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Pulmonary Eosinophilia in Helminthic Infection: A Comprehensive Review of Loeffler Syndrome Caused by Ascaris Lumbricoides

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Abstract

Background: Loeffler syndrome is a transient, eosinophilic pulmonary condition most commonly caused by the larval migration of helminths, particularly Ascaris lumbricoides. It manifests as a mild respiratory illness characterized by peripheral eosinophilia and transient pulmonary infiltrates. Objective: This review aims to provide a comprehensive overview of the etiology, pathophysiology, clinical features, diagnosis, treatment, complications, and public health implications of Loeffler syndrome associated with Ascaris lumbricoides. Methods: A detailed literature review was conducted focusing on the parasitic lifecycle, immune response, imaging findings, differential diagnosis, and treatment strategies for Loeffler syndrome and associated complications. Results: The syndrome typically resolves spontaneously within a few weeks, although severe cases or complications such as intestinal obstruction, hepatobiliary involvement, or Loeffler endocarditis may require targeted therapy. Antiparasitic treatment should be timed carefully to avoid exacerbation of pulmonary inflammation. Corticosteroids and supportive therapies may be used in select cases. Conclusion: Recognition of the parasitic etiology and characteristic clinical presentation is key to avoiding unnecessary interventions. Prevention through sanitation, deworming, and health education remains essential in endemic areas.

Keywords: Pulmonary Eosinophilia; Ascaris Lumbricoides; Helminthic Infection; Loeffler Syndrome

Abbreviations: LS: Loeffler Syndrome; EGPA: Eosinophilic Granulomatosis with Polyangiitis; HES: Hyper eosinophilic Syndrome; ABPA: Allergic Bronchopulmonary Aspergillosis; GI: Gastrointestinal; CT: Computed Tomography; IgE: Immunoglobulin E; IL-5: Interleukin 5; IL-13: Interleukin 13; PCR: Polymerase Chain Reaction; RR: Relative Risk; CI: Confidence Interval; PMN: Polymorphonuclear Leukocytes

Introduction

Loeffler syndrome is an uncommon, non-severe respiratory condition characterized by peripheral eosinophilia affecting the lungs, often triggered by parasitic infections [1]. The Syndrome manifests as a mild respiratory illness characterized by symptoms such as cough, wheezing, and dyspnea, which typically resolve within 6 to 8 weeks. However, this may lead to future complications, including intestinal obstruction and hepatobiliary manifestations [2]. The condition is named after Wilhelm Loeffler, who

first described the association between eosinophilic pneumonia and parasitic infections in 1932 [1]. The understanding of Loeffler syndrome dates back to early 20th-century observations linking pulmonary eosinophilia to helminthic infections [1]. Wilhelm Loeffler was the first to describe how parasitic larvae Ascaris lumbricoides, Strongyloides stercoral is, and hookworms (Ancylostoma duodenale and Necator americanus) trigger eosinophilic lung reactions during their migration [3]. Progressively, the advancements in diagnostic imaging and laboratory techniques have significantly improved the identification and management of the Syndrome [3]. Initially identified by Loeffler in 1932, the condition occurs when helminth larvae migrate through the lungs during their developmental phase, triggering a temporary immune response before continuing their life cycle [1].

Loeffler syndrome is primarily associated with helminthic infections that undergo pulmonary changes or phases [3]. Parasites such as Ascaris lumbricoides and Strongyloides stercoralis invade the human body through ingestion or skin penetration, subsequently traveling via the bloodstream to the lungs [3,4]. During each phase, the immune system responds by increasing eosinophil production, leading to pulmonary inflammation [3,4]. This reaction is typically self-limiting, resolving as the parasites migrate to their final destination in the gastrointestinal tract [1]. Loeffler syndrome is prevalent in tropical and subtropical regions, where parasitic infections thrive due to poor sanitation and inadequate healthcare [3]. An estimated 800 million to 1.2 billion people are infected with Ascaris lumbricoides, causing over 60,000 deaths annually, while 85% of cases remain asymptomatic [4]. According to Asfaw et al., the Global Burden of Disease 2019 estimated 594 cases per 100,000 people, 2,090 deaths, and 754,000 disability-adjusted life years due to ascariasis [2]. Helminthic infections disproportionately impact low-income communities, leading to malnutrition, cognitive impairment, and increased susceptibility to other diseases [2,3].

These infections persist in 149 tropical and subtropical countries, affecting over 1 billion individuals and costing developing economies billions annually [4]. Strategies to reduce the global burden of Loeffler syndrome include improvements in sanitation, deworming programs, and public health education [1]. Given the significant impact and potential complications associated with this condition, it is crucial to further investigate its pathophysiology, clinical manifestations, and current management strategies. Therefore, this review article aims to deepen our understanding of Loeffler syndrome and contribute to mitigating its burden on vulnerable populations worldwide.

Etiology and Life Cycle of Ascaris lumbricoides

Ascaris lumbricoides is a large intestinal roundworm and the most common helminthic parasite affecting humans globally. It is transmitted via the fecal-oral route, primarily through ingestion of embryonated eggs present in contaminated soil, water, or food. Young children, especially those under the age of five, are at greatest risk due to inadequate hygiene and frequent contact with contaminated environments [5,6]. The life cycle of A. lumbricoides begins when fertilized eggs are excreted in human feces and deposited into the soil. Under favorable environmental conditions-namely, moist, shaded, and warm soil-these eggs embryonate and become infective within approximately 10 to 15 days. Remarkably, these infective eggs can remain viable in the soil for up to ten years [6,7]. After ingestion by a human host, the eggs hatch in the duodenum, releasing larvae. These larvae penetrate the intestinal mucosa and enter the portal venous and lymphatic systems, migrating first to the liver and subsequently to the lungs within the first week of infection [7,8].

In the lungs, the larvae penetrate the alveolar walls and migrate into the alveolar spaces. This pulmonary phase typically occurs between 10 and 14 days after ingestion and represents a critical stage in the parasite's development. During this period, the host's immune response may result in a transient eosinophilic pneumonitis known as Loeffler syndrome. Clinical features include nonproductive cough, dyspnea, wheezing, and blood eosinophilia. Radiographic imaging often reveals transient pulmonary infiltrates, which correspond to larval migration through the lung parenchyma. Symptoms usually resolve spontaneously as the larvae ascend the tracheobronchial tree, are swallowed, and return to the gastrointestinal tract [5,7,9]. Upon reentering the small intestine-most commonly the jejunum and ileum-the larvae mature into adult worms within two to three weeks. Female worms typically measure 20-35cm in length, and males measure 15-30cm. When both sexes are present, fertilization occurs, and the gravid female releases approximately 200,000 eggs per day. These are subsequently excreted in the feces, thus completing the parasitic life cycle [6-8]. Unfertilized eggs, although excreted, are not infective. Adult Ascaris worms can survive in the human intestine for one to two years. In endemic settings with continuous environmental contamination and poor hygiene, this life cycle contributes to ongoing transmission and persistent public health challenges [6].

Pathophysiology of Loeffler Syndrome

Loeffler's syndrome is a self-limited, transient form of pulmonary eosinophilia that results from the host's immune reaction to the migration of helminthic larvae through the lungs. This pulmonary phase, which is integral to the parasites' life cycle, initiates a type I hypersensitivity response [10-14]. Upon entering the human body-either by ingestion or skin penetration-larvae travel via the bloodstream to the lungs as part of their developmental cycle. This pulmonary phase activates a type I hypersensitivity response characterized by the release of Th2 cytokines, notably interleukin-5 (IL-5) and interleukin-13 (IL-13). These cytokines promote the production and recruitment of eosinophils, resulting in a marked eosinophilic infiltration of the pulmonary interstitium and alveolar spaces [10-14]. Clinically, this immune-mediated process manifests as peripheral eosinophilia and transient pulmonary infiltrates seen on radiographic imaging. The inflam-

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mation increases alveolar permeability and can lead to mild respiratory symptoms such as cough and dyspnea. Importantly, this reaction is not caused by direct tissue damage from the parasites, but rather by an exaggerated immune response. As the larvae continue their migration into the gastrointestinal tract to mature, the pulmonary inflammation subsides spontaneously. This self-limited nature, often resolving within weeks, underscores the benign course of Loeffler syndrome in most patients [10-14].

Clinical Manifestations

Löffler syndrome typically appears 10-16 days after ingestion of Ascaris eggs, corresponding to the period of larval migration through the lungs. The same timeframe is noted for ingestion caused by Ancylostoma duodenale, Strongyloides stercoralis, and Necator americanus [15,16]. The syndrome is characterized by mild eosinophilic pneumonitis that tends to resolve spontaneously within 2-3 weeks, due to an immune response against migrating larvae in the lung tissue [15,17]. The most common respiratory manifestation is a dry, non-productive cough, sometimes accompanied by scant mucoid sputum [15,16]. Other common symptoms include fever, dyspnea, wheezing, and malaise. Less commonly, patients may report anorexia, myalgia, and urticaria, which reflect systemic allergic responses [16,18]. Physical examination may be unremarkable; however, lung auscultation can reveal fine crackles or wheezes. Crackles are more frequent in drug-induced pulmonary eosinophilia [17].

Diagnostic Approach

Löffler syndrome, also known as simple pulmonary eosinophilia, is a transient respiratory condition characterized by peripheral eosinophilia and pulmonary infiltrates. It typically presents 10–16 days after ingestion of Ascaris lumbricoides eggs, coinciding with the larval migration phase through the lungs. Patients often present with mild, self-limited respiratory symptoms, including non-productive cough, wheezing, and low-grade fever. A history of travel to or residence in tropical or subtropical regions with poor sanitation, or behaviors such as geophagia in children, increases the likelihood of exposure to Ascaris eggs [19,20-23]. Laboratory evaluation is pivotal in diagnosing Löffler syndrome. A complete blood count often reveals marked eosinophilia, with eosinophil counts frequently exceeding 500 cells/μL, reflecting a type 2 hypersensitivity immune response mediated by interleukin-5. Serum immunoglobulin E (IgE) levels are commonly elevated, although this finding is nonspecific. Stool examinations during the early pulmonary phase are typically negative, as Ascaris eggs do not appear in the stool until 6–8 weeks post-infection. Consequently, a negative ova-and-parasite test during the pulmonary phase does not exclude the diagnosis; repeat testing several weeks later is advised. Molecular diagnostics, such as polymerase chain reaction (PCR), can enhance sensitivity in detecting Ascaris DNA [20-22].

Imaging studies are central to diagnosis. Chest radiography often reveals transient, migratory, non-segmental pulmonary infiltrates, patchy opacities that may appear in different lung zones over a short period. High-resolution computed tomography (CT) enhances sensitivity and typically shows ground-glass opacities or peripheral consolidations, reflecting localized eosinophil-driven inflammation. These imaging patterns resolve spontaneously as larvae exit the lungs and the immune response subsides [24,25]. In certain cases, bronchoalveolar lavage may be performed, revealing a predominance of eosinophils, often above 25%, supporting the diagnosis. However, bronchoalveolar lavage is not routinely performed due to the self-limiting nature of the disease. Lung biopsy is rarely required unless ruling out alternative eosinophilic lung diseases [20]. The diagnosis of Löffler syndrome due to Ascaris lumbricoides involves a multifaceted approach, combining clinical evaluation, laboratory testing, imaging studies, and parasitological confirmation. Given the transient pulmonary manifestations and eosinophilic response induced by larval migration through the lungs, a high index of suspicion is crucial. Awareness of Löffler syndrome is critical, as misdiagnosis may lead to unnecessary interventions for other pulmonary conditions, underscoring the importance of recognizing its characteristic features [19,20].

Differential Diagnosis

There are numerous eosinophilic lung diseases that may resemble Löffler syndrome but can be distinguished based on key clinical aspects such as chronicity, severity, and the presence of systemic involvement. Eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss Syndrome, is characterized by eosinophilic vasculitis, chronic asthma, and extrapulmonary features such as neuropathy. Unlike the self-limited and benign course of Löffler syndrome, EGPA typically requires long-term immunosuppressive therapy due to its risk of irreversible organ damage [26]. Hyper eosinophilic syndrome (HES) is defined by persistent eosinophilia exceeding 1,500 cells/μL for more than six months, accompanied by evidence of end-organ injury, including pulmonary involvement [27]. This chronic and potentially progressive condition contrasts with the transient eosinophilia and spontaneous resolution observed in Löffler syndrome. Allergic bronchopulmonary aspergillosis (ABPA) may also present with overlapping features, particularly in patients with asthma. However, its distinguishing characteristics include central bronchiectasis, markedly elevated IgE levels, and recurrent exacerbations that typically require prolonged treatment with corticosteroids and antifungals [28].

Drug-induced eosinophilic pneumonitis may mimic Löffler syndrome radiographically but usually follows a subacute course and is preceded by exposure to a specific medication. Resolution generally occurs only after discontinuation of the offending agent, unlike the spontaneous improvement seen in Löffler [29]. Finally, certain parasitic infections, including Strongyloides, Ancylostoma, and Toxocara, can cause eosinophilic pulmonary infiltrates [30]. These conditions, however, are often associated with more pronounced systemic symptoms and a more prolonged disease

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course. Strongyloides can lead to chronic infection, and in immunocompromised individuals, may progress to hyper infection syndrome with severe pulmonary and gastrointestinal involvement [31]. Ancylostoma species, which are hookworms, can cause transient eosinophilic pneumonitis during larval migration, though they are more commonly associated with chronic blood loss and iron-deficiency anemia [30]. Toxocara infections, particularly in children, may result in visceral larva migrans, characterized by pulmonary infiltrates, hepatomegaly, fever, and marked eosinophilia [32]. These features help distinguish these infections from the typically benign and self-resolving course of classic Löffler syndrome.

Treatment and Management

The management of Loeffler syndrome is typically supportive, as the condition is generally self-limited and resolves within two to four weeks without the need for medical or surgical intervention. However, specific treatment may be warranted in cases with persistent symptoms, confirmed parasitic infection, or systemic complications such as endocardial involvement. The timing of antiparasitic therapy is crucial, as initiating treatment during the pulmonary migratory phase of helminths can exacerbate inflammation and potentially lead to pneumonitis [33-37]. Therefore, treatment is generally deferred until after the larvae have completed their pulmonary transit and reached the gastrointestinal tract, typically several weeks following exposure [33]. When indicated, antiparasitic agents such as albendazole and mebendazole are commonly used. Albendazole is administered orally at a dose of 400 mg once daily for three consecutive days, while mebendazole is given orally at 100 mg twice daily for three days. An alternative option includes ivermectin, typically administered as a single oral dose of 200 micrograms per kilogram, although some guidelines recommend higher doses in severe cases. These treatments are effective against Ascaris lumbricoides, Strongyloides stercoralis, and hookworms, which are the primary helminths associated with Loeffler syndrome. It is important to discontinue or postpone antiparasitic therapy if pulmonary eosinophilia is active, to reduce the risk of immune-mediated lung injury [33-38].

Corticosteroids are not routinely recommended in uncomplicated cases, but they may be considered in patients experiencing significant respiratory compromise or with complications such as Loeffler endocarditis [33,35,36]. In these instances, corticosteroids help reduce eosinophilic infiltration, suppress the release of cationic proteins, and prevent the formation of fibrosis and thrombi. A typical regimen may begin with dexamethasone for several days, followed by a course of prednisone at a dose of 40 to 60mg per day, tapered gradually over one to two weeks depending on clinical response [33-36]. Supportive therapies may also play a role, particularly in patients with symptoms. Nebulized bronchodilators, such as levalbuterol, in combination with inhaled corticosteroids like budesonide, can help alleviate respiratory discomfort and promote the resolution of bronchospasm. These treatments may also contribute to the resolution of skin lesions when used in conjunction with systemic eosinophilia [33-38]. In more severe cases, oxygen supplementation may be required to address hypoxemia. Despite the benign course of most cases, follow-up is essential to monitor for the rare progression to chronic eosinophilic lung disease or cardiac involvement [33-38]. There are no specific dietary requirements for patients with Loeffler syndrome, but prevention of recurrence and reduction of disease burden rely heavily on public health measures. Improvements in sanitation, access to clean water, routine deworming, and education on proper hygiene practices are essential, especially in endemic regions where parasitic infections are prevalent [33-38].

Prognosis and Complications

Löeffler syndrome is a relatively self-limited and benign syndrome, with respiratory symptoms occurring around four to sixteen days after ingestion of infective ascaris eggs, and decreasing within three to four weeks. Nevertheless, there does appear to be a dose-dependent effect; it seems to appear severe symptoms in cases of large inoculum or persistent exposure [39]. LS should be suspected as a differential diagnosis for asthma and community-acquired pneumonia, which is unresponsive to antibiotic therapy in individuals who live in an endemic region for parasitic diseases in developing countries [40]. The most common complication of ascariasis infection is intestinal obstruction, and the mechanism is due to mechanical obstruction by occlusion of the intestinal lumen resulting from the entanglement of many worms, mainly in the distal ileum. Patients with massive infestation may present with acute intestinal obstruction [41].

Complications of ascariasis in the hepatobiliary system are less frequent in children (5%) than in adults (53%), due to the small ductal caliber in childhood, although ascaris invasion is more common in children; lesions of the biliary system at this age appear in rare cases [42]. The presence of Ascaris in the biliary tract may cause acute disease, such as biliary colic, or severe disease, such as acute cholecystitis, acute cholangitis, acute pancreatitis, and even liver abscess. Hepatobiliary and pancreatic complications of ascariasis should be suspected when a patient infested with this worm presents symptoms like acute onset of abdominal pain, especially in the right upper quadrant, vomiting, mucocutaneous pallor, and elimination of worms in vomit or feces [43]. Another complication rarer and more fatal, is Loeffler endocarditis, which is an endomyocardial disorder thought to be caused by eosinophilic infiltration into the heart, resulting in cardiac damage. Loeffler endocarditis can cause several complications, such as progressive heart failure, systemic thromboembolic events, arrhythmias, and sudden cardiac death [43].

Prevention and Public Health Considerations

Based on the knowledge of transmission and lifecycle of Ascaris lumbricoides, it is evident that adopting measures such as improved sanitation and hygiene, widespread deworming initiatives, health education, and food and water safety significantly prevent

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infection, disrupt transmission cycles, and prevent future complications [44]. Access to adequate sanitation facilities is crucial in preventing the transmission of ascariasis. Massive investments in water and sanitation infrastructure, including improved sewage systems, latrines, and waste management practices, can minimize human contact with contaminated environments and reduce the spread of Ascaris eggs [44]. A study conducted among students in Kabul revealed that washing hands before eating and after using the toilet was associated with a reduced prevalence of Ascaris carriage in stool (p=0.0080) [45]. Widespread deworming with anthelmintic medications, such as albendazole or mebendazole, is a fundamental pillar of ascariasis control programs. It involves the periodic distribution of deworming medications to entire at-risk populations, regardless of individual infection status. This approach seeks to reduce the worm burden within communities and break the transmission cycle of Ascaris [44].

Community-based health education campaigns tailored to local cultural practices, norms beliefs can be sustained and be effective for behavior change [44]. Bi et al. conducted a study among Chinese children, which showed that children who received a more detailed health education were likely to wash their hands after defecation and had a lower incidence of soil-transmitted helminths [46]. Ready access to a clean source of water is paramount to the prevention of transmission. Access to clean sources of water leads to the adoption of safe hygiene practices such as handwashing and proper cooking of food [1]. For a 1 % increment in availability of filtered water in schools, there was a 1.1% decrease in the mean of ascariasis prevalence (RR 0.986, CI 95 % 0.979-0.993) [47].

Conclusion

Loeffler syndrome, although self-limiting in most cases, represents a significant intersection between infectious disease, immunology, and public health. Triggered primarily by the pulmonary migration of helminths such as Ascaris lumbricoides, it presents with characteristic respiratory symptoms and peripheral eosinophilia, often resolving without intervention. However, complications including intestinal obstruction, hepatobiliary involvement, and rarely Loeffler endocarditis can increase morbidity, particularly in settings with high parasite burden. A clear understanding of the disease pathophysiology, clinical course, and appropriate timing for antiparasitic treatment is essential for effective management. Moreover, differentiating Loeffler syndrome from other eosinophilic pulmonary diseases prevents misdiagnosis and overtreatment. Public health measures, including improved sanitation, routine deworming, access to clean water, and health education, remain foundational in reducing the global burden of this condition. As the epidemiological landscape of parasitic diseases evolves, continued awareness and research into Loeffler syndrome are critical for improving patient outcomes and informing prevention strategies.

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