

OCCUPATIONAL ASTHMA IN SINGAPORE

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Articles on occupational asthma keep appearing in the international journals on respiratory medicine, occupational medicine, allergy and immunology. In recent years there has also been an increasing number of reports in our local journals. This trend is not just a reflection of increased academic interest in the subject. Occupational asthma is now recognised as the most common occupational respiratory ailment in the industrialised countries⁽¹⁾. In recent years, occupational asthma has overtaken asbestosis and silicosis to become the most common occupational lung disease in Singapore. To date, there are about 50 confirmed cases of occupational asthma in the official statistics on occupational diseases in Singapore⁽²⁾. With about five to ten cases confirmed each year, the condition is probably under-recognised and under-reported and the possible reasons for this have been discussed in a previous paper⁽³⁾.

What is occupational asthma? It has been defined rather narrowly eg "asthma induced by sensitisation to an agent inhaled at work"⁽⁴⁾. More recently, it has been defined as: "a disease characterised by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace"⁽⁵⁾. The latter definition is much wider eg the mechanism ie sensitisation is not mentioned. Often the actual mechanism is not known eg isocyanate induced asthma⁽⁶⁾. Sometimes, it is not possible to identify the exact agent that is causing the asthma but there is evidence of work-related airway obstruction as shown by the serial peak expiratory flow rate (PEFR) monitoring record covering periods at work and away from work. These cases would have been excluded by the earlier definition. Recently, there have been reports of occupational asthma in patients with pre-existent asthma^(7,8). The presence of pre-existent asthma should not preclude the diagnosis of occupational asthma, if it can be shown that the patient develops asthmatic attacks as a result of exposure to the working environment. In Singapore, occupational asthma is defined as asthma caused or aggravated by the work environment.

What are the common causes of occupational asthma in Singapore? Isocyanate is the most common cause, accounting for about 30% of the confirmed cases⁽²⁾. This is also the most common cause in the developed countries⁽⁶⁾. The reasons given were that this group of chemicals are very reactive and that they are widely used in industry. They are used as hardeners or curing agents in a polyurethane resin

system eg in the manufacture of foam mattresses and cushions and in paints, varnishes and adhesives. Occupations at risk are workers producing foam products, spray painters and insulation workers. Other common causative agents in Singapore are colophony resin in solder fumes, pharmaceutical products, welding fumes, amines and acid anhydrides (chemicals used in the manufacture of various resins), wood dusts etc.

Why should occupational asthma be investigated and confirmed? The management of any case of asthma should include dealing with aetiological factors whenever feasible. Medical treatment alone would not adequately control the asthma of a patient who is being provoked each time he is exposed to a particular allergen in his workplace. Furthermore, it is now known that the duration of symptoms after the onset of exposure influences the persistence of asthma once subjects are no longer exposed to the offending agent⁽⁴⁾. Most patients with occupational asthma are young and recommending a job transfer may affect their career prospects and pay⁽⁹⁾. It may be reasonable to base a diagnosis of pneumoconiosis on a history of exposure to mineral dust and a chest radiograph. The likelihood of having occupational asthma in the presence of asthma on the one hand and exposure to a potential causal agent on the other is not sufficiently high to make a diagnosis⁽¹⁾.

While the history alone may not be a satisfactory means of diagnosing occupational asthma⁽¹⁰⁾, it is certainly a very important screening tool. If occupational asthma is not suspected based on the clinical history, no further investigations would be carried out. One question should be asked in the history: whether symptoms improve when away from work and recur when back to work. Otherwise a case of occupational asthma may simply be labelled as "bronchial asthma" and "known case of bronchial asthma" on the follow-up visits.

How should occupational asthma be confirmed? Ideally, it should be based on a serial PEFR record showing evidence of reversible airway obstruction at work and improvement away from work together with documentation of exposure to a known cause of occupational asthma in the workplace or a positive bronchial provocation test (BPT) to the specific agent. Many studies have demonstrated a high sensitivity and specificity of the serial PEFR in the diagnosis of occupational asthma⁽¹¹⁻¹⁴⁾. There was a time when physicians were reluctant to carry out specific BPTs in Singapore. Today, it is an established tool in the investigation of occupational asthma. Specific BPTs should whenever possible be carried out to document new causes of occupational asthma. As a result, our doctors have been able to report four new agents not previously reported or documented by specific BPTs⁽¹⁵⁻¹⁸⁾. The specific BPT is still regarded by many as the "gold standard"⁽¹⁾.

The investigation of occupational asthma often involves a team eg the respiratory and occupational physicians, an occupational health nurse, an industrial hygienist (to

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measure the exposure) and sometimes a toxicologist or microbiologist etc (to identify the specific allergen). Perhaps the most important member of the team is the alert primary health care physician, the ward medical officer or the physician in a chest clinic who is in a strategic position to identify suspected cases of occupational asthma. Early diagnosis and removal may affect the prognosis of the case. The outcome can be most satisfying.

REFERENCES

1. Malo JL. The case for confirming occupational asthma: Why, how much, how far? (editorial). *J Allergy Clin Immunol* 1993; 91:967-70.
2. Lee HS. Occupational asthma in Singapore. Presented at the Cleveland Clinic Foundation Update, 9-10 Jul 1993, Singapore.
3. Lee HS, Phoon WH, Wang YT, Poh SC, Cheong TH, Yap JCH, et al. Occupational asthma in Singapore – A review of cases from 1983 to 1990. *Singapore Med J* 1991; 32:398-402.
4. Newman Taylor AJ. Occupational asthma. *Postgrad Med J* 1988; 64:505-10.
5. Bernstein IL, Chan-Yeung M, Malo JL, Bernstein DI. *Asthma in the workplace*. New York: Marcel Dekker, 1993.
6. Vandeplass O, Malo JL, Saetta M, Mapp CE, Fabbri LM. Occupational asthma and extrinsic alveolitis due to isocyanates: current status and perspectives. *Br J Ind Med* 1993; 50:213-28.
7. Gannon PFG, Burge PS, Benfield FGA. Occupational asthma due to polyethylene shrink wrapping (paper wrapper's asthma). *Thorax* 1992; 47:759.
8. Chan-Yeung M, McMurren T, Catonio-Begley F, Lam S. Occupational asthma in a technologist exposed to glutaraldehyde. *J Allergy Clin Immunol* 1993; 91:974-8.
9. Gannon PFG, Weir DC, Robertson AS, Burge PS. Health, employment, and financial outcomes in workers with occupational asthma. *Br J Ind Med* 1993; 50:491-6.
10. Malo JL, Ghezzi H, L'Archeveque J, Lagier F, Perrin B, Cartier A. Is the clinical history a satisfactory means of diagnosing occupational asthma? *Am Rev Respir Dis* 1991; 143:528-32.
11. Burge PS, O'Brien IM, Harries MG. Peak flow rate records in the diagnosis of occupational asthma due to colophony. *Thorax* 1979; 34:308-16.
12. Burge PS, O'Brien IM, Harries MG. Peak flow rate records in the diagnosis of occupational asthma due to isocyanates. *Thorax* 1979; 34:317-22.
13. Cote J, Kennedy SM, Chan-Yeung M. Sensitivity and specificity of PC20 and PEFr in cedar asthma. *J Allergy Clin Immunol* 1990; 85:592-8.
14. Perrin B, Lagier F, A'rcheveque L, Cartier A, Boulet LP, Cote J, et al. Occupational asthma: validity of monitoring peak expiratory flow rates and non-allergic bronchial responsiveness as compared to specific inhalation challenge. *Eur Respir J* 1992; 5:40-8.
15. Lee HS, Wang YT, Yeo CT, Tan KT, Ratnam KV. Occupational asthma due to tylosin tartrate. *Br J Ind Med* 1989; 46:498-9.
16. Lee HS, Yap I, Wang YT, Lee CS, Tan KT, Poh SC. Occupational asthma due to unheated polyvinylchloride resin dust. *Br J Ind Med* 1989; 46:820-2.
17. Chee CBE, Lee HS, Cheong TH, Wang YT, Poh SC. Occupational asthma due to hexahydrophthalic anhydride – a case report. *Br J Ind Med* 1991; 48:643-5.
18. Ng TP, Tan WC, Lee YK. Occupational asthma in a pharmacist induced by chlorella, a unicellular preparation. *Respir Med* 1994; 88: 555-7.