SHORT COMMENTARY

Mucosal Inflammation in Allergic Rhinitis and Bronchial Asthma - Two Sides of a Coin

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A mutual relationship between allergic rhinitis (AR) and bronchial asthma (BA) was observed for the past decades. This was not surprising due to the common mucosal immune system for the nose, lungs, eyes, skin, and gastrointestinal tract. If an immune response against certain allergen arises at one site of the mucous system, the effector immune cells then exert the same immune response at other sites of the mucosa. Thus, AR and BA are closely connected diseases and mucosal inflammation is not restricted to the mucosa of upper or lower airways. Moreover, AR is a risk factor for the development of BA, as well as AR, could aggravate ongoing bronchial inflammation.[1] Usually, AR precedes BA and could be the first manifestation of an allergic respiratory disease. However, in most cases, AR and BA present simultaneously. The crucial role of AR in BA was demonstrated in 1999 when the World Health Organization decided to highlight the connection between them. Furthermore, an evidence-based guideline was created - Allergic Rhinitis and its Impact on Asthma (ARIA). ARIA goals not only to increase the awareness of AR and to conduct an effective treatment worldwide but also to restrict the impact of AR as a risk factor for developing BA.[2]

AR and BA are common allergic diseases and a number of epidemiological studies clearly show the simultaneous presence of them. However, the explanation for that observation lays on the demonstrated similar dysfunction of the upper and lower airways and the common pathogenesis. Another proof for their mutual relationship is that the treatment of AR in patients with concomitant BA alleviates the symptoms of the latter, as well as reduces the risk of severe asthma exacerbations and hospitalization.[3,4]

It was shown that allergic inflammation of lower airways may facilitate the onset of AR in subjects with respiratory symptoms (i.e., dyspnea and wheezing) even before diagnosing with BA. When non-asthmatic patients with AR underwent bronchial allergen challenge, a reduction in nasal inspiratory flow with a simultaneous increase of the nasal symptoms was observed. Moreover, the number of eosinophils, eotaxin-positive cells, and interleukin-5 expression in the nasal mucosa was increased, whereas the number of mast cells was decreased.[3] Interestingly, even some patients denied preexisting asthma; >20% of newly diagnosed BA patients reported "respiratory problems" in childhood which could be due to the undiagnosed BA.

One of the most important common features of AR and BA is the mucosal inflammation, involving immune cells and mediators. Nevertheless, the early and late phases of allergic response in AR and BA, triggered by causative agents, are similar. To establish and confirm the nasal-bronchial relationship, Braunstahl *et al*. examined the expression of adhesion molecules in the nasal and bronchial mucosa after a nasal provocation test with an allergen. At a 24th h after the nasal exposure, an increase in eosinophil counts was found not only in the nasal but also in lung mucosa. Increased expression of intercellular adhesion molecule-1, vascular cell adhesion molecule-1, and E-selectins in the nasal and bronchial tissue of AR patients after nasal provocation in patients with AR leads to generalized airway inflammation including both upper and lower airways.[1] Clinically, mucosal inflammation is manifested with obstruction of the bronchi and increased secretion of mucus in the respiratory airways. Taking into account the common mucosal immune system of the upper and lower airways, the impact of the so-called minimal persistent inflammation is extended on the mucosa of the whole respiratory tract. In conclusion, allergen provocation of the nose or bronchi leads to generalized

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inflammation of the respiratory tract and this is clinically significant. Despite the similar inflammatory infiltrate in AR and BA, the degree of inflammation may vary. In patients with moderate-to-severe asthma, eosinophilic infiltration is more pronounced in bronchi than in the nose while patients with mild asthma have a similar degree of inflammation. This is the underlying reason for the more pronounced remodeling of the airways of the bronchi than in the nasal mucosa.[3]

All these evidence suggest that in patients with AR and BA could be employed a common therapeutic approach, except the limitation of allergenic exposure, such as using similar drugs and/or allergen-specific hyposensitization. It is essential to follow-up the AR patients for developing asthma symptoms, to treat simultaneously upper and lower airways, and to educate patients and their relatives. Concomitant diseases, such as rhinosinusitis, conjunctivitis, BA, should be diagnosed and treated early along with AR. Furthermore, there is evidence that the treatment of AR may influence the control of asthma in patients with both respiratory diseases.[4,5]

In conclusion, the similar pathophysiological mechanism and mucosal inflammation, along with the concomitant occurrence of AR and BA, suggest the benefit of using the same therapeutic strategy for these patients including biologic therapy.

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