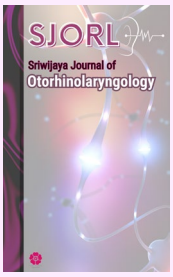

e-ISSN: 2987-131X

Sriwijaya Journal of Otorhinolaryngology (SJORL)

Journal website: https://phlox.or.id/index.php/sjorl

Analysis of the Immunobiological Aspects of Allergic Rhinitis

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ARTICLE INFO

Keywords:
Allergic rhinitis
Eosinophil
Inflammation
Interleukin
Histamine

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.59345/sjorl.v1i2.92

A B S T R A C T

Allergic rhinitis is characterized by intricate interactions between the immune system and specific allergens. The first stage starts with sensitization, a time when people who are vulnerable to allergens make an immune response to it, which usually includes making IgE antibodies that are specific to that allergen. Mast cells and Th2 cells have a pivotal role in the allergic response by releasing inflammatory mediators like histamine, interleukin-4 (IL-4), IL-5, and IL-13. Mast cells are the primary source of histamine, which induces characteristic symptoms of allergic rhinitis, including rhinorrhea, pruritus, sneezing, and nasal congestion. In addition, eosinophils, a specific subtype of leukocyte, accumulate in the nasal tissue and contribute to persistent inflammation. The stimulation of eosinophils by cytokines, such as IL-5, can lead to tissue damage and the extension of inflammation. This procedure initiates a recurring pattern of allergic reactions that might intensify with each successive encounter with the allergen. The immunobiology of allergic rhinitis is characterized by the combined processes of sensitization, IgE synthesis, the release of inflammatory mediators, and tissue destruction.

1.Introduction

Allergic rhinitis is a prevalent ailment that can have a substantial impact on an individual's quality of life. Allergic rhinitis is an inflammatory response in the nasal passages triggered by exposure to certain allergens, such as pollen, dust mites, animal dander, or mold spores. Understanding the immunobiological component involved in the allergic reaction is crucial when it comes to allergic rhinitis. The immunobiology of allergic rhinitis encompasses the intricate interplay between the immune system and the antigens responsible for inducing allergic responses. The immune system is responsible for recognizing and reacting to allergens by releasing substances that induce inflammation, such as histamine, leukotrienes, and cytokines. These substances then lead to the symptoms of allergic rhinitis. A comprehensive comprehension of various facets of immunobiology is crucial for the advancement of efficient diagnostic, therapeutic, and preventive approaches for allergic rhinitis. This study will examine fundamental elements of the immunobiology of allergic rhinitis, encompassing the mechanisms behind the immune response to allergens, the involvement of specific immune cells such as mast cells and Th2 cells, and their effects on nasal tissues and structures. Improved understanding of this component is expected to promote the development of targeted and innovative therapeutic approaches.[1-5]

Induction of sensitivity and initial immune reaction

When an individual is exposed to a specific allergen, sensitization occurs, leading to a sequence of reactions that are crucial in the formation of allergic rhinitis. The process occurs when humans encounter allergens, which can originate from many sources, like pollen, dust mites, animal dander, or mold spores. Exposure to this allergen may not always result in an allergic reaction, and not everyone will develop sensitivity. Following exposure, dendritic cells, which are a type of immune cell, have a crucial function in identifying the allergen. Dendritic cells phagocytose allergens and subsequently display them to T cells (T lymphocytes) within the immune system. The dendritic cells activate T cells, particularly T-helper (Th) cells, which then initiate additional immunological responses.

Within the framework of allergic rhinitis, T-helper type 2 (Th2) cells play a distinctive function and undergo excessive activation. When Th2 cells become activated, they make and release interleukin-4 (IL-4). This makes B cells make immunoglobulin E (IgE). IgE is an antibody subtype that exhibits specificity towards particular allergens. Mast cells, a specific form of immune cell, and basophils, a particular kind of white blood cell, subsequently bind to the generated IgE. Furthermore, IgE has the ability to attach to IgE receptors located on the outer membrane of nasal epithelial cells. Susceptible individuals may undergo a process known as sensitization over a period of time and repeated exposures. Sensitization is the process in which the body starts to react excessively to an allergen, leading to the development of symptoms of allergic rhinitis following further exposure.[6-9]

Mast cells and histamine release

Mast cells are essential elements in the allergic response and have a pivotal function in the pathophysiological processes of allergic rhinitis. Mast cells release various inflammatory mediators, such as histamine, in response to exposure to allergens. Mast cells reside in various tissues, including the nasal mucosa. Mast cells possess IgE receptors on their surface, which specifically attach to IgE antibodies generated during sensitization to allergens. Subsequent exposure to allergens that are attached to IgE on mast cells triggers the release of histamine and other inflammatory substances by mast cells into their surroundings. The mediators involved in inflammation and allergic rhinitis symptoms encompass leukotrienes, prostaglandins, and cytokines.[10-12]

Histamine induces vasodilation, hence augmenting blood circulation to the affected region. This can lead to inflammation and erythema of the nasal mucosa. Histamine additionally enhances blood vessel permeability, facilitating the movement of fluid and blood cells from the blood vessels into the surrounding tissues. This might result in rhinorrhea and the excessive production of mucus. Histamine induces smooth muscle contractions in the blood vessel walls and the adjacent smooth muscle tissue. This might lead to nasal congestion and exacerbate the symptoms of nasal congestion. Histamine activates sensory nerve endings, inducing pruritus and initiating the sneezing reflex as the body's response to eliminate the bothersome allergen. Upon repeated exposure to the same allergen, previously activated mast cells may exhibit a heightened and more rapid response, thus reinforcing the cycle of allergic and inflammatory reactions. By understanding the important role that mast cells play and how histamine affects allergic rhinitis, we can make medicines that target these substances specifically. Antihistamine therapy aims to mitigate the effects of histamine and can effectively alleviate the symptoms of allergic rhinitis. Ongoing research aims to explore alternative treatment targets to better regulate allergic responses and minimize their detrimental effects on the quality of life for individuals with allergic rhinitis.[13,14]

T-helper 2 (Th2) cells and proinflammatory cytokines

T-helper type 2 (Th2) cells play a crucial role in promoting and controlling allergic reactions in allergic rhinitis. Th2 cells, which are a component of the immune system, have the function of responding to allergens by releasing certain pro-inflammatory cytokines. After exposure to an allergen, Th2 cells

activate and gather in the affected region, such as the nasal mucous membranes. Th2 cells secrete many cytokines, such as interleukin (IL)-4, IL-5, and IL-13, when exposed to allergens. IL-4 has a crucial function in promoting the synthesis and secretion of immunoglobulin E (IgE) by B cells. Subsequently, the IgE molecules attach to mast cells and basophils, priming them for the subsequent reaction to the allergen. IL-5 facilitates the movement and stimulation of eosinophils, a specific subset of leukocytes that tend to accumulate in regions of inflammation. Eosinophils have a significant impact on chronic inflammation and the destruction of tissues. IL-13 is involved in promoting alterations in epithelial cells and nasal mucous membranes. This can lead to heightened mucus production, excessive secretion of mucous glands, and alterations in tissue morphology.[15,16]

Th2 cells generate proinflammatory cytokines that induce intricate alterations in the nasal mucous membranes. Symptoms such as rhinorrhea, pruritus, sneezing, and nasal obstruction may arise due to excessive mucus production, edema, and increased inflammatory cell growth. Allergic responses can perpetuate through a continual cycle of immune responses initiated by Th2 cells and proinflammatory cytokines. Continual exposure to an allergen can enhance and sustain the allergic reaction at an elevated intensity. By understanding how Th2 cells and proinflammatory cytokines work in allergic rhinitis, we can start making treatments that target reducing the activity of Th2 cells or blocking the effects of certain cytokines. Ongoing research is investigating medications that can interfere at this phase of the allergic response, with the aim of offering a more efficient therapeutic approach for individuals with allergic rhinitis.[17]

Inflammatory cascade reaction

Eosinophils play a significant role in the pathogenesis of the chronic inflammation observed in allergic rhinitis. Eosinophils gather in nasal tissue and cause inflammation when triggered by certain stimuli, including cytokines like IL-5. Eosinophils derive from the bloodstream and migrate to inflammatory tissues. Eosinophils can migrate to the nasal mucous membranes in response to allergen exposure in cases of allergic rhinitis. Interleukin-5 (IL-5) is a crucial cytokine that promotes the generation, development, and movement of eosinophils. Allergic rhinitis involves the production of IL-5 by Th2 cells, which then leads to the activation of eosinophils. Upon reaching the nasal tissue, eosinophils release a range of inflammatory mediators, including enzymes and proteins, which have the potential to harm the tissue. These mediators can potentially exacerbate the persistent inflammatory process, a defining trait of allergic rhinitis.[14,15]

Eosinophils in an activated state have the ability to inflict harm upon the nasal mucous membranes and the adjacent tissue structures. This process can give rise to symptoms such as rhinorrhea, nasal congestion, and nasal discomfort. Eosinophils, despite their potential harm as part of the inflammatory response, also contribute to the body's defense against allergens seen as a threat. Eosinophils possess chemicals that can eliminate allergies or engage in phagocytosis, the absorption and processing of allergens. Activated eosinophils have the ability to extend and intensify the inflammatory process in allergic rhinitis. Continual exposure to allergens can sustain persistent levels of eosinophilic activity and inflammation. Gaining insight into the function of eosinophils in allergic rhinitis is crucial for developing treatments that might regulate eosinophilic activity and diminish persistent inflammation. Currently, researchers are focusing on developing medications that specifically target IL-5 or other pathways that activate eosinophils. The goal is to alleviate symptoms and minimize the long-term consequences of allergic rhinitis for individuals affected by it.[16]

The function of IgE

Immunoglobulin E (IgE) is an antibody type that has a key role in the mechanism of the allergic reaction in allergic rhinitis. Frequently, exposure to an allergen leads to the generation of specific IgE antibodies targeting that allergen, accompanying the sensitization process. During the subsequent allergic response, mast cells and basophil cells attach to IgE molecules, intensifying the allergic reaction when the

person is exposed to the allergen again. Following sensitization to an allergen, B cells (B lymphocytes) become activated and start generating specific IgE antibodies targeting the allergen. IgE is present in the bloodstream and has the ability to attach itself to the outer layer of mast cells and basophils. Mast cells and basophils possess a distinct surface receptor called the FcεRI receptor, which specifically binds to IgE. The IgE attached to this receptor functions as a "sensor," ready to react to specific allergens. Upon re-exposure to the specific allergen, mast cells and basophils with pre-attached IgE molecules bind to the allergen. Allergens binding to IgE on mast cells and basophils release inflammatory mediators like histamine and leukotrienes.[18]

Mast cells and basophils release inflammatory mediators, leading to a fast and potent allergic reaction. For instance, released histamine induces vasodilation, heightened vascular permeability, and contraction of smooth muscles, resulting in the distinctive symptoms of allergic rhinitis. This response initiates a loop that has the potential to intensify the allergic response when exposed again, as the process of sensitization and the creation of IgE antibodies persist. IgE plays a pivotal part in the pathophysiology of allergic rhinitis and makes a substantial contribution to the symptoms experienced by those affected by this condition. Figuring out what role IgE plays in allergic reactions is the first step in making treatments that target IgE, like desensitization therapy or immunomodulatory therapy, which aim to lower IgE production and stop allergic reactions. Research aims to explore more efficient and groundbreaking therapeutic methods for controlling allergic rhinitis by elucidating these pathways.[19]

2. Conclusion

Allergic rhinitis is characterized by intricate interactions between the immune system and specific allergens. The basic process starts with sensitization, which is when people who are susceptible to an allergen make an immune response to it. This usually involves making IgE antibodies that are specific to the allergen. Mast cells and Th2 cells have a pivotal role in the allergic response by releasing inflammatory mediators like histamine, interleukin-4 (IL-4), IL-5, and IL-13. Mast cells are the primary source of histamine, which induces characteristic symptoms of allergic rhinitis, including rhinorrhea, pruritus, sneezing, and nasal congestion. In addition, eosinophils, a specific subtype of leukocyte, accumulate in the nasal tissue and contribute to persistent inflammation. Cytokines, such as IL-5, activate eosinophils, leading to tissue damage and the prolongation of inflammation. This procedure initiates a recurring pattern of allergic reactions that might intensify with each successive encounter with the allergen. When someone has allergic rhinitis, their immune system reacts by making them more sensitive, making IgE, releasing inflammatory chemicals, and then damaging tissue.

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