British Journal of Medicine & Medical Research
4(35): 5513-5518, 2014

SCIENCEDOMAIN *international*
www.sciencedomain.org

A Case of Chronic Cough with Progressive Breathlessness in a 32 Year-old Male Health Worker – Tuberculosis?, Allergic Bronchitis?, Asthma?

Gauri Billa[1*] and Karan Thakkar[2]

[1]Urban Health Post, NMMC, Kopar Khairane, India.
[2]Urban Health Post, NMMC, Ghansoli, India.

Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Case Study

Received 9th June 2014
Accepted 3rd July 2014
Published 30th July 2014

ABSTRACT

Introduction: Although tuberculosis is hyper-endemic in India and is responsible for a huge proportion of respiratory morbidity, adequate workup should be conducted to rule out other differential diagnosis wherever applicable.
Case Report: A 32 year old male health worker was suffering from productive cough and gradually increasing breathlessness since three months. The investigations conducted were a sputum analysis and a chest x-ray, both of which were normal and hence he was treated as a case of allergic bronchitis. Subject presented to us after three months with no relief. We further investigated him and found severe eosinophilia in the peripheral blood, a positive anti-filarial antibody and a negative triple stool test for ova and parasites. He was treated with diethylcarbamazine and albendazole+ivermectin combination. The patient responded well and had no complaints at the end of the 4 week treatment.
Discussion and Conclusion: The subject should have been evaluated by conducting a basic investigation like a complete blood count. Delay in treatment of cases of tropical pulmonary eosinophilia can lead to permanent respiratory morbidity.

**Corresponding author: Email: gauribilla@gmail.com;*

Keywords: Severe eosinophilia; tropical pulmonary eosinophilia; Eosinophilic pneumonia; Loeffler's syndrome; albendazole plus ivermectin; diethylcarbamazine.

1. INTRODUCTION

Tuberculosis (TB) is hyper-endemic in India, especially in regions with urban slums due to high density of the population and poor living conditions. The high risk groups are immuno-compromised individuals, close contacts of sputum positive tuberculosis (TB) cases, health workers etc. Although it contributes to a major chunk of cases with chronic cough and/or breathlessness, one should consider an alternative diagnosis every now and then. The same was highlighted in the case of a 32 year old health worker whose case is presented below.

2. CASE PRESENTATION

A 32 year old male health worker, non-smoker, with no addictions, came with the chief complaints of cough since three months and gradually worsening breathlessness. His cough was productive with mucoid sputum. He had breathlessness which had increased gradually from grade 0 to grade II over a period of three months, as per the Modified Medical Research Council Dyspnea scale [1]. There was no history of fever, loss of appetite, weight loss, night sweats, chest pain, pedal oedema, orthopnea, paroxysmal nocturnal dyspnoea, skin rash, abdominal pain or recent history of travel.On examination, the only positive findings were expiratory rhonchi/wheeze over both the lung fields. Surprisingly, until now, he had only been investigated by ordering analysis of sputum samples for *M. tuberculosis* (MTB) and a chest x-ray, both of which were normal.

Before presenting to us, he had been treated for allergic bronchitis and an upper respiratory tract infection with a fixed dose combination of Levocetrizine plus Montelukast and the antibiotics: Amoxcillin-Clavulinate and Cephalosporins, sequentially, but none of his symptoms had improved.

We ordered a complete blood count (CBC) and an erythrocyte sedimentation rate (ESR). His reports showed leucocytosis with a total white blood cell count (WBC) of 15,600 /mm3, 41% Eosinophils – Absolute count: 6400/µl, 35% Neutrophils, 15% Lymphocytes and an ESR of 55 at the end of first hour. There are a myriad of differential diagnoses for eosinophilia (Table 1) [2,3].

We further ordered a peripheral smear for blood cell morphology, liver and renal function tests, serum Ig E levels, anti-nuclear antibody levels, triple stool examination for parasites and ova and an anti-filarial antibody test. His serum IgE levels were elevated at 2120 IU/ml (normal is up to 380 IU/ml), and the anti-filarial specific IgG and IgE antibody test was positive. The patient was diagnosed as a case of Tropical Pulmonary Eosinophilia [4,5].

We started him on a course of tablet Diethylcarbamazine 100 mg twice a day for four weeks, a fixed dose combination of tablet Albendazole (400 mg) plus Ivermectine (6 mg) once a day for six days, syrup Salbutamol 4 mg thrice a day and tablet Loratadine 10 mg once a day for ten days. After seven days, the patient showed marked reduction in cough and breathlessness and the rhonchi had almost disappeared. A repeat CBC after a month revealed a total WBC count of 9,000/mm3 and the eosinophils were reduced to 8%.

3. DISCUSSION

Hypereosinophilia is defined as an absolute eosinophil count above 5000/µl [6]. The various causes of elevated eosinophils are given in Table 1. Although, any level of eosinophilia can be seen with allergic diseases and asthma, it is very important to exclude more serious systemic diseases like, Eosinophilic Leukaemia, Churg-Strauss syndrome, autoimmune diseases etc. [7].

Table 1. Various causes of peripheral blood eosinophilia

A) Primary eosinophilia
1. Hematologic and Neoplastic Disorders • Chronic eosinophilic leukemia • Acute myelogenous leukemias most commonly, B cell ALL • Lymphomas (particularly Hodgkin's, T- and B-cell lymphomas) • Systemic mastocytosis • Tumor associated o Adenocarcinomas o Squamous cell carcinomas * o Large cell lung carcinomas o Transitional cell carcinoma of the bladder
B) Secondary eosinophilia
1. Infectious Diseases • Parasitic infections * • Tropical Pulmonary Eosinophilia* • Allergic bronchopulmonary aspergillosis* **2. Allergic or Atopic Diseases** • Drug induced eosinophilias* • Atopic disorders* • Asthma* **3. Immunologic** • Churg-Strauss vasculitis (allergic granulomatosis or allergic granulomatous angiitis)* • Rheumatoid arthritis • Eosinophilic fascitis • Primary Immunodeficiency Diseases (HyperIgE syndrome, Omenn's syndrome) • Graft-versus-host-Disease **4. Endocrinologic Disorders** • Hypoadrenalism **5. Others** • Irradiation • Atheroembolic Disorders • Sarcoidosis*
C) Idiopathic
1. Idiopathic hypereosinophilia syndrome 2. Acute and Chronic Eosinophilic Pneumonia*

Being a health care worker from a congested metropolitan city of India where tuberculosis is hyper-endemic, the patient was initially investigated for MTB. But when these reports were

normal, he should have been investigated further for other possible causes of chronic cough. A basic and very informative test that was not ordered till three months was a CBC which could have given valuable information about his disorder. Finally, he was diagnosed as a case of TPE based on the following criteria [4,5].

History suggestive of nocturnal symptoms mainly cough and dyspnoea, Pulmonary infiltrates on chest radiograph (this criteria was not satisfied in our patient), Leukocytosis with peripheral eosinophilia > 3000/µl, Elevated serum IgE and filarial specific IgG and IgE, and clinical improvement with DEC.

TPE can be treated with DEC and 'Albendazole plus Ivermectin' combination [5,8]. Although some studies have shown that the cure rate of DEC is more than 90%, other studies have shown that in chronic cases it could be as low as 60% to 80 % despite a three week course of DEC [9-11]. The chances of relapse after DEC therapy are said to be about 20% in one year and DEC itself is not very effective in cases of relapse [9]. Hence, we decided to start the patient on 'Albendazole plus Ivermectin' combination in addition to DEC. The added advantage of this combination was that it would also be effective in cases of helminth induced respiratory eosinophilia. Steroids are said to have a beneficial role in the management of TPE but their dose and the regimen needs to be established by clinical trials [4]. Steroids are also known to have a significant and rapid eosinopenic effect [12]. We had started treatment for TPE based on the reports of the CBC when the report of the stool tests was not yet available. There have been cases of disseminated strongyloidosis, especially, in immuno-compromised individuals and those on steroids, leading to sepsis due to dissemination of the gram negative enteric bacteria [13]. By the time the stool report came as negative, six days of treatment had been completed and the patient was feeling significantly better and hence we didn't start him on steroids. At the end of one month of DEC and a six day course of Albendazole plus Ivermectin, the patient was alright and his physical examination was normal.

4. CONCLUSION

There was a substantial delay in ordering a CBC in this patient, which led to a delay in starting specific treatment. It is a known fact that long standing untreated TPE can lead to permanent pulmonary damage and resultant interstitial lung disease and long term morbidity [9,14,15]. The key message is that after preliminary evaluation of a patient for MTB, other differential diagnosis should be considered and evaluated so as to start the appropriate treatment at the earliest.

CONSENT

All authors declare that 'written informed consent was obtained from the patient for publication of this case report.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Hsu KY, Lin JR, Lin MS, Chen W, Chen YJ, Yan YH. The modified medical research council dyspnoea scale is a good indicator of health-related quality of life in patients with chronic obstructive pulmonary disease. Singapore Med J. 2013;54(6):321–7. [Internet]. [cited 2014 May 30];
   Available from: http://www.ncbi.nlm.nih.gov/pubmed/23820542
2. Fulkerson PC, Rothenberg ME. Targeting eosinophils in allergy, inflammation and beyond. Nat Rev Drug Discov. 2013;12(2):117–29. [Internet]. [cited 2014 May 28] Available:http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3822762&tool=pmcentrez&rendertype=abstract
3. Nutman TB. Evaluation and differential diagnosis of marked, persistent eosinophilia. Immunol. Allergy Clin North Am. 2007;27(3):529–49. [Internet]. [cited 2014 May 30] Available:http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2099264&tool=pmcentrez&rendertype=abstract
4. Vijayan V. Immunopathogenesis and treatment of eosinophilic lung diseases in the tropics. In: Sharma O, editor. Lung Biol Heal Dis Trop lung Dis. 2nd ed. New York: Taylor and Francis. 2006;195–239.
5. Mullerpattan JB, Udwadia ZF, Udwadia FE. Tropical pulmonary eosinophilia-a review. Indian J Med Res. 2013;138(3):295–302. [Internet]. [cited 2014 May 31].
   Available:http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3818591&tool=pmcentrez&rendertype=abstract
6. Weller PF. The immunobiology of eosinophils. N Engl J Med. 1991;324(16):1110–8. [Internet].
   Available from: http://www.nejm.org/doi/full/10.1056/NEJM199104183241607
7. Singh V, Gomez V, Swamy S, Vikas B. Approach to a case of eosinophilia. Indian Jouranl od Aerosp Med. 2009;53(2):58–64. [Internet].
   Available from: http://medind.nic.in/iab/t09/i2/iabt09i2p58.pdf
8. Kappagoda S, Singh U, Blackburn BG. Antiparasitic therapy. Mayo Clin Proc. 2011;86(6):561–83. [Internet]. [cited 2014 May 31].
   Available:http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3104918&tool=pmcentrez&rendertype=abstract
9. Udwadia F. Pulmonary eosinophilia: Progress in pulmonary research. In: Herzog H, editor. Pulm Eosinophilia Prog Pulm Res 7th ed. Basel: S Karger. 1975;35–155.
10. Rom WN, Vijayan VK, Cornelius MJ, Kumaraswami V, Prabhakar R, Ottesen EA, et al. Persistent lower respiratory tract inflammation associated with interstitial lung disease in patients with tropical pulmonary eosinophilia following conventional treatment with diethylcarbamazine. Am Rev Respir Dis. 1990;142(5):1088–92. [Internet]. [cited 2014 May 31] Available from: http://www.ncbi.nlm.nih.gov/pubmed/2173455
11. Vijayan VK, Rao K V, Sankaran K, Venkatesan P, Prabhakar R. Tropical eosinophilia: clinical and physiological response to diethylcarbamazine. Respir Med. 1991;85(1):17–20. [Internet]. [cited 2014 May 31]
    Available from: http://www.ncbi.nlm.nih.gov/pubmed/1901660
12. Altman LC, Hill JS, Hairfield WM, Mullarkey MF. Effects of corticosteroids on eosinophil chemotaxis and adherence. J Clin Invest. 1981;67(1):28–36. [Internet]. [cited 2014 May 31].
    Available:http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=371568&tool=pmcentrez&rendertype=abstract

13. Namisato S, Motomura K, Haranaga S, Hirata T, Toyama M, Shinzato T, et al. Pulmonary strongyloidiasis in a patient receiving prednisolone therapy. Intern Med. 2004;43(8):731–6. [Internet]. [cited 2014 May 31].
Available from: http://www.ncbi.nlm.nih.gov/pubmed/15468976
14. Alberts WM. Eosinophilic interstitial lung disease. Curr Opin Pulm Med. 2004;10(5):419–24. [Internet]. [cited 2014 May 31]
Available from: http://www.ncbi.nlm.nih.gov/pubmed/15316442
15. Vijayan VK. Tropical pulmonary eosinophilia: Pathogenesis, diagnosis and management. Curr Opin Pulm Med. 2007;13(5):428–33. [Internet]. [cited 2014 May 31]
Available from: http://www.ncbi.nlm.nih.gov/pubmed/17940489

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sciencedomain.org/review-history.php?iid=616&id=12&aid=5573