

Occupational asthma caused by iroko wood

J. AZOFRA¹ & J. M. OLAGUIBEL²

¹Allergy Unit, H. Ntra. Sra. de Covadonga, Oviedo, and ²Allergy Department, C. S. La Fe, Valencia, Spain

We report on a patient with occupational asthma caused by iroko. BPT induced an early and late response with a long recovery period, and absence of inhibition on pretreatment with cromoglycate and beclomethasone. Three months after leaving his job, the patient was symptom-free, but non-specific bronchial hyperreactivity was still demonstrated.

Key words: iroko wood; asthma

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Occupational asthma has been described after exposure to different types of wood dusts, such as western red cedar (1), African maple (7), iroko (12), ramin (8, 9), and mahogany (13).

With occupational agents, a bronchial provocation test (BPT), simulating in the laboratory the conditions met at work, is a valuable diagnostic tool. In this challenge, three types of response have been described: early, late, and a combination of the two.

This article describes a patient with occupational asthma caused by iroko wood dust. BPT produced a dual response with a long recovery period, and showed the failure of both cromoglycate and beclomethasone in the prevention of both types of responses to the test.

CASE REPORT

A 28-year-old asymptomatic non-smoker, male carpenter, began working in August 1985 with iroko wood. Five months later he had episodes of cough, wheezing and dyspnea as well as nasal and ocular itching, sneezing and rhinorrhea and each time he was exposed to iroko dust. On seven occasions, coinciding with work days, he required emergency assistance owing to dyspnea. During weekends the symptoms subsided, and after 3 months off work, he was asymptomatic.

A physical examination at this time was normal. White blood cell count was 5,600/mm³, with 3% eosinophils. Chest X-ray and spirometry were normal. Total IgE was 356 IU/ml.

Three months after work avoidance, a bronchial provocation test with methacholine, using the 2 min tidal breathing method with a DeVilbiss 646 nebulizer (output 0.20 ml/min) showed a PC₂₀ (FEV₁) of 4 mg/ml. Skin prick tests with a battery of 30 antigens, including molds, mites, pollens, foods, and cat and dog epithelia were negative.

A skin prick test with an extract of iroko wood (Bencard; Betchworth, Surrey, UK) was also negative. Skin prick and intradermal tests with our own iroko wood dust extract (10% v/w in PBS) were also negative.

The patient being asymptomatic and treatment-free after 3 months away from work, four BPT were done at intervals of at least 1 week (Fig. 1).

First BPT: The patient was challenged 3 min with iroko wood dust, tipping it from one tray into another. PEF was recorded as shown in Fig. 1: the best of three measurements was recorded. A $\geq 25\%$ decrease in PEF from baseline on this day was considered positive.

In the first BPT, PEF showed a decrease of 29% at 15 min, with a further decrease of 34% at 9 h, and a slow return to baseline in the next 4 days.

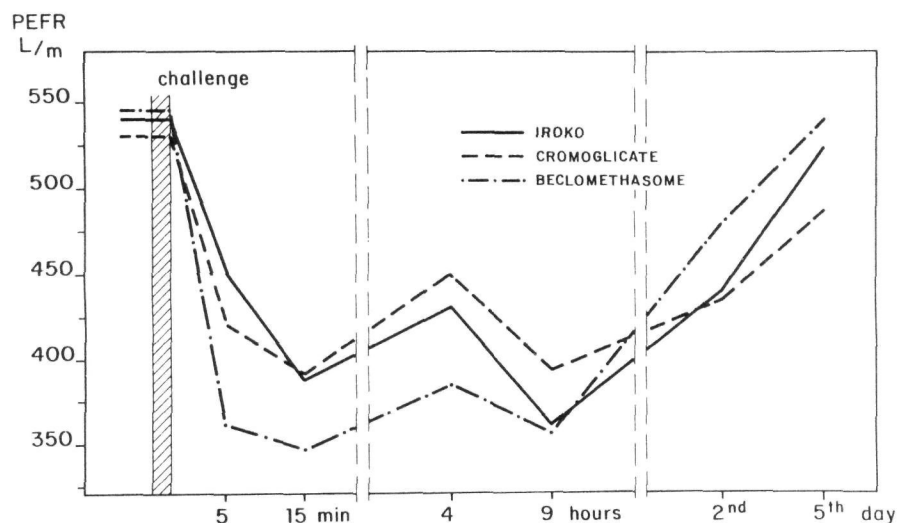


Fig. 1. PEF recordings.

Second BPT: The same as described above, but with a premedication of 40 mg cromoglycate (spinhaler) 10 min before the challenge. PEF showed a decrease of 27% at 15 min and 26% at 9 h. There was again a slow return to baseline within the next 4 days.

Third BPT: With the same method, but the patient was given 300 µg beclomethasone (MDI), 30 min before challenge. Again a significant reduction in PEF was noted: a decrease of 40% at 15 min, and of 29% at 9 h. The recovery pattern was similar to the previous BPTs.

Fourth BPT: A similar protocol but with lactose powder instead of iroko wood dust. There was no modification of PEF (maximum decrease 8% occurring on the following day).

In all cases, on the day following challenge the PEF was recorded three times daily (each time recording the best of three), and the mean is as shown in Fig. 1.

As controls, three unexposed asthmatic patients were provoked in the same way with iroko wood dust, and showed no reactions in the next hours or days.

DISCUSSION

We describe a patient with rhinitis and asthma associated with work exposure to iroko wood dust.

Data from clinical history, BPT and the fact that the patient become asymptomatic after

leaving the job, seem to confirm the diagnosis of occupational asthma. The non-irritating nature of iroko wood dust was demonstrated by the negative BPTs in three non-related asthmatic patients.

Pickering et al. (12) described a patient with iroko wood related asthma. After BPT, their patient showed only a late response. Their patient was also non-atopic, but in contrast to our patient, continued to suffer mild symptoms even after he had stopped working. This patient had been working with iroko for 12 months. Colas et al. (5) also described a patient with iroko asthma showing an early and late response on BPT. No data are given on development.

Our patient reacted to three challenges with the same dual pattern, thus arguing against a toxic or irritant mechanism: an early response (decrease at 15 min), a partial recovery (at 4 h), and a later response (at 9 h) with a slow return to baseline within 4 days.

As indicated by Cockcroft in a recent review (3), challenges with occupational agents, compared with classic allergens, show more frequent and variable late reactions, occasionally with a longer period of recovery, as exemplified in our case.

Cromoglycate, given in a dose of 40 mg prior to the BPT, was unable to inhibit either the early or the late response. In the case cited by Pickering (12), cromoglycate before provocation did not inhibit the late response. These results

are contrary to the general assessment that cromoglycate usually abrogates the early and late responses (11), although as expected, this is not a universal finding (6). When pretreating the patient with beclomethasone dipropionate (300 µg) 30 min before challenge, there was no inhibition of the response. However, inhibition of the late response has been demonstrated with beclomethasone at a similar dose (200 µg) (4, 10). We could not explain why both pretreatment drugs failed to inhibit the early and late responses in our patient, other than that this type of effect is not expected to occur in all cases. However, it is possible that a more permanent prophylactic treatment might prevent provoked asthma attacks. In this regard, it is not possible to draw any conclusions about pathogenesis, granted that both drugs, cromoglycate and beclomethasone, are able to prevent responses irrespective of the demonstration of an IgE mechanism, as it occurs in some cases of occupational asthma where there is no conclusive proof of its being IgE-mediated (2).

In our patient, we could not demonstrate an IgE mechanism, as illustrated by the negative skin tests to iroko wood extracts, although we are aware that it does not exclude an IgE mediated disease, considering the difficulty in obtaining good quality and standardized occupational allergens. The same result was obtained by Colas in the skin test (5). However, a positive prick test was described in the Pickering report (12).

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Address:

J. Azofra Garcia

Muñoz Degrain 20, E-C

33007, Oviedo

Spain

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