Occupational Rhinitis and Asthma due to Cedroarana (Cedrelinga catenaeformis Ducke) Wood Dust Allergy

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Abstract. We describe a case of occupational rhinitis and asthma in a 46-year-old carpenter who presented nasal and bronchial symptoms after cedrorana (*Cedrelinga catenaeformis Ducke*) wood dust exposure. Skin prick tests (SPT) with a battery of common allergens and different kinds of wood, were positive to cedrorana and grass pollen and negative to the other wood extracts. Nasal provocation and exposure challenge tests with *Cedrorana* wood dust also gave a positive reaction. IgE-immunoblotting showed two bands of 45 and 78 kDa respectively. This is the first reported case of occupational rhinitis and asthma due to *Cedrorana* wood dust where an IgE mediated mechanism has been found.

Key words: Allergy. Cedrorana. Occupational rhinitis. Occupational asthma. Wood dust.

Resumen. Describimos un caso de rinitis y asma ocupacional en un carpintero de 46 años de edad que presentaba síntomas nasales y bronquiales tras la exposición al polvo de madera de Cedrorana (Cedrelinga catenaeformis Ducke).

Las pruebas cutáneas, mediante prick-test, con una batería de alérgenos comunes y distintos extractos de madera, fueron positivas frente al extracto de Cedrorana y al polen de gramíneas, siendo negativas para el resto de las maderas. La provocación nasal y el test de exposición con el polvo de madera de Cedrorana fueron positivos. El Inmunoblotting reveló dos bandas de 45 y 78 kDa respectivamente. Este es el primer caso descrito de rinitis y asma ocupacional debido al polvo de madera de Cedrorana, donde se ha encontrado un mecanismo mediado por IgE.

Palabras clave: Alergia. Cedrorana. Rinitis ocupacional. Asma ocupacional. Polvo de madera

Introduction

Exposure to different kinds of wood sawdust has been shown to cause skin and respiratory symptoms in carpenters. An immunologic mechanism has been suggested in some cases; nevertheless an IgE-mediated sensitization has not always been elucidated [1-4]. We report a case of occupational rhinitis and asthma in a carpenter after exposure to *Cedorana* (*Cedrelinga catenaeformis Ducke*) wood dust.

Case Description

A 46-year-old male had been working in his carpentry for 20 years where he was exposed to different kinds of wood (pine, oak, teak) daily, and sometimes to *Cedrorana* sawdust.

Since 2002, the patient reported episodes of rhinitis after working with cedrorana wood. Nasal symptoms disappeared in some hours and were not related to the inhalation of the other wood dust. One year later, he

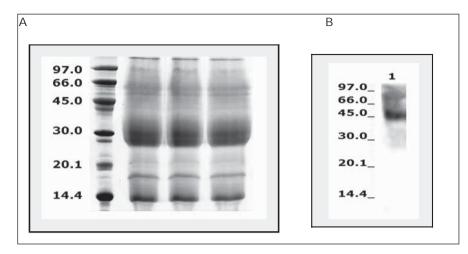


Figure. A) SDS-PAGE: Lane (M) marker; (1) Cedrorana wood. B) IgE-Inmunoblotting: Lane (1) Cedorana wood.

presented an episode of rhinitis, cough and dyspnea, immediately after cedrorana wood dust exposure. His symptoms improved after treatment with \$2-agonists and corticosteroids. The carpenter had been suffering from seasonal rhinoconjunctivitis for five years, but he never had complained of bronchial symptoms.

Skin prick tests (SPT) were performed with a battery of common allergens in our area. Commercial extracts of different kinds of wood (pine, oak, iroko, teak, sapeli, chestnut) and a *Cedrorana* wood dust extract were also tested at 10 mg/ml of dry weight (Diater, Spain).

SPT with cedrorana extract was also performed in 10 control patients (6 atopic and 4 non atopic). Two of the atopic patients were carpenters unexposed to *Cedrorana* wood. SPT were positive in our patient to cedrorana extract (wheal of 15×9 mm), grass and olive pollen, and were negative to the other wood extracts. Test results were negative in all control subjects.

A nasal provocation test with cedrorana extract at 5 mg/ml was also carried out by depositing a drop of cedrorana extract on the inferior turbinate mucosa of the nose using a cotton wool swab. We considered a true positive response when allergen caused at least two of the following criteria: 5 sneezes, rhinorrea, nasal blockage and a decrease in nasal peak inspiratory rate flow (NPIFR) greater than 20% [5].

In our subject the test with cedrorana was positive. We obtained an immediate response with rhinitis and a 20% decrease in nasal airflow after allergen challenge, with no late response.

An exposure test was performed by asking the patient to tip cedorana sawdust from one tray to another. The aerosolized sawdust was inhaled for 20 minutes in a 20 m2 room. Hourly peak expiratory flow rate (PEFR) was monitored in the next 24 hours (although the patient was allowed to sleep normally during the night), to detect a late response. Twenty minutes after the test, the patient presented rhinitis, cough and dyspnea, with a 27% decrease in forced expiratory volume in 1 second (FEV1). No late reaction was detected. A challenge test also performed as a control in an allergic asthmatic patient was negative.

Methacholine bronchial challenge testing was performed outside the pollen season, 24 hours before and after the cedrorana exposure test. The test was performed using a MB-3 dosimetre, with 25 mg/ml inhalations of methacholine solution. The patient inhaled a cumulative dose of 250 mg (Chatham abbreviated protocol). Results were negative both before and after the cedrorana exposure test.

Total and specific IgE to cedrorana was determined with enzymatic method. Protein extracts of cedroarana were separated by sodium dodecyl sulphate-polyacrilamide gel electrophoresis (SDS-PAGE) and SDS-PAGE immunoblotting was performed with serum from our patient sensitized to cedroarana according to the method of Moreno-Ancillo et al [6]. Total IgE was 663 kU/L (Pharmacia Immuno-Cap System, Uppsala, Sweden), and Specific IgE was 2.7 IU/L (EAST: HYTEC, HYCOR Biomedical Ltd., UK). IgE-Immunoblotting showed two bands of 45 and 78 kDa respectively (Fig.1).

Discussion

To our knowledge, this is the first reported case of occupational rhinitis and asthma due to cedrorana wood dust allergy where an IgE mediated mechanism has been found.

Two bands of 45 and 78 kDa, respectively, recognized from the serum of the patient could be involved.

Our results show that cedrorana wood dust can cause occupational respiratory disease in exposed workers. Allergy to this wood dust should be considered in individuals who present similar symptoms and exposure histories.

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