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Anti-IgE Therapy for Allergic Rhinitis

Kalyan Nagulapalli Venkata*

Research Associate, Loker Hydrocarbon Institute, University of Southern California, USA

Abstract

Although skin prick testing in AR patients is positive (showing the production of antigen-specific Immunoglobulin E), there is no correlation with total circulating IgE levels. There was no correlation between circulating IgE levels and skin prick tests or laboratory testing for specific IgE. Omalizumab binds to uncomplicated IgE more strongly in humans than Fc-epsilon. Omalizumab works by lowering IgE levels and decreasing the development of FceRI receptors (which bind IgE) in mast cells and basophils, resulting in reduced mast cell recruitment and responsiveness and, as a result, less eosinophilic infiltration and activation. Anti-IgE treatment with omalizumab may reduce the lifespan of mast cells and induce dendritic cells to produce less FcRI.

Keywords: Allergic dermatitis • Multimorbidity • Hypermethylation • Anti-IgE

Introduction

The morbidity of allergic disorders has risen rapidly in recent decades, becoming a serious public health concern. Allergic illnesses not only harm patients' physical and psychological health, but they also impose a significant economic cost. Allergic illnesses are a group of diseases caused by immune system hypersensitivity to certain environmental chemicals. Common allergic illnesses in individuals include asthma, allergic rhinitis (AR) and allergic dermatitis (AD). The growing understanding of allergic disorders has aided in recognising the critical function of the interleukin (IL) family in the onset and progression of allergic diseases. IL-37, a new member of the IL-1 family, significantly suppresses both innate and acquired immunity. The human IL-37 gene is found on human chromosome 2q12-13, near the regulatory areas for the IL-1 and IL-1 genes. In human monocytes and macrophages, IL-1, IL-1 and IL-37 are all transcribed at the same time in response to LPS stimulation. The uniqueness of this location may be connected to IL-37's immunomodulatory activity. Unlike other members of the IL-1 family, the IL-37 gene is not found in mice or chimps. As a result, many IL-37 investigations are based on IL-37 transgenic mice (IL-37tg mice) and recombinant IL-37 (rIL-37) protein administration in a mouse model.

Literature Review

Asthmatic morbidities the pathogenesis of allergic asthma and other allergy diseases has been conclusively linked to IgE. Multiple genetic and environmental factors interact to affect illness manifestation and result in varied and frequently coexisting phenotypes in IgE-mediated allergy disorders. Complex allergic illnesses including asthma, rhinitis, conjunctivitis and food allergy may be linked with allergen-specific IgE and nonallergic processes that can coexist in the same patient (referred to as allergic comorbidity cluster or multimorbidity) and share causative pathways. The Mechanisms of the Development of ALLergy programme (MeDALL) was launched as an FP7 European Union initiative to investigate novel mechanisms of allergy beginning from early infancy through young adulthood. In the MeDALL study, IgE sensitization was associated with approximately 38% of allergic multimorbidities; additionally, rather than being the sole factor for multimorbidity, IgE sensitization was suggested to be a component of a broader phenotypical presentation of patients characterised by polysensitization and multimorbidity, which was associated with the frequency, persistence and severity of allergic symptoms.

Discussion

A typical AR history includes sneezing, rhinorrhea, nasal blockage and nasal irritation. Cough, postnasal drip, irritation and weariness are other frequent symptoms. Some individuals may report irritation of the palate and inner ear. Ocular symptoms of ARC may include itching, tearing and burning. Symptoms in younger children may include snorting or sniffing, throat clearing and coughing. Children may generate a clicking sound as they rub their tongue on their itchy palate to soothe this pruritic discomfort. Once the history and physical point to AR, finding particular IgE positivity may be useful in confirming the diagnosis. Particular IgE determination is appropriate when establishing an allergic cause for a patient's symptoms, confirming or excluding specific allergic causes for a patient's symptoms, or determining specific allergen sensitivity to advise avoidance measures or IT is required. Skin testing for particular antigens may be performed safely in the allergy clinic and produces findings in 20 minutes with high sensitivity and specificity. When assessing individuals with nasal allergic responses following allergen challenge testing, specific blood IgE testing demonstrates equivalent sensitivity to skin testing [1-7].

Conclusion

Exposure to allergens can cause epigenetic changes in the form

***Address for Correspondence:** Kalyan Nagulapalli Venkata, Loker Hydrocarbon Institute, University of Southern California, USA, E-mail: kalyanvenkata@gmail.com*

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of increased histone deacetylation, increased DNA methylation and changes in miRNA levels in respiratory epithelial cells and peripheral CD4+ cells in the host system, which can change the cytokine profile and dysregulate the balance of T regulatory cells, resulting in the development of allergic rhinitis symptoms. Controlling histone acetylation with histone deacetylase inhibitors, DNA hypermethylation with DNA methyl transferase inhibitors and post-transcriptional gene expression with miRNA mimetics may all aid in the treatment of allergic rhinitis.

Conflict of Interest

There are no conflicts of interest by author.

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