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ORIGINAL ARTICLE

Correlation between nasal eosinophils and nasal airflows in children with asthma and/or rhinitis monosensitised to house dust mites

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Abstract
Background: Allergic rhinitis and asthma due to mite sensitisation are diseases which are frequently associated and characterised by persistent inflammation. In the present study, we aimed to investigate the relationship between nasal airflows and nasal eosinophils in patients with asthma and/or rhinitis due to house dust mite sensitisation.
Methods: Twenty-four children with both rhinitis and asthma (R + A), 13 children with rhinitis and no asthma (R) and 10 non-allergic healthy children were evaluated prospectively. The patients belonging to the first two groups had moderate–severe grade of nasal obstruction. Total nasal symptom scores, peak nasal inspiratory flows (PNIFs) obtained by anterior rhinomanometry, skin prick tests, nasal eosinophils and FEV1 values were all assessed.
Results: Percentages of nasal eosinophils and PNIFs in patients with R + A and R ($r = -0.415$, $p = 0.04$) were found to be statistically significant and to have an inverse correlation. Skin prick tests were also significantly correlated with nasal eosinophils and PNIFs ($r = 0.372$, $p = 0.01$ and $r = -0.306$, $p = 0.04$, respectively). Both PNIFs and nasal eosinophils of patients with R + A were significantly correlated with FEV1 values ($r = -0.641$, $p = 0.001$ and $r = 0.548$, $p = 0.007$, respectively).
Conclusion: In this study, a close relationship was demonstrated between eosinophil infiltration and nasal airflows in children having asthma and/or rhinitis monosensitised to mites. Additionally, the significant association found between FEV1 values and nasal eosinophils or PNIFs supported the close link of upper and lower airways.

Introduction

Allergic rhinitis (AR) is a common disease associated with significant morbidity. Studies have shown that as many as 10% of children and 20–30% of adolescents have AR.[1,2]

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The interaction between allergens and IgE-mast cells plays a triggering role, but the immediate phase is followed by a delayed-type inflammatory phase and, if the allergenic stimulus persists, a chronic and sustained inflammation ensues.[3] Allergen exposure activates mast cells resulting in the release of mediators and cytokines capable of inducing inflammatory cell recruitment (including eosinophils, neutrophils, and Th2 cells) and their activation at the target organ level.[4]

The hallmark of allergic inflammation is eosinophilic infiltration, as its presence in the nasal smear is suggestive of allergic aetiology.[5]

Patients with allergic rhinitis experience sneezing, nasal itching, rhinorrhoea and nasal blockage after exposure to the relevant allergen. Nasal obstruction constitutes a leading symptom and may be roughly evaluated subjectively, by the perception of the passage of air throughout the nose, and objectively, by measuring nasal airflow by rhinomanometry.[6] Allergic inflammation, mucosal congestion, and mucus hypersecretion contribute to cause nasal obstruction.[7] Allergic inflammation induces a mucosal swelling. Vasodilatation causes engorgement of sinusoidal capacitance vessels. Rhinorrhoea and increased mucus production contribute to impair nasal airflow.

Many patients have coexisting upper and lower airway disease, and estimates show that 60–78% of patients who have asthma have coexisting AR.[8]

The aim of this study was to investigate the relationship between nasal eosinophil counts and nasal airflows in children with asthma and/or rhinitis monosensitised to house dust mites.

Methods

Patients

The patients for this study were selected among those who were referred to the Pediatric Allergy and Immunology Clinic on the basis of perennial nasal and/or bronchial symptoms. Three study groups were formed: allergic rhinitis and asthma (R + A, $n = 24$); allergic rhinitis, with no asthma (R, $n = 13$); and non-allergic healthy control children (C, $n = 10$). All children in the first two groups were sensitised to only house dust mites. Allergy was assessed by skin prick tests. The children were tested with a panel of common inhalant allergens (*Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, grass mix, tree mix, mould mix, *Alternaria* species, *Cladosporium* species, eucalyptus, olive and dander) (Allergopharma, Reinbeck, Germany). A mean wheal diameter greater than 3 mm was considered positive. Methacholine challenges of patients with R were negative.[13] Controls had a negative skin prick test, and were not hyperresponsive to methacholine. Asthmatic patients fulfilled the criteria for mild persistent asthma according to GINA guidelines.[9] Diagnosis of persistent allergic rhinitis was based on criteria in ARIA[10] consensus statement.

The study was approved by the ethical committee of the University Hospital and each patient and their caregivers gave written informed consent.

Study design

The study was conducted when patients were symptomatic. The patients who met the following criteria were eligible for inclusion in the study: age between 6 and 15 years; history of asthma and/or perennial allergic rhinitis due to perennial allergen exposure for the previous 2 years; a minimum total symptom score greater than 6 at baseline, particularly with moderate-to-severe nasal obstruction.

Exclusion criteria were as follows: the presence an inhalant allergen sensitisation other than mites in skin prick tests; acute or chronic upper respiratory tract infections within 30 days of the study; anatomic nasal disorders (i.e., septum deviation); nasal polyps; use of antibiotics; use of nasal, inhaled or oral corticosteroids in the previous 4 weeks; use of antihistamines, antileukotrienes, and long-acting β_2-agonists in the previous week.

Nasal symptoms

The four symptoms of rhinitis were assessed: nasal obstruction, itching, sneezing and rhinorrhoea. Each symptom was evaluated using the 4-point scale. 0 = absent, 1 = mild (symptom was present but not troublesome), 2 = moderate (symptom was frequently troublesome but did not interfere with daily activity or sleep) and 3 = severe (symptom has interfered with daily activity or sleep). The sum of the individual symptom scores was referred to as total nasal symptom score.

Rhinomanometry

Active anterior rhinomanometry was performed according to the criteria of the Committee Report on Standardisation of Rhinomanometry.[6] A Rhinospir 165 rhinomanometer (Sibelmed, Barcelona, Spain) was used to measure nasal airflows. Patients wore a tight-fitting face mask and, with the mouth closed, breathed through one nostril. A sensor, placed to the contralateral nostril, recorded data on prenasal and postnasal pressures via airflow and pressure transducers. The rhinomanometer was connected to a personal computer; the signals of transnasal airflows and resistances were recorded in an $X-Y$ mirror image. Nasal airflow was reported as the sum of recorded airflow through the right and left nostrils in millilitres per second at a pressure difference of 150 Pa across the nasal passage.

Nasal cytology

Nasal cytological specimens were obtained by using a cytological brush as described in previous reports.[11,12] Briefly, the brush was introduced in the middle meatus of the nose and turned carefully for 360°; then, it was immediately placed in a polystyrene plastic tube containing 5 mL phosphate-buffered saline (PBS) and incubated for 30 min after shaken vigorously in the solution. The tubes were centrifuged at $400 \times g$ for 10 min. After the supernatant was discarded, differential cell count was performed on cytospins (Cytospin 4, Shandon Corp., Pittsburgh, PA, USA) stained using