

CORRESPONDENCE

Nasal inflammation and reversible bronchial obstruction in asymptomatic allergic children

M. A. Tosca¹, G. Ciprandi² and M. Silvestri¹

¹Pulmonary and Allergy Disease Paediatric Unit, IRCCS G.Gaslini Institute, Genoa, Italy and ²DIMI, University of Genoa-Azienda Ospedaliera Universitaria San Martino, Genoa, Italy

In their article, Amorim et al. [1] demonstrate, with a cross-sectional study in adult patients with moderate-severe asthma, a convincing association between nasal and sputum eosinophilia and a link between the former and the bronchodilator response, i.e. post-salbutamol forced expiratory volume in 1 s (FEV₁). These results are in agreement with recent studies that showed strong similarities in tissue inflammatory changes in asthma and rhinitis, further supporting the concept that upper and lower airways should be considered a unique entity influenced by common physiologic processes [2, 3]. The authors suggest that the evaluation of upper airway inflammation may reflect and provide an additional insight into the lower airway involvement and propose evaluation of nasal eosinophilia as a surrogate for sputum analysis in these patients. The possible clinical relevance of their findings is also reinforced by the demonstration that the increase in the bronchodilator response was associated with an increase in the odds of nasal eosinophilia. The complex relationship between lower airway eosinophilic inflammation and expiratory flow limitation or bronchial hyperresponsiveness is still unclear [4]. However, it is accepted that eosinophils may induce relevant airway damage, being the source of proteases and toxic reactive oxygen species, and the relevance of nasal, in addition to bronchial, allergic inflammation as the main trigger for bronchial involvement has been considered by several studies performed in allergic patients [5]. We recently evaluated a group of 40 children (24 males and 16 females, mean age 7.2 years), with persistent allergic rhinitis and a history of mild intermittent asthma, all mono-sensitized to house dust mite. All children were in a stable condition, i.e. without nasal and bronchial symptoms and without any anti-inflammatory or anti-allergic medication in the previous 4 weeks. Pulmonary function, measured at baseline and after a bronchodilation test, and nasal brushing for assessing upper airway inflammation were performed in all children. Basal spirometry [FEV₁ 91.83% of predicted (SE 1.80), FVC 92.38% of predicted (SE 1.7)] and after 200 mcg salbutamol inhalation [FEV₁ 95.79 (SE 1.92), FVC 93.67 (SE 1.82)], parameters were within normal values in the majority of the patients. The nasal eosinophil proportion (expressed as

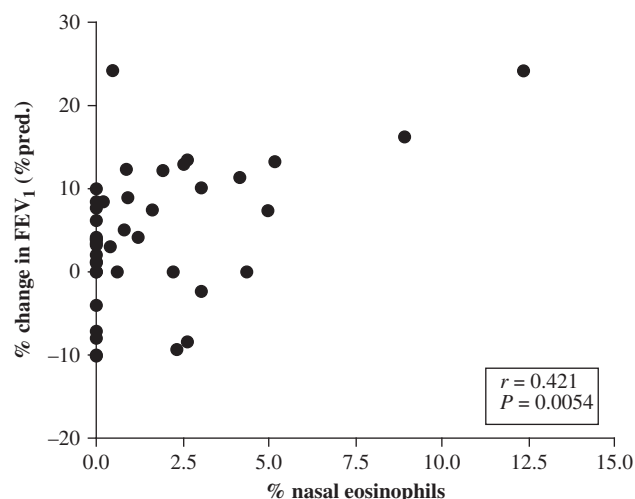


Fig. 1. Relationship between nasal eosinophils and forced expiratory volume in 1 s (FEV₁) reversibility in a group of 40 allergic children.

percentage of nasal cells, including ciliated epithelial cells, recovered by brushing) was 0.435 (SE 0–2.55) [median values (lower quartiles–upper quartiles)]. Although we detected an increase in FEV₁ values $\geq 9\%$ only in 14 children and an eosinophil % $\geq 2\%$ in 14 children, a moderate and significant relationship between nasal eosinophils and reversibility after the bronchodilation test ($r = 0.421$, $P = 0.039$) was found as shown (Fig. 1). These preliminary results demonstrate that, also under stable conditions, a positive relationship may be detected between a subclinical degree of eosinophil nasal inflammation related to ‘low-level’ mite exposure [6] and reversible bronchial obstruction, suggesting a relevant pathophysiological link between these phenomena also in asymptomatic patients.

References

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Response by Ana Luisa Godoy Fernandes

A. L. G. Fernandes

Department of Medicine, Respiratory Division, Federal University of São Paulo, São Paulo, Brazil

Thank you for the comments regarding our paper 'Nasal eosinophilia: an indicator of inflammation in asthma'. Tosca and colleagues, in their study, have also shown a significant relationship between nasal eosinophils and reversible bronchial obstruction, in allergic children.

It is crucial to maintain surveillance in airway inflammation to guide us to a better-tailored treatment. Nasal cytology is a tool that has been used for a long time to measure nasal inflammation [1]. It not only represents a local disease but also it might be able to measure inflammation of the lower airways as a surrogate.

Our study showed an agreement between nasal (recovered by blowing) and sputum eosinophilia, which was interpreted as a predictor of airway inflammation in adult asthma patients under regular treatment. It is relevant that even in stable conditions, the percentage of bronchodilator response was an independent variable associated with nasal eosinophilia [2].

Tosca and colleagues highlighted a similar association between nasal eosinophilia (recovered by brushing) and reversible bronchial obstruction, in asymptomatic children with mild or intermittent asthma. It is also in favour of the probable link between nasal eosinophilic inflammation and bronchial response. The relationship between rhinitis and bronchial hyperresponsiveness has already been described in others scenarios as in cases of positive bronchoprovocative test in allergic rhinitis patients [3].

It is noteworthy to mention that there are important differences in methodology and definitions between our studies such as the definition of nasal eosinophilia and positive bronchodilator response. Our study had different

cut-off points for nasal eosinophilia and bronchodilator response, which results in different level of evidence. We consider that the cut-off point of 2% for nasal eosinophilia is borderline (nasal brush) and 9% increasing in forced expiratory volume in 1 s (bronchial response) is near to the normal range for children [4]. Another important point is that both study populations have important differences in age, severity of disease, medicine usage, although they recognize the same connection linking inflammation to bronchial response.

It is time to test the nasal cytology normal standards, as a tool to understand airway pathophysiology and as a predictor to guide how to monitor asthma control.

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