

CASE REPORT

Occupational asthma due to exposure to chengal wood dust

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Background	Chengal is a resistant rainforest hardwood that is commonly used in South-East Asia for the construction of boats, bridges, house structures and furniture. It contains oligostilbenoids, phenolics, quinones and resins.
Aims	To describe a case of occupational asthma (OA) related to chengal wood dust.
Methods	The patient was evaluated clinically for asthma in relation to occupational history, serial peak flow monitoring and specific inhalation challenge (SIC) test.
Results	Peak flow monitoring showed significant deterioration during exposure to chengal wood dust at work. SIC test resulted in an isolated immediate asthmatic reaction.
Conclusions	Exposure to chengal wood dust can lead to OA and possibly rhinitis. The underlying mechanism should be investigated.
Key words	Dust; occupational allergy; occupational asthma; rhinitis.

Introduction

Wood dust from many tree species is known to cause occupational asthma (OA) (1). To the best of our knowledge, OA caused by chengal wood dust has never been reported. We report a case seen at our occupational lung disease clinic.

Chengal is the common name of *Neobalanocarpus hemeii*, a genus of the subfamily of rainforest tree, Dipterocarpoideae (Family: Dipterocarpaceae) (2). The hardwood is highly resistant to decay and is used in construction of boats, railway sleepers, bridges, poles, flooring, windows and door frames in South-East Asia. The resin of this tree is also used for dammar gum (2, 3).

Case report

A 41-year-old male who had been a carpenter for more than 20 years was seen for suspected OA. He had no family or past history of allergy, rhinitis, eczema or asthma.

In his 20 years as a carpenter, he had worked with different woods without any health problems. He first noticed

that he had rhinitis whenever he was exposed to chengal wood dust about a year prior to presentation to us. Six months after this, the rhinitis was accompanied by cough and chest tightness whenever he was exposed to chengal wood dust, while involved in a house renovation project. The symptoms were partially relieved with bronchodilator and steroid inhalers which were prescribed for him after he visited the hospital emergency department with acute asthma. He became asymptomatic for a month when he returned to his home country for a vacation. Upon return to work, he had another acute attack and was transferred to a different worksite where he did not need to work with chengal wood. However, he still had to return to the house renovation project whenever there was a shortage of manpower. His symptoms recurred whenever he was there.

In the house renovation project, chemically treated and non-treated chengal wood was used to replace structures destroyed by termites. He had to saw and plane the wood using hand-held electrical tools. No paint, glue or varnish was used in the house.

The patient's chest X-ray was normal. A differential blood count revealed eosinophils of 6.1% (normal range 0–6%). Spirometry showed moderate obstruction with a forced expiratory volume in 1 s (FEV₁)/forced vital capacity ratio of 61%. The FEV₁ rose 66% from 1.59 L (54% of predicted value) to 2.63 L after inhalation of a beta 2 agonist.

Serial peak flow monitoring showed a fall in peak expiratory flow rate (PEFR) of 40 and 28% on Days 12 and

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23, respectively (Figure 1). These days corresponded to the times when he was deployed to work on chengal wood in the house renovation project.

Informed consent was obtained from him to carry out a specific inhalation challenge (SIC). Prior to the SIC, his peak flow rate was monitored without medications and noted to be stable for 2 weeks while he was working with other woods. The SIC was conducted in a hospital room using untreated willow wood (also used in the workplace) as a control. He cut the wood into pieces with his saw, simulating workplace exposure. No asthma, rhinitis or significant diurnal variations were noted (Figure 2).

The following day, a SIC was carried out using untreated chengal wood. He developed rhinitis, cough and dyspnoea within 10 min of sawing the wood. By 30 min, PEFR fell 35% from the baseline of 540 L/min, and FEV₁ fell 38% from baseline of 2.98 L, respectively (Figure 2). Both returned to baseline levels within 2 h after nebulization and remained stable up to 48 h.

There were 11 other workers performing the same job. One of them had also been diagnosed with asthma and was noted to have an eosinophilia of 13.6%. However, he had returned to his home country and was unavailable for further investigation. A survey was conducted on 10 of his other co-workers. Nine of them reported symptoms of rhinitis, sore throat or cough whenever they inhaled the chengal wood dust. None had a rash, prolonged cough or dyspnoea. Spirometry conducted during their working hours was normal. All workers were not wearing respirators and housekeeping was poor.

His employer was informed of the hazard of wood dusts. As work is carried out at different worksites, the institution of permanent engineering control measures was not possible. The company stopped using chengal wood for their projects. The workers were provided with suitable respirators and advised on good work practices.

The patient's symptoms improved and he was maintained on low-dose steroid inhaler and steroid nasal spray.

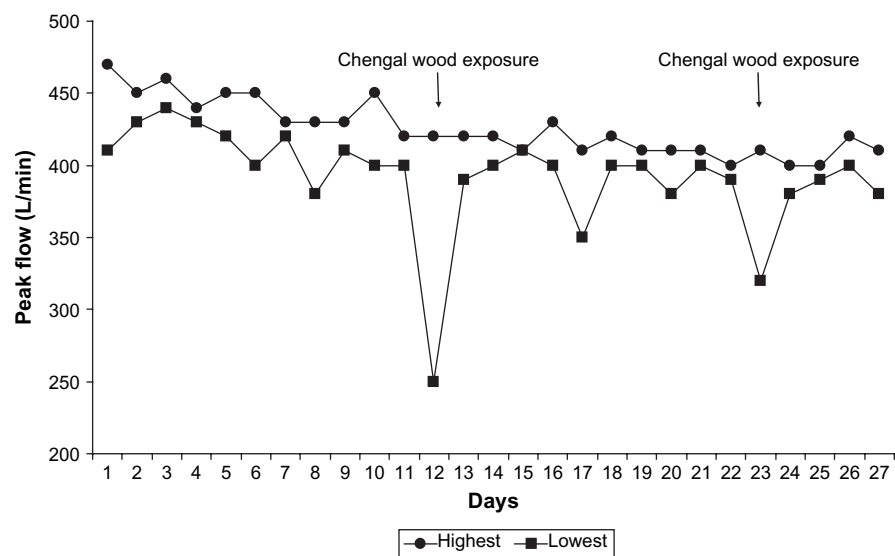


Figure 1. Peak flow monitoring at work and at home.

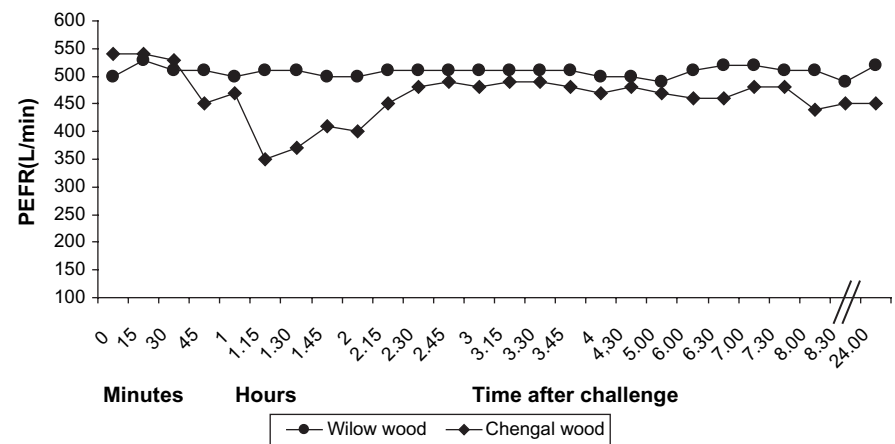


Figure 2. PEFR monitoring during SIC with willow wood (control) and chengal wood dust.

A repeat lung function test 6 months later was normal. There were no subsequent health complaints from the other workers.

Discussion

In our practice, we had encountered patients attributing their respiratory symptoms to chengal wood dust but were not able to carry out a full investigation. This is the first time we were able to do so. The patient's history, PEFR monitoring and SIC confirmed that he had OA. There was a likelihood that he had occupational rhinitis given his history and the high prevalence (90%) of his co-workers experiencing rhinitis.

We carried out a SIC on him using untreated chengal wood in a non-workplace environment to exclude other possible causes in the workplace. The result showed he had developed rhinitis and asthma from exposure to the chengal wood dust.

We were however unable to ascertain the mechanism by which rhinitis and bronchial asthma were induced by chengal wood dust. The patient was not keen for other tests and we did not have the facilities to extract the wood components for tests. Definitive diagnostic tests for occupational rhinitis like rhinomanometry, acoustic rhinometry and peak nasal inspiratory flow (4) were not conducted though his history and symptoms during the SIC were suggestive.

From our observation, the latency of sensitization appeared short (within a year) and rhinitis and asthma developed after short-term exposure. Chengal wood is not known to cause skin allergy associated with type IV hypersensitivity. A past study of 479 wood sanders who were patch tested with chengal wood dust did not reveal a positive result (5). In this patient, the isolated immediate response to the SIC is suggestive of a type 1 hypersensitivity to a component of the wood dust.

The chengal tree (*N. hemeii*) belongs to the subfamily of Dipterocarpaceae. Bioactive constituents extracted from this subfamily include oligostilbenoids, phenolics, quinones and resins (2). Quinones, phenolics and resins have been reported to cause skin allergy and less commonly asthma (6,7,8). We are uncertain which of these components could be responsible for the hypersensitivity.

In conclusion, OA to chengal wood dust should be considered in individuals who present with similar symptoms and history of exposure. Information on the biological activities of the constituents of chengal wood has been growing due to economic interest especially since oligostilbenoids have been discovered to have antioxidant activ-

ities. It would also be useful to explore the allergenic properties of its constituents as well.

Users of this wood should also be warned of its allergenic properties and take the necessary precautionary measures.

Key points

- The wood dust of the tropical hardwood tree, chengal (*N. hemeii*), can cause occupational rhinitis and occupational asthma.
- Workers exposed to chengal wood dust should be evaluated for occupational asthma if they develop respiratory symptoms.
- Workers with occupational asthma due to chengal wood dust should be removed from further exposure.

Conflicts of interest

None declared.

References

1. Chan-Yeung M. Western red cedar and other wood dusts. In: Bernstein IL, Chan-Yeung M, Malo JL *et al*, eds. *Asthma in the Workplace*. New York: Marcel Dekker, 1993; 503–532.
2. Seo EK, Kinghorn D. Bioactive constituents of the family Dipterocarpaceae. In: Atta-ur Rahman, ed. *Studies in Natural Products Chemistry (Part D)*. Amsterdam: Elsevier, 1988; 23: 531–560.
3. Lim SC, Gan KS, Choo KT. *Timber Notes—Heavy Hardwoods (I) in Timber Technology Bulletin*. Kuala Lumpur: Timber Technology Centre (TTC), FRIM, 1998;8: 4.
4. Denyse G, Desrosiers M, Castano R. Occupational rhinitis. *Curr Opin Allergy Clin Immunol* 2006;6:77–84.
5. Gan SL, Goh CL, Lee CS, Hui KH. Occupational dermatosis among sanders in the furniture industry. *Contact Derm* 1987;17:237–40.
6. Estlander T, Jolanki R, Alanko K, Kanerva L. Occupational allergic contact dermatitis caused by wood dusts. *Contact Derm* 2001;44:213–217.
7. Hausen BM. Sensitizing capacity of naturally occurring quinones V. 2,6-Dimethoxy-p-benzoquinone: occurrence and significance as a contact allergen. *Contact Derm* 1978;4: 204–213.
8. Chan-Yeung M, Malo JL. Aetiological agents in occupational asthma. *Eur Respir J* 1994;7:346–371.