# TROPICAL PULMONARY EOSINOPHILIA- AN ELABORATE CASE SERIES

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## INTRODUCTION

Pulmonary infiltrates with eosinophilia (PIE) syndromes is a motley group of diverse entities, which share common features of chest opacities and peripheral blood eosinophilia. Tropical pulmonary eosinophilia (TPE) is a PIE prevalent worldwide, which has continued to fox pulmonologists as well as radiologists for years, due to the varied clinico-radiological manifestations. This often leads to delayed diagnosis, engendering significant morbidity. Hence, acquiring holistic information about all the clinical and radiologic manifestations of the entity becomes vital. This article attempts to do the same using a case series format.

## CLINICAL DIAGNOSIS

Filariasis is a rampant malady worldwide particularly so in the Asian countries with manifestations ranging from asymptomatic microfilaremia to chronic obstructive lymphatic elephantiasis. The heterogeneous group of disorders presenting with pulmonary infiltrates on radiographs and peripheral eosinophilia on blood examination are clubbed under the umbrella term, pulmonary infiltrates with eosinophilia (PIE) syndromes.

## DIFFERENTIAL DIAGNOSIS

The term PIE syndrome includes six entities namely simple pulmonary eosinophilia, prolonged pulmonary eosinophilia, TPE, allergic bronchopulmonary aspergillosis, Churg Strauss syndrome and Hypereosinophilic syndrome.[1,2] Table 1 depicts the spectrum of PIE syndromes.[3] Tropical pulmonary eosinophilia (TPE) is a conspicuous member of this spectrum. TPE was first described in 1940 and was labelled as "pseudo tuberculosis with eosinophilia" due to the classic radiographic finding of military nodules and the striking eosinophilia on haematological examinations. It is common in the tropics especially the Indian subcontinent.[4]

Though this entity has been considered a benign condition for years, it was eventually realized that TPE can traumatize the lung parenchyma irrevocably and can prove to be debilitating if not treated punctually. However, its diagnosis can sometimes be difficult owing to the varied clinico-radiological manifestations and its potential to mimic many other pulmonary conditions. We hereby depict the clinical and radiological spectrum, successfully diagnosed in a multidisciplinary setting.

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## PRESENTATION OF CASES

### CASE 1

#### Clinical Details

A 20-years old, non-smoker man, presented with a 4-months history of fever, cough with mucoid sputum and dyspnoea. Chest auscultation revealed bilateral crackles.

#### Laboratory Investigations

There was a high absolute eosinophil count (AEC) of 25972/mm3. Serum immunoglobulin E (S IgE) was raised (2820 U).

#### Radiology

The chest radiograph (CXR) showed bilateral micronodular opacities. High resolution computerized tomography (HRCT) showed ill-defined centrilobular nodules and branching opacities scattered throughout the lung parenchyma bilaterally without any zonal predominance. Mild tubular bronchiectasis with peribronchial thickening was also seen in both the lungs (Figure 1A, 1B).

#### PFT

Spirometry showed a restrictive abnormality with forced vital capacity (FVC) of 2.68 L (65% predicted); forced expiratory volume in first second (FEV1) 2.33 L (65% predicted); and FEV1/FVC ratio 87%.

#### Follow Up

The patient was symptomatically better with a significant decline in serum IgE to 450 U, AEC to 14098 with improvement in FEV1 to 2.72(75%) and FVC to 3.13 (76%).

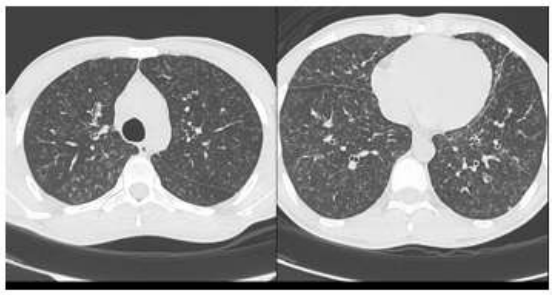

Figure 1A

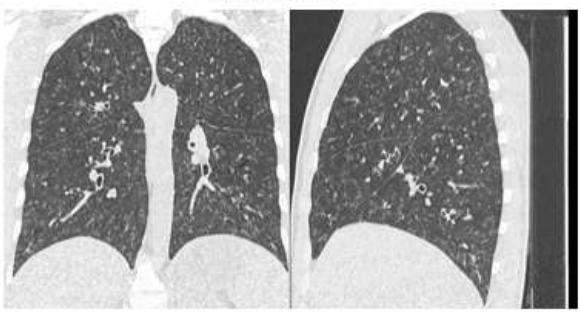

Figure 1B

Figure 1. The axial (A) HRCT lung images show widespread ill-defined centrilobular nodules and branching opacities scattered throughout the lung parenchyma bilaterally with mild tubular bronchiectasis and peribronchial thickening also seen. The coronal and sagittal reconstructed (B) images show the widespread lesions better and without any zonal predominance.

**CASE 2**

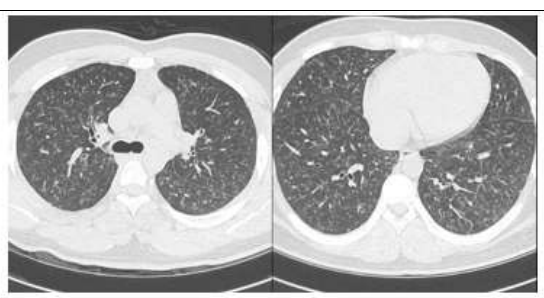

Figure 2A

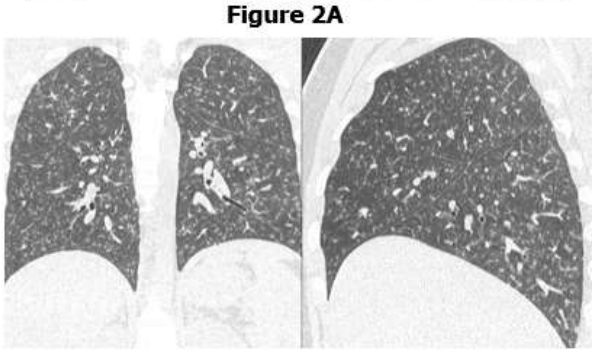

Figure 2B

Figure 2. The axial (A) HRCT lung images show diffuse ill-defined centrilobular nodules and opacities in both the lungs. The coronal and sagittal reconstructed (B) images shows the diffuse lesions, without any zonal predominance.

**Clinical Details**

A 15-years old, non-smoker man, presented with a 2-months history of fever and cough with mucoid sputum. His vital parameters were normal with pulse oximetry saturation of 98% on room air. Post exercise desaturation was present. Chest auscultation did not reveal crackles or rhonchi.

**Laboratory Investigations**

A high absolute eosinophil count (AEC) of 40070/mm3 was found. S.IgE was raised (2754 U)

**Radiology**

CXR showed bilateral micronodular opacities. HRCT chest showed diffuse ill-defined centrilobular nodules and opacities in both the lungs (figure 2A & 2B).

**PFT**

Spirometry showed a restrictive abnormality with FVC of 3.11 L (68% predicted); FEV1 2.19 L (58% predicted); and FEV1/FVC ratio 85%.

**Follow Up**

The patient was asymptomatic with a decline in serum IgE to 2280 U, AEC to 12000 with improvement in FEV1 to 2.19(58%) and FVC to 3.26(72%).

**CASE 3**

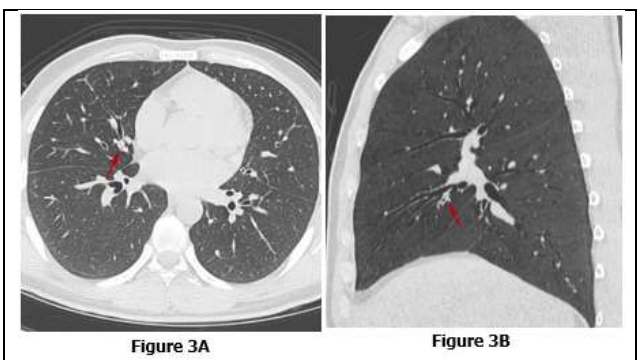

Figure 3A Figure 3B

Figure 3. The axial (A) HRCT lung and sagittal MinIP reconstructed images shows mild bronchial prominence in both the lungs with patchy mucoid impaction (arrows) in the right middle lobe.

**Clinical Details**

A 29-years old, non-smoker man, presented with a 10-years history of episodic cough and dyspnoea. He was treated as bronchial asthma at peripheral health centres with minimal relief. Chest auscultation was normal.

**Laboratory Investigations**

They revealed a high absolute eosinophil count (AEC) of 3105/mm3. S.IgE was raised (4000 U).

**Radiology**

CXR was within normal limits. HRCT chest showed mild bronchial prominence in both lungs with patchy mucoid impaction in the right middle lobe (figure 3A & 3B).

**PFT**

Spirometry was within normal limits with FVC of 2.95 L (76% predicted); FEV1 2.76 L (82% predicted); and FEV1/FVC ratio 94%. Filarial antibody test was positive while peripheral smear showed absence of microfilaria.

**Follow Up**

The patient was asymptomatic with a decline in serum IgE to 1220 U, AEC to 240 with improvement in FEV1 to 2.83(84%) and FVC to 3.02(78%)

**CASE 4**

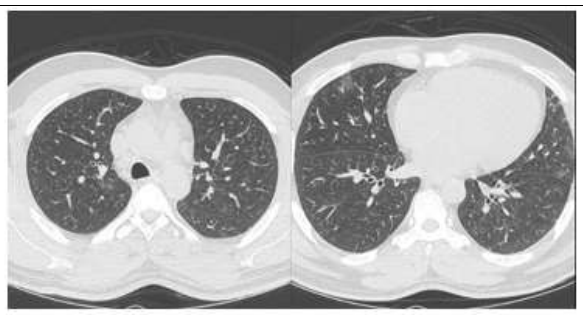

Figure 4A

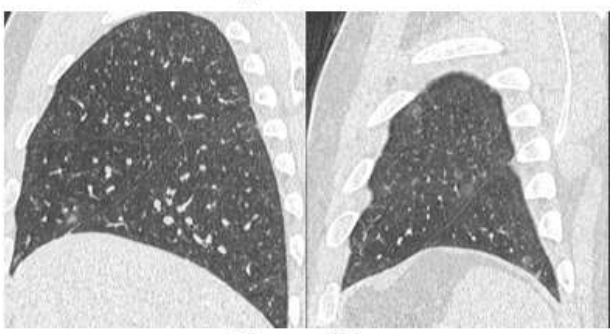

Figure 4B

Figure 4. The axial (A) HRCT lung images show widespread ill-defined centrilobular nodules and opacities in both the lungs with few larger bronchocentric ground-glass opacities also seen in the right middle lobe and the lingula. The sagittal reconstructed (B) images show the diffuse lesions in both the upper and lower lobes.

**Clinical Details**

A 25-years old, non-smoker man, presented with a 10-years history of episodic cough and dyspnoea. He was treated as bronchial asthma by multiple physicians with minimal relief. Chest auscultation was normal.

**Laboratory Investigations**

They revealed a high absolute eosinophil count (AEC) of 17500/mm3. S.IgE was raised (4704 U).

**Radiology**

CXR was within normal limits. HRCT chest showed widespread ill-defined centrilobular nodules and opacities in both the lungs. Few larger bronchocentric ground-glass opacities are also seen in the right middle lobe and the lingula (figure 4A & 4B).

**PFT**

Spirometry was restrictive abnormality with FVC of 2.96 L (64% predicted); FEV1 2.54 L (64% predicted); and FEV1/FVC ratio 86%.

**Follow Up**

The patient was symptomatically better with a significant decline in serum IgE to 1582 U, AEC to 12006 with improvement in FEV1 to 3.18(80%) and FVC to 3.70(80%).

**CASE 5**

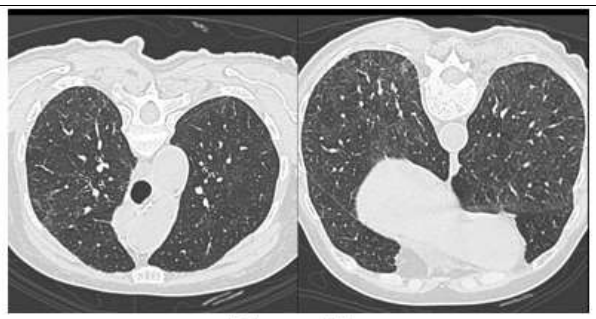

Figure 5A

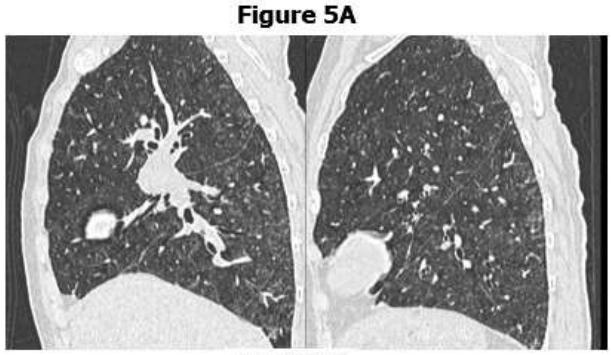

Figure 5B

Figure 5. The axial (A) HRCT lung images show ill-defined intralobular interstitial and septal thickening with peripheral distribution and lower zone predominance in the lung parenchyma bilaterally. The sagittal reconstructed (B) images shows apico-basal gradient with subpleural sparing.

**Clinical Details**

A 75-years old, non-smoker woman, presented with a 1-year history of dry cough and progressive dyspnoea. Her vital parameters were normal with pulse oximetry saturation of 91% on room air. Post exercise desaturation was present. Chest auscultation revealed bilateral crackles.

**Laboratory Investigations**

They revealed an absolute eosinophil count (AEC) of 4, 704/mm3. S.IgE was raised (16, 780 U). Filarial antibody test was positive while peripheral smear showed absence of microfilaria.

**Radiology**

CXR showed bilateral reticulonodular opacities. HRCT chest showed interstitial fibrosis in both the lungs with peripheral

distribution and lower zone predominance. Areas of sub pleural sparing were also seen (figure 5A & 5B).

**PFT**

Spirometry showed a restrictive abnormality with forced vital capacity FVC of 0.57 L (32% predicted); FEV1 0.49 L (34% predicted); and FEV1/FVC ratio 85%.

**Follow up**

The patient was symptomatically better with a significant decline in serum IgE to 1460 U, AEC to 680 with improvement in FEV1 0.87(60%) to and FVC to 1.03(58%).

The summary of clinical and laboratory details of five cases is illustrated in Table 2.

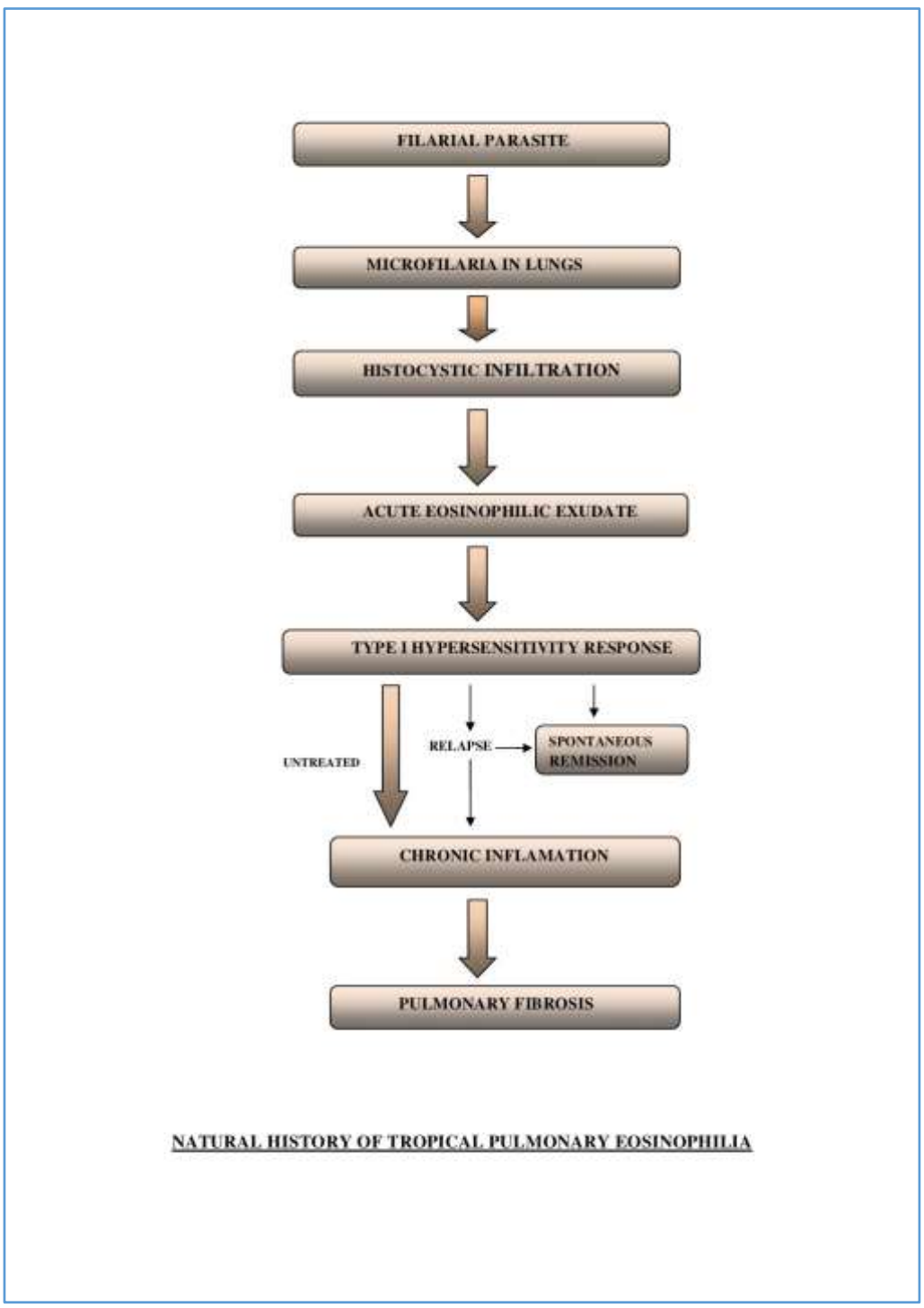


***Figure 6. Natural History of Tropical Pulmonary Eosinophilia***

| Diseases | Pathology | Clinical history | Laboratory | Spirometry | CXR |
|---|---|---|---|---|---|
| Simple Eosinophilia/ Loeffler's Syndrome | Response to Ascaris lumbricoides | Acute Symptoms, Persist for Less Than A Month | Transient Eosinophilia, Raised S. IgE | Normal | TAbn |
| Prolonged Pulmonary Eosinophilia | Response to Other Worms | Persistent Symptoms, Last for More Than One Month till Six Months | Eosinophilia, Raised S. IgE | Normal, Obstructive Abnormality | Abn |
| Tropical Pulmonary Eosinophilia | Type 1 Hypersensitivity Response to Microfilaria | Subacute-Chronic Symptoms | Eosinophilia More Than 3000, S. IgE More Than 1000 IU | Obstructive, Restrictive, Mixed Abnormality | Abn |
| Allergic Bronchopulmonary Aspergillosis | Response to Aspergillus fumigatus | Severe Persistent Symptoms | Eosinophilia, S. IgE More Than 1000 IU | Obstructive Abnormality | Abn |
| Churg Strauss Syndrome | Allergic Granulomatosis | Allergic, Eosinophilic, Vasculitic Phases | Eosinophilia with Neurological Involvement | Obstructive, Restrictive Abnormality | Abn |
| Hyper Eosinophilic Syndrome | Clonal Eosinophilia | Diagnosis of Exclusion | Eosinophilia More Than 1500 Over Six Months | Restrictive Abnormality | Abn |

***Table 1. Spectrum of Peripheral Infiltrates and Eosinophilia Syndrome***

CXR- Chest X-Ray, TAbn- Transient Abnormality, Abn- Abnormal, N-Normal

| Case No. | | | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---|---|---|---|---|---|---|---|
| Age | | | 20 | 15 | 29 | 25 | 75 |
| Gender | | | Male | Male | Male | Male | Female |
| Duration of Symptoms | | | 5 months | 2 months | 10 years | 10 years | 1 year |
| Predominant Symptoms | | | Episodic cough | Episodic cough | Episodic cough | Episodic cough | Episodic Cough and Exertional Dyspnoea |
| History of Atopy | | | Present | Present | Present | Present | Absent |
| Chest X-Ray Reticulonodular Opacities | | | present | present | absent | absent | present |
| Antifilarial Antibody | | | positive | positive | positive | positive | positive |
| Peripheral Smear for Microfilaria | | | negative | negative | negative | negative | negative |
| AEC (per cu mm) | | Pre | 25972 | 40070 | 3105 | 17500 | 4704 |
| | | Post | 14098 | 12000 | 240 | 12006 | 680 |
| Serum (IU) Immunoglobulin E | | Pre | 2820 | 2754 | 4000 | 1582 | 16780 |
| | | Post | 450 | 2280 | 1220 | 340 | 1460 |
| Spirometry | FVC L (%) | Pre | 2.68(65) | 3.11(68) | 2.95(76) | 2.96(64) | 0.57(32) |
| | | Post | 3.13 (76) | 3.26(72) | 3.02(78) | 3.70(80) | 1.03(58) |
| | FEV1 L (%) | Pre | 2.33(65) | 2.19(58) | 2.76 (82) | 2.54(64) | 0.49(34) |
| | | Post | 2.72(75) | 2.77(73) | 2.83(84) | 3.18(80) | 0.87(60) |
| | FEV1/FVC | | 87 | 85 | 94 | 86 | 85 |
| HRCT Thorax | Centrilobular Nodules | | Present | present | Absent | Present | Absent |
| | Septal Thickening | | Absent | Absent | Absent | Absent | Present |

***Table 2. Clinical and Laboratory Details of Five Cases of Tropical Pulmonary Eosinophilia***

AEC- Absolute eosinophil count, HRCT- High resolution computed tomography

| | |
|---|---|
| 1 | Infections Like Tuberculosis |
| 2 | Hypersensitivity Pneumonitis |
| 3 | Respiratory Bronchiolitis |
| 4 | Tropical Pulmonary Eosinophilia |
| 5 | Vasculitis |
| 6 | Pneumoconiosis |
| 7 | Pan-Bronchiolitis |
| 8 | Metastasis |

***Table 3. Differential Diagnosis of Diffuse Centrilobular Nodules***

| Sl. No. | Criteria |
|---|---|
| 1 | Resident of Tropics |
| 2 | Absolute Eosinophil Count More Than 3000/$mm^3$ |
| 3 | Serum Immunoglobulin Level More Than 1000 IU |
| 4 | Elevated Titres of Anti-Filarial Antibodies |
| 5 | Absence of Peripheral Blood Microfilaria |
| 6 | Response to Diethylcarbamazine |

***Table 4. Diagnostic Criteria for Tropical Pulmonary Eosinophilia***

**PATHOLOGICAL DISCUSSION**

TPE is a PIE that stems from a hypersensitivity response to the microfilariae unshackled by the parasites Wuchereria bancrofti and Brugia malayi. TPE is known to be a benign self-limiting disease with a distinct natural history. Udwadia FE et al reported pioneer studies that have helped understand the pathophysiology of the disease.[5-9] The mature filarial parasites situated in the human lymphatics periodically release microfilaria in the pulmonary microcirculation. These microfilariae are antigenic and propagate a pathological cascade of events in the pulmonary system. The immediate response as to any antigen is characterised by histiocytic infiltration attempting at destruction of the microfilaria presented to the macrophages and histocytes. This is followed by mobilisation of eosinophils into the lung causing an acute eosinophilic exudate. Subsequently over about six months or so, various cytochemical factors released mount hypersensitivity Type I immune response against the microfilaria. There is no peripheral blood eosinophilia identifiable till this stage. With this, there may be spontaneous remission of the disease and immunity preventing further infections. In some cases, there may be relapses on repeated exposure of the microfilaria. Otherwise the pathological events progress over six months to two years giving rise to a mixed cell reaction which involves, histiocytes, eosinophils, epithelioid cells with peripheral blood eosinophilia. This is the usual phase when patients report with increasing symptoms, visit a health care facility and are diagnosed as cases of TPE. Untreated/unreported disease progresses over two to five years to fibroblast activation and proliferation leading to pulmonary fibrosis and interstitial lung disease. Though this causes disability, the fibrosis is patchy, non-progressive and does not lead to hypoxemia, pulmonary hypertension and cor-pulmonale. The natural history is depicted in Figure 6.

**DISCUSSION OF MANAGEMENT**

Otteson highlighted five distinct clinical types of this spectrum with varying nature of severity, namely TPE, chronic lymphatic obstructive pathology, filarial fever, asymptomatic microfilaremia, and possible cryptic infection in endemic individuals.[10] TPE customarily exhibits a male preponderance and also shows a predisposition for the second and third decades of age as seen in our case series too. However, extremes of age have also been reported as seen in one of our patients, who was an elderly lady. It is ubiquitous as far as organ involvement is concerned, however the pulmonary system is the one most commonly involved. TPE may present with nocturnal cough, haemoptysis, dyspnoea, fever, night sweats, chest pain and nasal catarrh. Constitutional symptoms in the form of body ache, malaise, anorexia and weight loss may also be encountered. There was heterogeneity in clinical manifestations amongst our five cases too. The duration of symptoms ranged from 2 months to 10 years. Also, the nature of symptoms and manner of presentation was varied. Two patients had asthma like presentation, two presented in a sub-acute manner mimicking a pulmonary infection whereas one patient exhibited clinical features suggestive of interstitial lung disease in form of dry cough progressive dyspnoea and exercise desaturation. Thus, even in a small case series of a handful of cases we observed intriguing variations in the clinical manifestations. Spirometry in TPE may be normal, or have an obstructive pattern, a restrictive pattern or a combination of both, depending on the extent of airway involvement, airway hyper-reactivity and the interstitial or lung parenchymal scarring. Spirometry in our patients by clinical and radiological correlation showed a restrictive abnormality with poor bronchodilator reversibility. The radiological spectrum of TPE is varied. The radiograph is usually abnormal when the diagnosis is made, however the findings are non-specific. The typical appearance is of a reticular or reticulonodular pattern with lower zone predominance.[11] Approximately 20% of patients may have a normal radiograph.[12] CT scan shows the nodules better and the typical pattern consists of widespread ill-defined centrilobular nodules and opacities scattered throughout the lung parenchyma bilaterally.[13,14] Table 3 shows the differential diagnosis of diffuse centrilobular nodules and opacities. The cases 1, 2 and 4 showed this typical picture on HRCT. In addition, tubular bronchiectasis with peribronchial thickening and patchy areas of air trapping can also be seen, as seen in the case 3. Consolidation, cavitation, pleural effusion and lymphadenopathy can sometimes be seen, but are uncommon.[15] The radiological findings often resolve after treatment and mild residual lesions may remain.[16] If the disease is not adequately treated, it may progress to interstitial lung disease with fibrosis.[17] Case 5 describes an elderly patient, symptomatic for a longer duration, who presented with interstitial fibrosis. TPE mimics many conditions, the commonest being bronchial asthma.[18]

Our third and fourth cases had bronchial asthma as a comorbid condition and TPE was an associated incidental finding. Cavitation, pleural effusions, pneumonitis, masslesions[19] and interstitial lung diseases[20] have been chronicled in literature. It may even be misconstrued as tuberculosis[21] especially in countries where tuberculosis is rampant. The diagnosis is confirmed with the help of Donohugh criteria.[22] They include residence in tropics, presence of eosinophil count of more than 3000 per cumm, absence of microfilaria in the peripheral blood, raised serum immunoglobulin E (IgE) more than 1000 U, elevated titres of microfilarial antibodies and response to diethylcarbamazine.[23] The diagnostic criteria are given in table 4.

Arsenic compounds were used initially following which DEC was recommended.[24,25] DEC is prescribed at the dose of 6 mg/kg/day in three divided doses for duration of 21 days. In persistent/ resistant cases corticosteroid therapy may be used. Response to therapy is by and large good with complete clinical and radiological response in most cases. The laboratory and lung function response to therapy can however be variable. None of our patients exhibited outright normalisations of their laboratory parameters while only one patient showed complete normalisation of lung function values. Failure to understand this phenomenon can result in unwarranted prolonged therapies. Also relapses despite of adequate therapy have been reported in literature[26] warranting a retreatment or alternative therapies like steroids and ivermectin.[26] The pulmonary fibrosis stage of disease does not respond to DEC and is managed with pulmonary rehabilitation with vaccinations, physiotherapy, comorbidity optimisation and long-term oxygen therapy.

**FINAL DIAGNOSIS**

Thus, TPE remains a challenging clinical and radiological entity. Radiology and clinical and laboratory correlation in a multi-disciplinary setting help avoid invasive lung biopsies and usually allows an accurate diagnosis to be made.

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