

HYPOTHESIS**ALLERGIC BRONCHO-PULMONARY HELMINTHIASIS
(ABPH)**

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ABSTRACT**Based on current literature, an aetiological classification of pulmonary eosinophilia due to helminths including tropical eosinophilia is proposed.****Introduction**

Infestations with helminths (cestodes, nematodes and trematodes) are the commonest causes of pulmonary eosinophilia in tropical countries (1-3). Other causes of pulmonary eosinophilia include Allergic Broncho-Pulmonary Aspergillosis (ABPA) (4), chronic eosinophilic pneumonia (5), cryptogenic pulmonary fibrosis (6), Wegener's granulomatosis (7), lymphomatoid granulomatosis (8) eosinophilic granuloma of lung (9), the Churg-Strauss syndrome (10), drug hypersensitivity reactions (11) and infections such as brucellosis (12), coccidioidomycosis (13), corynebacterium pseudotuberculosis (14) and tuberculosis (15).

Pulmonary eosinophilia due to helminths :

One of the commonest forms of pulmonary eosinophilia in tropical countries is due to hypersensitivity reaction to an occult form of filariasis termed as tropical (filarial) eosinophilia (16) or Tropical Eosinophilia (TE) (17-20). TE presents with symptoms suggestive of bronchial asthma such as cough, dyspnoea, nocturnal wheezing and chest discomfort, occasionally associated with such constitutional symptoms as weight loss, anorexia and fever (17,21). Physical examination reveals rhonchi and rales (21). Chest radiographs show a variety of abnormalities, including diffuse miliary mottling, pleural effusions, and uncommonly cavitation and large areas of consolidation (22). High levels of blood and lung eosinophils with elevated serum and lavage fluid levels of IgE and filaria - specific IgE and IgG antibodies (17, 23, 24) had also been demonstrated. Pulmonary function studies had shown airway obstruction in addition to reduced lung volumes and reduced diffusing capacity (21, 25-27). Following a three -week course of diethylcarbamazine (DEC), there was incomplete reversal of clinical, haematological, radiological and physiological changes

one month after starting treatment (25). In conformity with these findings, a chronic respiratory tract inflammation as evidenced by a persistent eosinophilic alveolitis with lower respiratory tract inflammatory cells spontaneously releasing exaggerated amounts of O_2 and H_2O_2 has also been demonstrated (28). It had also been shown that, at one year, patients with eosinophilic alveolitis had a significantly reduced diffusing capacity despite three weeks treatment with DEC (21). It had also been reported that bronchiectasis could occur during and persist after clinical attacks of TE (29). Therefore, there is an urgent need to undertake controlled clinical trials in TE to assess the role of corticosteroids in preventing the occurrence of irreversible radiological and lung function changes.

A similar syndrome of pulmonary eosinophilia with respiratory symptoms can result from infestations with other helminths such as *Ascaris* (30), *Ancylostoma* species (31), *Strongyloides stercoralis* (32) and *Toxocara* species (33, 34). Currently available immunological investigations of antibody assays (35) may not differentiate the eosinophilic syndromes due to various helminthic infections because of antigenic similarities. Because of the probable broad-spectrum anti-helminthic action of DEC, patients with pulmonary eosinophilia due to various helminthic infections respond to treatment with DEC (Personal observation). Thus it appears that the syndrome of tropical eosinophilia is an end result of a common pathogenesis resulting from infestations with various helminths. Though the syndrome of Tropical eosinophilia is common in tropical and sub-tropical countries, it had also been described in subjects from temperate countries on exposure to appropriate infections in endemic areas (36). Therefore, it will be ideal to have a better name for this syndrome based on aetiology and research should be directed to identify tests that may differentiate various types of pulmonary

eosinophilia.

Allergic broncho-pulmonary mycosis :

A similar disease resulting from a hypersensitivity reaction to fungal antigens (*Aspergillus* species) has been termed Allergic Broncho-Pulmonary Aspergillosis (ABPA)(4). ABPA also manifests as asthma with elevated serum levels of total IgE and specific antibodies to aspergillus antigens (37). Pulmonary function tests had revealed both obstructive and restrictive ventilatory defects (38). Irreversible lung changes including proximal bronchiectasis can occur if ABPA is left untreated (39). Since other fungi such as *Candida* (40, 41), *Helminthosporium* (42), *Stemphyllium* (43) and *Curvaluria* (44) can cause a similar syndrome, it was suggested that the term "Allergic Broncho-Pulmonary Fungal diseases" was a better name for the disease (45) (Table 1).

TABLE 1

Allergic Broncho-Pulmonary Mycoses (45)

1. Allergic Broncho-Pulmonary Aspergillosis (4).
2. Allergic Broncho-Pulmonary Candidiasis (40).
3. Allergic Broncho-Pulmonary Helminthosporiasis (42).
4. Allergic Broncho-Pulmonary Stemphyliosis (43).
5. Allergic Broncho-Pulmonary Curvulariasis (44).

Members in parenthesis refer to articles quoted in reference

Comparison of ABPA and TE :

Thus, there appears to be a similarity in pathogenesis of pulmonary eosinophilia resulting from hypersensitivity reactions to fungi and helminths. A comparison of two diseases viz. ABPA and TE which are prototypes of pulmonary eosinophilia due to fungi and helminths are shown in Table 2.

TABLE 2

Comparison of ABPA and TE

	ABPA	TE
1. Features of Asthma	+	+
2. Blood and Sputum eosinophilia	+	+
3. Lung Eosinophilia	+	+
4. Chest Radiographic infiltrations	+	+
5. Total IgE	↑	↑
6. Specific IgG and IgE	↑	↑
7. Pulmonary Function	Obstruction +	Obstruction +
	Restriction	Restriction
8. Irreversible lung damage including bronchiectasis	Can occur	Can occur
9. Antigens	Fungus	Helminths
10. Treatment	Corticosteroids	DEC

? Corticosteroids

Both result from exaggerated immune-responses to respective antigens as evidenced by elevated levels of total IgE and specific antibodies (17,35,37). Both have bronchial and lung parenchymal abnormalities as revealed by similar pulmonary function abnormalities (25,38). These may be due to type I and type III hypersensitivity reactions. Lung eosinophils in both diseases can cause irreversible lung changes if left untreated (17,29,39). Therefore, it is suggested that the term "Allergic Broncho-Pulmonary Helminthiasis" is a better term, for pulmonary eosinophilia due to helminthic infections. Terminologies that are appropriate for pulmonary eosinophilia resulting from various helminthic infections are given in Table 3.

TABLE 3

**Allergic Broncho-Pulmonary Helminthiasis
Suggested list of Infestations**

1. Allergic Broncho-Pulmonary Filariasis
2. Allergic Broncho-Pulmonary Ascariasis
3. Allergic Broncho-Pulmonary Ancylostomiasis
4. Allergic Broncho-Pulmonary Strongyloidiasis
5. Allergic Broncho-Pulmonary Toxocariasis

Tropical Eosinophilia can then be termed as "Allergic Broncho-Pulmonary Filariasis".

In case of an incorrect diagnosis in a patient with pulmonary eosinophilia due to strongyloidiasis and if this patient is treated with steroids, the parasite may become disseminated (46). This emphasises the need for an aetiological diagnosis in patients with pulmonary eosinophilia.

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