

## LETTERS

### **Bronchial hyperresponsiveness and airway inflammation markers in nonasthmatics with allergic rhinitis**

*To the Editor:*

We read with interest the article by POŁOSA *et al.* [1] in which methacholine and adenosine monophosphate (AMP) responsiveness and induced sputum cell counts were measured in 12 subjects with allergic rhinitis due to Parietaria pollen. The subjects had no symptoms of asthma and apparently no cough. However, if a provocative concentration of methacholine causing a 20% fall in forced expiratory volume in one second  $\leq 8$  mg·mL<sup>-1</sup> by the method they use is abnormal, eight of the 12 subjects had hyperresponsiveness and might have had asthma, if asthma is defined by variable airflow limitation [2]. Such variable airflow limitation has been identified in similar subjects by abnormal diurnal variation of peak expiratory flow (PEF) or hyperresponsiveness to physical stimuli like hyperventilation of cold dry air [3]. Also, they could have had symptoms of variable airflow limitation but failed to recognize, or to admit to, these [4].

All of the rhinitic subjects had sputum eosinophilia. Are the authors sure there was no contamination with postnasal drip? Assuming there was none, if abnormal sputum cell counts are used to diagnose bronchitis, they all had an eosinophilic bronchitis. Such an eosinophilic bronchitis has previously been reported in subjects with asymptomatic methacholine hyperresponsiveness [5] and in patients with a chronic cough without hyperresponsiveness to methacholine or AMP [6–8]. Treatment with inhaled steroid or avoidance of the cause [9] can reverse the eosinophilia and improve the degree of airway responsiveness.

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### **Diagnostic utility of eosinophils in pleural fluid**

*To the Editor:*

On the basis of their study MARTÍNEZ-GARCÍA *et al.* [1] have determined that pleural eosinophilia at initial thoracentesis cannot be considered as a predictor of an underlying benign disease.

We would like to report the result of our own series. We studied the prevalence of eosinophilia in 720 consecutive samples of pleural fluid. Eosinophilic pleural effusions were identified in 50 patients. Malignant underlying conditions were diagnosed in 12 cases and benign aetiologies were found in 38.

There was no difference in the prevalence between eosinophilic and noneosinophilic effusions according to the different diagnoses except for malignant (noneosinophilic 39.7%, eosinophilic 24%;  $p < 0.04$ ), parapneumonic (18.6% versus 34%;  $p < 0.001$ ) and traumatic effusions (2.4% versus 18%;  $p < 0.001$ ).

Although the presence of eosinophilia lessens the likelihood of malignancy in our experience, we share the opinion of MARTÍNEZ-GARCÍA *et al.* [1] that pleural eosinophilia cannot be considered as a predictor of a benign disorder. The high prevalence of malignant pleural effusions in our series marks this disorder as a common cause of pleural eosinophilia.

We conclude that the presence of pleural eosinophilia should not deter clinicians from pursuing malignancy in case of clinical suspicion.

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