Clinical Characteristics, Diagnosis, and Management of Tropical Pulmonary Eosinophilia in Respiratory Practice-An Updated Review for Laboratory Professionals

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Abstract:

Background: Tropical pulmonary eosinophilia (TPE) is a rare, immunologically mediated hypersensitivity reaction to microfilarial antigens, primarily caused by *Wuchereria bancrofti* and *Brugia* species. It manifests with nocturnal cough, dyspnea, wheezing, and marked peripheral eosinophilia, and may progress to fibrosis without early treatment.

Aim: This review aims to summarize current knowledge on the clinical characteristics, diagnosis, pathophysiology, and management of TPE, highlighting its relevance in both endemic and non-endemic regions.

Methods: The article synthesizes historical descriptions, epidemiological data, immunopathological insights, diagnostic criteria, and therapeutic strategies for TPE, drawing from contemporary literature and clinical experience.

Results: TPE results from an exaggerated type I hypersensitivity response to microfilariae trapped in the pulmonary vasculature, triggering intense eosinophilic inflammation and elevated IgE. Although endemic to tropical regions, increased global travel has expanded its geographic distribution. Diagnosis relies on characteristic symptoms, eosinophilia often >3,000/μL, elevated IgE, radiologic infiltrates, positive antifilarial antibodies, and marked clinical response to diethylcarbamazine (DEC). DEC at 6 mg/kg/day for 21 days remains the treatment of choice, producing rapid clinical improvement, though relapse occurs in up to 20% of patients. Adjunct corticosteroids may be required for persistent inflammation. Untreated disease risks progression to chronic fibrosis and pulmonary hypertension.

Conclusion: Early diagnosis and prompt DEC therapy result in excellent outcomes for most patients. Failure to treat can lead to irreversible structural lung changes, highlighting the need for clinical vigilance—especially in non-endemic regions where cases may be missed.

Keywords: Tropical pulmonary eosinophilia, filariasis, eosinophilia, diethylcarbamazine, pulmonary fibrosis, hypersensitivity.

INTRODUCTION:

Tropical pulmonary eosinophilia represents a distinct hyperresponsive pulmonary disorder that arises as an immunologic reaction to microfilariae retained within the pulmonary microvasculature and interstitial tissues. The condition reflects an exaggerated host immune response rather than direct parasitic invasion of lung parenchyma. The term was formally introduced by Weingarten in 1943 to describe a characteristic respiratory manifestation associated with lymphatic filariasis, a helminthic infection caused by filarial nematodes. Earlier clinical descriptions by Frimodt-Moller in 1940 referred to a similar presentation as "pseudotuberculosis with eosinophilia," highlighting the resemblance of its pulmonary features to tuberculous disease while emphasizing the marked peripheral eosinophilia that distinguished it from mycobacterial infection [1][2]. This historical evolution in terminology reflects the progressive clarification of its etiological basis and immunopathological mechanisms. Clinically, tropical pulmonary eosinophilia typically develops insidiously, with patients experiencing a gradual onset of systemic and respiratory manifestations. Common presenting features include low-grade fever, paroxysmal nocturnal cough, progressive dyspnea, and episodic wheezing. The nocturnal predominance of cough is considered characteristic and may relate to circadian variation in immune and inflammatory responses. Although the disorder is most frequently encountered in regions where filarial infections are endemic, patterns of global migration and increased international travel have contributed to rising recognition in non-endemic, industrialized settings [3]. This epidemiological shift underscores the importance of maintaining diagnostic awareness beyond traditionally affected geographic areas.

Lymphatic filariasis itself is recognized as a neglected tropical disease with a substantial global burden, primarily affecting populations in tropical and subtropical climates. Its most widely recognized clinical sequela is elephantiasis, resulting from chronic lymphatic obstruction. The World Health Organization estimates that approximately 120 million individuals worldwide are infected with lymphatic filarial parasites; however, fewer than 1% of affected individuals develop tropical pulmonary eosinophilia. This relatively low proportion suggests that host-specific immunological factors play a critical role in determining susceptibility to the pulmonary syndrome. From a broader perspective, pulmonary eosinophilic syndromes are categorized according to underlying etiologic mechanisms into extrinsic and intrinsic forms. Extrinsic causes are attributable to identifiable external agents, including certain pharmacologic agents such as sulfonamides, phenytoin, phenobarbital, and carbamazepine, as well as infectious pathogens including parasitic, fungal, and mycobacterial organisms. In contrast, intrinsic causes arise from primary inflammatory or immunologic disorders, such as Churg-Strauss syndrome, eosinophilic granuloma, chronic eosinophilic pneumonia, and idiopathic hypereosinophilic syndrome. Within this classification framework, tropical pulmonary eosinophilia is considered an extrinsic eosinophilic lung disease, as its pathogenesis is directly linked to an identifiable parasitic trigger [1][2][3].

Etiology

Tropical pulmonary eosinophilia represents an immunologically mediated pulmonary disorder that develops as a consequence of an exaggerated type I hypersensitivity reaction to circulating microfilariae that become sequestered within the pulmonary microvasculature and interstitial

tissues. Rather than resulting from direct tissue invasion or destructive parasitic proliferation in the lungs, the syndrome reflects a heightened host immune response to parasitic antigens. The entrapped microfilariae trigger intense eosinophilic inflammation, elevated immunoglobulin E levels, and the release of inflammatory mediators that contribute to airway hyperreactivity and parenchymal involvement. This immunopathological cascade explains the marked peripheral eosinophilia and respiratory manifestations that characterize the condition. The etiologic agents implicated in human lymphatic filariasis include the filarial nematodes Wuchereria bancrofti, Brugia malayi, and Brugia timori. These helminths primarily reside within the lymphatic system of the human host, where adult worms establish chronic infection and produce microfilariae that circulate in the bloodstream. While lymphatic obstruction and chronic lymphedema represent the classical consequences of filarial infection, a subset of infected individuals develops pulmonary hypersensitivity in response to microfilarial antigens. Among these species, Wuchereria bancrofti accounts for the majority of global infections and is most frequently associated with tropical pulmonary eosinophilia, although Brugia species can also contribute to disease pathogenesis [4][5]. Transmission of lymphatic filariasis occurs through the bite of infected mosquitoes, which function as biological vectors in the parasite's life cycle. When a mosquito feeds on an infected individual, it ingests circulating microfilariae that subsequently mature into infective larvae within the vector. These larvae are then transmitted to another human host during subsequent blood meals. The principal mosquito genera responsible for transmitting Wuchereria bancrofti include Culex, Anopheles, and Aedes species [4][5]. The efficiency of transmission is influenced by ecological, climatic, and socioeconomic factors that support sustained vector breeding and human exposure. Consequently, endemicity remains closely linked to tropical and subtropical environments where vector populations are abundant and control measures are limited.

Epidemiology

Tropical pulmonary eosinophilia is encountered predominantly in tropical and subtropical regions where lymphatic filariasis remains endemic. The highest concentration of reported cases originates from the Indian subcontinent, Southeast Asia, parts of South America, and several regions of Africa, reflecting the geographic distribution of filarial transmission. These areas provide favorable ecological conditions for mosquito vectors and sustain ongoing transmission cycles. Although the disorder is traditionally associated with endemic zones, its geographic boundaries are no longer confined to these settings. Increasing global mobility, labor migration, tourism, and population displacement have facilitated the appearance of cases in North America, Europe, and other non-endemic Western countries. As a result, clinicians practicing outside classical endemic areas must maintain awareness of this entity, particularly when evaluating patients with unexplained eosinophilia and respiratory symptoms who have relevant travel or migration histories. The risk of developing tropical pulmonary eosinophilia varies according to host immune status and prior exposure to filarial antigens. Individuals who reside permanently in endemic regions often develop partial immunologic tolerance or modulation due to repeated exposure to microfilariae. In contrast, nonimmune individuals, including travelers or recent migrants to endemic areas, appear more susceptible to developing the exaggerated hypersensitivity response that characterizes the pulmonary syndrome. This observation supports the concept that immunologic priming and host-parasite interactions influence disease expression. Epidemiologic data also demonstrate a marked sex disparity, with males affected approximately four to seven times more frequently than females. The reasons for this difference are not fully defined but may relate to occupational exposure patterns, behavioral factors, or sex-based immunologic variation. The condition most commonly

presents in young adults, an age group that may experience higher exposure risk or more robust immune reactivity. Furthermore, disease severity tends to be greater in immunocompromised individuals, in whom dysregulated immune responses may amplify pulmonary inflammation and clinical manifestations [4].

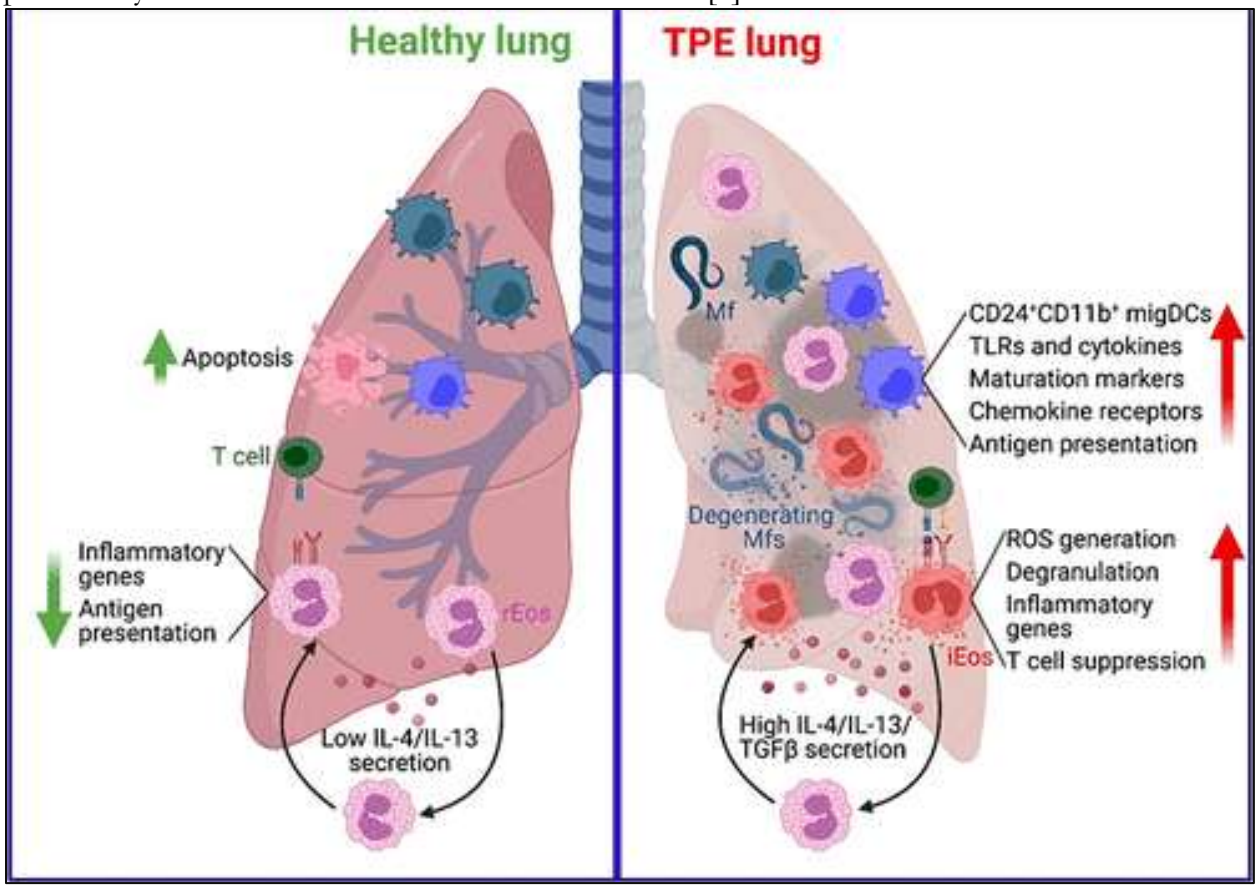

Fig. 1: Tropical pulmonary eosinophilia.

Pathophysiology

The pathophysiological mechanisms underlying tropical pulmonary eosinophilia are complex and not yet fully elucidated, despite extensive investigation. Multiple hypotheses have been advanced to explain the immunologic and inflammatory processes that define this syndrome. Central to all proposed mechanisms is the pivotal role of eosinophils as effector cells mediating both host defense and tissue injury. The initiating event is believed to involve the periodic release of microfilariae from the lymphatic system into the peripheral circulation. These circulating microfilariae become mechanically entrapped within the pulmonary microvasculature due to their size and host immune interactions. Their sequestration within the lung triggers an intense local and systemic immune response characterized by marked eosinophilic activation. Following antigen recognition, eosinophils are recruited in large numbers to the pulmonary interstitium and alveolar spaces. Once activated, they undergo degranulation and release a spectrum of cytotoxic and proinflammatory mediators, including eosinophilic cationic protein, eosinophil-derived neurotoxin, major basic proteins, and eosinophil peroxidase. These substances contribute to the destruction and clearance of microfilariae, serving a protective antiparasitic function. However, the same mediators exert deleterious effects on pulmonary tissues, promoting epithelial injury, interstitial inflammation, and airway dysfunction. This dual role of eosinophils explains the paradox of effective parasitic

clearance accompanied by progressive pulmonary damage. Complement activation and antibody-mediated opsonization further enhance microfilarial elimination. Antifilarial antibodies bind circulating parasites and facilitate their recognition by immune effector cells. Among eosinophil-derived mediators, major basic protein-2 has been implicated in the development of airway hyperresponsiveness. Cytokine signaling plays a critical regulatory role in this process. Interleukin-4 promotes airway hyperactivity and supports eosinophil activation, whereas interferon-gamma exerts an inhibitory influence that may modulate excessive inflammation. The immunologic profile in tropical pulmonary eosinophilia is characterized by an exaggerated T helper 2 (TH2) response within both systemic and pulmonary compartments. This response leads to elevated concentrations of IL-4, IL-5, and IL-13, along with increased production of filarial-specific IgG, IgM, and IgE antibodies and profound peripheral and pulmonary eosinophilia [6]. IL-4 enhances eosinophil priming and activation, while IL-13 upregulates adhesion molecules on endothelial cells, facilitating eosinophil migration into lung tissue.

Specific parasitic antigens appear to amplify this hypersensitivity cascade. Bm22-25, a dominant antigen derived from the infective L3 larval stage of Brugia malayi, has been shown to stimulate IgE synthesis in affected individuals. Structural similarities between gamma-glutamyl transpeptidase expressed on human pulmonary epithelial cells and that found on the surface of infective larvae suggest a potential mechanism of molecular mimicry. This resemblance may intensify immune cross-reactivity and sustain inflammatory injury. Experimental studies have also identified acidic, calcium-independent phospholipase A2 as a key lysosomal regulator in the inflammatory pathway of tropical pulmonary eosinophilia, supporting its role as a central mediator in disease progression [7]. Pharmacologic intervention provides insight into the inflammatory mechanisms involved. Diethylcarbamazine therapy effectively suppresses the intense eosinophilic alveolitis characteristic of the disorder, indicating that reduction of microfilarial antigenic stimulation can attenuate downstream immune activation [8][9]. In addition to eosinophil-derived toxins, inflammatory cells in the lower respiratory tract release reactive oxygen species such as superoxide and hydrogen peroxide. These oxidants contribute to epithelial damage and may participate in both acute inflammatory exacerbations and chronic structural alterations [10]. Consequently, short courses of corticosteroids may be required in selected cases to dampen excessive inflammatory mediator production and limit pulmonary injury.

Histopathology

The histopathological progression of tropical pulmonary eosinophilia reflects the evolving immunologic and inflammatory response to microfilarial antigens within the pulmonary tissue. The earliest detectable change involves infiltration of histiocytes into the lung parenchyma, which correlates with the initial clinical manifestations of cough, dyspnea, and wheezing. This accumulation of macrophage-lineage cells represents the host's initial attempt to contain and process trapped microfilariae, establishing the foundation for subsequent inflammatory cascades. Following the initial histiocytic response, there is a pronounced eosinophilic interstitial infiltration. Eosinophils accumulate in large numbers and may form localized aggregates, leading to the development of eosinophilic abscesses, eosinophilic granulomas, or eosinophilic bronchopneumonia. These lesions reflect the dual role of eosinophils in mediating parasitic clearance and inducing collateral tissue injury. The cytotoxic proteins released by degranulating eosinophils contribute to epithelial and interstitial damage, perpetuating the inflammatory response and contributing to airway hyperreactivity. Between six months and two years from disease onset, histopathology reveals a more complex mixed-

cell infiltrate. This includes histiocytes, eosinophils, epithelioid cells, and lymphocytes, indicating a transition from acute eosinophilic inflammation to a more organized, chronic immune response. The persistence of antigenic stimulation during this period sustains inflammation and increases the risk of tissue remodeling. In cases where the condition remains untreated, ongoing inflammatory injury may progress to pulmonary fibrosis. Fibrotic changes reflect irreversible deposition of extracellular matrix and disruption of normal alveolar architecture, which can result in chronic respiratory impairment and reduced pulmonary function [4]. The histopathological spectrum of tropical pulmonary eosinophilia thus mirrors the clinical course, highlighting the importance of early diagnosis and intervention to prevent long-term sequelae [4].

History and Physical

The clinical history of patients with tropical pulmonary eosinophilia is essential for establishing a suspicion of the disease, particularly in individuals with relevant epidemiologic exposure. Most patients report either residence in or recent travel to regions where filarial infections are endemic. These regions include parts of South and Southeast Asia, Africa, and South America, reflecting the distribution of mosquito vectors capable of transmitting filarial parasites. A thorough medication and dietary history is equally important, as alternative causes of pulmonary eosinophilia must be considered. Certain infections, dietary exposures, or pharmacologic agents can precipitate conditions such as Loeffler syndrome, which also presents eosinophilic pulmonary involvement secondary to parasitic infection. Differentiating tropical pulmonary eosinophilia from these conditions relies on integrating clinical, laboratory, and exposure data. The onset of tropical pulmonary eosinophilia is generally insidious, with characteristic respiratory and systemic manifestations. Patients often present with a paroxysmal, predominantly nocturnal dry cough, which may be accompanied by dyspnea of variable severity. Systemic symptoms such as low-grade fever, malaise, anorexia, and unintentional weight loss frequently accompany the pulmonary features. Peripheral blood eosinophilia is typically marked, often exceeding 3,000 cells per microliter, and serves as a key laboratory indicator supporting the diagnosis. Rare extrapulmonary findings, including hepatosplenomegaly or lymphadenopathy, may be observed in some cases [11]. Physical examination findings reinforce the clinical suspicion. Chest auscultation commonly reveals wheezing and crepitations, which may mimic asthma in presentation. While pulmonary signs are predominant, abdominal palpation in select patients may detect mild organomegaly, and examination of the cervical and axillary regions may reveal enlarged lymph nodes. These findings reflect the underlying immune-mediated inflammatory response triggered by trapped microfilariae. The combination of epidemiologic exposure, characteristic symptomatology, laboratory eosinophilia, and supportive physical findings provides a foundation for accurate diagnosis and guides subsequent confirmatory testing and therapeutic intervention [11].

Evaluation

The evaluation of a patient suspected of having tropical pulmonary eosinophilia requires a comprehensive approach integrating laboratory, radiologic, and functional assessments. Prompt and accurate evaluation is critical, as delayed diagnosis can allow progression of interstitial inflammation to irreversible pulmonary fibrosis, resulting in chronic respiratory compromise [12]. The diagnostic workup begins with detailed laboratory studies, including a complete blood count. Leukocytosis accompanied by pronounced eosinophilia, typically exceeding 3,000 cells per microliter, represents the cardinal hematologic finding and provides the first objective evidence of systemic immune activation. This eosinophilic response reflects the host's hypersensitivity to microfilarial antigens and is a critical marker for disease severity

and activity. Stool examination plays a complementary role by excluding alternative parasitic infections that can produce eosinophilic pulmonary syndromes. Identifying non-filarial helminths such as Strongyloides or Schistosoma species is essential because these pathogens require distinct therapeutic approaches. In addition, quantitative serum immunoglobulin testing is frequently performed, as elevated immunoglobulin E (IgE) levels are consistently observed in tropical pulmonary eosinophilia and correlate with the intensity of the immunologic response. Confirmation of the diagnosis often involves serologic detection of filarial-specific antibodies using indirect enzyme-linked immunosorbent assays (ELISA). A significant rise in antibody titers against Wuchereria bancrofti or Brugia species supports the diagnosis and helps differentiate it from other causes of pulmonary eosinophilia [13].

Radiologic assessment is an integral part of the evaluation. Chest radiography may reveal reticulonodular patterns, miliary mottling predominantly in the middle or lower lung zones, or interlobular septal thickening. Despite these typical findings, up to 20% to 30% of patients may demonstrate normal radiographs in early disease, and advanced cases may reveal fibrotic changes. Computed tomography (CT) scanning is reserved for patients with atypical or inconclusive radiographic findings. CT imaging can demonstrate features such as bronchiectasis, mediastinal or hilar lymphadenopathy, and pleural effusions, providing additional anatomical detail that guides clinical decision-making. Pulmonary function tests (PFTs) commonly demonstrate a mixed ventilatory pattern characterized by a predominant restrictive defect with variable obstructive elements. Mild hypoxemia may also be detected, reflecting impaired gas exchange due to interstitial inflammation. Bronchoalveolar lavage, when performed, typically reveals a marked eosinophilic predominance in alveolar and interstitial samples, confirming active pulmonary involvement [11]. Clinicians must remain vigilant for serological cross-reactivity, as high antifilarial IgG titers can result from exposure to nonfilarial helminths. Such cross-reactivity can mimic tropical pulmonary eosinophilia-like syndromes and necessitates additional, more specific diagnostic testing to accurately determine the underlying etiology [14]. The established diagnostic criteria for tropical pulmonary eosinophilia combine epidemiologic exposure, clinical manifestations, laboratory evidence of eosinophilia and elevated IgE, radiologic pulmonary infiltrates, and a favorable clinical response to diethylcarbamazine therapy. Collectively, these evaluations provide a robust framework for accurate diagnosis, guiding both timely intervention and monitoring of therapeutic outcomes [11][12][13].

Treatment / Management

The management of tropical pulmonary eosinophilia is primarily guided by prompt initiation of antiparasitic therapy following diagnostic confirmation. Diethylcarbamazine remains the cornerstone of treatment due to its potent activity against both microfilariae and adult filarial worms. The recommended dosing regimen is 6 mg/kg/day, administered in two to three divided doses for a total duration of 21 days [15]. Clinical response to diethylcarbamazine is typically rapid and pronounced, with alleviation of fever, cough, dyspnea, and wheezing often occurring within days of treatment initiation. Resolution of peripheral eosinophilia and improvement in pulmonary function further support the effectiveness of this intervention. This dramatic therapeutic response is considered a hallmark of tropical pulmonary eosinophilia and can serve as both a diagnostic and prognostic indicator. However, approximately 5% of patients may exhibit a suboptimal response, and relapse occurs in up to 20% of cases, highlighting the need for careful follow-up and potential retreatment in selected patients. In addition to antiparasitic therapy, adjunctive use of corticosteroids may be warranted, particularly in cases characterized by chronic airway inflammation or persistent

symptoms. Corticosteroids exert anti-inflammatory effects that mitigate the damage caused by activated inflammatory cells in the lower respiratory tract, which release reactive oxygen species such as superoxide and hydrogen peroxide. These oxidants contribute to interstitial inflammation, airway hyperreactivity, and early fibrotic changes. By suppressing these mediators, corticosteroids can reduce tissue injury and improve symptom control in patients with prolonged or severe disease [16][17][18][19]. Prior to corticosteroid therapy, it is essential to rule out coexisting Strongyloides infection, as immunosuppressive treatment in the presence of threadworm can precipitate life-threatening disseminated disease.

Alternative pharmacologic options have been explored, though data remain limited. Ivermectin has demonstrated efficacy against microfilariae, whereas albendazole primarily targets adult filarial worms. Despite their theoretical utility, there is a lack of published clinical data supporting their routine use in tropical pulmonary eosinophilia. In regions where diethylcarbamazine is unavailable, doxycycline administered at 200 mg orally daily for four to six weeks, combined with corticosteroids, has been proposed as a viable alternative. This regimen targets the endosymbiotic Wolbachia bacteria within filarial worms, indirectly impairing parasite survival and reducing antigenic stimulation of the host immune system [20].

Long-term management also includes monitoring for relapse, evaluating pulmonary function, and assessing for residual interstitial changes. Patients may require repeated courses of therapy or extended corticosteroid treatment if symptoms persist. Preventive strategies, including vector control and community-based filariasis elimination programs, complement individual patient management by reducing exposure to infective mosquitoes and the overall incidence of tropical pulmonary eosinophilia. Comprehensive management, integrating pharmacologic treatment, careful monitoring, and preventive measures, remains essential to reduce morbidity and preserve long-term pulmonary function in affected individuals [16][17][18].

Differential Diagnosis

When evaluating a patient with suspected tropical pulmonary eosinophilia, it is critical to consider a range of differential diagnoses, as several other conditions can produce overlapping respiratory and systemic manifestations. Parasitic infections other than filariasis, including Strongyloides stercoralis, Toxocara species, and Ascaris lumbricoides, may lead to pulmonary eosinophilia through similar immunologic mechanisms. Loeffler syndrome, which arises from transient pulmonary migration of helminth larvae, can mimic the nocturnal cough, dyspnea, and eosinophilic response observed in tropical pulmonary eosinophilia. Distinguishing these conditions requires detailed travel or exposure history, stool examination, serologic testing, and, in some cases, molecular diagnostics to identify the specific parasitic etiology. Non-infectious causes of pulmonary eosinophilia must also be considered. Bronchial asthma frequently presents episodic wheezing, cough, and dyspnea, but typically lacks extreme peripheral eosinophilia. Allergic bronchopulmonary aspergillosis, often associated with recurrent pulmonary infiltrates and elevated IgE, may be distinguished through Aspergillus-specific IgE testing and imaging findings. Acute and chronic eosinophilic pneumonias, which can develop independently of infectious triggers, present with diffuse pulmonary infiltrates, systemic symptoms, and significant eosinophilic infiltration on bronchoalveolar lavage, requiring histopathologic or imaging confirmation. Systemic immunologic disorders such as Churg-Strauss syndrome (eosinophilic granulomatosis with polyangiitis) and DRESS syndrome (drug reaction with eosinophilia and systemic symptoms) must also be considered. These conditions can present with pulmonary involvement, systemic eosinophilia, and multi-organ manifestations, but are typically differentiated based on clinical course, medication history, serologic markers, and histopathology. Miliary tuberculosis, although primarily

infectious, can occasionally present with diffuse lung involvement and systemic symptoms, requiring microbiologic or molecular testing to rule out. Allergic rhinitis, while common, rarely produces the high-grade eosinophilia characteristic of tropical pulmonary eosinophilia. Careful integration of clinical, laboratory, and imaging findings is essential to differentiate these conditions, ensure accurate diagnosis, and avoid inappropriate therapy [21][22].

Treatment Planning

The cornerstone of tropical pulmonary eosinophilia management is targeted antiparasitic therapy, with diethylcarbamazine representing the primary pharmacologic intervention. The standard dosing regimen is 6 mg/kg/day, administered in two to three divided doses over a 21-day period. This agent exhibits dual activity against both microfilariae and adult filarial worms, directly addressing the source of antigenic stimulation responsible for pulmonary hypersensitivity. Rapid clinical improvement following initiation of diethylcarbamazine, including resolution of fever, cough, wheezing, and peripheral eosinophilia, is considered characteristic and confirms both diagnosis and therapeutic efficacy. Adjunctive interventions may be required in patients with persistent respiratory symptoms, chronic inflammation, or evidence of early interstitial involvement. Short courses of corticosteroids can reduce airway inflammation, limit oxidative tissue injury, and provide symptomatic relief, particularly in patients with evidence of ongoing lower respiratory tract inflammation. In areas where diethylcarbamazine is unavailable, alternative regimens such as doxycycline combined with corticosteroids have been advocated to target filarial endosymbionts and reduce microfilarial burden indirectly. Other antiparasitic agents, including ivermectin and albendazole, may theoretically provide additional coverage, although clinical evidence supporting their routine use remains limited. Treatment planning must also include patient education regarding adherence, potential relapse, and monitoring for complications, as approximately 20% of patients may experience recurrence, necessitating re-evaluation and possible retreatment. The combination of targeted pharmacologic therapy, adjunctive anti-inflammatory measures, and careful follow-up constitutes a comprehensive treatment strategy for tropical pulmonary eosinophilia [22].

Toxicity and Adverse Effect Management

While diethylcarbamazine is highly effective, its administration is associated with a range of adverse effects that require careful monitoring and management. Common side effects include fever, headache, gastrointestinal disturbances such as nausea, vomiting, abdominal discomfort, as well as pruritus. These reactions are reported in up to 25% of patients and may persist even after completion of therapy [12]. The underlying mechanisms are thought to involve immunologic reactions to dying microfilariae, resulting in transient systemic and inflammatory responses. Management of these adverse effects primarily involves supportive care. Antipyretics and analgesics may be administered to alleviate fever and headache, while antihistamines can reduce pruritus and associated discomfort. Gastrointestinal symptoms can be managed with dietary modifications, antiemetic agents, and adequate hydration. Clinicians should counsel patients regarding the expected course of side effects, emphasizing that these reactions are typically self-limited and do not indicate treatment failure. In rare cases of severe hypersensitivity or systemic reaction, temporary discontinuation or dose adjustment of diethylcarbamazine may be necessary, under close supervision. Regular follow-up during and after therapy is recommended to monitor for both therapeutic response and adverse effects, ensuring safe and effective resolution of tropical pulmonary eosinophilia symptoms while minimizing patient morbidity.

Prognosis

The prognosis of tropical pulmonary eosinophilia is generally favorable when the condition is identified early and treated promptly with diethylcarbamazine. Most patients exhibit a dramatic clinical response, with rapid resolution of nocturnal cough, dyspnea, and systemic symptoms, alongside normalization of peripheral eosinophil counts. Early intervention often prevents irreversible pulmonary damage and allows near-complete restoration of lung function. Despite this, studies have shown that a subset of patients may develop persistent mild interstitial lung disease even after appropriate therapy. This residual parenchymal involvement can manifest as subtle functional limitations, mild dyspnea on exertion, or radiographic abnormalities. Long-term follow-up is therefore recommended to monitor pulmonary function and detect early signs of chronic disease progression. Relapse is another concern, with approximately 20% of patients experiencing recurrence within five years. This may be due to incomplete eradication of microfilariae, ongoing exposure in endemic regions, or individual variations in immune response. Pulmonary hypertension represents a significant adverse prognostic indicator. In patients who develop pulmonary hypertension, the condition is frequently irreversible and associated with increased morbidity due to the sustained hemodynamic burden on the right heart. Such patients require careful long-term monitoring and management to mitigate the risk of progressive heart failure. Overall, while the majority of patients respond well to standard therapy, prognostic outcomes are closely linked to the timing of diagnosis, adequacy of treatment, and the presence of complications such as pulmonary hypertension or chronic interstitial changes. Early recognition and intervention remain critical to optimizing long-term outcomes in tropical pulmonary eosinophilia [23].

Complications

Tropical pulmonary eosinophilia carries a risk of significant complications, particularly if diagnosis and treatment are delayed. The chronic inflammatory response to trapped microfilariae can lead to structural remodeling of the lung parenchyma, resulting in pulmonary fibrosis. Fibrotic changes impair gas exchange and contribute to restrictive ventilatory defects, which may persist even after the eradication of the filarial infection. Chronic bronchitis is another potential sequela, characterized by persistent cough, mucus production, and intermittent wheezing, which can progress to chronic respiratory failure in severe or prolonged cases. Pulmonary hypertension is a less common but serious complication that may arise from embolization of destroyed microfilariae into the pulmonary vasculature. This leads to increased pulmonary vascular resistance, right ventricular strain, and, in advanced cases, cor pulmonale. Patients with pulmonary hypertension often experience exercise intolerance, progressive dyspnea, and increased risk of cardiovascular morbidity. Early identification and management of tropical pulmonary eosinophilia are therefore essential to prevent these long-term consequences. In addition to cardiopulmonary complications, persistent eosinophilic inflammation may predispose patients to secondary infections, further compromising respiratory function. Comprehensive monitoring, early therapeutic intervention, and ongoing pulmonary assessment are crucial for minimizing the risk of these complications and improving patient quality of life [22].

Consultations

Following the diagnosis of tropical pulmonary eosinophilia, multidisciplinary consultations are essential to ensure comprehensive care. Infectious disease specialists play a central role in confirming the filarial etiology, guiding appropriate antiparasitic therapy, and monitoring for potential treatment failures or relapses. Pulmonologists provide expertise in evaluating the extent of pulmonary involvement, interpreting radiographic and functional studies, and

managing chronic respiratory complications such as fibrosis or pulmonary hypertension. Collaboration with public health experts is equally important, particularly in advising on preventive measures, assessing exposure risks, and coordinating community-based interventions to reduce filarial transmission. Engaging these specialists facilitates early recognition of disease progression, timely initiation of therapy, and integration of preventive strategies. Moreover, consultation ensures that patients receive individualized care tailored to the severity of their symptoms, underlying comorbidities, and potential complications. This multidisciplinary approach enhances diagnostic accuracy, guides long-term monitoring, and supports patient adherence to therapeutic regimens. By combining the expertise of infectious disease experts, pulmonologists, and public health professionals, healthcare teams can optimize outcomes, minimize morbidity, and contribute to broader efforts to control filarial infections in endemic regions [22][23].

Patient Education

Prevention and patient education are critical components in managing tropical pulmonary eosinophilia, particularly for individuals traveling to or residing in filarial-endemic regions. Effective prevention focuses on minimizing exposure to mosquito vectors, the primary means of filarial transmission. Travelers and residents should employ insecticide-treated bed nets, permethrin-impregnated clothing, and other vector-control measures to reduce the risk of mosquito bites. Additionally, public health awareness campaigns play a vital role in educating communities about the transmission cycle, early recognition of symptoms, and the importance of prompt medical evaluation for febrile or respiratory complaints. Patient education should emphasize adherence to preventive strategies, recognition of early symptoms such as nocturnal cough, dyspnea, and systemic manifestations, and the necessity of completing prescribed diethylcarbamazine therapy. Individuals should also be informed about potential relapses and the importance of follow-up evaluations, including laboratory monitoring and pulmonary function testing. In endemic regions, education should extend to community-level interventions, including environmental control of mosquito breeding sites and participation in mass drug administration programs for filariasis. By integrating personal protective measures with community-based public health strategies, the incidence of tropical pulmonary eosinophilia can be significantly reduced, and early recognition and treatment can improve clinical outcomes [21][22][23].

Other Issues

Effective control of lymphatic filariasis directly impacts the incidence of tropical pulmonary eosinophilia. The World Health Organization recommends an integrated elimination strategy, combining annual administration of ivermectin with diethylcarbamazine or albendazole to populations in endemic areas. Given the longevity of adult worms, therapy must be continued for four to six years to interrupt the transmission cycle effectively. Vector control measures targeting mosquito populations further enhance these interventions by reducing exposure to infective bites. Early diagnosis and treatment remain critical to prevent irreversible pulmonary sequelae. Chronic tropical pulmonary eosinophilia can lead to fibrosis and long-term respiratory failure despite therapy. Key clinical indicators include a history of travel to filarial-endemic regions, nocturnal cough, dyspnea, marked peripheral eosinophilia often exceeding 3,000/μL, elevated IgE levels above 1,000 U/L, positive filarial antibodies, radiologic evidence of pulmonary infiltrates, and a favorable response to diethylcarbamazine [15]. Recognizing these features ensures timely intervention and prevents progression to chronic respiratory impairment.

Enhancing Healthcare Team Outcomes

Optimal management of tropical pulmonary eosinophilia requires an interprofessional approach, particularly in regions where the condition is rare. Clinicians and nurses play a key role in early identification through comprehensive history-taking, noting recent travel to endemic areas, characteristic nocturnal cough, dyspnea, wheezing, and significant eosinophilia. Early diagnostic evaluation, including serum IgE and antifilarial antibody testing, coupled with stool examinations to exclude other parasitic infections, ensures accurate diagnosis and timely initiation of therapy. Infectious disease specialists and microbiologists provide confirmatory testing and help distinguish tropical pulmonary eosinophilia from other eosinophilic pulmonary syndromes. Pharmacists contribute to safe administration of diethylcarbamazine, monitoring for adverse effects and drug interactions. Nurses and clinicians provide patient education, reinforce adherence, and monitor clinical response. Strong communication and care coordination among team members enhance patient outcomes by facilitating early intervention, minimizing complications, and ensuring continuity of care. This collaborative model ensures patient-centered management and maximizes safety, efficacy, and long-term pulmonary health for individuals affected by tropical pulmonary eosinophilia [23].

CONCLUSION:

Tropical pulmonary eosinophilia represents a unique pulmonary hypersensitivity syndrome with a generally favorable prognosis when recognized early and treated promptly. The disease is driven by an exaggerated immune response to microfilarial antigens, resulting in marked eosinophilia, nocturnal cough, dyspnea, and diffuse inflammatory injury of the lung parenchyma. Diethylcarbamazine therapy remains highly effective, offering rapid symptomatic and hematologic improvement, and serving as both a diagnostic and therapeutic tool. Despite its responsiveness to treatment, TPE poses significant risks when diagnosis is delayed. Chronic pulmonary inflammation may progress to irreversible fibrosis, restrictive ventilatory defects, and, in severe cases, pulmonary hypertension—an important adverse prognostic factor associated with long-term morbidity. Additionally, relapse rates approaching 20% underscore the need for diligent follow-up, assessment of ongoing exposure risks, and patient education, particularly in endemic areas. As global travel continues to rise, clinicians in non-endemic regions must maintain awareness of TPE when evaluating patients with unexplained eosinophilia and respiratory symptoms. Early recognition, multidisciplinary cooperation, and prompt initiation of therapy remain essential to preventing long-term complications and preserving lung function. Overall, TPE highlights the complex interplay between infectious exposure and host immune response, reinforcing the importance of vigilance, timely treatment, and sustained public health measures.

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