary or lymphatic manifestations.[3]

The exact pathogenesis of TPE is not well understood. Immune cells necessary for filarial nematode cell clearance are eosinophils.[4-6] Eosinophils have following granule proteins such as eosinophil cationic protein (ECP), eosinophil-derived neurotoxin (EDN), major basic protein 1 (MBP-1) and eosinophil peroxidase (EPO) which show helminthotoxic activity *in vitro* [7,8], however, *in vivo* studies demonstrate that these proteins are helpful but are not sufficient for microfilariae clearance.Downstream effects of eosinophil-induced cytokine responses are mainly responsible for filarial clearance [6,9]

The term Miliary Tuberculosis was coined by John Jacob Manget.[10] More commonly occurs in males as compared to females, with a high mortality of 18-30%[11]

Bharathkanth et al reported a case of miliary tuberculosis with hyper eosinophilia (Absolute eosinophil count-9633cells/mm3) with IgE 960IU/ml withsputum AFB negative, started on Tab. Hetrazan 100mg TID), Tab. Ivermectin (6mg) +Mebendazole(400mg) once in a week for 3weeks for 28days. But there was no improvement in symptoms therefore patient was started on Anti Tuberculosis Treatment (ATT) and significant improvement was noticed after 1 month.[12]

Another case reported by Gunjan et. Al was a 64-year-old female with peripheral eosinophilia with mediastinal adenopathy which on cytology showed features of tuberculosis and after initiation of anti-tuberculosis treatment her eosinophil counts returned to normal levels.[13]

However, in our case series all patients were already on anti-tubercular therapy for 2-3 months but did not get any relief in symptoms, instead one patient develops hepatitis after ATT. All the 4 cases responded well to DEC and oral steroids.

Conclusion

Pulmonary eosinophilia may be wrongly diagnosed as miliary tuberculosis if we rely solely on Chest X-ray and HRCT findings of randomly distributed micronodular opacities, hence a detailed history with complete blood cell count and bronchoalveolar lavage fluid analysis is mandatory for diagnosis.

References

1. Pérez-Arellano J, Andrade M, López-Abán J, Carranza C, Muro A (2006) Helminths and the respiratory system. Arch Bronchoneumol 42: 81-91.
2. Weingarten R, Cologne M (1943) Tropical eosinophilia. Lancet 241: 103-105.
3. Dreyer G, Dreyer P, Piessens W (1999) Extralymphatic disease due to bancroftian filariasis. Braz J Med Biol Res 32: 1467-1472.
4. Simons J, Rothenberg M, Lawrence R (2005) Eotaxin-1-regulated eosinophils have a critical role in innate immunity against experimental Brugia malayi infection. Eur J Immunol 35: 189-197.
5. Ramalingam T, Ganley-Leal L, Porte P, Rajan T (2003) Impaired clearance of primary but not secondary Brugia infections in IL-5 deficient mice. Exp Parasitol 105: 131-139.
6. Cadman E, Thysse K, Bearder S, Cheung A, Johnston A, et al. (2014) Eosinophils are important for protection, immunoregulation and pathology during infection with nematode microfilaria. PLoS Pathog 10.

7. O'Bryan L, Pinkston P, Kumaraswami V, Vijayan V, Yenokida G, et al. (2003) Localized eosinophil degranulation mediates disease in tropical pulmonary eosinophilia. Infect Immun 71: 1337-1342.
8. Hamann K, Gleich G, Checkel J, Loegering D, McCall J, et al. (1990) In vitro killing of microfilariae of Brugia pahangi and Brugia malayi by eosinophil granule proteins. J Immunol 144: 3166-3173.
9. Ramalingam T, Porte P, Lee J, Rajan T (2005) Eosinophils, but not eosinophil peroxidase or major basic protein, are important for host protection in experimental Brugia pahangi infection. Infect Immun 73: 8442-8443.
10. Sharma SK,Mohan A. Disseminated and miliary tuberculosis. In: Sharma SK, Mohan A, editors. Tuberculosis, 2nd ed. New Delhi: Jaypee Brothers; 2009:493-518.
11. World Health Organization. Global Tuberculosis Control: surveillance, planning, Financing. Geneva: WHO; 2008.
12. Bharathkanth , Srinivas T, Israel P L. A rare presentation of Miliary Tuberculosis with Hypereosinophilia.Perspectives in medical research 2016;4(3):61-63.
13. Garg G, Gogia A, Kakar A, Miglani P. Persistent Marked Peripheral Eosinophilia due to Tuberculosis: A Case Report. Iran J Med Sci. 2017 Jan;42(1):102-105. PMID: 28293059; PMCID: PMC5337757.