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Local Allergic Rhinitis and its Relation to Allergic Rhinitis

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Introduction

Allergic rhinitis (AR) is triggered after allergen specific IgE and T helper cells recognize inhalant allergen in the environment. Some studies have shown that allergen exposure is protective against IgE-mediated allergic disorders [1-3], while some strongly suggest that exposure to allergen increase the risk of developing allergy [4-7]. The divergence of views may be attributed to the variety of the intensity and persistence of allergen exposure in the environment.

Our previous study demonstrated dose- and duration-related effects of allergen exposure on the development of allergic diseases by evaluating the allergic nasal response, OVA-specific IgE (OVA-sIgE) in serum and nasal lavage fluid (NALF), and nasal histology in mice treated with different doses and sensitized duration of allergen exposure. Nasal sensitization and challenge of mice with different doses and sensitized duration of OVA were able to induce the symptoms of AR in the study. Moreover, there was a dose related effect on eosinophil accumulation in the nasal mucosa. Allergic nasal response and histologic examination revealed the success in the establishment of AR mouse model. The OVA treatment led to the induction of OVA-sIgE in serum and nasal lavage fluid, but prolonged sensitization decreased circulating levels of OVA-sIgE. Taken together, different patterns of allergen exposure had significant effects on the development of AR.

According to the pattern of sensitization to the aeroallergens, rhinitis patients were classified into 3 groups at present: (1) AR, who was symptomatic with systemic atopy; (2) local allergic rhinitis (LAR), who was symptomatic with positive local but negative skin test/systemic sIgE; and (3) non-allergic rhinitis (NAR), who was symptomatic without systemic atopy. LAR is characterized by one or more of AR nasal symptoms and localized sensitization at nasal mucosa, which distinguishes it from AR and NAR. In clinical practice, the diagnosis of AR is mostly based on clinical manifestations and supported by systemic atopy (positive skin test/serum specific IgE). Patients who have similar symptoms to those of AR without detectable systemic atopy are diagnosed with NAR. But owing to the negative results in traditional test for systemic atopy, most LAR patients were misdiagnosed as NAR. It was reported that LAR was a common upper airway disease that affected 25.7% of the Spanish rhinitis population [8]. A significant number of rhinitis patients previously diagnosed with NAR might actually have had LAR.

In our previous study, the mice were intranasally sensitized with 400 or 25 μg OVA for 5 or 14 days, respectively. Interestingly, we found mice treated with 25 μg OVA sensitized for 14 days could manifest the major features of human LAR, such as allergic nasal symptom and localized but not systemic sIgE. LAR was likely to occur if the allergen is exposed repeatedly at low dose.

Despite the identification of LAR, little is known about its pathogenesis and relation to AR. The epidemiological investigation showed that LAR patients had the similar clinical characteristics with those of AR patients [8]. LAR not only had similar clinical magnification, but also similar histologic changes to typical AR. Prominent eosinophilic accumulation was observed in the LAR mice, suggesting that LAR share the same histologic changes with AR. The main difference between AR and LAR is the detectable levels of sIgE in serum. System atopy could be detected in AR patients, whereas not in LAR patients. LAR, instead, has localized sIgE at nasal mucosa. It is pointed out that LAR was the first step in the development of AR [9]. However, the view that LAR might be recently acquired allergy is only partly accepted, for there were rhinitis symptoms, negative serum sIgE and IgE producing B cells in nasal tissues, but localized sIgE antibody, the most important biomarker for LAR, was absent in that study.

Since there were no detectable levels of systemic sIgE in LAR, it was inferred that localized sIgE might have originated solely from B lymphocytes residing in the nasal mucosa [10,11]. Though a significant amount of sIgE is expressed locally in the nasal submucosal tissue [12,13], the reason why its production is restricted only in the nose instead of being distributed systemically remains unknown. One of the hypotheses is that environmental aeroallergens, as they are inhaled, are "captured" by the nasal mucosa, where the production of local sIgE is provoked and immune response is restricted, resulting in undetectable or even no sIgE in the serum.

There may be another explanation for localized sIgE. On one hand, in sensitization phase repetitive low-dose allergen exposure induced immunological tolerance and decreased sIgE level in serum, as demonstrated in our previous study. On the other hand, circulating sIgE is dynamic in the challenge phase of AR progress. To investigate whether circulating level of OVA-sIgE is also associated with OVA challenge, the 25 μg OVA-treated and sensitized for 14 days mice were challenged with OVA on 2 or 5 consecutive days, respectively. OVA-sIgE levels were reduced in serum and tended to increase in the NALF after 5 allergen challenges (data not published), suggesting that sIgE response may undergo dynamic change after repetitive OVA exposures and serum sIgE may be distributed locally to nasal mucosa during the challenge phase. A more plausible explanation for lack of evidence of systemic atopy in LAR would seem to be that very few sIgE remaining in the blood after repetitive low-dose allergen exposure made it a false impression of the absence of circulating sIgE.

Seemingly, different patterns of allergen exposure have an important role in the degree of inducing allergic nasal response. The establishment of mouse model is indicative of influence of allergen exposure pattern on developing AR and LAR. LAR was not a distinct entity but rather a status that AR might evolve to under the circumstances of allergen exposure repeatedly at low dose. Whether

other factors in allergen exposure might affect the local allergic inflammation in LAR remains unknown. The subtle relationship between AR and LAR still requires further investigation in human and animal models.

References

1. Du Toit G, Katz Y, Sasieni P, Mesher D, Maleki SJ, et al. (2008) Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. J Allergy Clin Immunol 122: 984-991.
2. Katz Y, Rajuan N, Goldberg MR, Eisenberg E, Heyman E, et al. (2010) Early exposure to cow's milk protein is protective against IgE-mediated cow's milk protein allergy. J Allergy Clin Immunol 126: 77-82.
3. Wang Y, McCusker C (2006) Neonatal exposure with LPS and/or allergen prevents experimental allergic airways disease: development of tolerance using environmental antigens. J Allergy Clin Immunol 118: 143-151.
4. Wahn U, Lau S, Bergmann R, Kulig M, Forster J, et al. (1997) Indoor allergen exposure is a risk factor for sensitization during the first three years of life. J Allergy Clin Immunol 99: 763-769.
5. Brussee JE, Smit HA, van Strien RT, Corver K, Kerkhof M, et al. (2005) Allergen exposure in infancy and the development of sensitization, wheeze, and asthma at 4 years. J Allergy Clin Immunol 115: 946-952.
6. Torrent M, Sunyer J, Muñoz L, Cullinan P, Iturriaga MV, et al. (2006) Early-life domestic aeroallergen exposure and IgE sensitization at age 4 years. J Allergy Clin Immunol 118: 742-748.
7. Celedón JC, Milton DK, Ramsey CD, Litonjua AA, Ryan L, et al. (2007) Exposure to dust mite allergen and endotoxin in early life and asthma and atopy in childhood. J Allergy Clin Immunol 120: 144-149.
8. Rondón C, Campo P, Galindo L, Blanca-López N, Cassinello MS, et al. (2012) Prevalence and clinical relevance of local allergic rhinitis. Allergy 67: 1282-1288.
9. Kato Y, Akasaki S, Muto-Haenuki Y, Fujieda S, Matsushita K, et al. (2014) Nasal sensitization with ragweed pollen induces local-allergic-rhinitis-like symptoms in mice. PLoS One 9: e103540.
10. Kim DY, Fukuyama S, Nagatake T, Takamura K, Kong IG, et al. (2012) Implications of nasopharynx-associated lymphoid tissue (NALT) in the development of allergic responses in an allergic rhinitis mouse model. Allergy 67: 502-509.
11. Schryver ED, Devuyst L, Derycke L, Dullaers M, Zele TV, et al. (2015) Local immunoglobulin e in the nasal mucosa: Clinical implications. Allergy Asthma Immunol Res 7: 321-331.
12. Yoshida T, Usui A, Kusumi T, Inafuku S, Sugiyama T, et al. (2005) A quantitative analysis of cedar pollen-specific immunoglobulins in nasal lavage supported the local production of specific IgE, not of specific IgG. Microbiol Immunol 49: 529-534.
13. Eckl-Dorna J, Pree I, Reisinger J, Marth K, Chen KW, et al. (2012) The majority of allergen-specific IgE in the blood of allergic patients does not originate from blood-derived B cells or plasma cells. Clin Exp Allergy 42: 1347-1355.