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An overview on allergy-associated asthma management

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DESCRIPTION

A clinical syndrome known as asthma is characterised by airway inflammation, increased bronchial responsiveness, and episodic reversible blockage. Inflammatory cells, their mediators, the airway epithelium and smooth muscle, and the nervous system interact in a complex way to cause asthma. These interactions can cause the asthma patient to experience symptoms of shortness of breath, wheezing, coughing, and tightness in the chest in genetically predisposed individuals.

There are two categories of etiologies for asthma triggers: allergic and nonallergic. Seasonal pollen, mould spores, dust mites, and animal allergens are examples of aeroallergens. Smoke, scents, cold air and weather, chemicals, medications (such as aspirin and other NSAIDs), beta-blockers, exercise, hormonal changes (such as pregnancy, menstruation), and bisulfite food additives can all be non-allergic causes of asthma. Sinusitis, nasal polyposis, gastro-esophageal reflux disease (GERD), and allergic rhinitis are asthma co-morbidities.

Genetic variations may affect an individual's predisposition to developing asthma as well as their response to treatment. Between and among racial and ethnic groups, there is substantial genetic variation, but the issue is complicated by significant concurrent economic, cultural, and environmental differences, including location.

Airway inflammation, a defining characteristic of the asthmatic lung, is significantly induced by environmental exposure in sensitised individuals. Although different routes are used by different stimuli to cause inflammation, the end result is always a rise in bronchial reactivity.

It is commonly known that allergies play a significant role in asthma. For instance, early childhood dust mite exposure has been linked to the development of asthma and probably atopy. Antigens from mites and cockroaches are widespread, and studies have demonstrated that exposure and sensitization enhance the morbidity of asthma.

Asthma episodes are brought on by allergies in 60–90%of children and in 50% of adults. Skin test results are positive (immediate) in 75%-85% of asthma patients. This sensitivity is connected to disease activity in youngsters.

Although aeroallergens cause the majority of asthmatic symptoms, some persons also have symptoms in response to non-allergic stimuli. Approximately 3–10% of asthmatics have NSAID sensitivity. An estimated 5%-10% of asthmatics have airway illness brought on by their jobs or industries. Many people experience symptoms following viral respiratory tract infections.

Avoiding allergens and taking other measures to manage the environment are doable and successful. With the avoidance of environmental allergens, symptoms, lung function test results, and Airway Hyperreactivity (AHR) improve. Clinical improvement can be attained by eliminating even one allergen out of many. Patients routinely disregard these precautions, nevertheless.

Asthma's genesis is probably complex. Asthma predispositions in individuals may be influenced by genetic factors. Responses to drugs may also be influenced by genetics. Impaired responses to inhaled, short-acting beta-agonist inhalers have been linked to variation in the beta-adrenergic receptor gene of the Arg-Arg type.

CONCLUSION

In the past decade, there have been significant advances in the phenotypic classification of asthma. Asthma can be viewed as a Type 2 (T2 high) subtype, which implicates signaling of IL-4, IL-13, Il-5, IgE and other inflammatory cascade pathways. Proposed biomarkers of T2 asthma include serum IgE, blood and lung eosinophils, exhaled nitric oxide, and others. Allergic asthma is considered a T2-high form of asthma. Less is known about non-T2 asthma, but it is marked by the absence of these

biomarkers. Asthma can also be broadly categorized as eosinophilic versus non-eosinophilic. Asthmatics with eosinophilic disease may have very high levels of eosinophils in the blood and/or sputum. Asthma COPD Overlap (ACO) is also an overlap phenotype seen in patients with clinical features of both asthma and COPD.