EDITORIAL

RHINITIS AS A PRECURSOR FOR ASTHMA

It is well known that almost all patients with allergic asthma simultaneously present symptoms of rhinitis, although the severity of the underlying disease may minimize the annoyance produced by nasal symptoms, which are scarcely noticeable in some patients. In contrast, only 12-20 % of patients with allergic rhinitis suffer from asthma[1,2]. These epidemiological data are in agreement with the idea that, because the airway mucosa is a functional unit, for asthma to occur, bronchial lability (bronchial hyperreactivity) must exist, a condition not found in many patients with rhinitis. Moreover, in many cases, the atopic disposition of these patients is obvious from other allergic manifestations, such as atopic dermatitis or food allergy, demonstrating the immune system's unified response[3].

The respiratory mucosa shows structural and functional homogeneity in all areas where it is found with the exception of greater vascularization in the nasal area. Its specific function lies in providing a defense against the noxious agents that so abundantly penetrate through the airways. The entire inner layer of the airways participates in the ciliary defense system with which the epithelial cells are endowed, in addition to the various mucosal glands and cells of the immune system, present in the subepithelial layer throughout the mucosa[4]. Likewise, it can present a unified response, although in different ways, against allergens in individuals with an atopic disposition or in other circumstances. For this reason, allergic airway disease is commonly manifested by symptoms that affect the entire mucosa (the frequent co-occurrence of asthma and rhinitis, rhinosinusitis or rhinoconjunctivitis), although frequently only a partial stretch is affected, with symptoms exclusive to the upper airway being distinguished (rhinitis, rhinosinusitis, rhinopharyngitis). In contrast, involvement of the lower airways (tracheobronchitis, asthma) is always accompanied by symptoms of rhinitis or rhinopharyngitis.

Pediatric experience shows that many children begin with symptoms of rhinitis and that months or years later some children manifest symptoms characteristic of the lower respiratory tract until finally they have an asthmatic crisis. A similar phenomenon occurs in adults, above all when the process is due to occupational overexposure to contaminants or allergens. In both cases, predisposition to progression of the disease (rhinitis/asthma) should be investigated to establish appropriate preventive treatment.

Several studies published some time ago[5-8], in which bronchial sensitivity was evaluated through methacholine or histamine challenge in patients whose sole allergic manifestation was rhinitis, led to the suspicion that to a large extent disease progression depends on the existence of bronchial lability because it was found that not all patients with rhinitis showed bronchial lability; consequently the possibility that they would develop asthma would only depend on excessive exposure to allergens or harmful substances that would give rise to inflammation of the respiratory mucosa with the consequent increase in bronchial reactivity (acquired hyperreactivity).

Other recent studies have confirmed and added to these already known findings, that is, the functional unity of the respiratory mucosa as the substrate of allergic reaction in both processes (rhinitis and asthma) as well as the anatomical-physiological bases that distinguish them[1,9-12]. The importance of these studies is undoubted since they help to establish the therapeutic measures required to prevent, as far as possible, the development of asthma in patients with rhinitis in whom challenge with methacholine, histamine or other current methods reveal bronchial lability.

Maximizing anti-allergic measures in the patient's home or workplace should be an adequate general rule but is inadequate given the difficulty of completely eliminating or sufficiently reducing aeroallergens. Topical or systemic medication merely reduces or prevents symptoms but does not prevent disease progression. Hence the advisability of establishing a treatment that could modify the anomalous immune response characteristic of allergic reaction, such as immunotherapy, a procedure highlighted in the above-mentioned review articles, and corroborated by other authors[13,14] and which could even reduce bronchial reactivity, as confirmed by Pichler et al[15] and Grembale et al[16] among other authors.

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REFERENCES

1. Danielsson J, Jenssen M. The natural course of allergic rhinitis during 12 years of follow-up. Allergy 1997;52:331-4.
2. Schoenwetter WF. Allergic rhinitis: epidemiology and natural history. Allergy Asthma Proc 2000;21(1):1-6.
3. Kulig M, Bergmann R, Tacke U, Whan U, Guggenmoos-Holzmann I and the MAS Study Group, Germany. Long-lasting sensitization to food during the first two years precedes allergic airway disease. Pediatr Allergy Immunol 1998;9:61-7.
4. Jacoby DB. Role of the respiratory epithelium in asthma. Research in Immunology 1997;148:48-58.
5. Townley R, Ryo U, Kolotin B, Kang B. Bronchial sensitivity to methacholine in current and former asthmatic and allergic rhinitis patients and control subjects. J Allergy Clin Immunol 1975;56:429-37.
6. Baraza MC, Soto M, Monferrer R, Martín-Mateos MA, Muñoz-López F. Hiperreactividad bronquial en atópicos no asmáticos. Estudio preliminar (abstract). Rev Esp Alergol e Inmunopatol Clin 1986;1(3):50.

7. Muñoz-López F. Aplicaciones del estudio de la hiperreactividad bronquial inespecífica en patología alérgica respiratoria. Arch Pediat 1987;38:373-80
8. Leone C, Teodoro C, Pelucchi A, Mastropasqua B, Cavigioli G, Marazzini L et al. Bronchial responsiveness and airway inflamation in patients with nonallergic rhinitis with eosinophilia symdrome. J Allergy Clin Immunol 1997;100:775-80
9. Linneberg A, Henrik Nielsen N, Frølund L, Madsen F, Dirksen A, Jørgensen T. The link between allergic rhinitis and allergic asthma: a prospective population-based study. The Copenhagen Allergy Study. Allergy 2002;57:1048-52.
10. Togias A. Rhinitis and asthma: evidence for respiratory system integration. J Allergy Clin Immunol 2003;111:1171-83.
11. Bousquet J, Vignola AM, Demoly P. Links between rhinitis and asthma. Allergy 2003;58:691-706.
12. Bousquet J, van Cauwenberge P, Khaltaev N. ARIA Workshop Report: Allergic rhinitis and its impact on asthma. J Allergy Clin Immunol 2001;108 (Suppl.):S147-S333.
13. Valovirta E. Capacity of specific immunotherapy in prevention of allergic asthma in children: the Preventiva Allergy Treatment Study (PAT). J Invest Allergol Clin Immunol 1997;7:369-70.
14. Möller C, Dreborg S, Ferdouse HA, Halken S, Høst A, Jacobsen L, et al. Pollen immunotherapy reduces the development of asthma in children with seasonal rinoconjuntivitis (the PAT-Study). J Allergy Clin Immunol 2002;109:251-6.
15. Pichler CE, Marquardsen A, Sparholt S, Løwenstein H, Bircher A, Bischof M et al. Specifc immunotherapy with *Dermatophagoides pteronyssinus* and *D. farinae* results in decreased bronchial hyperreactivity. Allergy 1997;52:274-83.
16. Grembiale RD, Camporota L, Naty S, Tranfa CM, Djukanovic R, Marsico SA. Effects of specific immunotherapy in allergic rhinitic individuals with bronchial hyperreactiveness. Am J Respir Crit Care Med 2000;162:2048-52.