Respiratory Allergic Disorders

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KEYWORDS

• Allergic • Asthma • Allergic bronchopulmonary aspergillosis
• Hypersensitivity pneumonitis

KEY POINTS

- Allergic asthma is a chronic, reversible, bronchoconstriction influenced by an allergic trigger.
- Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction due to bronchi colonization by *Aspergillus* species.
- Hypersensitivity pneumonitis (HP) refers to lung inflammation from airborne environmental antigens, such as dust and mold.

ALLERGIC ASTHMA

Introduction

Asthma is described as a chronic, reversible, inflammatory disease, accompanied by airway hyper-responsiveness and bronchoconstriction.[1–3] Common symptoms include wheezing, dyspnea, chest tightness, and coughing.[1–3] The number of individuals afflicted with asthma continues to rise, with worldwide prevalence at approximately 300 million individuals.[1,4]

Asthma can be classified as allergic versus nonallergic types. Several different asthma triggers have been identified, including exercise, tobacco smoke, infections, cold air, and allergens.[2,3] Allergic asthma, previously referred to as extrinsic asthma, is the term used to describe the influence of allergens in the development of asthma.[5,6] Conversely, nonallergic asthma, formerly intrinsic asthma, should be considered when allergic etiology is ruled out after careful history, physical examination, and allergy testing.[6,7]

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Epidemiology/Pathophysiology

Allergic asthma patients tend to have earlier age of onset in comparison to nonallergic asthmatics (15.8 ± 1.3 vs 32.2 ± 2.3).[5] Boys and men are more prone to the allergic asthma subtype, whereas girls and women are more likely to have nonallergic asthma. One study concluded nonallergic asthma patients have a male-to-female ratio of 0.8 to 1.2.[5] Smoking status does not seem to differ between allergic versus nonallergic asthmatics.[5]

Both genetic and environmental factors play a role in the development of atopy and asthma, with several common gene loci identified among allergic asthmatics.[8] The presence of maternal asthma has been thought to play a role to a larger degree than paternal asthma.[9] One theory suggests that the combination of genetic susceptibility and in utero maternal exposures induces more of a type 2 helper T cell (Th2) immunity response, thus increasing the risk of asthma from allergic sensitization early in life.[9,10] With infants spending more time indoors than outdoors, potential indoor allergens are also thought to lead to allergic sensitization and asthma in select high-risk children.[1] Early childhood exposure to domestic animals, however, has been shown to add a protective effect against the development of atopy and wheezing.[11]

Allergic asthma is initiated by the production of antigen-specific T cells to allergens.[12] The allergic asthma pathway is triggered on re-exposure of the allergen after the initial sensitization.[13] The term, *atopy*, refers to the immunoglobulin E (IgE) antibody production to low doses of allergens.[5,6] In allergic asthma, allergens cross-link IgE on the surface of mast cells, resulting in the release of histamine, prostaglandins, and cytokines.[6,13,14] In turn, Th2 cell recruitment, increased mucus production, and bronchoconstriction are seen.[6,13,14] Cytokines that participate in the inflammatory response and the asthma pathway include interleukin (IL)-4, IL-13, IL-5, and IL-9.[6,10,15]

In patients with allergic asthma, acute infections often result in exacerbations of asthma. It has been suggested that respiratory syncytial virus bronchiolitis in infancy can give rise to asthma, with subsequent amplification of allergic inflammation.[16] Another example is the increased number of asthma exacerbations related to rhinovirus infection in patients with high IgE titers to allergens, such as dust mites.[17]

An overwhelming association with concurrent allergic rhinitis has been seen among individuals with allergic asthma, with allergic rhinitis commonly preceding the onset of asthma.[2,3,7,11,18] Allergic rhinitis serves as an independent risk factor for future development of asthma, due to the similar Th2 immune cell involvement, triggering mast cell and eosinophil involvement.[7,15] Allergic rhinitis may also lead to increased mouth breathing, thus increasing exposure of the lower respiratory system to allergens unable to be filtered by the nose.[11] Comorbid asthma with allergic rhinitis has been linked to increased emergency room visits and asthma attacks in comparison to individuals with asthma alone.[7]

Repeated allergen exposure subsequently results in recurring inflammation of the lungs and bronchial hyper-reactivity.[3,17] Many different allergens have been cited as potential triggers for patients with allergic asthma, including dust mites, cockroach residue, furred animals, molds, and pollen.[2,3,19] Food allergy is not a common trigger for allergic asthma symptoms.[20] New investigations using mouse models are also analyzing the potential role of atrial natriuretic peptide on the respiratory system, particularly involvement inducing a Th2 immune cell response during acute allergic asthma.[21]

Clinical Presentation/Diagnosis

On diagnosis of asthma, a clinician should attempt to differentiate between allergic and nonallergic asthma. Hay fever and seasonal exacerbations of asthma favor