Journal of Clinical Respiratory: Open Access

Pathophysiology and Diagnosis of Allergic Rhinitis Mediators

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Commentary

Received: 15-Jun-2023, ManuscriptNo. JCROA-23-102593; **Editor assigned:** 20-Jun-2023, Pre QC No. JCROA-23-102593 (PQ); **Reviewed:** 04-Jul-2023, QC No. JCROA-23-102593; **Revised:** 19-Jul-2023, Manuscript No. JCROA-23-102593 (R); **Published:** 16-Aug-2023, DOI: 10.4172/jclinresp.5.2.002

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Citation: Ahmed H, Pathophysiology and Diagnosis of Allergic Rhinitis Mediators. J Clin Resp. 2023;5:002.

DESCRIPTION

The pathophysiology of rhinitis is well defined for allergic infectious some medication related and select systemic disease associated rhinitis syndromes. The pathophysiology of allergic rhinitis stems from the degranulation of mast cells and the subsequent mucosal recruitment of inflammatory cells, particularly eosinophils. The role of mast cell degranulation has been confirmed by nasal allergen challenge, nasal lavage with analysis of mediators, nasal cytology and nasal biopsy. Inflammation characterized by recruitment of eosinophils into the nasal mucosa, is an essential component of the pathology of allergic rhinitis. The symptoms of allergic rhinitis result from the combined effects of inflammatory cell recruitment and of the actions of mediators on receptors like H1 receptor, leukotrienes specifically LTD4 with the cysteinyl L receptor.

The mediators released from mast cells are responsible for the acute symptoms of allergic rhinitis, primarily itching and sneezing. The mucosal inflammation is primarily a result of eosinophil immigration, activation and persistence due largely to factors released by the mast cell. The mast cell degranulates when high affinity IgE receptors are cross-linked by antigen (allergen). IgE specific for a causal allergen is bound to the mast cell *via* the high affinity IgE receptor, enabling the triggering of degranulation on exposure to specific allergen. The production of specific IgE is a result of the complex interaction of genetic predisposition and the environment. Exposure to environmental allergens, which is a risk factor for sensitization, does not result in uniform immune responses, even in subjects with similar or even identical, genetic backgrounds. Modulation of the IgE response depends on variables such as the type of allergen, the route and dose of exposure, the timing of exposure (*e.g.*, childhood versus adulthood) and concomitant or preceding exposure to infectious organisms or adjuvants, such as endotoxin. Genetic factors affect the epitope or specific portion of the anti-gen to which the individual responds (some epitopes are more likely to evoke an IgE response) as well as the immunologic regulation that modulates the tendency to produce IgE. Interactions between antigen-presenting cells, such as dendritic cells and B lymphocytes, T-regulatory cells (T), group 2 Innate Lymphoid Cells (ILC2). Epithelial cells and T helper 1-(Th1-) and Th2-like cells, determine the probability of specific IgG antibody formation versus IgE antibody formation versus tolerance to a specific allergen. To further complicate the understanding of this process, individuals may simultaneously be sensitized and tolerant to different allergens, for example, dust mite and cat, emphasizing that antigen properties, variation in exposure characteristics and genetic factors regulate individual antigen responses. Finally, the blood concentration of specific IgE for a selected allergen or the magnitude of a skin test response with allergen does not generally correlate with the severity of symptoms on exposure to that allergen but rather the likelihood that the allergen is contributing to symptoms. Thus, a simple, unifying explanation of the allergic response or a measurable parameter that will consistently predict symptoms is not available.

The diagnosis of allergic rhinitis is presumptive until specific allergic sensitivity is identified by epicutaneous or percutaneous testing or *in vitro*-specific IgE testing. Immediate wheal and flare skin tests remain the most cost-effective means of identifying specific IgE. The value of intradermal allergy testing is primarily to exclude the diagnosis with negative results, with positive intradermal results providing only tenuous support for a diagnosis of allergic rhinitis. The evidence of specific IgE should be correlated with exposure and symptoms to support the diagnosis. Identifying environmental factors that trigger nasal symptoms is important in distinguishing allergic rhinitis from non-allergic or mixed rhinitis (components of both allergic and nonallergic rhinitis). Congestion is also one of the most commonly occurring symptom and the secretions are clear, white or cloudy mucus type.