ISSN:2308-5703
Impact factor:13.5 *International Multidisciplinary Online Journal* September, 2026

Recurrent Infections in Children and Allergic Predisposition: Diagnosis and Approach

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doi:https://doi.org/10.5281/zenodo.22832518

ABSTRACT: Recurrent infections represent one of the most frequent concerns prompting medical consultation in pediatric practice. While many episodes reflect normal exposure in early childhood, a substantial proportion of affected children exhibit an underlying allergic predisposition that both mimics and exacerbates infectious processes. Allergic inflammation of the respiratory and cutaneous barriers impairs local defense mechanisms, alters cytokine profiles, and increases susceptibility to viral and bacterial pathogens. Conversely, repeated infections can amplify type 2 immune responses and promote the development or persistence of atopic disease. This review synthesizes current understanding of the bidirectional relationship between recurrent infections and allergic predisposition in children. Emphasis is placed on clinical differentiation between physiologic childhood infections, atopic disease, and true inborn errors of immunity. Diagnostic strategies encompassing detailed history, physical examination, allergen-specific testing, immunoglobulin quantification, and selective immunologic evaluation are outlined. Therapeutic approaches integrate allergen avoidance, anti-inflammatory treatment of the atopic component, targeted infection prevention, and judicious use of immunomodulatory interventions. Recognition of the interplay between allergy and infection enables more precise diagnosis, reduces unnecessary antibiotic exposure, and improves long-term respiratory and allergic outcomes. Multidisciplinary collaboration between pediatricians, allergists, and immunologists remains essential for optimal management.
KEY WORDS: recurrent infections; children; allergic predisposition; atopic disease; diagnosis; immune dysregulation; microbiome; management

INTRODUCTION

Recurrent infections constitute a common clinical presentation in pediatric medicine and generate considerable anxiety among caregivers as well as diagnostic challenges for clinicians. In the first years of life, children experience multiple episodes of upper and lower respiratory tract infection as part of normal immune maturation and environmental exposure. However, when infections become unusually frequent, severe, or prolonged, underlying predisposing factors must be considered. Among these factors, allergic predisposition occupies a central position. Allergic rhinitis, asthma, and atopic dermatitis not only produce symptoms that closely resemble infectious processes but also create a microenvironment favorable to pathogen colonization and invasion.

Epidemiologic data indicate that up to thirty percent of children evaluated for recurrent upper respiratory tract infections harbor underlying atopic disease. The inflamed respiratory epithelium of atopic individuals demonstrates increased adherence of viruses and bacteria, reduced production of antiviral interferons, and a shift toward type 2 cytokine dominance that impairs effective pathogen

clearance. At the same time, early-life infections themselves can influence the trajectory of allergic sensitization through effects on the developing immune system and the gut and airway microbiota.

The clinical importance of recognizing this association extends beyond accurate diagnosis. Children with untreated allergic inflammation often receive repeated courses of antibiotics, contributing to antimicrobial resistance and further microbiome disruption. Conversely, failure to identify true primary immunodeficiency in a minority of patients may delay life-saving interventions. Therefore, a structured diagnostic approach that systematically distinguishes among normal childhood infection patterns, allergic disease, anatomic or chronic conditions, and inborn errors of immunity is required.

The present article examines the pathophysiologic links between recurrent infections and allergic predisposition, reviews the relevant literature, discusses diagnostic strategies, presents synthesized findings from contemporary studies, and outlines practical management principles. The goal is to provide clinicians with a coherent framework for evaluating and treating affected children.

LITERATURE REVIEW

Multiple investigations have established a clear association between atopic status and increased frequency of respiratory infections in childhood. Children with allergic rhinitis and asthma produce lower quantities of interferon-gamma in response to viral stimuli, rendering them particularly vulnerable to rhinovirus, respiratory syncytial virus, and other common respiratory pathogens [1, 2018, 112]. Rhinovirus has been recovered from approximately seventy-five percent of asthma exacerbations, illustrating the intimate relationship between viral infection and atopic airway inflammation [2, 2025, 4] Bacterial co-infection is also more prevalent among atopic children. One comparative analysis demonstrated Mycoplasma pneumoniae involvement in 27.1 percent of atopic subjects versus only 4.9 percent of non-atopic controls [2, 2025, 5]. The inflamed mucosa of allergic rhinitis facilitates bacterial adherence and impairs mucociliary clearance, thereby promoting recurrent sinusitis and otitis media.

Early-life infection burden has itself been linked to subsequent atopic outcomes. Prospective cohort studies reveal that higher numbers of respiratory infections in the first two years of life correlate with increased risk of atopic dermatitis, allergic rhinitis, and asthma later in childhood [3, 2026, 8]. This bidirectional relationship is mediated in part by alterations in the gut microbiota. Delayed maturation of the infant gut microbiome, characterized by reduced abundance of Bifidobacterium species and elevated oxidative metabolic activity, precedes the clinical appearance of allergic diagnoses at five years of age [4, 2023, 5]. Similar microbial imbalances have been documented in infants with food-protein-induced enterocolitis syndrome and other allergic phenotypes [5, 2024, 12].

The diagnostic challenge is compounded by the existence of monogenic disorders that present with both severe atopy and recurrent infections. Primary atopic disorders and certain inborn errors of immunity, such as hyper-immunoglobulin E syndromes and signal transducer and activator of transcription pathway defects, may initially be mistaken for ordinary polygenic allergy [6, 2021, 15]. Clinical red flags including early-onset severe eczema, failure to thrive, opportunistic infections, family history of consanguinity, and markedly elevated immunoglobulin E levels should prompt consideration of genetic evaluation [7, 2025, 22].

Risk-factor analyses further differentiate the determinants of atopic disease from those of recurrent respiratory tract infections. Family history of atopy strongly predicts allergic outcomes, whereas daycare attendance and environmental tobacco smoke exposure more prominently increase

infection frequency [8, 2020, 3172]. Vitamin D and vitamin A deficiency, low birth weight, and passive smoking emerge as independent predictors of recurrent respiratory infections in large real-world cohorts [9, 2026, 7].

Selective immunoglobulin A deficiency, the most common primary immunodeficiency, frequently coexists with allergic disease and predisposes to recurrent sinopulmonary infections. Aggressive management of the allergic component is recommended because allergic inflammation further compromises mucosal barrier function [10, 2026, 3].

Collectively, the literature underscores that allergic predisposition and recurrent infection are interwoven rather than mutually exclusive phenomena. Accurate diagnosis therefore requires simultaneous consideration of both processes.

DISCUSSION

The pathophysiologic interface between allergy and infection centers on epithelial barrier dysfunction and immune polarization. In atopic individuals the respiratory epithelium exhibits reduced tight-junction integrity, diminished production of antimicrobial peptides, and preferential secretion of type 2 cytokines. These changes facilitate viral entry and replication while simultaneously suppressing the type 1 interferon responses necessary for viral clearance. Once infection is established, the resulting inflammation further amplifies type 2 responses, creating a self-perpetuating cycle.

Diagnostic evaluation begins with a meticulous clinical history that quantifies the frequency, severity, and anatomic localization of infections, together with the presence of classic atopic symptoms such as pruritus, seasonal exacerbation, and response to antihistamines or inhaled corticosteroids. Physical examination seeks allergic stigmata (allergic shiners, nasal crease, eczema) as well as signs of chronic disease or immunodeficiency (failure to thrive, organomegaly, absent tonsils). Laboratory investigation proceeds in a stepwise fashion. Initial testing includes complete blood count with differential, total immunoglobulin levels, and allergen-specific immunoglobulin E directed by history. Persistently elevated immunoglobulin E exceeding two thousand international units per milliliter, profound hypogammaglobulinemia, or unusual pathogen profiles warrant referral for advanced immunologic and genetic assessment [11, 2022, 48].

Management strategies must address both components of the problem. Optimal control of allergic inflammation through allergen avoidance, topical or systemic anti-inflammatory agents, and, when indicated, allergen immunotherapy reduces mucosal edema and restores barrier function, thereby decreasing infection susceptibility. Prophylactic antibiotics may be considered for selected children with documented antibody deficiency during high-risk seasons, but their prolonged use should be avoided in pure atopic disease. Vaccination according to standard schedules remains essential, including annual influenza immunization. Emerging evidence supports the potential of early microbiome modulation through specific probiotic strains or human-milk oligosaccharides to promote immune tolerance and reduce both infection frequency and allergic sensitization [4, 2023, 9].

In severe or refractory cases, biologic agents targeting type 2 pathways (interleukin-4 receptor antagonists, anti-immunoglobulin E monoclonal antibodies) simultaneously improve allergic control and may lower infection-related exacerbations. Multidisciplinary care involving pediatricians, allergists, and, when necessary, immunologists ensures that neither the infectious nor the allergic dimension is neglected.

RESULTS

Synthesis of available observational and interventional data yields several consistent findings. Approximately thirty percent of children referred for evaluation of recurrent upper respiratory tract infections ultimately receive a diagnosis of atopic disease rather than primary immunodeficiency [12, 2018, 132]. Atopic children demonstrate significantly higher rates of documented viral and bacterial respiratory pathogens compared with non-atopic peers, with rhinovirus predominance reaching seventy-five percent during exacerbations [2, 2025, 4]. Early-life infection burden shows a dose-dependent association with later atopic diagnoses, independent of antibiotic exposure and filaggrin genotype [3, 2026, 10]. Delayed gut microbiota maturation at one year of age is uniformly associated with atopic dermatitis, asthma, food allergy, and allergic rhinitis at five years [4, 2023, 6]. History of allergy confers an odds ratio greater than five for subsequent recurrent respiratory tract infections in large pediatric cohorts [9, 2026, 8]. Stepwise laboratory evaluation beginning with immunoglobulin quantification and allergen-specific testing correctly classifies the majority of children and identifies the minority requiring advanced immunologic work-up [11, 2022, 50]. Aggressive treatment of coexisting allergic inflammation in children with selective immunoglobulin A deficiency reduces the frequency of subsequent infections [10, 2026, 4].

These synthesized results confirm the clinical relevance of the allergy–infection interplay and support the diagnostic and therapeutic algorithms proposed in contemporary guidelines.

CONCLUSION

Recurrent infections in children frequently reflect an underlying allergic predisposition that both mimics and potentiates infectious processes. Recognition of this relationship allows clinicians to move beyond repeated antibiotic courses toward comprehensive management that addresses barrier dysfunction, type 2 inflammation, and environmental triggers. A structured diagnostic pathway distinguishing physiologic infection patterns, atopic disease, and true immunodeficiency protects children from both under-diagnosis of serious immune defects and over-treatment of ordinary allergic conditions. Therapeutic success depends on simultaneous control of allergic inflammation and prevention of infection, ideally within a multidisciplinary framework. Future research should focus on refined biomarkers of the allergy–infection interface, personalized microbiome interventions, and long-term outcome studies of integrated management strategies. Implementation of the principles outlined in this review promises to reduce morbidity, minimize antimicrobial resistance, and improve the respiratory and allergic health of affected children.

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