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WHEN MITE ALLERGY MIMICS HELMINTH SENSITIZATION: CROSS-REACTIVE IGE TO *ASCARIS LUMBRICOIDES* IN A HIGHLY ATOPIC CHILD

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Abstract

Sensitization to helminth antigens detected by specific IgE assays may occasionally reflect immunological cross-reactivity rather than true parasitic infection, particularly in highly atopic individuals.

A 5-year-old boy was hospitalized for the fourth time because of severe bronchial obstruction and recurrent wheezing. Laboratory findings showed mildly elevated leukocyte count without eosinophilia. Specific IgE testing revealed strong sensitization to house dust mites (*Dermatophagoides pteronyssinus* and *D. farinae*, both class 6) and elevated *Ascaris lumbricoides*–specific IgE (class 4).

Stool examination for intestinal parasites was negative. A previous *Enterobius vermicularis* infestation treated months earlier was reported, with no clinical or laboratory evidence of current or past *Ascaris* infection. Given the strong dust mite sensitization and absence of evidence for ascariasis, the detected *Ascaris*-specific IgE was interpreted as a result of IgE cross-reactivity, most likely mediated by shared pan-allergens such as tropomyosin. Prior *Enterobius* infestation was considered an unlikely contributor to the observed IgE profile.

This case highlights the risk of overinterpreting helminth-specific IgE results in highly atopic children and underscores the importance of clinical correlation.

In the context of severe house dust mite sensitization, *Ascaris lumbricoides*–specific IgE positivity most likely reflected allergen cross-reactivity rather than true parasitic infection.

This case highlights the importance of interpreting helminth-specific IgE results within the clinical context to avoid diagnostic error and inappropriate management.

Key words: Asthma, IgE cross-reactivity, Ascaris lumbricoides, *Dermatophagoides pteronyssinus*, *Dermatophagoides farina*.

Introduction

Asthma is a prevalent chronic childhood disease, affecting approximately 10.2% of children globally [1]. Pediatric asthma is a heterogenous diseases, however, more than 80% of cases exhibit an atopic phenotype, frequently associated with sensitization to house dust mites (HDM), particularly Dermatophagoides pteronyssinus and Dermatophagoides farinae [2,3].

On the other hand, it is well established that HDM and *Ascaris lumbricoides* shared conserved invertebrate pan-allergens, including tropomyosin and other homologous proteins, which may induce IgE cross-reactivity [4].

Consequently, detection of specific *Ascaris* IgE in children with strong HDM sensitization may not necessarily indicate true helminth sensitization and should be interpreted cautiously within the clinical and epidemiological context.

Case Report

We report the case of a 5.5-year-old child with diagnosed asthma who had experienced recurrent episodes of bronchial obstruction since the age of 14 months, requiring several hospitalizations. The child had a history of atopic dermatitis in early childhood and a positive family history of asthma.

The patient was admitted due to sudden, recurrent, short-lasting, and variable wheezing episodes. Six months prior to admission, the child experienced abdominal pain, and stool examination was positive for *Enterobius vermicularis*, for which albendazole therapy was administered for three days.

On admission, the child was afebrile, pale, mildly ill-appearing, and mildly tachypneic and tachycardic, with normal oxygen saturation. Lung auscultation revealed vesicular breathing with prolonged expiration, diffuse expiratory wheezing, and coarse crackles bilaterally.

Total IgE was markedly elevated (1770 IU/mL). Peripheral eosinophilia was absent. Specific IgE testing demonstrated strong sensitization to *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae* (>100 IU/mL, class 6), as well as positive IgE to *Ascaris lumbricoides* (32 IU/mL, class 4). Stool examination for parasites was negative. Laboratory investigations showed mild leukocytosis (16.6×10^9/L), while the other parameters were within reference ranges.

Additional investigations, including chest radiography, microbiological testing, echocardiography, and bronchoscopy, did not reveal structural or infectious causes of recurrent wheezing.

Treatment included intravenous fluids, inhaled bronchodilators, and systemic corticosteroids. During the final three days of hospitalization, no wheezing or bronchial obstruction was observed. The patient remained afebrile throughout the hospital stay and was discharged with inhaled corticosteroid therapy (fluticasone) and scheduled for outpatient follow-up.

Discussion

This case highlights a diagnostic pitfall in highly atopic children: the potential misinterpretation of helminth-specific IgE results caused by immunological cross-reactivity between house dust mite allergens and helminth antigens. Cross-reactivity between conserved invertebrate proteins, particularly tropomyosin, is known to induce IgE cross-reactivity between house dust mites and nematodes [4,5].

In a study by Valmonte and colleagues, 70% of allergic individuals demonstrated positive IgE reactivity to *Ascaris lumbricoides*, while 20–28% of individuals with ascariasis showed IgE reactivity to house dust mites [4].

Furthermore, *A. lumbricoides* extract inhibited up to 92% of the binding of mite-specific IgE in sera from allergic individuals, supporting the presence of shared antigenic determinants. These proteins, particularly tropomyosin (Der p 10 / Asc l 3), exhibit substantial structural homology and similar IgE-binding epitopes [4 6].

Similar findings were reported by Acevedo et al., who demonstrated that *A. lumbricoides* antigens can inhibit up to 79.3% of *Dermatophagoides pteronyssinus*-specific IgE binding [7]. This cross-reactivity is attributed to several shared allergens, including tropomyosin and glutathione-S-transferase. The authors also suggested the ABA-1 polyprotein as a more specific marker of *A. lumbricoides* infection, as no cross-reactive equivalent has been identified in mite extracts [7].

The present case involves a 5.5-year-old boy with strong sensitization to house dust mites, demonstrated by very high specific IgE levels to *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae* (both class 6), and a clinical presentation consistent with an atopic asthma phenotype. In such highly atopic individuals, characterized by pronounced Th2 polarization and elevated total IgE production, cross-reactive IgE antibodies may bind homologous parasite antigens in extract-based assays.

Therefore, the detection of elevated *A. lumbricoides*-specific IgE (class 4) in the absence of eosinophilia, parasitological evidence, or clinical manifestations of ascariasis strongly suggests immunological cross-reactivity rather than true infection.

A previous infection with *Enterobius vermicularis* was considered as a potential explanation for the elevated *A. lumbricoides*-specific IgE.

However, several factors argue against this possibility. First, *E. vermicularis* infection typically induces only a weak systemic IgE response [8,9], and the infestation in our patient was remote and successfully treated, making persistent parasite-specific IgE unlikely. Second, *E. vermicularis* does not prominently express the well-characterized cross-reactive pan-allergens shared between house dust mites and *A. lumbricoides* [10].

Third, there was no eosinophilia or clinical evidence suggestive of ongoing helminth infection. Compared with HDM-related cross-reactivity, this explanation therefore appears considerably less plausible.

Conclusion

In highly house dust mite–sensitized children, detection of *Ascaris lumbricoides*-specific IgE may reflect immunological cross-reactivity rather than true helminth sensitization or infection.

Awareness of this phenomenon is particularly important in non-endemic settings to avoid unnecessary diagnostic procedures or anti-helminthic therapy and to ensure appropriate management of the underlying allergic airway disease.

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