

Letter to the Editor

Usefulness of induced sputum in investigating occupational asthma with normal responsiveness to methacholine: A case report

To the Editor:

Current international guidelines suggest stopping investigations of occupational asthma (OA) in the case of normal nonspecific bronchial hyperresponsiveness (NSBH) in a subject currently exposed to the suspected agent(s) in the workplace.¹ Noninvasive markers of inflammation have been increasingly used in the diagnosis and management of obstructive airway diseases over the past 15 years. Induced sputum (IS) analysis is a noninvasive method for studying airway inflammation. The monitoring of sputum eosinophil counts has been shown to be useful in the investigation of OA.^{2,3}

We report the case of a 33-year-old man, an exsmoker, who was referred to our allergy unit for evaluation of work-related respiratory symptoms. He had been working as a stock handler in a food industry for 12 years. Six months before our investigations, he started a job as a technician in an electric industry. Dyspnea, productive cough, chest tightness, and wheezing appeared a few days after starting this work, with significant improvement during weekends and holidays. Respiratory symptoms appeared a few hours after starting work, persisting at home during the evening. The worksheets showed exposure to cyanoacrylate through the inhalation route at the workplace, without exposure to any other known sensitizer.

The patient underwent the common diagnostic pathway of OA.¹ Skin prick test responses to common aeroallergens were positive for house dust mites, several pollens, latex, and cat's epithelium (although he did not own a cat). These findings were confirmed based on specific IgE count. The result of a reversibility test with albuterol was negative. Peak expiratory flow (PEF) monitoring and methacholine (Mch) challenge during periods at work and off work were in the normal range.

According to current diagnostic algorithms for the diagnosis of OA,¹ investigations should have been stopped at this step (normal Mch assessment at work), but because of the

suggestive occupational history, the patient was submitted to IS challenge.³ IS was carried out 2 days after the last exposure at the workplace and showed bronchial eosinophilia (27.7%; normal value, <2%).² Therefore we performed a specific inhalation challenge (SIC) with cyanoacrylate. The SIC was carried out 3 months later, when the patient had not been exposed at the workplace for 2 months. On day 1, the patient was exposed to isobutyl acetate (control day). Twenty-four hours later, an IS test was carried out, showing 11.7% eosinophils. On day 3, SIC with cyanoacrylate was carried out by using the occupational method.^{4,5}

The patient was exposed to cyanoacrylate by painting a paper surface with the cyanoacrylate glue (occupational-like method). A total of 8 mg of cyanoacrylate was used, which, in an inhalation chamber of 7.46 m³, corresponded to the time-weighted average limit (1 mg/m³). The subject was exposed in the inhalation chamber for 30 minutes.

Both on the control day and during specific exposure, spirometric and PEF results were measured before and 5, 15, 30, and 60 minutes after the end of exposure, then hourly for 7 hours and after 24 hours (Fig 1). A late response with a maximum 16% FEV₁ decrease at 300 minutes and a 25% PEF decrease 10 hours after the end of exposure was registered. At the same time points, the patient reported respiratory symptoms (dyspnea, productive cough, chest tightness, and wheezing). The day after SIC, the results of Mch challenge were negative (FEV₁ PD₂₀ >2376 µg before and after SIC), whereas IS showed an increase in total cellular count (2640 cells/mg after SIC vs 1320 cells/mg before SIC) and eosinophilia (54.5%), as shown in Fig 2. The serum eosinophil cationic protein level was 26.4 ng/mL before and 30.7 ng/mL after the specific challenge.

The decrease in FEV₁ and PEF after SIC and the reproducibility of respiratory symptoms referred at work after cyanoacrylate exposure confirmed the diagnosis of OA caused by cyanoacrylate. The diagnosis was supported by the results of IS.⁶

The case we have described underlines the importance of assessing airway inflammation by means of IS in the diagnostic pathway of patients with OA when NSBH to Mch is normal before

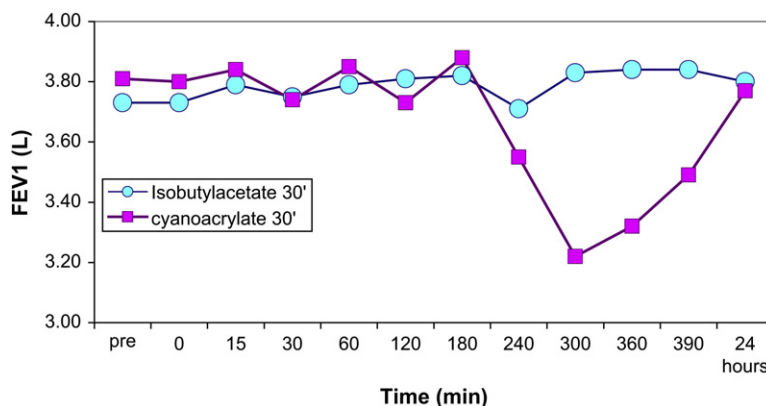


FIG 1. Time course of FEV₁ (in liters) during SICs.

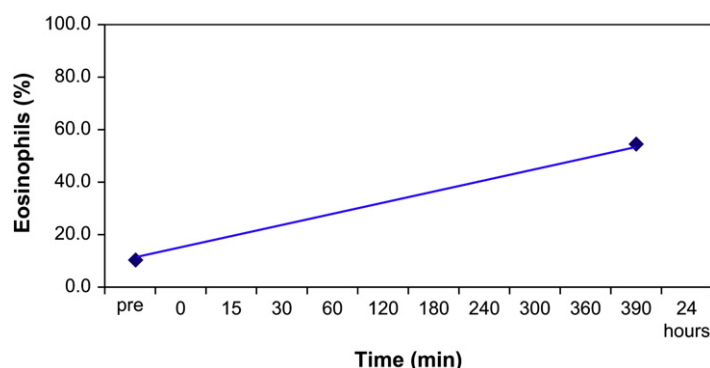


FIG 2. Time course of eosinophils during SICs.

excluding the possibility of OA. In our case the latency period from onset of exposure and onset of symptoms was relatively short for sensitization, but this finding has previously been reported for the same agent,⁷ underlining the high sensitizing properties of cyanoacrylate.

We suggest that airway inflammation is an early marker of OA that is more sensitive than NSBH, especially when agents with high sensitizing properties are involved. The IS test should be included in the diagnostic pathway of OA.

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