

Brief communications

Occupational asthma caused by imbuia wood dust

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As the patterns of occupational asthma change in parallel with the changing nature of industrialization, exotic wood dusts are being incriminated in the causation of this disease.¹ We describe another wood species, namely imbuia (Brazilian walnut), or *Phoebe porosa* (family: Lauraceae), as a cause of asthma in a joinery worker. Imbuia wood is imported by the local woodworking industry from South America for the production of furniture. To our knowledge, this is the first case published.

CASE REPORT

A 43-year-old non-smoking woman was first seen with a 4-month history of unproductive cough and chest tightness associated with wheezing and rhinitis. Her symptoms were precipitated by exposure to imbuia wood dust at work. Treatment with an inhaled β_2 -agonist and a steroid had partially relieved her symptoms. No personal or familial history of atopy was present. The only physical finding was a scattered wheeze.

The patient had been a hand sander in the furniture industry for 10 years, and in this particular joinery for 4 years. The joinery produced and assembled wood panels from chipboard (bonded wood chips or fibers). The process began with the application of veneer of various wood types (imbuia, sapelli, meranti, oak, or pine) to the chipboard, with urea-formaldehyde resin as an adhesive. The panels were then passed through an enclosed sanding machine, a cutting machine, and an assembly station. Thereafter, the wood was sandpapered by the patient; it was then sprayed in a booth where a paint lacquer and an acid catalyst were used, before it was returned to her for re-sanding. Although the patient used a conventional

Abbreviations used

FVC: Forced vital capacity
PEFR: Peak expiratory flow rate

oronasal polypropylene face mask, she reported that the dust penetrated the mask.

The chest roentgenogram was normal, with no evidence of hyperinflation or infiltrates. Spirometry showed marked obstruction and a brisk response to bronchodilator. Initial values were: FEV₁, 1.40 L (52% of predicted value); forced vital capacity (FVC), 2.02 L (60% of predicted value); FEV₁/FVC ratio, 69%; and peak expiratory flow rate (PEFR), 191 L/min (58% of predicted value). The FEV₁ improved to 1.92 L (37% increase), and the FVC to 2.59 L (28% increase) after inhalation of a β_2 -agonist.

Serial PEFR measurements over a 2-week period showed substantial variability (between 320 and 440 L/min) with no specific work-related pattern.

The complete blood count demonstrated no eosinophilia, and intradermal tests showed no positive response to any of a battery of 10 common aeroallergens (Hollister-Stier, Spokane, Wash.).

Precipitating IgG antibodies to ethanol (25% concentration) but not aqueous extracts of the imbuia wood dust were detected by using 1% agarose Ouchterlony gels and concentrated serum from the patient (Fig. 1). The gels were stained with 1% Coomassie Brilliant Blue, and on counterstaining two bands of precipitating antibodies to the imbuia wood proteins were identified. None of the eight other workers who were similarly exposed in the sanding operation at the joinery but were free of symptoms demonstrated IgG antibodies to the wood extract. Furthermore, no specific IgE antibodies to imbuia antigens were detected with polyacrylamide gel electrophoresis and Western blotting with anti-IgE monoclonal antibodies.

The patient underwent bronchial challenge to meranti wood dust as a control, which was achieved by sieving the wood shavings in a manner simulating the workplace exposure. No significant response was elicited (Fig. 2). Two weeks later, on challenge with imbuia

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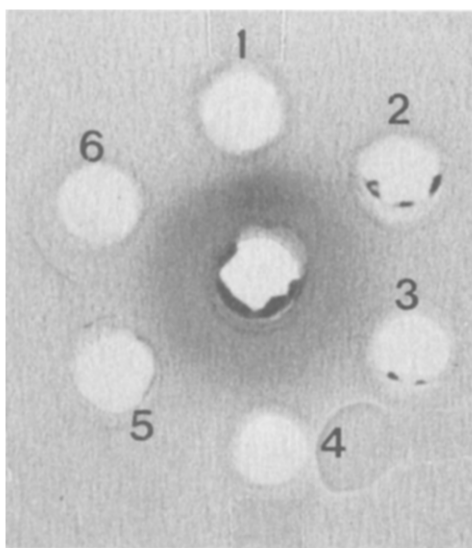


FIG. 1. Ouchterlony gel containing six wells of increasing dilutions of the patient's serum ranging from neat (well 1) to 1:32 dilutions (well 6). The central well contained concentrated imbuia wood extract. Two bands of precipitating antibodies are visible between neat serum (well 1) and the central well. They did not occur with dilutions of the serum. No precipitating bands were visible with identical Ouchterlony gels with sera from eight workers who were free of symptoms.

wood dust, she demonstrated an immediate bronchospastic response (Fig. 2). Her FEV₁ declined from 1.89 to 1.50 L (a 21% fall) within 1 hour of exposure, and to 1.26 L (a 33% fall from baseline) 3 hours later. She regained her pre-exposure level within 1 hour after nebulization with a β_2 -agonist and went home after a 24-hour period of PEFR monitoring without showing a late response.

The patient's condition improved considerably after she elected to quit her job. Spirometry 2 months later showed a 43% improvement in FEV₁ over the value when the patient was first seen (FEV₁ from 1.40 to 2.00 L, FVC from 2.02 to 2.88 L, PEFR from 191 to 350 L/min). At follow-up 18 months later, her asthma persisted but her FEV₁ remained stable during maintenance therapy (FEV₁, 1.92 L; FVC, 2.77 L; PEFR, 322 L/min). She was still unemployed.

DISCUSSION

Various studies have documented occupational asthma caused by exotic wood dusts.¹ There have been no reports of asthma caused by imbuia wood (Brazilian walnut), although asthma caused by Central American walnut (*Juglans olanchana*), a member of a different family, has been described.² This report describes a case of occupational asthma in a nonatopic joinery worker exposed to imbuia wood dust for 4 years.

The pathogenesis of bronchial hyperreactivity induced by wood dust exposure is poorly understood. It has been suggested that the presence of alkaloids, acids, or other natural constituents in the wood dust are responsible.¹ The reaction may also be due to the interactive effects of dusts and finishes or adhesives used in the making of the furniture. In this case, three possible causative agents were initially considered, namely urea-formaldehyde resin, the formaldehyde-containing lacquer, or one of the exotic woods. The positive bronchial challenge test response to imbuia but not to meranti wood (treated with the same paint lacquer and formaldehyde resin), together with the presence of precipitating IgG antibodies to imbuia wood proteins in the patient's serum, supports the diagnosis. A degree of hypersensitivity to other components used in the process cannot be entirely excluded, however.

It is now accepted that the symptoms of occupational asthma may persist despite removal from exposure. Studies among snow-crab processors have suggested that the mean time to the plateau of improvement for pulmonary function after removal from exposure is 1 year.³ The poor prognostic factors in this patient included: 4-year exposure history; moderately impaired lung function (FEV₁, 52% of predicted) on initial assessment; and significant specific bronchial hyperreactivity, inferred from the 33% decline in FEV₁ during the bronchial challenge test.⁴

Eighteen months after leaving her job, the patient was still unemployed and had suffered considerable loss of earnings because occupational asthma was not compensable in South Africa at the time. Studies have shown that although removal from exposure may be beneficial in terms of symptoms and lung function, it is inevitably associated with loss of income.⁵ Gannon et al.⁵ have therefore suggested that compensation be directed toward assisting relocation and retraining rather than providing inadequate compensation for disability, which is difficult to assess in patients with asthma.

In conclusion, as the importation of exotic woods for industrial and vocational purposes increases, more cases of wood dust hypersensitivity will undoubtedly be recognized.² The reporting of such sentinel cases is essential in building the database of exotic wood dusts known to provoke respiratory symptoms. More research into the pathogenesis of bronchial hyperreactivity caused by these woods is needed in order to develop valid diagnostic modalities,

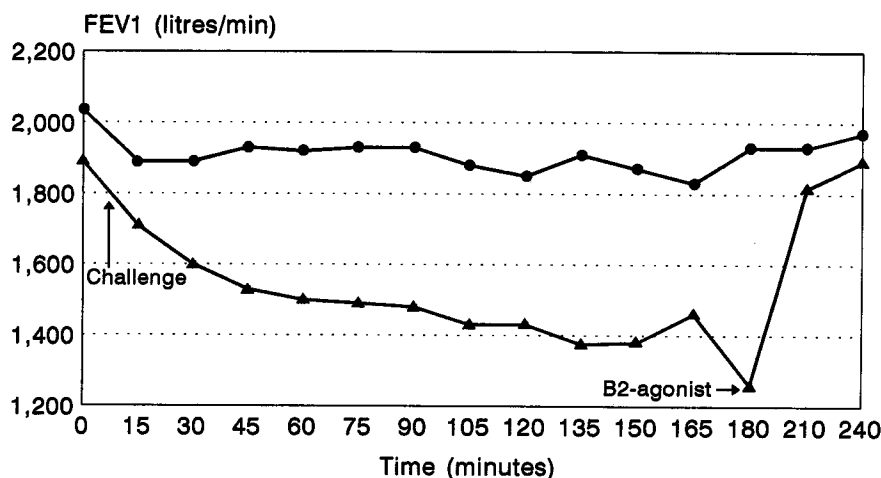


FIG. 2. Bronchoprovocation test with imbuia (▲) and meranti (●) wood dust.

including immunologic tests, to assist in the often difficult task of confirming a diagnosis of occupational asthma.

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Hypersensitivity pneumonitis: Problems in diagnosis

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Hypersensitivity pneumonitis (HP) is an immunologically mediated lung disease, caused by expo-

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Abbreviation used

HP: Hypersensitivity pneumonitis

sure to an inhaled antigen. Pigeon breeder's disease is a form of HP resulting from inhalational exposure to avian antigens. We have been seeing more cases of undiagnosed HP recently; this may