Inhibition of nasal mucosal eosinophils after immunotherapy is associated with a decrease in interleukin-13 mRNA and vascular cell adhesion molecule-1 expression

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Background: Grass pollen immunotherapy is highly effective in reducing seasonal hay fever symptoms and medication requirements. Clinical improvement is accompanied by a reduction in nasal mucosal eosinophils, although the mechanism is unknown.

Methods: Nasal biopsies were taken from 37 adults before immunotherapy and during the peak pollen season following 2 years treatment. Biopsies were processed for immunohistochemistry for CCR3, adhesion molecules (intercellular adhesion molecule (ICAM)-1 and vascular cell adhesion molecule (VCAM)-1) and apoptotic cells (terminal deoxyribonucleotidyl transferase-mediated dUTP-digoxigenin nick end-labeling; TUNEL), as well as for interleukin (IL)-4 and IL-13 mRNA-positive cells. Results were compared with eosinophil numbers in the nasal mucosa.

Results: Analysis of the clinical data confirmed that the proportion of patients who showed greater than 60% improvement in symptoms (47 and 15%) and in rescue medication (79 and 10%) were significant for the immunotherapy group compared with placebo group ($P < 0.03$ and $P < 0.02$, respectively). Seasonal increases were observed for VCAM-1 expression ($P = 0.05$) and IL-13 mRNA-expressing cells ($P < 0.05$) in the placebo group, but not in the immunotherapy group. The differences for VCAM-1 expression achieved significance between groups ($P = 0.05$). There was no significant difference in either ICAM-1 expression or in the number of CCR3+ cells, TUNEL+ apoptotic cells and IL-4 mRNA-expressing cells.

Conclusion: Successful grass pollen immunotherapy was associated with inhibition of seasonal increases in nasal eosinophils, IL-13 mRNA-expressing cells and VCAM-1 expression, but no change in CCR3 expression or in the number of apoptotic cells. The reduction in eosinophils after immunotherapy may be due to suppression of eosinophil recruitment to the nasal mucosa rather than enhanced apoptosis.

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