

The impact of allergic rhinitis on asthma

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The nose as protector of the airways

Why do we have a nose? Certainly not only for smell or esthetic purposes as most people like to think. It's no coincidence that the nose is located at the beginning of the respiratory tract. The nose plays an important role in air-conditioning and host defense. While breathing through the nose, the inhaled air is heated and humidified due to close contact with the vascular network in the nasal mucosa. Moreover, the nose has an impressive filter capacity: inhaled particles, 10 µm and larger, get trapped in the nose and will be transported with the mucus to the oropharynx. Micro-organisms and allergens in sensitized people can activate resident inflammatory cells leading to a localized immune response. The initial response is characterized by mast cell degranulation and T-cell activation. These two cells release adhesion molecules, cytokines, chemokines and vasoactive products. This results in the recruitment of leukocytes, such as neutrophils, basophils and eosinophils, from the circulation to the exposed area. Therefore, the nose is the first line of defense in protecting the lower airways from potentially harmful agents. In allergic rhinitis, nasal function is often impaired which can affect the lower airways as well.

The association between allergic rhinitis and asthma

Allergic rhinitis and asthma frequently occur together and they share a common genetic background. The prevalence of allergic rhinitis and asthma varies all over the world and is associated with a "westernized" lifestyle. Allergic rhinitis patients have an increased risk to develop asthma, especially when bronchial hyperresponsiveness (BHR) is present. In the majority of the allergic rhinitis patients, the rhinitis symptoms start before the onset of asthma. At least 80% of all asthmatic patients have rhinitis. The epidemiological data are the foundations on which the united airways concept is build.

The immunopathological aspects of allergic rhinitis and asthma have been extensively studied in the past decade. Allergic rhinitis and asthma are characterized by a similar inflammatory response. In allergic rhinitis patients without asthma inflammatory changes have been demonstrated in the lower airways. In asthmatic patients without rhinitis, on the other hand, increased numbers of eosinophils are present in nasal mucosa. The number of nasal eosinophils is positively correlated with the degree of BHR. In conclusion, mucosal inflammation is present in the entire airway of patients with allergic rhinitis and/or asthma.

The mechanisms of nasobronchial interaction

Allergen provocation studies in animals and human have provided a useful model to better understand the crosslink between nose and lungs. Nasal provocation in allergic rhinitis subjects induced bronchial inflammation characterized by increased endothelial expression of adhesion molecules, such as VCAM, and an influx of eosinophils and basophils. Vice versa, segmental bronchial provocation in the same subset of allergic patients showed similar results in nasal mucosa. The inflammatory changes were accompanied by airway symptoms and decreased airway patency.

Although several mechanisms of nasobronchial interaction have been proposed in the past (altered breathing pattern, nasobronchial reflex and aspiration of nasal contents), to date most evidence points towards a systemic pathway. Inflammatory mediators and cells, such as IL-5 and eosinophils, increased after nasal or bronchial allergen challenge, suggesting that the systemic circulation is involved in the interaction between upper and lower airways. This has profound impact on daily clinic. It stresses the importance of a diagnostic strategy involving both ends of the respiratory tract. The systemic link between allergic rhinitis and asthma provides a rationale for a systemic approach targeting the underlying allergic disorder.

Suggestions for further reading:

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