

Case Report

Allergy to an occupational allergen (Sapelli wood) in a child

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The case is presented of a child who developed rhinoconjunctivitis, angioedema and asthma by sensitization to Sapelli wood, which was used in his father's carpentry. Positive skin-prick test, high levels of specific immunoglobulin E by ELISA and a positive conjunctival challenge test suggest a type I hypersensitivity mechanism to this wood. This is the first case report of sensitization to Sapelli wood and it confirms that occupational allergen exposure as Sapelli wood may also cause sensitization in a child.

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Abebay or Sapelli (*Entandrophragma cylindricum*) is a tree highly distributed and cultivated in tropical Africa that may grow up to 60 m high with 1 m width. Sapelli wood is widely used in carpentry for exterior and interior structures due to its resistance, durability and attractive appearance.

Western red cedar wood dust and others have been recognized as causes of occupational asthma (OA) (1), and may cause a variety of other occupationally related lung disorders including hypersensitivity pneumonitis, organic dust syndrome, chronic bronchitis, mucous membrane irritation syndrome, allergic conjunctivitis, rhinitis and dermatitis (2).

We report here a child who experienced several allergic reactions while handling Sapelli wood. To clarify the relationship between some woods and the symptoms, we measured specific IgE and performed a conjunctival challenge test.

Case report

A 9-year-old boy with no clinical history of atopy or asthma experienced nasal and ocular itching,

upper lip and eyelid edema, stuffiness of the nose, cough, shortness of breath, wheezing and hoarseness when he handled pieces of wood at home. Chest auscultation revealed tachypnea and disseminated wheezing. A fast initial response to treatment with anti-histamines, β -agonist and corticosteroids was observed at the emergency department. Only stuffiness of the nose remained and gradually improved in one week. He suffered three episodes that required emergency assistance in the autumn of 1990. His father was a carpenter and the child usually played with woods at the carpentry or home every day. He suffered a similar reaction when his father was changing his clothes at home after having handled Sapelli wood at the carpentry in December 1990. Interestingly, from January 1991, his father was transferred to work in a different country and the boy stopped being exposed to woods. He did not suffer any more asthmatic reactions. Two years later, the child started to show symptoms of allergic rhinitis and asthma by grass pollen in the spring of 1993.

He was first seen by our allergy service in February 1991. A physical examination at that

time was normal. Chest roentgenogram revealed peri-bronchial inflammatory changes. Two months later, forced basal spirometry revealed an FEV₁ of 1.92 l (109%), FVC of 2.28 l (105%) and FEV₁/FVC of 84%. No fall in FEV₁ followed the bronchial challenge with histamine.

Skin prick test (SPT) and serum-specific IgE were negative for common inhalant allergens except for grass pollen which was slightly positive. We prepared several extracts in our laboratory following the Chan-Yeung method (3) using some woods that the father usually handled at the carpentry (Iroko – *Chlorophora excelsa*; Central American walnut – *Juglans olanchana*; African maple – *Triplochiton scleroxylon*; and Sapelli) and sawdust from his workplace. Final solutions had a protein concentration of 33 µg/ml (Iroko), 80 µg/ml (walnut), 28 µg/ml (African maple), 61 µg/ml (Sapelli) and 14 µg/ml (sawdust). Positive SPT responses were only obtained with Sapelli extract at 1:1 (7 × 5 mm), 1:10 (5 × 4 mm) and 1:100 (4 × 4 mm), and sawdust at 1:1 (7 × 4 mm), 1:10 (5 × 3 mm) and 1:100 (4 × 3 mm). Saline solution was used as negative control. The positive control was histamine hydrochloride 10 mg/ml (5 × 4 mm). The same SPTs performed in eight atopic subjects and seven non-atopic controls were negative.

Total serum IgE was 756 kU/l (CAP method, Pharmacia, Uppsala, Sweden). High levels of allergen-specific IgE antibodies to Sapelli (12 151 cpm; atopic control 93 cpm) and sawdust (12 164 cpm; atopic control 134 cpm) were obtained by radio-allergosorbent test (RAST) (Phadebas RAST, Pharmacia). RAST with other woods gave negative results. Sapelli and sawdust completely abolished specific IgE binding to Sapelli extract (RAST inhibition), probably because the presence of Sapelli particles in the sawdust.

Serial dilutions of Sapelli extract were prepared for the conjunctival challenge test. Informed consent was obtained from his parents. Intense itching, erythema and epiphora with mild edema were obtained 4 min after the administration one-drop of antigen 1:1000 (0.0061 µg/ml) into the conjunctival sac. The conjunctival provocation test was negative with grass pollen allergen. None of the control subjects (six atopic patients and four healthy subjects) had a positive response even with the undiluted Sapelli (6.1 µg/ml). Specific bronchial challenge was not carried out.

Discussion

OA has become the most common form of occupational lung disease in many industrialized countries (4). Immediate skin testing and serum IgE antibodies are not usually useful for diagnosis (5), so that specific bronchial challenge is often necessary to confirm OA. Because of the high cost and the risks and technical problems, the use of these tests is reserved to document new causative substances for OA, for medical/legal purposes and when there is uncertainty about the diagnosis of OA. Wood sensitization in children has not been reported previously, perhaps because it is difficult to confirm the diagnosis (4).

We report the case of a child who developed rhinoconjunctivitis, angioedema and asthma by sensitization to Sapelli dust. Positive immediate skin test reactivity, serum-specific IgE and conjunctival challenge test suggest a type I hypersensitivity mechanism to this wood. To the best of our knowledge, this is the first case report of sensitization to Sapelli wood.

The patient had a chronic low-level exposure to wood and sawdust in his environment. This and other factors, including airway size, family history of asthma, serum IgE levels, and exposure to allergen sources or passive smoking, may have enhanced the risk of the child for developing asthmatic symptoms. Interestingly, OA was not suffered by the carpenter. Probably, he is an antigen carrier to home, the 'workplace' of the son. Wood sensitization is included in occupational asthma and since the prevalence of asthma is increasing, we consider that occupational allergens are an under-estimated source in children.

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