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Occupational asthma

Occupational asthma

M Abramson, M R Sim

Early cessation of exposure is important

Occupational asthma is the commonest form of occupational lung disease in many Western countries,[1–4] having overtaken the pneumoconioses in these countries owing to improved control of exposure to silica, asbestos, and coal dust. The reported incidence ranges from 13 per million workers in South Africa[5] to 174 per million workers in Finland.[6] It has been estimated that occupational factors may be responsible for 15% of all cases of adult onset asthma.[7] The financial costs of occupational asthma in the US alone were estimated at between $1.1 and $2.1 billion in 1996.[8]

Although occupational causes are relatively uncommon, they are important because, unlike most other forms of asthma, occupational asthma is eminently preventable. However, one of the challenges in prevention is the fact that there are several hundred known causes arising from many occupations in most major industries.[3 4] This is one of the reasons why prevention strategies are often unsuccessful.[9] To be successful, clinicians and occupational health practitioners need to be actively involved with the primary, secondary and tertiary prevention of occupational asthma.

Primary prevention is about the maintenance of safe working conditions and avoiding exposure to known sensitisers and irritants. A good example with which many readers would be familiar comes from the healthcare industry. A recent systematic review found evidence that substituting powdered latex gloves with low protein powder free gloves or latex free gloves greatly reduced latex aeroallergen, sensitisation, and asthma in healthcare workers.[10] This evidence was rated as SIGN level 2+, indicating that it came from well conducted observational studies with a low risk of bias or confounding and a moderate probability that the relationship was causal.

Secondary prevention of occupational asthma involves screening of workforces at risk. Workers known to be exposed to asthmagenic agents should undergo regular health surveillance. Cases of occupational asthma need to be identified early because continuing exposure results in worse symptoms, a faster decline in lung function, and a poorer prognosis.[3] Clinicians usually only become involved with diagnosis, management and rehabilitation—that is, tertiary prevention of occupational asthma. Respiratory symptoms from unrecognised or undertreated asthma cause work related respiratory disability among young adults in many countries.[11] While a key principle of management, removal from exposure often entails loss of job with the consequent socioeconomic disadvantages.

In this issue of *Thorax* Anees and colleagues report a study of the decline in forced expiratory volume in 1 second (FEV_1) in a series of patients with occupational asthma in Birmingham.[12] The authors found that FEV_1 was declining by 101 ml/year before removal from occupational exposure. Following removal from exposure, FEV_1 actually improved by 12.3 ml and subsequently declined by only 27 ml/year, a rate similar to what would be expected in a working age population. The authors admitted the likelihood of selection bias and the fact that they could not always be certain precisely when removal from exposure had occurred. The lack of an effect of current smoking on decline in FEV_1 was surprising and might be due to a ''healthy smoker'' effect.

Nonetheless, this paper is important because it adds to the body of evidence that early cessation of exposure improves the outcome in occupational asthma. It is well known that the likelihood of improvement or resolution of symptoms or of preventing deterioration is greater in workers who have no further exposure to the causative agent.[3] It is also known that the likelihood of improvement or resolution of symptoms or of preventing deterioration is greater in workers who have shorter duration of symptoms before avoidance of exposure.[3] Symptoms and non-specific bronchial hyperresponsiveness (BHR) to methacholine can persist for 10 years after removal from exposure in patients with occupational asthma caused by toluene di-isocyanate.[13] A more favourable prognosis was associated with less BHR at the time of diagnosis.[13]

Pharmacological management of occupational asthma is similar to non-occupational asthma. The observational study by Anees *et al* did not detect a benefit on FEV_1 from inhaled steroids.[12] However, a randomised controlled crossover trial of inhaled beclomethasone 1000 µg/day found small effects on symptoms, peak flow, and quality of life in patients with occupational asthma.[14] The beneficial effects were more pronounced if steroids were given early after diagnosis. Another small cohort study found that the same dose of inhaled steroid together with long acting bronchodilators seemed to prevent deterioration over 3 years among workers with mild to moderate occupational asthma who were still exposed to the causal agent.[15]

The paper by Anees and colleagues[12] provides further support that patients with occupational asthma should be removed from further exposure to the causal agent as soon as the diagnosis is confirmed. This may require retraining and/or alternative duties. Early cessation of exposure will improve symptoms and avoid the excessive loss of lung function that could result in earlier onset of respiratory disability. There is a place for treatment with inhaled steroids and long acting bronchodilators, but this should not be at the expense of continuing exposure, cessation of which must be the first line of management.

There is a pressing need for better evidence from randomised controlled trials of both pharmacological and non-pharmacological management of patients with occupational asthma. Effective health surveillance of exposed workers and early detection and removal of affected workers should be an essential aim for governments and practitioners concerned with the prevention of this important cause of occupational disease.

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Theophylline for COPD

Theophylline for COPD

P J Barnes

Reinstatement in the light of new evidence?

Theophylline has been used as a bronchodilator in the treatment of COPD for over 70 years, but has lost popularity as better tolerated and more effective bronchodilators have been introduced. However, new insights into the molecular action of theophylline have raised the possibility that this old drug may come back into favour as an anti-inflammatory treatment and may even reverse steroid resistance in COPD.[1] A paper by Hirano *et al* in this issue of *Thorax* provides further support for the anti-inflammatory effects of theophylline in patients with COPD.[2]

CURRENT USE OF THEOPHYLLINE IN COPD

In the major guidelines for the treatment of COPD, theophylline is relegated to a third line bronchodilator after inhaled anticholinergics and β_2 agonists. Nevertheless, it is recognised that theophylline is a useful treatment in patients with severe COPD as its withdrawal leads to significant clinical worsening of the disease.[3] Many older clinicians have been convinced by its clinical value in severe disease.

THEOPHYLLINE AS A BRONCHODILATOR

Traditionally, theophylline was used as a bronchodilator in the treatment of airway disease but, to achieve significant bronchodilatation comparable with