

Hypersensitivity to wood dust

T. A. A. Skovsted*, V. Schlünssen, I. Schaumburg, P. Wang, P. S. Skov

Key words: asthma; hypersensitivity; wood dust.

• **WORKERS** exposed to wood dust have an increased risk of suffering from bronchitis, asthmatic symptoms, or impairment of lung function (1). Exposure to different kinds of wood dust may cause asthma and rhinitis (2). Few attempts have been made to document type I allergy to dust from pine wood, which is frequently used in the Danish furniture industry (3, 4).

A 49-year-old man gradually developed work-related episodes of wheezing during his 4-year employment in a furniture factory using mainly pine wood. During the last year, he also suffered from nasal congestion and rhinorrhea. He was a nonsmoker without history of allergy and with no respiratory symptoms prior to his exposure to wood dust.

There was no eosinophilia, and Phadiatop and total IgE were not increased (Pharmacia CAP System, Sweden). Forced basal spirometry and radiographic examination of the lungs were normal. Monitoring of peak expiratory flow rate (PEFR) was done during working days and days off work. The patient measured PEFR every 3 h except during sleep.

On the suspicion of a type I allergic reaction to wood dust, the patient was tested by beech, oak, and pine extracts by skin

prick test (SPT) and basophil histamine release (HR).

Wood from pine, beech, and oak was extracted overnight in PBS, pH 7.6. The wood extracts were concentrated 100 times in a high-molecular-weight extract (>10 kDa cutoff) by ultrafiltration. Low-molecular-weight extracts (<10 kDa) consisted of run-through from the ultrafiltration. For the SPT, the wood-extract stock solution was filtered under sterile conditions through a 0.22-mm filter and mixed with glycerol to a 50% (v/v) final concentration. The sterile quality of the wood extract was tested and confirmed for all the extracts of pine, beech, and oak.

Histamine released from peripheral blood cells was measured by the glass fiber method (RefLab, Copenhagen, Denmark). Diluted extract was used in the following final concentrations: 1:10, 1:35, 1:100, 1:350, 1:1000, and 1:3500 v/v. The histamine content in the wells was analyzed in duplicate by the OPT method (5). A histamine release of >15 ng/ml washed blood was considered positive.

To exclude unspecific histamine release by the different extracts, we performed identical release experiments on blood from two healthy, nonallergic individuals.

SPT were carried out in duplicates with extracts of wood dust from beech, pine, and oak and 13 common inhalation allergens including pollen, dust mites, epithelia, and storage mites (ALK-Abelló, Denmark). The SPT were carried out for high-molecular (>10 kDa) as well as low-molecular (<10 kDa) wood-dust extracts. The positive control was histamine hydrochloride 10 mg/ml, and the negative control was PBS in 50% glycerol. A reaction was considered

positive when the mean wheal was at least 3 mm.

Fluctuations in PEFR above 200 l/min were measured, consistent with the diagnosis of asthma. No difference in average PEFR was seen on working days compared to days off work, but the daily fluctuation in PEFR was greater on working days (37% compared to 25%). SPT with 13 common allergens were negative. No positive reactions were found to any of the low-molecular (<10 kDa) wood-dust extracts in the SPT and HR test.

SPT with high-molecular >10 kDa extracts showed a positive result for pine and beech. SPT to oak was negative. The HR test was positive to pine with a histamine peak at 20 ng/ml. No positive reaction was observed in the two controls to any of the wood extracts in either the SPT or the HR test.

This case report indicates that an IgE-mediated allergic reaction might explain some of the respiratory reactions to wood dust. After he left the wood industry, the patient's symptoms, as well as his need for medication, decreased. There seems to be a divergence between the results of the SPT and the HR test. This difference could be due to the fact that the SPT is more sensitive than the HR test.

* Department of Clinical Biochemistry
Slagelse Central Hospital
4200 Slagelse
Denmark

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Rofecoxib should be tried in NSAID hypersensitivity¹

E. Enrique, A. Cisteró-Bahima*,
M. M. San Miguel-Moncín, R. Alonso

Key words: COX-2 inhibitors; nimesulide; NSAIDs hypersensitivity; rofecoxib.

● NONSTEROIDAL anti-inflammatory drugs (NSAIDs) are frequently involved in nonallergic reactions, and constitute a major problem, as these drugs are commonly used in medical treatment (1).

Valid alternatives for NSAID-sensitive patients present a challenge to allergists. Nimesulide has recently been reported to be a useful alternative choice in these patients (2), being tolerated in 85.4% of those with hypersensitivity to NSAIDs (3). When nimesulide also is not tolerated, few drug alternatives are available. Two patients with hypersensitivity to NSAIDs and nimesulide

who showed good tolerance to rofecoxib are reported.

Case 1. A 32-year-old woman with a history of dust-mite rhinitis and allergy to fruits suffered several episodes of eyelid angioedema after taking acetylsalicylic acid (ASA), ibuprofen, and naproxyn. An oral challenge test with nimesulide was performed, and eyelid angioedema appeared a few minutes after a dose of 100 mg had been administered. Five months later, a single-blind oral challenge test to rofecoxib 25 mg b.i.d. for 3 days was performed with good tolerance.

Case 2. A 29-year-old man with a history of cholinergic urticaria suffered several episodes of generalized urticaria and lip angioedema after ingesting ASA and metimazol. He also reported a similar episode on the third day of a 100-mg b.i.d. course of nimesulide. An oral challenge test with nimesulide was performed and elicited the same response 12 h after the 100-mg dose. The same oral challenge test at the same doses as used in case 1 was performed with good tolerance.

Although the mechanisms implicated in hypersensitivity to NSAIDs remain unknown, most adverse responses are related to cyclooxygenase (COX) inhibition, particularly the COX-1 isoenzyme (1).

Currently available NSAIDs are more potent inhibitors of COX-1 than COX-2, and provoke or exacerbate bronchial asthma, rhinitis, and urticaria/angioedema in sensitive patients. Nevertheless, some NSAIDs, such as nimesulide, preferentially inhibit COX-2 (3), and others, such as rofecoxib and celecoxib, are truly selective COX-2 inhibitors (4, 5). Thus, as these drugs reduce inflammation with lower inhibition of the production of physiologically active eicosanoids, the risk of side-effects decreases (1).

To our knowledge, these are the first cases to be reported, and they suggest that rofecoxib may be an alternative in NSAID-

sensitive patients who also do not tolerate nimesulide. Further oral challenge tests to rofecoxib in nimesulide-sensitive patients are required.

* Paseo de la Bonanova 69
22 planta
E-08017 Barcelona
Spain
Tel. 34-93-417 80 00
Fax: 34-93-417 19 95
E-mail: acb01b@nacom.es

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U-EPX and severity of asthma

K. Reichenberg

Key words: asthma; child; peak expiratory flow rate; quality of life; severity of illness index; urinary eosinophil protein X.

● THE relationship between asthma severity and urinary concentration of eosinophil