Easily missed?

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A 22 year old man thought he might have hay fever. Several months later his general practitioner diagnosed asthma. It later emerged that his symptoms were associated with his work in a bakery, and he was referred to an occupational lung disease specialist who diagnosed occupational asthma.

**What is occupational asthma?**

Occupational asthma is caused by an immune reaction to specific agents that are inhaled in the workplace. It differs from asthma that is aggravated at work by non-specific factors—for example, exposure to dusts or fumes, cold air, physical exertion, and even shift patterns.

Offer all adults with suspected occupational asthma referral to an occupational lung disease specialist. Diagnosis is based on a history of exposure to a sensitising agent and immunology tests, peak flow measurements, and specific challenge tests if appropriate.

**How common is occupational asthma?**

About one in seven cases of adult onset or recrudescent asthma will have a link with work, but it is unknown what proportion is “occupational asthma.”

About 400 causative agents are recognised, although fewer than a dozen are responsible for most cases. These include proteins (“high molecular mass” agents) that induce a specific IgE response and certain reactive chemicals (“low molecular mass” agents) for which the mechanism is often less clear (table 1).

**Why is it missed?**

GPs may not routinely explore the role of occupation in patients with asthma or rhinitis. In a UK case series of almost 100 patients with occupational asthma, the mean interval between presentation to primary care and referral for specialist assessment was four years. An audit of primary care electronic records found that, of 400 people with adult onset asthma, 14% had their occupations recorded, and 2% had documentation of the effect of work at their most recent asthma reviews. Because there is generally an asymptomatic “latent” period after first exposure to the sensitising agent, GPs and patients may not make the link with work (see “How is it diagnosed?”); furthermore, they may be unaware of the need to refer. Finally, patients may be reluctant to link their symptoms to their work for fear of losing employment.

**Why does this matter?**

Prognosis is better if cases are detected early. Occupational asthma has a clear, defined trigger and identifying and removing it has the potential to improve symptoms or render the patient asymptomatic. This is unique in asthma because the patient may show no residual evidence of non-specific bronchial hyper-responsiveness. Delayed diagnosis carries a risk that the disease will not respond to usual asthma treatment at best, and of fixed airflow obstruction and persistent symptoms at worst.

**How is it diagnosed?**

**Clinically**

Rhinitis is always seen in occupational asthma caused by proteins and usually precedes chest symptoms. Patients might say: “ever since I started this job I’ve had a cold” or “I seem to have developed hay fever.” Latency is an important feature: occupational rhinitis and asthma usually start 6-18 months after...
first exposure (or change in exposure), and asthma symptoms are usually mild initially. For all adults with new symptoms of rhinitis, wheeze, or airflow obstruction, check for an occupational cause by asking standard screening questions (box 1). A systematic review found a sensitivity of 58-100% for occupational asthma in people referred to a specialist clinician who agreed that “symptoms are better on days away from work.” Have a high index of suspicion in those with high risk jobs (see table 1). However, its use as a positive or negative predictive question in the community is unclear.

Consider referring patients who answer “yes” to either question in box 1 to a specialist occupational respiratory unit to investigate for occupational asthma. There are about 15 occupational lung disease specialists across nine centres in the UK.

**Investigations**

Serial peak flows—measured at least four times a day (preferably 2-3 hourly) for a minimum of three weeks, on days at and away from work—have a sensitivity and specificity of around 80% and 90%, respectively, in patients being assessed for occupational asthma (see fig 1). This investigation may be instigated in primary care but is best interpreted by specialists. A different characteristic pattern is also seen for those with work aggravated asthma, with evidence of poorer control on both days at work and days away from work.

Immunological testing with specific IgE assays or skin prick tests against relevant workplace sensitising agents is offered to most of those referred. The accuracy (and availability) of testing varies by sensitising agent, and results must be interpreted along with the clinical picture and peak flow summary to confirm or refute the working diagnosis. Specific bronchial provocation testing, the gold standard, may be needed in difficult cases, but it is not widely available.

**How is it managed?**

Avoidance of further exposure to the causative antigen is the cornerstone of management. How this is achieved depends on workplace circumstances, and liaison with the occupational health provider, if there is one, will be necessary. Exposure control starts with substitution of the sensitising agent, if possible, or redeployment of the employee to another area without exposure. Other options include the use of respiratory protective equipment. Residual asthma symptoms may be managed with inhalers according to standard guidelines.

After a diagnosis of occupational asthma, about 25% of employees continue working in their current job, but a third are unemployed for up to six years afterwards. People with occupational asthma are eligible to claim industrial injuries disabilities benefit in the United Kingdom. One case generally heralds others in the same workplace.

Contributors: JF and PC conceived the idea; HP provided a primary care perspective. All authors contributed equally to drafting and revising the manuscript and approved the final version. JF is guarantor.

Competing interests: We have read and understood BMJ policy on declaration of interests and declare the following interests: PC is chair of the research working group for the Industrial Injuries Advisory Council, which advises the UK government on its industrial injuries disablement benefit scheme, and is a member of the workplace health expert committee, which advises the Health and Safety Executive on current and future occupational hazards. HP is a member of the executive and steering committee for the British Thoracic Society/Scottish Intercollegiate Guideline Network British asthma guidelines. JF has no interests to declare.

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Box 1: What to ask patients

In patients with adult onset or reappearance of childhood asthma, ask:
• Are you better on days away from work?
• Are you better on holiday?

What you need to know
• Occupational asthma differs from pre-existing or work aggravated asthma
• The diagnosis will be missed if it is not considered by non-specialists
• Specialist referral is needed for diagnosis and management

Education into practice

Do you ask patients with asthma or rhinitis about the connection between their symptoms and work?

How patients were involved in the creation of this article

One of our patients (a woman with baker’s asthma) reviewed the article favourably. She emphasised that it took several years for her diagnosis to be made and that consequently she had to retire and find alternative employment, which has been financially difficult; she is left with persistent symptoms of asthma.

Table 1 | Common causes of occupational asthma

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Agent</th>
<th>Molecular mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baker</td>
<td>Wheat flour, α amylase</td>
<td>High</td>
</tr>
<tr>
<td>(Laboratory) animal worker</td>
<td>Animal proteins</td>
<td>High</td>
</tr>
<tr>
<td>Detergent powder manufacturer</td>
<td>Biological enzymes</td>
<td>High</td>
</tr>
<tr>
<td>Fish and seafood processor</td>
<td>Fish proteins</td>
<td>High</td>
</tr>
<tr>
<td>Hairdresser</td>
<td>Persulphates (in bleaching mixtures)</td>
<td>Low</td>
</tr>
<tr>
<td>Paint sprayer</td>
<td>Di-isocyanates</td>
<td>Low</td>
</tr>
<tr>
<td>Foam manufacturer</td>
<td>Di-isocyanates</td>
<td>Low</td>
</tr>
<tr>
<td>Electronic solderer</td>
<td>Colophony fume</td>
<td>Low</td>
</tr>
<tr>
<td>Woodworker</td>
<td>Hardwood (tropical) dusts</td>
<td>Low</td>
</tr>
</tbody>
</table>
Fig 1 Peak expiratory flow diary suggesting occupational asthma. The minimal variation on days away from work (shaded white) contrasts with the 20-30% variability on workdays (shaded blue)