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Allergic rhinitis and Asthma Pathophysiology on Human health

Aria Jenny*

Department of Otolaryngology, ospital University Donostia, San Sebastian, Spain.

***Corresponding author**: Aria Jenny, *Department of Otolaryngology, Hospital University Donostia, San Sebastian, Spain,* E-mail: AriaJenny21@gmail.com

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Allergic rhinitis (AR) is one of the most common chronic illnesses, with prevalence rates ranging from 3% to 19% in different countries. Seasonal AR ('hay fever') symptoms and the more complex diagnostic category, perennial AR, are two different types of AR (PAR). According to one study, SAR affects about 10% of the general population, while PAR affects 10% to 20% of the population.

Asthma is also a heterogeneous disorder; descriptions of asthma are difficult to come by due to its ambiguity, which may represent a variety of phenotypes. Most clinical concepts go into detail about lung function symptoms (such as wheezing and trouble breathing), exacerbations, and, in certain cases, medication reaction (eg, high-dose corticosteroids).

Allergic Rhinitis and Asthma Pathophysiology

Allergic stimuli, non-allergic causes, or both (mixed rhinitis) may cause rhinitis. Depending on the form of rhinitis, the underlying mechanism that causes nasal symptoms varies. Allergic rhinitis is only seen in people who have a hereditary predisposition to allergies. Despite the fact that everybody is continually exposed to allergens in the community, only those patients who have the inherent potential to become sensitised develop symptoms.

Repeated exposure to aeroallergens causes B cells to activate and mature into plasma cells, which contain unique IgE antibodies in these hypersensitive people. IgE binds to various receptors on the basophil and mast cell surfaces. When the sensitizing allergen cross-links specific IgE bound to cells, the cells release or emit chemical mediators that cause allergic symptoms. Mast cells that have been activated release preformed histamine and produce new leukotrienes, prostaglandins, kinins, and other compounds. Because of increased vascular permeability, vasodilation, and increased mucus output, this mediator release causes an immediate hypersensitivity reaction with itching, sneezing, and congestion. Plasma protein leakage into the vascular system is a factor. After an allergen exposure, the release of inflammatory mediators continues, resulting in a late-phase reaction that prolongs nasal symptoms. During this late-phase reaction, cytokines and chemokines are released and produced, which attract more inflammatory cells.

Increased lymphocyte recruitment may be aided by the release of lymphokines such as thymic stromal lymphopoietin (TSLP). Mast cells are also essential for delivering late-phase reactants to inflamed areas. Depending on the nature of the condition, it is important to understand all of these pathways in order to target treatment and alleviate both bronchoconstriction and inflammation.

Reference

1. Bousquet J, Van Cauwenberge P, Khaltaev N (2001) Allergic rhinitis and its impact on asthma. J Allergy Clin Immunol. 108: S147–S334.
2. Nathan RA (2007)The burden of allergic rhinitis. Allergy Asthma Proc 28:3-9.
3. Berger WE (2004) Allergic rhinitis in children: diagnosis and management strategies. Paediatr Drugs 6: 233–250.