Occupational allergic contact dermatitis, rhinoconjunctivitis, and asthma caused by moabi wood dust

Title page

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Occupational allergic contact dermatitis, rhinoconjunctivitis, and asthma caused by moabi wood dust.

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Manuscript

Case report

A 57-year-old man was referred to the Clinic for Occupational and Environmental Medicine, University Hospitals Leuven, because of a suspicion of work-related asthma. He was a never-smoker with an unremarkable medical history, except for an anaphylactic reaction after the administration of a tetanus vaccine in his 20s. He was a never-smoker.

Recently, he had started working in a wood furniture factory, where his main tasks were machining (sawing and sanding) various types of tropical wood—including moabi (*Baillonella toxisperma*), sipo (*Entandrophragma utile*) and meranti (mix of various species)—which generated substantial amounts of airborne wood dust. Effective local exhaust ventilation was reportedly absent and he declared being often covered in wood dust. After 4 months at the job, he began to experience red eyes, rhinorrea, coughing and wheezing with progressive development of dyspnea (especially at night). In addition, he had erythema, pruritus and dermatitis of the hands, as well as facial and neck erythema. His rhinoconjunctivitis and asthma symptoms improved during the weekend and especially during a 10-day summer closure of the factory. He had noticed that the symptoms were particularly pronounced when he worked with moabi wood.

Spirometry revealed obstructive impairment (FEV_1 1.67L, 50%predicted; FEV_1/FVC 0.51) with reversibility after administration of salbutamol (FEV_1 2.35L, i.e. a 41% increase) and a high fractional exhaled nitric oxide (F_{ENO}) (50 ppb), thus confirming asthma, for which he received a corticosteroid/long-acting beta-agonist inhaler. Sequential peak flow measurements performed at home and at work during 3 months confirmed significant improvements of his peak expiratory flow on days off work (OASYS score 3.04).[1]

Total serum IgE was 380 IU/mL (normal < 100). Specific IgEs were positive for *Dermatophagoides pteronyssinus* and *D. farinae*, but negative for other common aeroallergens. Prick tests performed with moabi, sipo and meranti dusts (‘as is’), brought from the patient’s workplace, were all negative.

Given the concomitant skin symptoms, he was referred to the Antwerp Contact Allergy Unit, where patch tests with moabi, sipo and meranti dusts were performed, both semi-open and on patch (10% pet., applied on AllergEAZE test chambers [Smartpractice, Calgary, Canada]). Following an occlusion of 2 days, readings on day (D)2 remained entirely negative, yet positive (+) reactions were observed to moabi (semi-open and on patch) on D3 and D7 (**Figure 1**). No reactions occurred to the other wood types. One unexposed control patient showed no reactions to moabi dust 10% pet.

We concluded that the patient had occupational rhinoconjunctivitis, asthma and allergic contact dermatitis (ACD) caused by moabi. We advised avoidance of further exposure at work and applied for recognition as an occupational disease. As no unexposed job seemed available in the same factory, he was sent on sick leave. After four months off work his cutaneous symptoms had disappeared and his respiratory symptoms had improved—with only minimal residual dyspnea. His lung function had substantially improved but was still obstructive (FEV_1 2.36L, 70%predicted; FEV_1/FVC 0.58) with borderline reversibility after bronchodilation (FEV_1 2.53L or +7%) while F_{ENO} had returned to normal (17 ppb).

Discussion

Dust of tropical woods may irritate the skin, eyes and airways.[2] In addition, numerous wood species may cause skin or airway sensitization,[3] mostly in carpenters or wood industry workers. Occupational allergic contact dermatitis,[4,5] rhinitis,[4] and asthma[2] caused by moabi have been previously reported, but all published cases date from decades ago. Moabi wood is used for (high-quality) furniture, but also, because of its durability, for joinery and carpentry in exterior applications, such as in boat construction, architectural woodwork, and building facades.

In the present case, we confirmed the presence of occupational asthma by a suggestive clinical history—with symptom onset after a 4-month latency period and improvement during days off work—and by sequential peak flow measurements that showed a clear relation with working days.[6] Although the irritant properties of the moabi wood dust might have contributed to the work-related ocular and respiratory symptoms, the latency period between the beginning of exposure and the onset of these symptoms suggests an allergic mechanism. The positive patch test to moabi, having an allergic morphology and a clear crescendo reaction, along with two negative internal controls (meranti, sipo) and a negative test in an unexposed control patient, suggests contact allergy—including airborne ACD—provoked by this wood species.

It is unclear how the presence of this documented type IV hypersensitivity relates to the patient's respiratory symptoms. Although in some published cases of occupational rhinitis or asthma positive sIgEs or skin prick tests have suggested IgE-mediated (type I) sensitization to wood dust,[7] the mechanisms by which wood dust compounds cause respiratory allergies are ill understood, especially in case of wood-associated low molecular weight (LMW) sensitizers such as terpenes or plicatic acid.[8]

Woods causing ACD, often do so with an airborne pattern because of the presence of fine wood dust aerosols generated by various working processes.[3] Although respiratory symptoms have often been reported in published cases of occupational ACD to wood dust,[9]

concurrent diagnoses of occupational rhinitis or asthma have rarely been documented. Conversely, also, in studies on occupational asthma, the presence of ACD is rarely assessed. This may be due, in part, to “silo thinking” of organ specialists. In a recent large series of occupational asthma cases, Tsui *et al* found a high frequency of patch-test positive ACD among patients with occupational asthma induced by LMW sensitizers.[10] However, hitherto only few studies have systematically patch-tested patients with LMW-induced occupational asthma.[11] The present case suggests that concomitantly studying respiratory and skin allergies in wood workers in future research might be of interest.

References

1. Burge PS, Pantin CF, Newton DT, et al. Development of an expert system for the interpretation of serial peak expiratory flow measurements in the diagnosis of occupational asthma. Midlands Thoracic Society Research Group. *Occupational and Environmental Medicine*. 1999;56(11):758-764. doi:10.1136/oem.56.11.758

2. Hausen BM. *Woods Injurious to Human Health: A Manual*. Walter de Gruyter GmbH & Co KG; 2016.

3. Levy Y, Hausen BM. Woods. In: John SM, Johansen JD, Rustemeyer T, Elsner P, Maibach HI, eds. *Kanerva’s Occupational Dermatology*. Springer International Publishing; 2018:1-18. doi:10.1007/978-3-319-40221-5_73-2

4. Alemany-Vall R. Rhinitis and dermatitis caused by exotic woods. In: *Occupational Allergy*. Stenfert-Kroese; 1958:273.

5. Babisch G. *Berufsallergien in Der Holzindustrie. [Occupational Allergies in the Wood Industry] Doctoral Thesis*. 1966.

6. Nemery B. Occupational asthma for the clinician. *Breathe*. 2004;1(1):25.

7. Chan-Yeung M, Schlünssen V, Fishwick D, Malo JL. Western Red Cedar and Other Wood Dusts. In: Tarlo S, Vandenplas O, Bernstein D, Malo JL, eds. *Asthma in the Workplace*. 5th ed. CRC Press; 2021.

8. Wiggans RE, Evans G, Fishwick D, Barber CM. Asthma in furniture and wood processing workers: a systematic review. *Occupational Medicine*. 2016;66(3):193-201. doi:10.1093/occmed/kqv149

9. Estlander T, Jolanki R, Alanko K, Kanerva L. Occupational allergic contact dermatitis caused by wood dusts. *Contact Dermatitis*. 2001;44(4):213-217. doi:10.1034/j.1600-0536.2001.044004213.x

10. Tsui HC, Ronsmans S, Hoet PHM, Nemery B, Vanoirbeek JAJ. Occupational asthma caused by low-molecular-weight chemicals associated with contact dermatitis: a retrospective study. *The Journal of Allergy and Clinical Immunology: In Practice*. 2022;10(9):2346-2354.e4. doi:10.1016/j.jaip.2022.05.014

11. Kanerva L, Estlander T, Jolanki R, Keskinen H. Asthma from diisocyanates is not mediated through a Type IV, patch-test-positive mechanism. *Contact Dermatitis*. 2001;44(4):246-263. doi:10.1034/j.1600-0536.2001.440409-2.x

Figure

Figure 1: Positive patch test (+) on day (D)3 to moabi wood dust (10% pet.)