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Commentary

Effects and Causes of Allergic Airway Diseases

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DESCRIPTION

Allergic Airway Diseases (AAD) such as Bronchial Asthma (BA) and Allergic Rhino Conjunctivitis (ARC), are substantial health and financial burdens because of their detrimental effects on quality of life, including social interactions, school attendance, and work performance. Pollen is one of the main environmental variables producing respiratory allergy, and both hereditary and environmental factors have a role in the development of allergic airway illnesses. A distinctive feature of pollinosis is the repeating of symptoms during a certain pollen season, which differs between nations. Patients with respiratory allergies who are sensitive to both seasonal (such as pollen) and perennial allergens (such as House Dust Mites (HDM), cockroaches, and moulds) report worsening during the pollen season. Pollen allergenicity is mostly influenced by air pollution and climate change, despite the fact that the pollen season and the flowering season are associated. Pollution increases the amount of pollen allergenic proteins, particularly in metropolitan regions, and modifies these proteins into more allergenic components that cause allergies in susceptible people. On the other hand, the timing, length, geographic range, and dispersion of the pollen season are all impacted by climate change.

The allergic airway diseases share a tissue tropism, but they also have a unique pattern of inflammation that includes the build-up of eosinophils, type 2 macrophages, Innate Lymphoid Cells type 2 (ILC2), IgE-secreting B cells, and T helper type 2 (Th2) cells in the tissues of the airways, as well as the prominent production of type 2 cytokines like interleukin, IL-4, in addition to the major clinical manifestations of nasal obstruction, headache, hyposmia, cough, shortness of breath, chest pain, wheezing, and, in the most severe cases of lower airway disease, death, these factors and associated inflammatory molecules also cause characteristic remodelling and other changes of the airways that include goblet cell metaplasia, enhanced mucus secretion, smooth muscle hypertrophy, tissue swelling, and polyp formation.

The fact that allergic airway illnesses are syndromic and now come in a wide variety of physiological forms or endotypes shows that unique endogenous or environmental variables are responsible for how they manifest. But research from several angles has now connected these illnesses to a single infectious source-fungi-and to a molecular pathophysiology in which these microbes produce airway proteinases locally.

Importance of IgE in allergic rhinosinusitis, chronic rhinosinusitis and asthma

IgE is important in AAD, after Th2 cytokines cause the transformation from IgG to IgE, B cells control its release. It functions as an early-phase effector of the Th2 hypersensitive immune response and is a sign of atopy and allergen sensitization. Anti-IgE medicines have IgE as a therapeutic target. Total IgE levels in the serum that are high serve as a diagnostic indicator for AAD. Additionally, bronchial hyperreactivity is linked to total IgE. Total and serum IgE have a correlation with asthma severity, and serum-specific IgE is a biomarker for AR that can be predicted. Although its value as a predictor of treatment response is debatable, it is a trustworthy monitoring biomarker of the effectiveness of anti-IgE therapy. Serum-specific IgE is a reliable diagnostic to choose patients for AIT (Allergen Immunotherapy) treatment, but it is neither a prognostic nor a monitoring biomarker for AIT. AIT may have a potential predictive biomarker in the serum IgE/total IgE ratio.

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