

OCCUPATIONAL CONTACT URTICARIA AND EARLY-ONSET WORK-RELATED ASTHMA IN A LATEX-ALLERGIC WOODWORKER EXPOSED TO OBECHÉ WOOD

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ABSTRACT

Exposure to wood dust has been shown to cause allergic skin and respiratory symptoms in woodworkers. We describe a case of a 53-year-old latex-allergic woodworker presenting with work-related contact urticaria and subsequent upper and lower airway symptoms associated with obeche wood (*Triplochiton scleroxylon*) exposure. Clinical evaluation of the patient was suggestive of an early-onset, work-related asthma and latex allergy with possible cross-reactivity to obeche wood. The difficulties associated with investigating and managing workers with occupational allergy and asthma related to wood dust exposure are illustrated. It is recommended that workers with wood dust-related airway and skin symptoms should be investigated thoroughly using a more extensive repertoire of immunological and pulmonological tests in order to confirm the diagnosis and institute appropriate management of these patients.

Keywords: occupations contact urticaria; early-onset work-related asthma; latex-allergic woodworker; obeche wood

INTRODUCTION

Work in the furniture and wood-processing industries is associated with an increased prevalence of respiratory and skin symptoms and asthma.^{1,2} The evidence for a greater risk of impaired lung function with increasing exposure to wood dust is conflicting. Despite this clear link being reported in case reports and epidemiological studies, the underlying pathophysiological mechanisms remain poorly understood, although IgE-mediated reactions have been reported with some woods. Wood dust has been shown to cause irritation to mucous membranes, allergic sensitisation and asthma as a result of exposure to inhalable particles as low as 1 mg/m³.³ This report highlights the case of a woodworker who presented with work-related urticarial symptoms due to latex gloves and subsequently developed upper and lower airway symptoms associated with obeche wood exposure.

CASE PRESENTATION

A 53-year-old woman presented to the Occupational Medicine Clinic of Groote Schuur Hospital in Cape Town with a three-year

history of contact urticaria affecting her hands. This condition was initially associated with her use of latex gloves at work, but this was subsequently triggered by handling obeche wood. Handling other species of wood used in the factory had no effect (see Table 1). A few months following urticarial symptoms the patient developed work-related intermittent itchy, blocked or runny nose, chest tightness, wheezing and cough. She reported significant improvement of her symptoms during weekends and during annual leave, with a recurrence on returning to work. She had no history of childhood asthma, eczema or hay fever. She was a smoker with a ten pack-year smoking history. When she presented to the clinic, the patient had been on treatment with Budesonide inhaler 400 mcg twice daily (BD) and Salbutamol inhaler 200 mcg whenever required.

The occupational history revealed that for five years she had been working as a woodsander and stainer at a wooden shutter-making factory. The patient worked an eight-hour day for five days a week. She used oil-based wood stains (containing maleinised

linseed oil, medium aliphatic solvent naphtha, namely, petroleum and kerosene) and polyurethane varnishes (containing 1, 2, 4-Trimethylbenzene, cobalt carboxylate, ethylbenzene, white spirit, mesitylene, petroleum and xylene) for staining wood. She used powdered latex gloves while staining the wood. She sanded mainly meranti and obeche woods and was provided with respiratory protection (FFP2 respirators) while performing these tasks. Her previous work included clerical work for a plastics factory (three years) and woodsanding at a furniture-making factory (five years), but denied having any respiratory symptoms at the time. She could not recall the types of wood she had worked with in her previous position.

On clinical examination she was comfortable and not in respiratory distress. Her body mass index (BMI) was 25.30 kg/m². Her nasal turbinates were only 25% patent, but the rest of the ear and throat examination revealed no abnormality. On chest examination, she had no features of hyperinflation and no wheezing on auscultation. Her blood pressure was 120/80 mmHg and there was no evidence of cardiac failure. Examination of the other systems was unremarkable.

The spirometry results showed moderate airway obstruction with no significant reversibility of FEV₁ following bronchodilator administration. Her serial peak expiratory flow (PEFR) measurements showed a diurnal variation greater than 20%

and the Occupational Asthma Expert System (OASYS) work-effect index (WEI) computed was borderline with a value of 2.00 (a value >2.5 denotes a high probability of occupational asthma).⁴ The fractional exhaled nitric oxide (FeNO) level was 9 ppb, which was within normal limits (>50 is considered high – indicative of probable allergic-airway inflammation), while the chest radiograph did not reveal any underlying lung disease.

Skin-prick testing (SPT) with a standard panel of common aeroallergens was negative but the patient demonstrated a positive SPT to latex (see Table I). This was also demonstrated on specific immunoglobulin E (sIgE) testing showing mildly elevated sIgE to latex (k82) (0.54 kUA/L) but very low levels of recombinant latex allergen Hev b 6.02 and rHev b5. The levels of sIgE to obeche wood (sIgE = 0.19 kUA/L) was very low but relatively higher than meranti wood. The IgE to cross-reactive carbohydrate determinant (CCD) was below reliably detectable limits.

WORKPLACE WALKTHROUGH INSPECTION

A walkthrough inspection of the patient's factory (see Figure 2) revealed poor occupational hygiene measures in that settled wood dust was visible on the floor and on fixed surfaces. The wood dust easily became airborne during sweeping or dusting of the surfaces. Tasks associated with excessive wood dust generation in the factory included wood cutting, thicknessing,

TABLE I: SUMMARY OF THE PATIENT'S CLINICAL, PULMONOLOGICAL AND IMMUNOLOGICAL PROFILE

Demographic characteristics	Age	53 years
	Gender	Female
	Smoking status	Smoker
Exposure history	Exposure duration	Five years
	Job type	Wood sanding and staining
	Wood species causing symptoms	Obeche
Work-related symptoms	Urticaria	Yes (associated with obeche wood or NRL gloves)
	Ocular-nasal	Yes
	Asthma	Yes (three years duration)
Pulmonological assessment	Chest radiograph	NAD
	Spirometry	
	FEV ₁ pre-BD/post-BD, mL	1340/1470
	FVC pre-BD/post-BD, mL	2140/2190
	FEV ₁ /FVC pre-BD/post-BD, %	63/67
	FEV ₁ increase post-BD, mL (%)	130 (10% increase)
	Work-related changes on serial PEFR- OASYS Score	2.00
Immunological assessment (<i>in vivo</i>)	FeNO (away from work, at clinic)	9 ppb
	SPT to common aeroallergens	Negative
	SPT to occupational allergens	Positive (Latex extract)
	Allergen-specific IgE, kUA/L	
	Meranti	0.01
	Obeche	0.19
	CCD	0.00
	Latex (K82)	0.54
	Latex (K220- rHev b 6.02)	0.28
	Latex (K218- rHev b 5)	0.00

NAD: No abnormality detected; BD: bronchodilator; FEV₁: Forced expiratory flow in one second; FVC: Forced vital capacity; FeNO: Fractional exhaled nitric oxide; SPT: skin-prick test; CCD: cross-reactive carbohydrate determinant – horse radish peroxidase

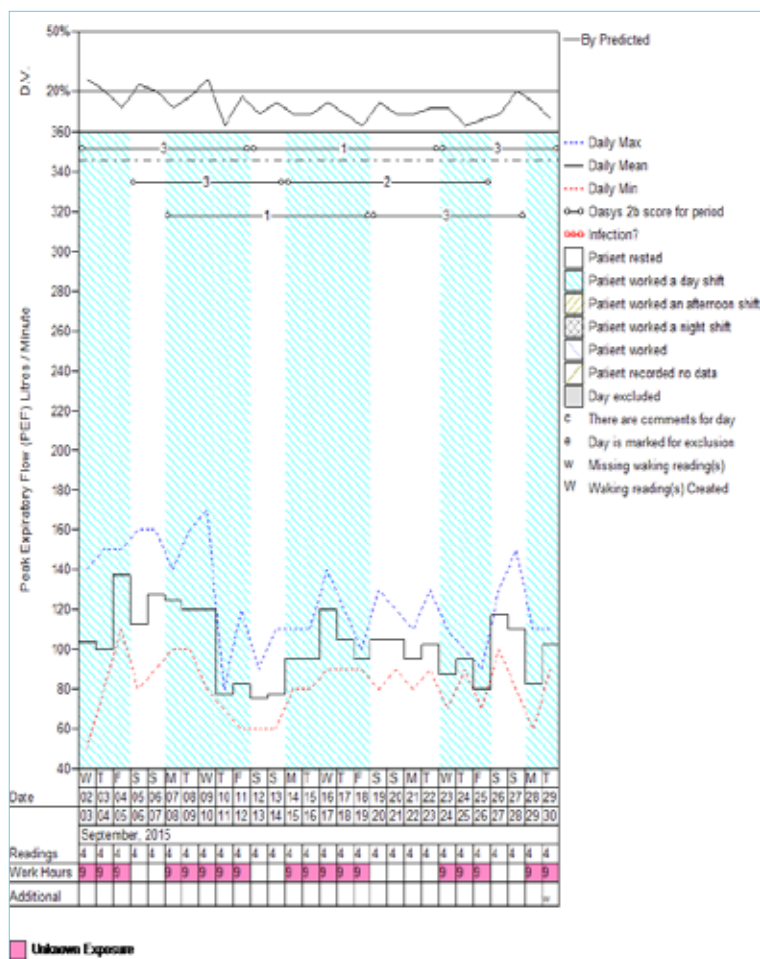


Figure 1: Serial expiratory flow recording of the patient over a four-week period (at work and away from work)



Figure 2a: A schematic layout of the factory floor



Figure 2b: Wood receiving section



Figure 2c: Wood cutting section

planing, moulding and sanding (see Figures 2a–d). Wood-staining used non-isocyanate polyurethane varnishes as well as oil-based wood stains that potentially had respiratory and skin-irritant properties. According to the report of an occupational hygiene survey conducted in the factory, the wood dust concentrations in the machining and sanding sections of the factory were found to be below the South African occupational

exposure limit for wood dust (5 mg/m^3) but nevertheless the machine operators (2.81 mg/m^3) and sanders (1.26 mg/m^3 and 1.65 mg/m^3) experienced relatively higher levels of inhalable wood dust than other jobs.

CLINICAL AND WORKPLACE MANAGEMENT

Since the patient was not optimally managed with budesonide and salbutamol inhaler treatment, a long-acting β -agonist



Figure 2d: Wood-planing and thicknessing section

(Formoterol 12 mcg twice daily) was added to her treatment regimen. Attempts to accommodate her in another section of the factory were unsuccessful since the factory was conducting wood machining, staining or spray painting in one open area. The patient was counselled on the importance of seeking alternative employment without exposures to the offending wood dust in addition to taking up a smoking cessation programme. However, since there were no alternative jobs available to the patient she chose to continue working in her current job despite experiencing intermittent work-related respiratory and skin symptoms that resulted in significant absenteeism. She was concerned that she would not be able to find alternative employment due to her age and level of education. The company was advised to provide her with powder-free non-latex gloves, which they duly implemented. This resulted in intermittent alleviation of her skin symptoms. A worker's compensation claim was initially submitted to the Worker's Compensation Fund in the Department of Labour according to the Compensation for Occupational Injuries and Diseases Act of 1993.⁵

DISCUSSION

This report highlights a case of a non-atopic woodworker with latex-allergic contact urticaria and features suggestive of early-onset asthma associated with obeche wood dust exposure.

Obeche is a softwood widely used for all purposes for which durability and strength are not very important but stamina is needed, such as windows and door frames.⁶ Inhalation of dust from obeche wood, is known to cause allergic sensitisation and asthma in exposed workers. It has been demonstrated that this dust has a higher protein content⁷ and therefore possesses greater allergic sensitisation potential resulting in contact urticaria, rhinitis and occupational asthma (OA).^{6,8–11} A 38 kDa class I chitinase obeche wood allergen, Trip s 1,

has been identified as the major allergen of this wood with a large proportion (70%) of symptomatic workers demonstrating sensitisation to this allergen.⁷ Other extractable protein patterns identified for obeche wood are in the range of 5–70 kDa with dominant bands present at 7, 12, 26, 28, 29, 30, 38 and 62–65 kDa.^{7,12}

Since no standardised wood allergen test solutions for assessing wood sensitisation exist, individual wood allergen extracts must be prepared and tested for each wood species. However, the protein content of the prepared extracts has been shown to vary depending on the tree growing conditions, geographical origin as well as drying and storage methods used.¹³ These limitations mean that it is difficult to assess sIgE-mediated wood sensitisation using the conventional SPTs. Adding to this complexity, recent studies have recommended that *in vitro* tests, particularly those involving plant allergens, should be evaluated for clinically irrelevant results caused by CCDs.^{13,14} These are asparagine-linked carbohydrate moieties of plant and insect glycoproteins found abundantly in the environment.¹⁵ Therefore, in order to confirm work-related wood sensitisation it is important to exclude sensitisation to these compounds. In this current study, the patient did not demonstrate sIgE sensitisation to CCDs (sIgE = 0 kUA/L) confirming that her sIgE to the latex and obeche wood allergens were not due to clinically irrelevant glycosensitisation.

The extent to which wood dust exposure causes allergic sensitisation and work-related asthma depends on the presence of environmental risk factors such as increased job duration and high wood dust exposures as well as host-associated risk factors such as genetic susceptibility, atopy, smoking and presence of upper airway symptoms.¹⁶ In this case study, the patient had worked in the wood industry for more than ten years, which included five years of exposure to obeche wood dust. She was involved in wood-machining and sanding tasks, which were demonstrated to produce excessive wood dust exposures of >1 mg/m³ according to the industrial hygiene report. Furthermore, the patient had an appreciable smoking history, which has been associated with increased risk of work-related asthma symptoms (OR 3.6, CI 1.7–7.6) and symptomatic bronchial hyper-responsiveness (OR 2.3, CI 1.0–5.4) in woodworkers.¹⁷ All these factors provide additional evidence for an increased risk of sensitisation and asthma.

In this case report, the patient presented with upper airway symptoms for several months prior to developing asthma. Rhinitis has consistently been reported as an independent risk factor for asthma development in atopic and non-atopic individuals.¹⁸ Workers with allergic rhinitis (AR) may therefore form an important population for secondary intervention to interrupt the natural history of allergic respiratory disease.¹⁹

Despite the strong history of work-related asthma symptoms, the spirometry showed mild airway obstruction with no significant reversibility. However, the serial PEFr measurements revealed a maximum single-day variability of 26%, consistent with a clinical picture of asthma (see Figure 1). Although the OASYS

WEI was borderline negative, this score does not rule out the presence of occupational asthma, because an analysis of data collected showed that 26% of patients with score of 2.0 and a suggestive history have OA.²⁰ FeNO has been shown to be useful in detecting eosinophilic airway inflammation and determining the likelihood of corticosteroid responsiveness.²¹ The fact that FeNO levels were within normal limits in this patient suggests that other mechanisms may also be present.

The patient also reported urticarial reactions associated with using powdered latex gloves. Interestingly, she subsequently developed urticaria and respiratory symptoms when working with obeche wood despite having very low sIgE to this wood. It is likely that these reactions could be due to possible cross-reactivity between natural rubber latex (NRL) and obeche wood. It is well known that many NRL-sensitised individuals show allergic reactions to a wide range of allergens originating from different plant families.²² The existence of cross-reactivity between proteins in NRL and obeche wood has been demonstrated to be largely due to a 38 kDa protein (Trip s 1), a chitinase, which is the most important cross-reacting obeche allergen. This allergen has been reported to share some IgE-binding epitopes with Hevein (Hev b 6.02), the major latex allergen from *Hevea brasiliensis*.^{6,7} A study conducted to identify obeche wood allergen and determine possible cross-reactivity reported that, while co-recognition or cross-reactivity of obeche chitinase was observed in individuals with primary latex sensitisation, the latex Hev b 6.02 allergen did not cause cross-reactivity in obeche wood sensitised patients.⁷ The natural history of urticarial symptoms, an elevated sIgE to latex (k82) (0.54 kUA/L) and relatively lower IgE to obeche wood (0.19 kUA/L) in this patient may suggest that primary sensitisation was due to NRL followed by cross-reactivity to obeche wood. Furthermore, component-resolved analysis of the latex sensitisation using individual recombinant latex allergens revealed that sIgE to Hev b 6.02 (0.28 kUA/L) were comparable to obeche wood sIgE (0.19 kUA/L). This may indicate possible cross-reactivity between obeche wood and Hev b 6.02 latex allergen, as previously suggested in the literature.⁶ Specific IgE-inhibition tests would need to be conducted with these allergens to objectively confirm this cross-reactivity.

The occupational hygiene survey conducted in the patient's workplace revealed that the total dust particulate concentrations in the machining and sanding sections were below the South African Occupational Exposure Limit (OEL) for wood dust of 5 mg/m³ as contained in the Hazardous Chemical Substances Regulations.²³ However, it is evident that this exposure limit is not protective since symptoms were reported by the patient at levels below this limit. A similar observation was reported in a previous study of spice-mill workers in South Africa.²⁴ The American Conference of Governmental Industrial Hygienists

(ACGIH) guideline (2013) recommends that the threshold limit value (TLV) for wood dust, other than western red cedar, of 1 mg/m³ should be used.²⁵ Using this limit, the wood dust concentrations measured during machining (2.81 mg/m³) and sanding (1.26 mg/m³ and 1.65 mg/m³) tasks were above this TLV. Furthermore, it has been recommended that apart from measuring the concentration of airborne wood dust, the allergen content of wood should also be quantified in order to determine the risk of sensitisation²⁶ since the total dust particulate may not be a good reflection of allergenic exposures. This was demonstrated in a study, which reported that the total obeche wood dust concentration in workplaces did not correspond to the assessed airborne allergen concentration despite the assay being highly sensitive. The assay was able to quantify obeche wood-allergen concentrations from 30 to 300 ng/mL.²⁷

This case study has demonstrated the difficulties associated with managing workers with OA in the South African setting. The patient opted to continue working despite intermittent asthma symptoms due to daily exposures to wood dust and other respiratory irritants at work. Early and complete removal from occupational exposure is an important factor in the management of OA and its prognosis.²⁸ However, removal from exposure seems to benefit only those who continue to be employed in alternative employment. Woodworkers exposed to western red cedar with OA who remained employed, particularly without any wood dust exposure, were shown to have better quality of life (QOL) scores compared to those quitting work, especially for OA-related reasons.²⁹

CONCLUSION

This report demonstrates a case of a latex-allergic woodworker with features of work-related contact urticaria, rhinitis and early asthma symptoms due to possible cross-reactivity with obeche wood. It illustrates the point that workers with wood dust-related airway and skin symptoms should be investigated thoroughly using a more extensive repertoire of immunological and pulmonological tests to confirm the diagnosis and institute appropriate management.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest.

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