CASE REPORT

Occupational eosinophilic bronchitis without asthma due to chloramine exposure

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Abstract

A case is discussed of eosinophilic bronchial inflammation without asthma due to chloramine T (CLT) exposure in a nurse. She reported a non-productive chronic cough on contact with CLT during workshifts. She had negative results of skin prick testing to CLT. However, sensitisation to CLT was confirmed by the presence of specific anti-chloramine IgE. Airway responsiveness to histamine was normal before and after CLT challenge. Eosinophil proportion in sputum was increased at 6 and 24 h after CLT challenge.

Key words

Chloramine; induced sputum; occupational eosinophilic bronchitis.

Introduction

The term eosinophilic bronchial inflammation without asthma is used by some authors to describe the condition suffered by patients with chronic respiratory symptoms suggestive of asthma, but with a normal lung function and signs of eosinophilic bronchial inflammation [1]. The causative agents involved in the development of this condition have rarely been described [2,3].

The present study is a case report of eosinophilic bronchitis related to workplace exposure to chloramine. Chloramine, the sodium salt of N-chloro-p-toluene sulphonamide [chloramine T (CLT)] or N-chloro-p-benzene sulphonamide (chloramine B) is used as a disinfectant in hospitals [4].

Case study

A 61-year-old non-smoking woman had been working as a hospital nurse since 1985. Ten years after she had started working, she developed a non-productive chronic cough without wheezing or dyspnoea. She cleaned the instruments with a CLT-containing disinfectant throughout this period. The data on the patient’s exposure were provided by the State Sanitary Inspectorate. She did not report any nasal, eye or skin symptoms at the workplace. She also substantially improved at weekends, as confirmed by the medical records supplied to the Nofer Institute by her general practitioner (GP).

Her family history was negative for allergies and asthma. The patient had been treated with an antitussive medication, with a poor response, and was not receiving any treatment for asthma. After she was diagnosed with CLT respiratory allergy, CLT exposure was discontinued and she was treated with budesonide (800 µg per day). After 1 month, her cough markedly improved, as documented on a symptom diary card.

The study was designed as a biphasic, crossover, single blind trial. In phase I, the subject was challenged with 0.9% saline as placebo and 7 days later, an allergen challenge with 2% chloramine was performed. The test consisted of painting chloramine solution onto a 2 m² piece of cardboard in a challenge chamber for 15 min.

Skin prick tests (SPT) were performed using common allergens (Allergopharma, Reibek, Germany) and CLT (0.1–1–10 mg/ml). The evaluation of specific serum IgE to CLT—k 85 was made using the UNI-CAP system (Pharmacia Diagnostics, Sweden). The results were expressed quantitatively in kU/l and considered positive at values higher than 0.35 kU/l.

We used a sputum processing method modified from the one described by Fahy et al. [5] and validated by Pizzichini et al. [6]. Induced sputum was obtained before, and 6 and 24 h after the challenges.

Bronchial response was assessed through serial monitoring of Forced Expiratory Volume in 1 s (FEV₁) before and 5, 30 min, 1, 2, 4, 5 and 24 h after the provocations. Histamine challenge was performed according to Cockroft et al. [7] before and 24 h after the chloramines and placebo challenges.

Results

The results of physical examination and chest radiography were normal. SPT to common and occupational
allergens were negative. Total serum IgE was 29 kU/l. Sensitization to CLT was confirmed by the presence of specific anti-chloramine IgE (class 2).

Serial peak expiratory flow (PEF) monitoring showed no significant daily variability at work or away from work, as confirmed by the medical records provided by the GP.

Spirometry revealed a FEV₁/FVC ratio of 78%, with no changes after salbutamol inhalation.

The patient developed a cough during and after CLT challenge, becoming more severe at 6 h after the challenge and lasting up to 24 h.

Baseline spirometry did not show any airway obstruction after CLT provocation (Figure 1). PEF fluctuations were less than 10% throughout a 24 h observation period.

The non-specific bronchial hyperreactivity (BHR) test with histamine was negative (PC₂₀ of 11 g/ml) before and after specific inhalation challenge (SIC) (PC₂₀ of 10.8 mg/ml). The eosinophil proportion in sputum was increased at both 6 and 24 h after SIC (Table 1).

Placebo provocation did not induce any changes in the clinical state, spirometric parameters or cellular findings in induced sputum (Table 1).

Discussion

The results of our study indicate that the patient developed eosinophilic bronchitis caused by CLT exposure at work. The presenting complaint was chronic dry cough since 1995, especially on contact with chloramine. Chronic cough seems to be the main symptom reported by patients with asthma-like inflammation [8].

Our patient was non-atopic and had negative results for SPT to CLT, but we found specific anti-chloramine IgE antibody in her serum. Atopy is not a well-documented risk factor for the development of allergy to CLT [9]. Palczynski et al. [9] found specific anti-chloramine IgE antibodies, using the Uni-Cap system, in four out of six subjects with CLT asthma. He noted a very high correlation between clinical symptoms and specific IgE antibodies in serum.

The SIC with CLT induced a less than 15% fall in FEV₁. The arbitrarily accepted criterion for a positive test is a 20% fall in the FEV₁, compared to the baseline value, after exposure to the causative agent [10].

Airway responsiveness to histamine was normal both before and after SIC. In our study, PC₂₀ higher than 8 mg/ml virtually rules out the presence of current symptomatic asthma. One should bear in mind that the BHR is not always apparent in patients with occupational asthma [11].

We found PEF fluctuations of less than 10% throughout a 24 h observation period after SIC. Serial PEF monitoring showed no evidence of asthma in a symptomatic subject either at work or after SIC, the diagnosis of occupational asthma is unlikely [12].

SIC combined with a morphological analysis of biological fluids appears to be a very useful tool for differential diagnosis of occupational airway sensitization.

CLT challenge resulted in an increase in eosinophil proportion in induced sputum, up to 8% at 6 h and 11% at 24 h, respectively. It is postulated that the limit for diagnosis of eosinophilic bronchitis should be 3% of eosinophils in the sputum [13]. The reference values and distribution of these cells in induced sputum of healthy subjects are not well-defined, however in Belda’s study, the proportion of eosinophils was 0.4%, and of neutrophils 37.5% [14]. The patient's cough markedly improved after she discontinued CLT exposure.
We suggest that once this diagnosis has been established, the patient’s clinical state and spirometric data should be monitored by a physician and the patient should be advised about the risk of developing bronchial asthma and counselled regarding their work. Further research is necessary to elucidate the natural history of this disease.

References


