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Chronic Eosinophilic Pneumonia As A Diagnostic Mimic of Tuberculosis : A Case Report.

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ABSTRACT

Background :
Pulmonary tuberculosis (PTB) is endemic in Sri Lanka, and many patients with chronic cough, constitutional symptoms and upper-zone opacities are treated empirically when microbiological confirmation is unavailable. This practice risks diagnostic anchoring and delayed recognition of important mimics. Chronic eosinophilic pneumonia (CEP) is an uncommon, steroid-responsive eosinophilic lung disease that can closely resemble PTB clinically and radiographically particularly when infiltrates involve upper lobes. A key clue is the presence of "flitting" (migratory) consolidations, supported by peripheral or bronchoalveolar lavage (BAL) eosinophilia.

Case report:
A 25-year-old woman presented with a 6-month dry cough and a 3-week history of low-grade evening fever with night sweats, anorexia, myalgia/arthralgia and 4-kg weight loss. Examination was unremarkable aside from low body mass index (16.2 kg/m²). Initial investigations showed elevated inflammatory markers (ESR 118 mm/h; CRP 36 mg/dL), mild eosinophilia (697 cells/µL), Mantoux induration 10 mm, negative sputum AFB smear and a right upper-zone opacity on chest radiograph. She was started on standard anti-tuberculosis treatment for clinically diagnosed PTB. Within one week, symptoms persisted with vomiting and a markedly raised CRP (174 mg/dL), rising eosinophils (3289 cells/µL) and repeat radiography showing resolution of the right-sided lesion with new left upper-zone consolidation. HRCT demonstrated bilateral apical peripheral consolidations with ground-glass opacities. Parasitic studies, filarial antibodies, HIV testing and ANCA were negative and IgE was elevated (620 IU/mL). Bronchoscopy was normal; BAL showed 90% eosinophils with negative AFB smear, GeneXpert, bacterial cultures, and pyogenic cultures. Anti-tuberculosis therapy was stopped and oral prednisolone 30 mg/day initiated leading to complete clinical and radiological resolution within two weeks.

Conclusion:
In tuberculosis-endemic settings, CEP should be considered when presumed PTB is bacteriologically unconfirmed, fails to improve on therapy and demonstrates migratory infiltrates with eosinophilia. Early BAL differential counts and prompt corticosteroid treatment can rapidly reverse disease and prevent unnecessary anti-tuberculosis exposure.

Keywords: ***Bronchoalveolar Lavage, Chronic Eosinophilic Pneumonia, Eosinophilia, Pulmonary Tuberculosis.***

INTRODUCTION

Pulmonary tuberculosis (PTB) remains a major health problem in Sri Lanka and other South Asian countries where a substantial proportion of patients present with chronic cough, constitutional symptoms and radiographic opacities that are at least in some cases treated as PTB on clinical grounds. Beyond its direct health impact, tuberculosis disproportionately affects socioeconomically vulnerable groups and continues to impose meaningful morbidity at working ages, reinforcing a low threshold for treatment initiation when clinical suspicion is high. In routine practice, clinicians frequently encounter patients with "TB-compatible" syndromes but incomplete microbiological corroboration, creating a clinical environment in which diagnostic anchoring is common and alternative causes of chronic pulmonary infiltrates may be under-recognized.[1]

Current standards emphasize microbiological confirmation of PTB whenever possible however, smear-negative and bacteriologically unconfirmed PTB remains a common problem and diagnostic challenge. This is particularly important when sputum production is scant, bacillary burden is low or access to timely mycobacterial culture and molecular testing is not widely available.

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In such cases, diagnostic reasoning often relies on symptom complexes (fever, night sweats, weight loss), immunological tests, and chest radiography—tools that are neither perfectly sensitive nor specific for PTB. The consequences of this approach are clinically important: empirical anti-tuberculosis treatment (ATT) can expose patients to drug toxicity, delay appropriate therapy for the true underlying disease, and confound subsequent evaluation when radiographic changes evolve under the influence of non-specific anti-inflammatory or antimicrobial effects.[2]

A variety of conditions can closely mimic PTB in both clinical presentation as well as imaging. These conditions may include malignancy, sarcoidosis, fungal infections and eosinophilic lung diseases. Eosinophilic pneumonias are particularly challenging because they may present with subacute systemic symptoms, persistent cough, elevated inflammatory markers and patchy consolidations that are easily interpreted as pulmonary tuberculosis. Moreover, peripheral blood eosinophilia may be absent early or dismissed as reactive, and radiographic patterns can overlap with post-primary PTB when upper-zone involvement is present. Conceptually, eosinophilic lung diseases represent a spectrum of disorders unified by pulmonary eosinophilic infiltration, with diagnosis resting on an integrated assessment of exposure history, parasitic evaluation where relevant, peripheral eosinophil counts, and bronchoalveolar lavage (BAL) or tissue eosinophilia.[3]

Chronic eosinophilic pneumonia (CEP), also termed idiopathic chronic eosinophilic pneumonia is an uncommon but highly steroid-responsive eosinophilic lung disease. It is characterized by constitutional symptoms, respiratory complaints and marked eosinophilic inflammation. Classic descriptions highlight a predilection for women and non-smokers and frequent co-occurrence with asthma. The characteristic imaging features include peripheral, often upper-lobe–predominant airspace opacities that may migrate over a period of time.[4] This "migratory" or "flitting" behavior is a critical discriminant in the differential diagnosis of consolidation. While tuberculous infiltrates may evolve gradually or cavitate, the appearance of new opacities with concomitant resolution of prior lesions should prompt immediate reconsideration of the diagnosis. High-resolution computed tomography can further support suspicion by demonstrating peripheral consolidations and ground-glass opacities, while BAL eosinophilia (commonly >25%) provides strong supportive evidence in the appropriate clinical context.[5]

Despite the availability of defining clinical, radiologic, and bronchoscopic clues, CEP remains underdiagnosed in tuberculosis-endemic settings where empirical ATT is a common response to constitutional symptoms and upper-zone opacities. The present case report aims to address this gap by illustrating CEP as a diagnostic mimic of PTB, emphasizing the value of longitudinal radiographic assessment and eosinophil-directed investigations (including BAL differential counts) in patients with suboptimal response to empirical ATT, and reinforcing timely corticosteroid therapy as a means to achieve rapid clinical and radiological resolution.

CASE REPORT

A 25-year-old girl presented to the clinic with a dry cough of six months duration. The cough showed no diurnal variation and was not associated with wheezing. There was no significant sputum production and no history of haemoptysis. Approximately three weeks prior to her initial clinic visit, she developed fever.

This was a low-grade evening pyrexia lasting for approximately two hours each day and was associated with profuse sweating. She also reported generalized arthralgia and myalgia. Although there was no associated vomiting, she complained of severe, progressive loss of appetite. This was accompanied by an unintentional weight loss of approximately 4 kg over a three-week period. There was no past history of tuberculosis and no known contact with patients diagnosed with tuberculosis.

She was employed as an executive in a finance company for the past two years, primarily engaged in clerical and administrative work. There was no occupational or environmental exposure of relevance, and no history of contact with individuals with tuberculosis. On examination, she was under-weight with a BMI of 16.2 kg/m. She was not pale and not icteric. There was no cervical or axillary lymphadenopathy. Systemic examination, including examination of the respiratory system, was normal.

Her white cell count was slightly elevated, with a mild elevation of eosinophils (697 cells/ml). The erythrocyte sedimentation rate (ESR) was 118 mm/Hr and the C-reactive protein (CRP) was 36 mg/dl. The Mantoux test showed an induration of 10 mm. A chest X-ray revealed right upper zone opacities (Figure 1).

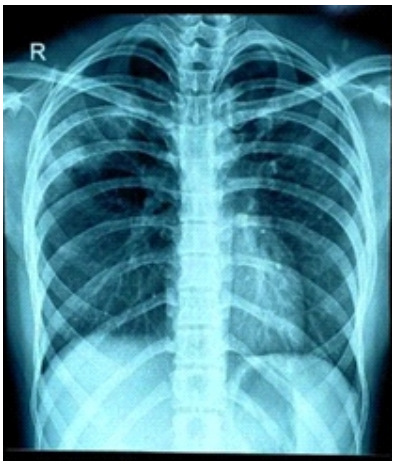

Figure 1: Chest X-ray taken on presentation

Sputum for acid fast bacilli (AFB) was negative and sputum for acid-fast bacilli (AFB) culture was arranged in the central culture lab. Other investigations, including urine analysis, blood cultures, two-dimensional echocardiography, abdominal ultrasonography, HIV testing, and anti-neutrophil cytoplasmic antibody (ANCA) testing, were negative.

Although available bacteriological investigations were negative, a diagnosis of clinically diagnosed pulmonary tuberculosis was made based on the combination of clinical features and radiological findings. The patient was commenced on standard anti-tuberculosis treatment (ATT), and bronchoscopy and bone marrow examinations were arranged. The index patient was admitted to the ward the following week for bronchoscopy; however, at the time of admission, she was symptomatic with vomiting and fever. The initial clinical suspicion was anti-tuberculosis treatment (ATT)–induced hepatitis. Liver function tests revealed a mild elevation. Serum glutamic oxaloacetic transaminase (SGOT/AST) was 42 U/L (normal range 10–40 U/L), serum glutamic pyruvic transaminase (SGPT/ALT) was 58 U/L (normal range 7–56 U/L), and serum bilirubin was 4.0 µmol/L (reference range 3–19 µmol/L). The C-reactive protein (CRP) was markedly elevated at 174 mg/dl.

Full blood count revealed a significant elevation in eosinophils (3289 mm^{-3}). A repeat chest X-ray demonstrated resolution of the previously noted right upper zone consolidation, with the appearance of a new consolidation in the left upper zone(Figure 2).

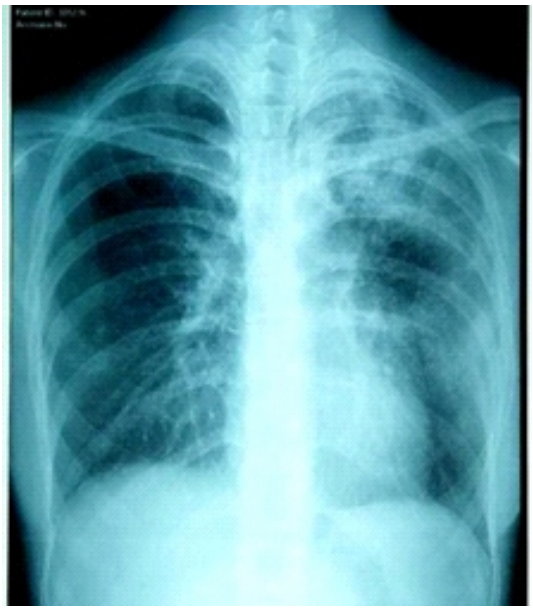

Figure 2: Chest X-ray showing resolution of right upper zone opacities and appearance of left upper zone consolidation.

High-resolution computed tomography (HRCT) of the chest revealed bilateral apical, peripheral consolidations with associated ground-glass opacities (Figure 3).

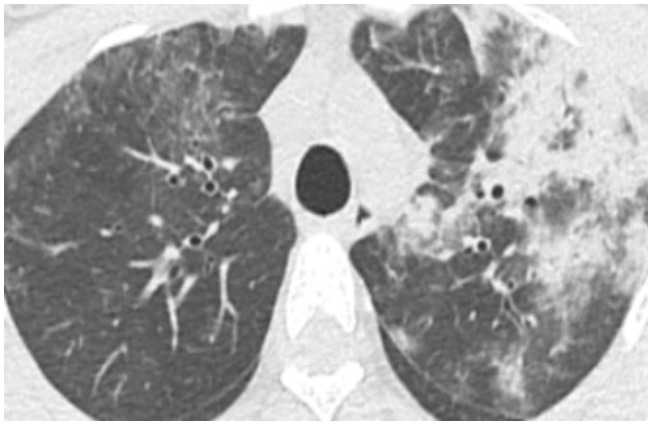

Figure 3: HRCT chest showing Bilateral peripheral consolidations with ground glass opacification (left more than right).

Stool examinations for ova and parasites were negative. Serum immunoglobulin E (IgE) levels were elevated at 620 IU/mL (reference range <100 IU/mL).The antineutrophil cytoplasmic antibody (ANCA) panel, including p-ANCA and c-ANCA, was negative. Filarial antibodies were also negative. Subsequently, a significant rise in the peripheral blood eosinophil count was noted on serial full blood counts.

Anti-tuberculosis treatment was discontinued, and the patient was commenced on broad-spectrum antibiotics after obtaining blood cultures. The diagnosis was reviewed, and bronchoscopy was performed three days later. Bronchoscopy was normal and bronchoalveolar lavage (BAL) demonstrated a white blood cell count of 2620 cell/mm^3, with 90% eosinophils, and a red blood cell count of 120 cell/mm^3. BAL investigations, including direct smear for acid-fast bacilli, GeneXpert testing, and pyogenic cultures, were negative.

A diagnosis of chronic pulmonary eosinophilia was made, and oral prednisolone was initiated at a dose of 30 mg/day. The patient showed rapid clinical improvement, with complete resolution of symptoms and radiological abnormalities within two weeks of initiating corticosteroid therapy.

DISCUSSION

Sri Lanka, being an endemic country for pulmonary tuberculosis, faces a significant public health burden. The diagnosis of pulmonary tuberculosis depends on microbiological confirmation. The tuberculin skin test (TST) is performed as a supportive investigation. However, it is not always possible to obtain bacteriological confirmation, and in such instances, the diagnosis is made on clinical grounds with supportive radiological evidence. Such decisions are made by a clinical expert, typically a consultant respiratory physician, and empirical treatment is initiated.

In these situations, patients should be monitored closely for a possible alternative diagnosis, especially if they do not show the expected improvement with anti-tuberculosis treatment (ATT)[3]. While empirical therapy may be lifesaving in true tuberculosis, it can lead to delay and inappropriate treatment if the underlying condition is misdiagnosed.[6]

The typical clinical features and radiological signs of pulmonary tuberculosis can be seen in many other conditions, such as eosinophilic lung diseases, sarcoidosis, lymphomas, and fungal infections. Eosinophilic lung diseases, including chronic eosinophilic pneumonia (CEP), tropical pulmonary eosinophilia (TPE), and eosinophilic granulomatosis with polyangiitis (EGPA), may present with similar constitutional symptoms, radiographic opacities, and chronic cough, leading to diagnostic confusion[7,8].

In tuberculosis-endemic countries such as Sri Lanka, clinicians frequently face the dilemma of treating pulmonary tuberculosis in the absence of microbiological confirmation. When patients present with classic symptoms - such as chronic cough, constitutional symptoms, and characteristic chest X-ray findings - a clinical diagnosis may appear justified, especially when diagnostic tests are inconclusive, inaccessible or delayed. The benefits of initiating treatment on clinical grounds include early disease control, prevention of complications, and reduction in community transmission.

The initial diagnosis of pulmonary tuberculosis was reconsidered when the patient demonstrated persistent symptoms despite empirical anti-tuberculosis therapy, along with a rising peripheral eosinophil count, persistently elevated C-reactive protein, and radiographic findings suggestive of “flitting” pulmonary infiltrates. These red flag features prompted consideration of an alternative diagnosis, namely eosinophilic lung disease.

Chronic eosinophilic pneumonia (CEP) is a rare, idiopathic eosinophilic lung disease characterized by marked eosinophilic infiltration of the alveolar spaces and interstitium. The incidence of CEP among interstitial lung diseases (ILDs) has been reported to be 0–2.7% in an ILD registry in Europe and 0.5–1.2% in an ILD registry in the United States[9]. CEP predominantly affects women and non-smokers, which aligns with the demographic profile of the patient in this case, although most reported cases typically involve middle-aged women. While this patient did not have a history of asthma, it is worth noting that approximately 50% of CEP patients report asthma, often of recent onset.

Patients with CEP usually experience a gradual onset of symptoms. Although this patient had symptoms for only two to three months at the time of presentation, most reported cases describe a symptom duration of around eight months by the time of diagnosis. Common presenting symptoms include cough and difficulty in breathing. Some patients may also present with wheezing, fever, loss of appetite, and even weight loss.

CEP is diagnosed based on clinical symptoms, laboratory findings, and exclusion of other eosinophilic lung diseases. The current working criteria include: 1) clinical symptoms lasting more than two weeks, 2) abnormal chest radiographic findings, 3) eosinophilia detected in bronchoalveolar lavage (BAL) fluid (usually >25%), peripheral blood eosinophilia, and/or evident eosinophilic infiltration in the lungs.

When bronchoscopy is performed, BAL fluid typically demonstrates eosinophilia exceeding 25%. Peripheral blood eosinophilia is commonly present and usually moderately elevated. In addition, histological or radiological evidence of eosinophilic infiltration in the lungs further supports the diagnosis.

It is crucial to exclude other causes with similar clinical and radiological features, including drug- and toxin-induced eosinophilic pneumonias, parasitic infections, allergic bronchopulmonary aspergillosis (ABPA), and eosinophilic granulomatosis with polyangiitis (EGPA). A thorough clinical assessment, exposure history, and relevant investigations are essential to differentiate CEP from these other conditions.[10]

Radiologically, chronic eosinophilic pneumonia (CEP) is characterized by peripheral, often bilateral, subpleural consolidations, predominantly involving the upper and middle lung zones. This pattern is frequently described as the "photographic negative" of pulmonary edema due to its peripheral distribution. These infiltrates tend to migrate over time, a feature rarely seen in other pulmonary diseases except eosinophilic conditions. The flitting nature of infiltrates is a hallmark that distinguishes CEP from other infections such as pulmonary tuberculosis, in which radiological abnormalities typically persist or progress unless adequately treated.[11]

High-resolution computed tomography (HRCT) provides superior resolution, revealing patchy ground-glass opacities, consolidations with air bronchograms, and subpleural ground-glass densities, as observed in this case. These findings supported a diagnosis of CEP and helped exclude differential diagnoses such as neoplasm.[12]

The natural history of CEP includes spontaneous remission in some cases, but immunosuppressive treatment is often required to prevent progression and fibrosis.[13]

The mainstay of chronic eosinophilic pneumonia (CEP) treatment is systemic corticosteroids, which induce rapid clinical and radiological improvement in the majority of patients. Prednisolone is typically started at 0.5–1 mg/kg/day, continued for 2–4 weeks, and gradually tapered over several months depending on clinical response and risk of recurrence.[14]

In this patient, oral prednisolone 30 mg daily was initiated, resulting in dramatic improvement with complete resolution of symptoms and radiological clearance within two weeks.

Although adrenal suppression is uncommon following a short course of high-dose corticosteroids (≤2 weeks), a gradual taper is often recommended in patients with CEP due to the significant risk of relapse. Relapses are common, occurring in up to 50% of patients, and may require prolonged maintenance therapy or additional immunosuppressive agents such as azathioprine or cyclophosphamide.[15] Biologic therapies, including anti-IgE antibody (omalizumab), anti-IL-5 antibody (mepolizumab), and anti-IL-5 receptor antibody (benralizumab), show promise in steroid-dependent or refractory cases for their steroid-sparing effects.[16]

Chronic eosinophilic pneumonia (CEP) generally has an excellent prognosis with timely corticosteroid treatment. Most patients achieve complete remission; however, long-term follow-up is necessary to monitor for relapses. Chronic, untreated CEP may progress to pulmonary fibrosis, resulting in irreversible lung damage and respiratory failure.[17]

CONCLUSION

This case highlights the importance of re-evaluating patients who fail to improve despite a clinical diagnosis of tuberculosis. Chronic eosinophilic pneumonia (CEP) is an important differential diagnosis in patients presenting with pulmonary infiltrates and constitutional symptoms, particularly in tuberculosis-endemic settings where clinical diagnosis of pulmonary tuberculosis is common.

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Author Contribution : KK: contributed to patient management, data collection, and manuscript drafting, **WD**: was involved in data compilation and literature review, and edited the manuscript, **JI**: performed data interpretation and critical revision of the manuscript, **AI**: supervised the study and approved the final manuscript for submission.

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