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REVIEW

If you want to cure their asthma, ask about their job

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Sero conversion

Summary Occupational asthma is the most common occupational respiratory disorder and accounts for 15% of cases of adult asthma. A recent systematic review of evidence and management has clarified patient care for General Practitioners (GPs) who are key professionals in early diagnosis.

Exposure to respirable agents in the work environment by means of dust, water aerosol or gases, causes an allergic sensitisation process in the respiratory tract. Initial rhinitis and night cough may progress to patterns of work-related wheezing from two weeks to six months after starting employment. The absence of symptoms while on holiday or sick leave suggests the diagnosis. Serial peak flow recordings show characteristic patterns. Smoking and atopy have a variable influence on whether a worker will develop the disease with exposure.

Early identification and removal from exposure is essential for the worker since it improves prognosis. Other workers will be at risk, and occupational hygienists are required to measure and improve the working environment by means of ventilation and extraction of toxic fumes.

Workplaces with workers who are at risk of occupational asthma, such as paint sprayers, food processors, welders and animal handlers, require health surveillance programmes for new and existing employees, as well as reinforcement of the more important primary safety measures of environmental monitoring and respiratory protection.

All clinicians responsible for asthma management need to be aware of the potential for occupational asthma in new cases of adult asthma or unexplained worsening of pre-existing asthma. Specialist help is required to confirm the diagnosis, which has substantial legal and economic implications for the worker and their employer.

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1. Introduction

New-onset adult asthma or aggravation of existing asthma should always raise the clinical question as to whether work is a contributing factor.

Occupational asthma accounts for up to 15% of all adult asthma and is the most commonly reported occupational respiratory disorder in Westernised industrial countries [1]. Occupational asthma has a poor prognosis unless it is identified early and managed effectively by removing the patient from exposure to the causative agent [2,3].

Health surveillance screening should be conducted at least annually and more frequently in the first two years of exposure. Workers who have pre-existing asthma and those who develop rhinitis require extra monitoring. Skin prick testing and immunological surveillance can help confirm the diagnosis together with the clinical history and lung function tests, but positive results by themselves do not indicate disease, merely sero-conversion. Workers with confirmed occupational asthma should be removed from exposure at the earliest opportunity and the whole workforce reassessed. Regular environmental measurements and prevention of allergen inhalation remain the cornerstones of prevention [2,3].

The chronic phase of occupational asthma is difficult to recognise clinically, and long-term exposure causes permanent asthma - in contrast to those individuals who are identified early and effectively cured of their asthma once removed from exposure [2,3].

A systematic review and publication of clinical guidelines by the British Occupational Health Research Foundation has been a welcome development in clinical management — for relevant web sites, refer to Box 1.

Box 1
British Occupational Health Research Foundation
http://www.bohrf.org.uk
Health & Safety Executive
http://www.hse.gov.uk/asthma
General Practice Airways Group
http://www.gpiag.org
OASYS and Occupational Asthma
http://www.occupationalasthma.com
BTS/SIGN Guidelines
http://www.sign.ac.uk

2. Who is at risk of developing occupational asthma?

The most commonly reported occupations leading to occupational asthma include:

Animal handlers
Laboratory workers

Box 2
High Molecular Weight Causative Agents

Prawn processing
Salmon processing
Bakery flour
Animal fur and urine
Biological detergent manufacture
Pharmaceuticals, e.g. amoxycillin production

Low Molecular Weight Causative Agents

Western Red Cedar asthma (plicatic acid)
Isocyanites (paint sprays and foams)

Bakers (supermarkets)
Chemical workers (isocyanites)
Food processing workers (prawn processors)
Nurses (latex gloves and endoscopy)
Paint sprayers (isocyanites)
Timber workers (wood dusts and resins)
Welders and solderers (fume colophony and fluxes)

Occupational asthma tends to occur in clusters of cases following the development of new work processes and failure to apply existing knowledge on safe working practices.

The respirable agent needs to be of respirable size in particle form, or suspended in water aerosol or gases. The allergic reaction tends to occur at bronchiolar level and the causative agents can broadly be separated into high molecular weight and low molecular weight agents (see Box 2).

3. Range of pathophysiology in occupational wheezing

The causative agent needs to be of respirable size to reach the right place in the lung. Large particles are blocked in the nose and don't become inhaled. Dust and mist particles are too large to reach the bronchioles. The pharmaceutical science of aerosol and dry powder inhaler production helps one to understand the concept of respirable size and lung deposition. A respirable particle size of 3—4 microns is likely to cause a reaction and we know that long thin asbestos fibres reach the alveoli. The classical pathophysiological process in occupational asthma is an IgE-mediated sensitisation which occurs at bronchial and bronchiolar level in the lungs. The allergen may be inhaled directly or suspended in water or gas.

Box 3: Pathophysiology of work-related wheezing

- Occupational asthma—IgE driven sensitisation
- Alveolitis — IgG mediated farmer's lung
- Irritant or corrosive (chlorine gas, powdered weed killer)

In the alveolitis of farmer's lung and malt worker's lung, fungal spores are inhaled to produce an IgG-driven immune reaction in the alveoli. Farmer's lung is often associated with systemic symptoms of shivers and general malaise in addition to breathlessness.

After irritant exposure due to chlorine inhalation, symptoms can follow from a single exposure, whilst in classical occupational asthma the respiratory symptoms are exactly the same as "normal" asthma after a period of sensitisation (Box 3).

4. Clinical history of occupational asthma

The onset of symptoms can be anything from two weeks to six months from the start of employment for an adult who has not been asthmatic before. In the early stages, rhinitis and a nocturnal cough might be the first symptoms. During the process of sensitisation to the inhaled allergen, there may be a few weeks or months when the patient feels worse when they go into work. However, a chronic phase quickly supervenes; the patient feels asthmatic all the time and it becomes difficult to distinguish a clear pattern of aggravation whilst at work. The predominant symptom at this stage is an improvement in symptoms at weekends and during days off, but this again progresses to a stage when there is no difference at weekends and the only noticeable improvement occurs during holidays and sickness absence.

Early identification and removal from exposure reduces the likelihood of the patient developing chronic asthmatic symptoms. Patients with occupational asthma can often find it difficult gaining alternative employment and can be economically disadvantaged. The possibility of industrial injuries compensation and litigation makes it essential for the clinician to document accurate clinical records.

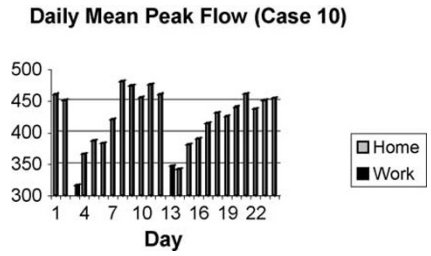

Figure 1

Investigations for Occupational Asthma

- Six-hourly peak flow recording
- Bronchial challenge test
- Reversibility with bronchodilators and steroids
- Specific IgG and specific IgE serology

5. Clinical investigations and treatments

The cornerstone of diagnostic investigation is the clinical history, as well as peak flow recordings [4] and demonstration of a specific antibody. The diagnosis of asthma is recorded in the conventional manner by demonstrating the reversibility of airway obstruction with salbutamol or steroids. Clarifying the clinical picture, prior to treatment, by measuring daily mean peak flows during time at work and away from work is valuable, as seen in Fig. 1.

Standard peak flow meters can be used to plot daily mean values related to work patterns or during 24-hour shift exposures. Computer programmes to analyse serial peak flow data are under development (http://www.occupationalasthma.com).

It may be feasible for the GP to sign the patient off work, treat with a course of oral steroids, and then ensure a week of normal peak flows without treatment before the patient returns to work.

The demonstration of specific IgE on serology testing is important, and when the patient is quickly identified and removed from exposure, the specific IgE disappears (for example, Fig. 2).

In contrast, delayed identification may cause specific IgE titres to remain raised for up to two years after exposure (for example, Fig. 3).

Bronchial challenge testing in the respiratory laboratory, and pre- and post-shift lung volumes with spirometry can all be used to gain objective evidence.

6. Confounding Clinical Variables

Exposure and sensitisation are required for a diagnosis of occupational asthma, but the influence of personal atopy is variable. With some causative agents, pre-existing atopy is an additional risk factor for developing occupational asthma [5] (e.g. detergent enzymes, laboratory animal asthma, isocyanites, bakery), whereas with other agents, cases are no more prevalent in atopic members of the population (e.g. gluteraldehyde, salmon, crab, platinum).

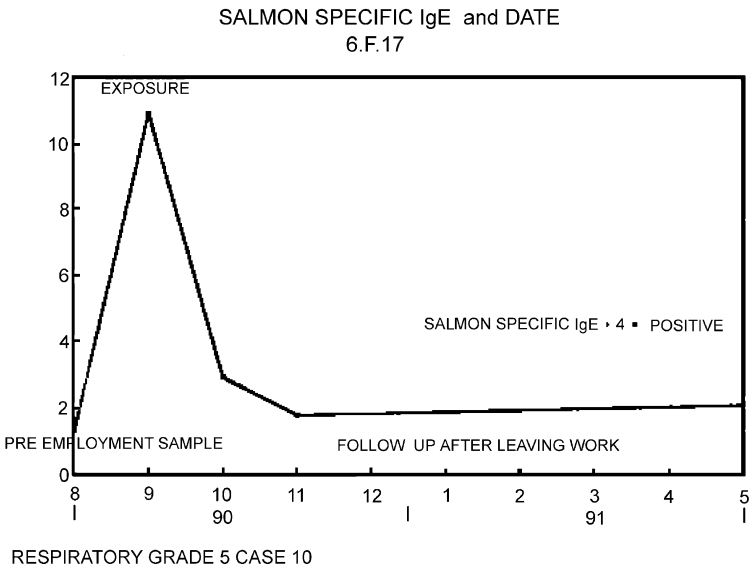

Figure 2

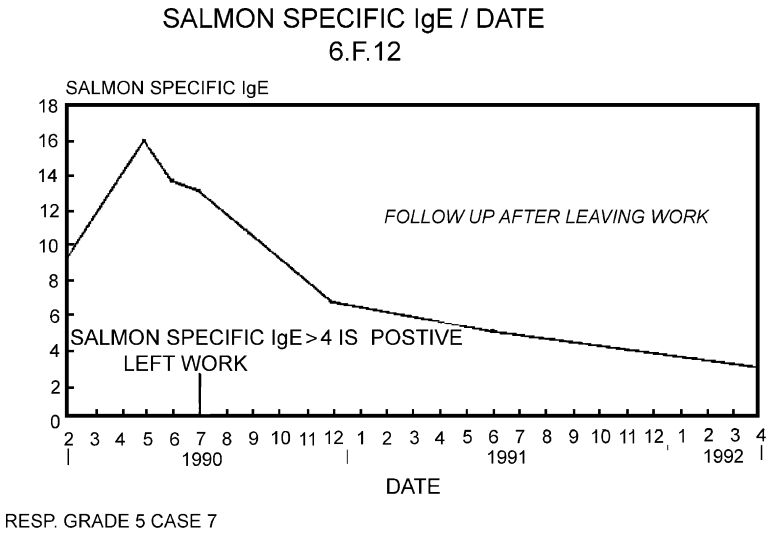

Figure 3

Similarly, smoking may have contradictory effects. In some causes of occupational asthma, smoking increases the likelihood of developing the condition [6] (e.g. isocyanites, platinum salts, salmon, snow crab) whereas, with other agents, smoking can be a protective variable [5] (e.g. bakers, farmer's lung, Plicatic acid, western red cedar asthma).

7. Clinical Case Histories From General Practice

7.1. New Factory

A new industrial salmon-processing plant was established in a small rural town and three new cases of adult-onset asthma presented to a local GP. Comprehensive study of the factory demonstrated 8% prevalence in the workforce, and water aerosol containing salmon protein from the automated gutting machines as the cause. Engineering modifications eliminated the aerosol and no new cases have been observed [6].

7.2. Reactive Airways Dysfunction

A 29-year old man presented acutely to his GP after inhaling weed killer dust, while employed by the local authority to clear a ferry slipway. Clinical examination noted respiratory distress which worsened over the following days and required admission and investigation. Lung volumes were reduced and severe symptoms of reactive airways dysfunction were noticed for at least two years; this meant that he was physically incapable of work due to breathlessness.

Ten years later, he now works in an office and his symptoms have gradually improved - but he still requires inhalers and can be compromised by winter respiratory infections. His lung volumes have been permanently damaged by a single episode of inhaling dust at work. He hadn't been issued with respiratory protection, despite the local authority having had a similar industrial accident at another regional depot one year previously.

8. Health & Safety

The potential to inhale toxic fumes or allergens in the workplace should be at the forefront of safety considerations for personnel managers and process engineers. Predictable hazards from car spray painting and welding need to be considered and engineered out by ventilation and extraction systems.

'Occupational hygiene' is the science of monitoring the environment of the workplace — for example, making measurements for respirable particles and checking air flow and ventilation systems. Occupational hygienists need to advise process engineers and factory designers on the modifications required to make factories safe.

In laboratory-animal asthma, occupational hygiene measurements have demonstrated that the antigenic challenge mainly comes from animal urine rather than the fur. Extraction of contaminated air and the provision of clean filtered air to the workers' breathing zone is preferable to having to wear face masks, which are often uncomfortable and ineffective. Compliance with personal protective equipment by workers always remains a difficult issue for factory managers.

Health surveillance of workers should be considered for all populations at risk of occupational asthma. Pre-employment screening questionnaires and baseline lung function can protect both employee and employer. However, there is no evidence-based justification to exclude from employment people with poorly discriminating factors such as atopy, a family or personal history of asthma, cigarette smoking or specific HLA phenotypes. Employees should be made aware of the risk of developing occupational asthma and the requirement to report early symptoms [2].

Health surveillance screening should be conducted at least annually and more frequently in the first two years of exposure. Workers who have pre-existing asthma and those who develop rhinitis require extra monitoring. Skin prick testing and immunological surveillance can help case confirmation together with a good clinical history and lung function tests, but positive results by themselves do not indicate disease, merely sero-conversion. Workers confirmed to have occupational asthma should be removed from exposure at the earliest opportunity and the whole workforce reassessed. Regular environmental measurements and prevention of allergen inhalation remain the cornerstones of prevention [2,3].

9. Legislation

The Control of Substances Hazardous to Health Regulations remain the cornerstone of legislation in occupational asthma. They require risk assessment and removal of noxious agents which have the potential to be inhaled. The RIDDOR regulations require employers to report industrial accidents and illnesses to the Health & Safety Executive. These notifications will trigger visits and assessments from HSE factory inspectors.

GPs should be encouraged to word sick-certificates with clear statements, e.g. "investigation of work-related asthma" or "suspected occupational asthma — report under RIDDOR".

10. What to do if you suspect a patient has occupational asthma

A GP has a responsibility to identify occupational asthma in the early stages and to seek specialist help. Obtaining an accurate clinical history and peak flow recordings are part of the GP's early role. Referral to a Chest Physician with an interest in occupational asthma or an Occupational Medicine specialist is advisable, given the medico-legal implications.

Patients are often very reluctant for the diagnosis to be suggested and made by their GP. However, the GP must always consider that other people are being potentially affected and there is a professional duty to pursue the problem and to notify the authorities. The GP's pre-existing relationship with the patient, and independence from the occupational health service, is an asset which is very important in individual case management. The patient may need to be persuaded over some time that they need to give up this particular employment. The patient's anger at the time can progress through to retrospective grateful appreciation years later when the asthma has resolved.

The GP needs to display patient understanding and meticulous record-keeping as their contribution, with occupational health and chest specialists, to improving the life of the patient. The prize of being able to cure occupational asthma is unique when compared to other forms of asthma, and GPs should always remain open to this possibility when reviewing adults with asthma.

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