SIMILAR ALLERGIC INFLAMMATION IN THE MIDDLE EAR AND THE UPPER AIRWAY

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Abstract. Otitis media with effusion (OME) is a chronic inflammatory disease of the middle ear space characterized by the accumulation of fluid. Previous investigations have suggested that the immunopathologic mechanism underlying the development of middle ear effusion in patients with allergy is largely due to the effects of T(H)2 mediators. The composition of the inflammatory substrate in the effusions of allergic otitis media is similar to the late-phase allergic response seen elsewhere in the respiratory tract, such as in asthma and in allergic rhinitis.

Key words: Otitis media with effusion, Eustachian tube, adenoid, IL-4, IL-5, IFN-g, eosinophil, allergy, united airways, inflammation.

Introduction. In the past decade, extensive research has supported the concept of a united airway in which an intimate interconnection exists between the upper and lower airways in allergic disease. The observed allergic inflammation is not confined to a specific target organ but rather is present in continuum with the common airway. Numerous cross-sectional studies have documented the frequent coexistence of allergic rhinitis and asthma: between 19% and 38% of patients with allergic rhinitis have coexisting asthma, a prevalence rate much higher than that in the general population.1,2 To date, no consistent

differences between the inflammatory profiles of upper and lower airways have been identified. The nasal mucosa of subjects with allergic rhinitis and the bronchial mucosa of atopic patients with asthma demonstrate similar cellular infiltrates and cytokine profiles, characterized by increased number of eosinophils, mast cells, and T-helper lymphocytes expressing TH2-type cytokines.3-5 Therefore, asthma and allergic rhinitis likely represent different clinical manifestations of a single inflammatory airway syndrome.

Objective: To determine whether the middle ear compartment may be a component of the united airways in allergic disease by comparing the inflammatory profiles of the middle ear to the upper airway.

Methods: Middle ear effusions, torus tubaris (Eustachian tube mucosa at the nasopharyngeal orifice), and adenoidal tissue biopsies were obtained from 45 patients undergoing simultaneous tympanostomy tube placement for OME and adenoidectomy for adenoid hypertrophy. The cellular and cytokine profiles of each site were investigated by using immunocytochemistry (elastase, CD3, major basic protein) and in situ hybridization (IL-4, IL-5, IFN-gamma mRNA). Atopic status was determined for each patient by using skin prick testing.

Results: Eleven of the 45 patients with OME (24%) were atopic. The middle ear effusions of atopic patients had significantly higher levels of eosinophils, T lymphocytes, and IL-4 mRNA + cells ($P < .01$) and significantly lower levels of neutrophils and IFN-gamma mRNA + cells ($P < .01$) compared with nonatopic patients. The nasopharyngeal tissue biopsies revealed similar cellular and cytokine profiles.

Conclusion: In atopic patients with OME, the allergic inflammation occurs on both sides of the Eustachian tube, both in the middle ear and in the nasopharynx. The results of this study support the concept that the middle ear may be part of the united airway in atopic individuals.

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