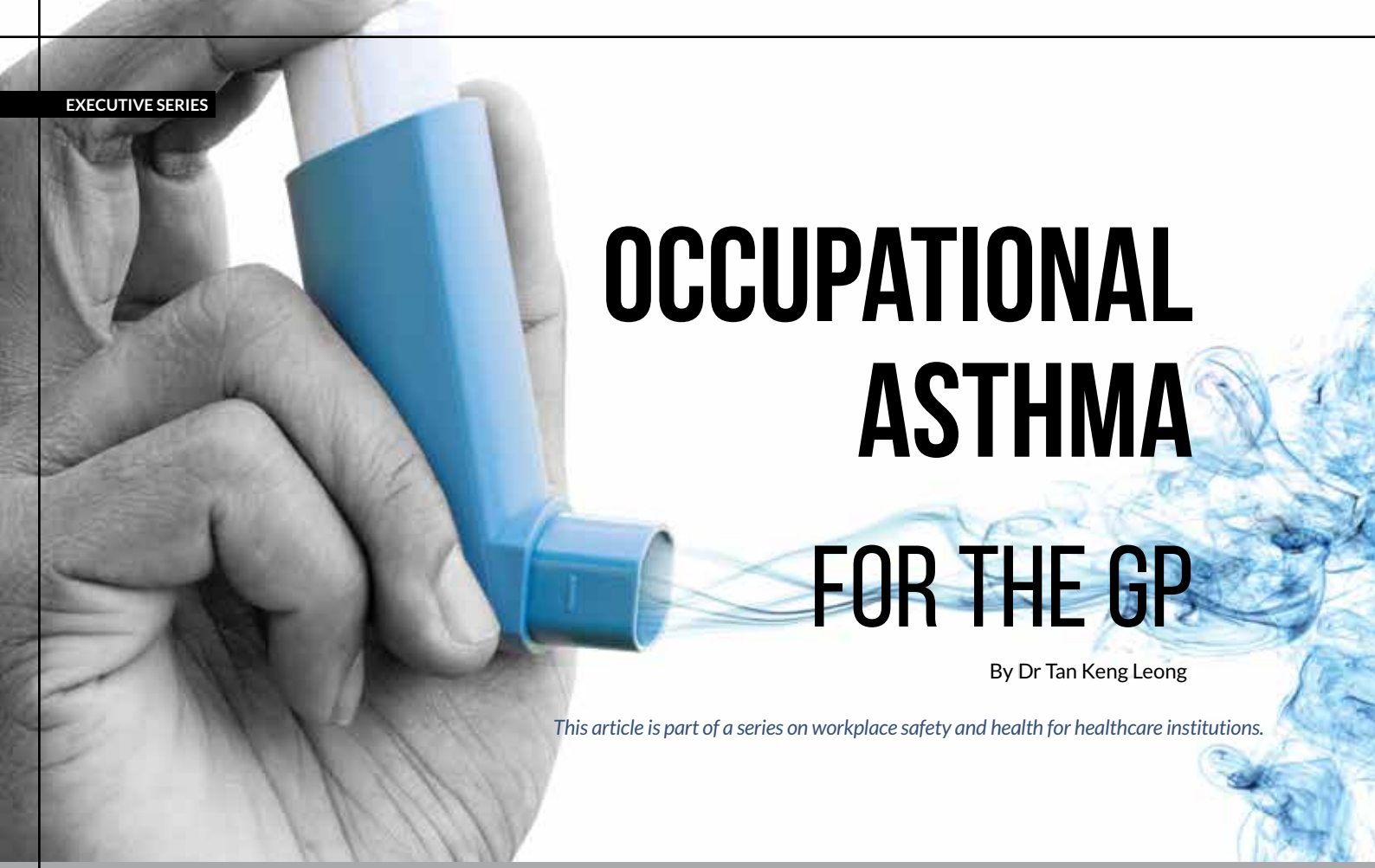

OCCUPATIONAL ASTHMA

FOR THE GP

By Dr Tan Keng Leong

This article is part of a series on workplace safety and health for healthcare institutions.

A 41-year-old who had been a carpenter for more than 20 years, presented with rhinitis, chest tightness and cough after exposure to chengal wood dust for one year. He had previously worked with different woods without any health problems. He was well during his month-long vacation when he was back in his home country. His symptoms recurred upon his return to work with chengal wood dust exposure. Serial peak flow monitoring showed significant drop in peak flows during workplace exposure to chengal wood dust. Specific inhalational challenge test resulted in an isolated immediate asthmatic reaction, thus confirming the first reported case of occupational asthma due to chengal wood dust.[1]

Key points

1. Occupational asthma is now the most common occupational respiratory disease in Singapore.
2. It is a legally notifiable occupational disease under the Workplace Safety and Health (WSH) Act and a compensable occupational disease under the Work Injury Compensation Act.
3. Appropriate management and prevention is important because of the medical, socio-economic and legal consequences.
4. Continued exposure to the causative agent may lead to permanent airway damage, resulting in persistent asthma even after removal from exposure.
5. Identification of the specific causative agent and early removal from exposure can prevent the risk of a severe or fatal asthmatic attack in the workplace.

Definition

Occupational asthma is defined as asthma due to conditions attributable to work exposures and not to causes outside the workplace.

Types

The two types of occupational asthma are distinguished by whether they appear after a latency period:

Sensitiser-induced asthma

This is characterised by a variable time (latency period) during which "sensitisation" to a specific agent present in the worksite takes place. The patient does not experience any respiratory problems during the latency period of weeks to years. Once sensitisation has occurred, the worker may be affected by very low concentrations of the offending agent.

Irritant-induced asthma

This occurs without a latent period after substantial exposure to an irritating dust, mist, vapour or fume (such as chlorine, sulphur dioxide or acid fumes). *Reactive airways dysfunction*

syndrome is a term used by some to describe irritant-induced asthma caused by short-term, high-intensity exposure (eg, an accidental spill or other high level respiratory irritant exposures).

Prevalence

Recent estimates suggest that 9% to 15% of adult asthmatics may have occupational asthma.[2] In Singapore, although the disease is common, it is likely to be under-diagnosed and under-reported.

Causative agents and occupations at risk

Substances that cause occupational asthma are classified either as high molecular weight or low molecular weight allergens. High molecular weight allergens include: products of animal, plant or microbial origin such as laboratory animal allergens, fish and seafood proteins, flour and detergent enzymes. Low molecular weight allergens include: chemicals and metallic agents such as acid anhydrides, antibiotics, isocyanates, western red cedar, amines, colophony and metals.

In Singapore, the most common causative agents reported were isocyanates (31%), colophony fluxes and solders (13%), welding fumes (9%) and wood dust (4%).[3] Isocyanates are the leading cause of occupational asthma in a number of other industrialised countries.[4] Some common causative agents of occupational asthma and the occupations at risk are summarised in Table 1.

Table 1: Common causes of occupational asthma and occupations at risk

Causative agents	Occupations
Isocyanates (eg, toluene diisocyanate)	• Polyurethane foam workers • Spray painters and varnishers • Insulation workers
Acid anhydrides (eg, phthallic anhydride)	• Chemical workers making or using polyester, epoxy, alkyl resins • Spray painters
Pharmaceuticals (eg, antibiotics, glutaraldehyde)	• Pharmaceutical technicians • Health care workers • Veterinary workers • Animal feed workers
Soldering flux, colophony	• Soldering operators
Welding fumes	• Welders
Wood dust	• Carpenters
Metals and their salts (eg, nickel, cobalt, chromium)	• Electroplaters • Welders • Machinists
Foodstuffs (eg, grain, soybean, flour)	• Food processing workers
Enzymes (eg, *Bacillus subtilis*)	• Bakers • Detergent workers • Pharmaceutical workers
Animal products (eg, dander, excreta, urine)	• Farmers • Zookeepers • Laboratory technicians

Diagnostic approach

Take a detailed medical and occupational history to assess current and previous job exposure to causative agents such as chemicals, proteins, organic dusts and animal products.

Knowledge of common causative agents and their associations with various occupations and industries, and information obtained from safety data sheet (SDSs) are often helpful. An SDS is a document that contains information on the potential hazards (health, fire, reactivity and environmental) and how to work safely with the chemical product. WSH Regulations require the employer to make the SDSs available to workers who may potentially be exposed to hazardous substances at the workplace.

Diagnosis

The diagnosis is made by:

1. Establishing the presence of asthma;
2. Demonstrating relationship between asthma symptoms and work; and
3. Establishing exposure to a specific causative agent.

A clinical diagnosis of asthma is made based on appropriate clinical history and evidence of reversible airflow obstruction. Symptoms include episodic breathlessness, wheezing, coughing or chest tightness, commonly in response to certain trigger factors.

A childhood history of asthma does not exclude the diagnosis of occupational asthma, as these patients may also become sensitised to a specific agent in the workplace.

Work-relatedness may be suggested based on: the history of improvement when away from work (eg, annual or maternity leave), and onset of symptoms during working periods.

Diagnostic pitfalls

Bronchial asthma

Occupational asthma may also present as chronic cough (eg, without episodic breathlessness or wheezing). Repeated absence from work due to frequent "bronchitis" is another typical presentation and should trigger the suspicion of occupational asthma.

Relationship to work

In occupational asthma, there is a temporal relationship between the asthma symptoms and exposure at work. For instance, the patient may report that asthma occurs soon after entering the workplace or when performing specific tasks. The patient may not necessarily be aware that the symptoms are temporally related to work as the asthma symptoms are commonly more pronounced in the evening, at night, or in the early morning (ie, outside the workplace). Symptoms (eg, wheezing) may be triggered by inhaled irritants, such as cigarette smoke, engine exhaust, strong odours, cold, exercise, which also frequently occur outside the workplace (eg, in pubs or during exercise). Therefore, a major pitfall in the clinical diagnosis of occupational asthma is that the patient with occupational asthma may have few symptoms during work and most symptoms outside the workplace.

Occupational asthma often responds well to appropriate medical treatment, at least initially. This may lead to the missed or late diagnosis of occupational asthma.

Hypersensitivity pneumonitis

Hypersensitivity pneumonitis may mimic occupational asthma. Numerous inciting agents have been described, including, but not limited to, agricultural dusts, bioaerosols, and certain reactive chemical species. Typical features of hypersensitivity pneumonitis include: respiratory and constitutional symptoms and signs, such as crepitations on chest examination, weight loss, coughing, breathlessness, febrile episodes, wheezing, and fatigue appearing several hours after antigen exposure; and reticular, nodular, or ground glass opacity on chest radiographs. In occupational asthma, typically, chest radiographs are clear and constitutional symptoms are absent.

Serial peak expiratory flow rate

Clinical history alone is generally not sufficient to make a definitive diagnosis of occupational asthma. Serial peak expiratory flow rate during periods at work and away from work is a useful tool for the objective documentation of work-relatedness.

Specific bronchial provocation test (specific inhalation challenge test)

A positive specific inhalation challenge test to the causative agent is considered the gold standard for the diagnosis of occupational asthma. A workplace challenge may sometimes be performed when it is not possible to perform controlled specific challenges in the laboratory.

Environmental monitoring

Environmental monitoring is useful in documenting exposure to the specific agent. It is also useful in the assessment of risk and effectiveness of industrial hygiene control measures based on the level of exposure. Personal air sampling of workers using portable collection devices at various workstations and different breathing zones may be performed to study the pattern of exposure at various locations in the plant. This is useful in establishing a non-exposed work area where the patient could be transferred to. In addition, high risk areas requiring further control measures can be identified.

> “IN OCCUPATIONAL ASTHMA, THERE IS A TEMPORAL RELATIONSHIP BETWEEN THE ASTHMA SYMPTOMS AND EXPOSURE AT WORK.”

Management

For all workers confirmed to have occupational asthma, permanent transfer to a job with absolutely no exposure to the causative agent is recommended as further exposure may trigger a severe or even fatal asthma attack.

The patient should be counselled with regard to the disease, role of medications (preventers and relievers), inhaler technique, avoidance of trigger factors and treatment compliance.

Pharmacological treatment

Pharmacological treatment is similar as for any patient with asthma. Short-acting inhaled beta2-agonists, taken as needed, are used in the treatment of acute asthma symptoms and exacerbations and in the prevention of exercise-induced bronchospasm. Persistent asthma is controlled with daily anti-inflammatory therapy (preventer medication, ie, inhaled glucocorticosteroids). The Global Initiative for Asthma guidelines for the management and prevention of asthma are available on their website at http://www.ginasthma.com.

Prevention

With delayed diagnosis and continued exposure, progressive deterioration in lung function may lead to persistent asthma symptoms. When the relationship of symptoms to work becomes less obvious, the diagnosis of occupational asthma becomes more difficult.

Eliminate workplace exposure by substituting the causative agent or totally enclosing the process. Early removal from exposure is associated with a better prognosis.

Reduce exposure to causative agents through the use of local exhaust ventilation systems, dilution ventilation, dust suppression and respiratory protection.

Notification and compensation

All registered medical practitioners are required to report any of the occupational diseases listed in the second schedule of the WSH Act (Table 2) within ten days from the diagnosis of the disease. Non-compliance with the reporting may result in a fine of up to $5,000 for first offence, and up to $10,000 or/and an imprisonment for second or subsequent offence.

All suspected cases of occupational asthma should be referred for further evaluation and management. The WSH (Incident Reporting) Regulations requires all medical doctors to notify such cases through http://www.mom.gov.sg/ireport/.

Conclusion

The possibility of occupational asthma should be considered in any adult patient with asthma. The physician plays an important role in the early recognition of possible cases of occupational asthma and in the prevention of further cases. ■

References

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Table 2: List of reportable occupational diseases in Singapore under the WSH Act

1. Aniline poisoning
2. Anthrax
3. Arsenical poisoning
4. Asbestosis
5. Barotrauma
6. Beryllium poisoning
7. Byssinosis
8. Cadmium poisoning
9. Carbarmate poisoning
10. Cataracts due to infrared, ultraviolet or X-ray radiation
11. Compressed air illness or its sequelae, including dysbaric osteonecrosis
12. Cyanide poisoning
13. Diseases caused by excessive heat
14. Diseases caused by ionising radiation
15. Glanders
16. Hydrogen sulphide poisoning
17. Lead poisoning
18. Leptospirosis or its sequelae
19. Liver angiosarcoma
20. Manganese poisoning
21. Mercurial poisoning
22. Mesothelioma
23. Musculoskeletal disorders of the upper limb
24. Noise-induced deafness
25. Occupational asthma
26. Occupational skin cancers
27. Occupational skin diseases
28. Organophosphate poisoning
29. Phosphorous poisoning
30. Poisoning by benzene or a homologue of benzene
31. Poisoning by carbon dioxide gas sequelae, including dysbaric osteonecrosis
32. Poisoning by carbon disulphide
33. Poisoning by carbon monoxide gas
34. Poisoning by oxides of nitrogen
35. Poisoning from halogen derivatives of hydrocarbon compounds
36. Silicosis
37. Toxic anaemia
38. Toxic hepatitis
39. Tuberculosis
40. Ulceration of the corneal surface of the eye from exposure to tar, pitch, bitumen, mineral oil (including paraffin), soot

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