Symptomatic therapies and immunomodulating therapies in allergic rhinitis.

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Introduction

The formation of an immune response to allergens that is mediated by immunoglobulin E (IgE) is what defines atopic allergic sensitization. Since its incidence has grown over time, allergic rhinitis (AR), an illness that affects 400 million people globally, constitutes a global problem. Asthma and allergic rhinitis frequently coexist, which compromises quality of life, productivity at work or school, and has a substantial financial impact. Recent research on the pathogenesis of allergic rhinitis points to disruption of the nasal epithelial barrier integrity as the source of aberrantly elevated Th2 cytokines, which are demonstrated to be the cause of allergic rhinitis [1].

Symptomatic therapy

Antihistamines, nasal or oral glucocorticoids, nasal decongestants, and leukotriene receptor antagonists are used as symptomatic therapies for allergic rhinitis and work as symptom-relieving medications. The most common first-line treatment for mild allergic rhinitis is an antihistamine; however, the first generations of antihistamines (such as diphenhydramine and hydroxyzine) are no longer advised due to a number of negative side effects on the central nervous system, anticholinergic side effects, and cardiac toxicity. The safer and more effective newer generation antihistamines (such cetirizine, loratadine, desloratadine, fexofenadine, rupatadine, and bilastine) should be used. Olopatadine, levocabastine, and azelastine are examples of novel intranasal antihistamines that guarantee enhanced drug delivery to nasal mucosa exposed to release mediators during allergic inflammation in allergic rhinitis [2].

Moreover, intranasal corticosteroids, which serve as the first-line drug therapy for allergic rhinitis, are beneficial for treating both mild and moderate-severe allergic rhinitis in both children and adults. Mometasone furoate, fluticasone propionate, triamcinolone acetonide, and ciclesonide are the only intranasal corticosteroids for children that are currently licenced. The combination medicines that helped individuals with allergic rhinitis have less severe symptoms were determined by a meta-analysis study. As compared to oral H1 antihistamines with intranasal corticosteroids, the meta-analysis showed that intranasal H1 antihistamines and intranasal corticosteroids combination therapy were superior [3].

The action of cysteinyl leukotrienes, a significant and potent allergic mediator that produces allergic inflammation and a variety of allergic symptoms such nasal congestion and mucus production, is then inhibited by leukotriene receptor antagonists (such as montelukast, zafirlukast, and pranlukast). Leukotriene receptor antagonists were found to be more effective than H1 antihistamines during overnight symptoms, but not during daytime symptoms, according to a meta-analysis research. Additional meta-analysis studies showed that combining leukotriene receptor antagonist medication with H1 antihistamines boosted the therapy's effectiveness in easing daytime symptoms.

Immunomodulating therapy

Instead of causing a change to an immunologically naive or unresponsive state, treatment for allergic rhinitis that targets immune modulation tries to modify the natural courses of allergic rhinitis. The topic of allergen immunotherapy for allergic rhinitis was the main emphasis of this section. A small percentage of individuals with allergic rhinitis do not respond to treatment with standard pharmacotherapy, thus disease-modifying therapeutic treatments such allergen immunotherapy are used. There are two ways to administer allergen immunotherapy: subcutaneously or sublingually. In the context of allergen immunotherapy for HDM, subcutaneous immunotherapy can cause a transient rise in FcRI expression on DCs but not on basophils, suggesting potential tolerogenic functions for IgE/FcRI signalling in DCs [4].

Targeting these indicators with allergen immunotherapy is attractive because basophils are significant mediators in triggering early phase reactions in allergic rhinitis. The threshold of basophil activation can be lowered after a year of subcutaneous immunotherapy for Parietaria, highlighting the value of allergen immunotherapy in treating disease and slowing the spread of the disease. During HDM-subcutaneous immunotherapy, high blood periostin levels were seen, suggesting that periostin may be a valuable biomarker in allergen immunotherapy. Moreover, serum levels of BAFF, IFN, IL10, and IL33 were found to be highly predictive of the effectiveness of subcutaneous immunotherapy in individuals with allergic rhinitis who had received treatment with it. The downstream signalling pathway of type 2 innate lymphoid cells and the Th1 immune response are tightly linked in allergic rhinitis [5].

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Conclusion

Significant unmet requirements to address these problems are created by the rising prevalence of allergic rhinitis and the fact that it is an incurable illness. Comorbid conditions for allergic rhinitis place an additional financial and medical burden on those who suffer from the condition. In addition, dual allergic rhinitis, a recently recognised allergic rhinitis phenotype, makes it more difficult to diagnose allergic rhinitis in individuals who only have a seasonal allergy and present nasal symptoms due to both perennial and seasonal allergens.

References

1. Steelant B, Seys SF, Boeckxstaens G, et al. Restoring airway epithelial barrier dysfunction: a new therapeutic challenge in allergic airway disease. Rhinology. 2016;54(3):195-205.
2. Steelant B, Seys SF, Van Gerven L, et al. Histamine and T helper cytokine–driven epithelial barrier dysfunction in allergic rhinitis. J Allergy Clin Immunol. 2018;141(3):951-63.
3. Sarah CO, Shukri NM, Ashari NS, et al. Zonula occludens and nasal epithelial barrier integrity in allergic rhinitis. PeerJ. 2020;8:e9834.
4. Li J, Wang H, Chen Y, et al. House dust mite sensitization is the main risk factor for the increase in prevalence of wheeze in 13-to 14-year-old schoolchildren in Guangzhou city, China. Clin Exp Allergy. 2013;43(10):1171-9.
5. Notkins AL, Lernmark Å. Autoimmune type 1 diabetes: resolved and unresolved issues. J Clin Invest. 2001;108(9):1247-52.

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