CASE REPORT

Occupational eosinophilic bronchitis in a foundry worker exposed to isocyanate and a baker exposed to flour

Fabio Di Stefano, Luca Di Giampaolo, Nicola Verna, Mario Di Gioacchino

Eosinophilic bronchitis without asthma may occur as a consequence of occupational exposure. The cases of a foundry worker and a baker who developed symptoms, respectively, due to exposure to isocyanate and flour, are reported. Cough was not associated with variable airflow obstruction or with airway hyper-responsiveness but was responsive to inhaled corticosteroids. The eosinophilia detectable in their sputum was causally related to the occupational exposure in the workplace. The examination of induced sputum should be used in addition to the objective monitoring of lung function for workers who have asthma-like symptoms in an occupational setting.

CASE REPORT 1

A 44-year-old man had worked in a foundry for 8 years. He used to smoke 10 cigarettes daily and had no history of atopy and asthma. He described his job as making cores and, from the safety data sheet he gave us, it was apparent that he was exposed to methylene diphenyl isocyanate (MDI). He had never had respiratory symptoms before the previous 6 months, when he started having a non-productive chronic cough without wheezing or dyspnoea. The cough worsened at work and waned during holidays. He was prescribed antiinflammatory medication by his general practitioner, with a poor response, and was not receiving any medication for asthma. Physical examination and a chest radiograph (prescribed by the general practitioner 2 months before) were normal. Peripheral blood count was normal. Table 1 shows the skin and blood tests for allergy, lung function and cellularity of sputum induced after the work shift. The methacholine challenge was normal at a maximal dose–response curve (provocative dose inducing a 20% fall in FEV1 >3200 μg). Table 1 shows the percentages of non-squamous epithelial cells in induced sputum.

CASE REPORT 2

A 41-year-old male baker, non-smoker, had been exposed to flour for 10 years. In the previous 2 years he had developed a non-productive chronic cough without wheezing or dyspnoea. The cough worsened at work and was very pronounced during holidays. He was prescribed antiinflammatory medication by his general practitioner, with a poor response, and was not receiving any medication for asthma. Physical examination and a chest radiograph (prescribed by the general practitioner 2 months before) were normal. Peripheral blood count was normal. Table 2 shows the skin and blood tests for allergy, lung function and cellularity of sputum induced after the work shift. The methacholine challenge was normal at a maximal dose–response curve (provocative dose inducing a 20% fall in FEV1 >3200 μg). Table 2 shows the percentages of non-squamous epithelial cells in induced sputum.

Abbreviations: FEV1, forced expiratory volume in 1 s; MDI, methylene diphenyl isocyanate
induction was performed before methacholine challenge. The methacholine challenge was normal at a maximal dose–response curve (provocative dose inducing a 20% fall in FEV1 >3200 μg). Table 2 shows the percentages of non-squamous epithelial cells in induced sputum.

### DISCUSSION
Occupational exposure to isocyanate and flour was the cause of eosinophilic bronchitis in cases 1 and 2, respectively. The condition was characterised by work-related changes in sputum eosinophils that were significant and reproducible. Causative...
agents of eosinophilic bronchitis in the workplace have rarely been established. To date, eosinophil bronchitis has been causally related to occupational agents only in the two patients described here and in two other published case reports.\textsuperscript{4, 5} Challenge exposure to latex gloves in a nurse\textsuperscript{4} and acrylates in a worker\textsuperscript{5} resulted in a marked increase in sputum eosinophilia in the absence of airflow obstruction and bronchial hyper-responsiveness. In a cross-sectional health survey conducted on a mushroom farm,\textsuperscript{3} eosinophilic bronchitis was among the causes of chronic cough but a causal relationship between exposure to a specific occupational agent and sputum eosinophilia was not reproducibly demonstrated with specific occupational bronchial challenge.

It is not known why these patients do not have airway hyper-responsiveness despite the eosinophilic bronchial inflammation, nor whether they will progress to typical occupational asthma. A recent prospective follow-up study of patients with eosinophilic bronchitis not related to occupational exposure showed that recurrence of the disease, after an initial remission obtained with inhaled corticosteroids, was associated in some cases with the development of asthma or chronic airflow obstruction.\textsuperscript{6} Several hypotheses have been proposed to explain the eosinophilic bronchitis inflammatory response which does not cause bronchial hyper-responsiveness.\textsuperscript{7, 8} To examine the missing link between airway inflammation and airway hyper-responsiveness, the intricate relationships between inflammatory cells (eosinophils, lymphocytes, neutrophils), resident cells (mast cells, epithelial cells, macrophages), inflammatory mediators, cytokines and neuropeptides (substance P, neurokinin A, calcitonin gene-related peptide) secreted by the bronchial sensory nerves and bronchial smooth muscle cells need to be sustained. Although eosinophilic bronchitis does not meet the current definition of asthma, it should be regarded as an occupationally induced condition when work-related changes in sputum eosinophils are significant and reproducible. The examination of induced sputum should be part of the diagnostic algorithm for workers who have asthma-like symptoms in an occupational setting. It is a further diagnostic tool which complements the objective monitoring of lung function during periods at work and away from work, as well as before and after specific bronchial challenges with occupational agents.

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Competing interests: None declared.

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Received 21 April 2005
Accepted 30 June 2005
Published Online First 29 July 2005

\textbf{REFERENCES}

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Thorax 2007 62: 368-370 originally published online July 29, 2005
doi: 10.1136/thx.2005.045666

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