

ORIGINAL ARTICLE

Standards of care for occupational asthma: an update

David Fishwick,¹ Christopher Michael Barber,¹ Lisa M Bradshaw,¹ Jon G Ayres,² Richard Barraclough,³ Sherwood Burge,⁴ Jonathan M Corne,⁵ Paul Cullinan,⁶ Timothy Laszlo Frank,⁷ David Hendrick,⁸ Jennifer Hoyle,⁹ Andrew D Curran,¹⁰ Robert Niven,³ Tony Pickering,³ Peter Reid,¹¹ Alastair Robertson,⁴ Chris Stenton,⁸ Christopher J Warburton,¹² Paul J Nicholson¹³

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¹Centre for Workplace Health, Sheffield, UK

²University of Birmingham, Birmingham, UK

³North West Lung Centre, Manchester, UK

⁴Heartlands Hospital, Birmingham, UK

⁵Queen's Medical Centre, Nottingham, UK

⁶Royal Brompton Hospital, London, UK

⁷General Practice Research Unit, Manchester, UK

⁸Royal Victoria Infirmary, Newcastle upon Tyne, UK

⁹North Manchester General Hospital, UK

¹⁰Centre for Workplace Health, Buxton, UK

¹¹Western General Hospital, Edinburgh, UK

¹²Aintree Chest Centre, University Hospital Aintree, Liverpool, UK

¹³British Occupational Health Research Foundation, Society of Occupational Medicine and Faculty of Occupational Medicine, London, UK

Correspondence to

Professor D Fishwick, Centre for Workplace Health, Respiratory Function Unit, A Floor, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK; d.fishwick@sheffield.ac.uk

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ABSTRACT

Background The British Thoracic Society (BTS) Standards of Care (SoC) Committee produced a standard of care for occupational asthma (OA) in 2008, based on a systematic evidence review performed in 2004 by the British Occupational Health Research Foundation (BOHRF).

Methods BOHRF updated the evidence base from 2004–2009 in 2010.

Results This article summarises the changes in evidence and is aimed at physicians, nurses and other healthcare professionals in primary and secondary care, occupational health and public health and at employers, workers and their health, safety and other representatives.

Conclusions Various recommendations and evidence ratings have changed in the management of asthma that may have an occupational cause.

BACKGROUND

The British Thoracic Society (BTS) Standards of Care (SoC) Committee produced a standard of care for occupational asthma (OA) in 2008,¹ based on a systematic evidence review performed in 2004 by the British Occupational Health Research Foundation (BOHRF).² BOHRF updated the evidence base from 2004 to 2009 in 2010.³

This article summarises the changes in evidence and is aimed at physicians, nurses and other healthcare professionals in primary and secondary care, occupational health and public health, and at employers, workers and their health, safety and other representatives.

STANDARD OF CARE UPDATE

It is not intended, nor should it be taken to imply, that these amendments to the SoC override existing legal obligations, for example the Health and Safety at Work Act 1974, the Management of Health and Safety at Work Regulations 1999, the Equality Act 2010, the Control of Substances Hazardous to Health Regulations 2002 and other relevant legislation.

General comments

The recent evidence supports the estimate that occupational factors account for one in six cases of adult asthma. The range of incidence for cases of OA has been upwardly revised to between 12 and 300 cases/million workers/year. Under-identification of OA persists.⁴

Key messages

What is the key question?

► How does new evidence influence the assessment of asthma that may have an occupational cause?

What is the bottom line?

► Certain recommendations and ratings have changed.

Why read on?

► Read on to update and refresh your management approach to asthma that may have an occupational cause.

All the following statements are referenced to the 2010 BOHRF review,³ as details of the evidence review update, full references to statements in this document, an audit tool and case management can be found in the associated online supplement.

Prevention and health surveillance

Further evidence supports the role of health surveillance for identifying OA at an earlier stage, although screening questionnaires have significant false-negative response rates. Developing a workplace culture that supports workers to report symptoms accurately is key, as is workers' knowledge of a plan of action were they to report various work-related respiratory complaints.

Forced expiratory volume in 1 s (FEV₁) and forced vital capacity (FVC) measured to screen for OA are likely to detect few cases that would not otherwise be detected by respiratory questionnaire alone.

Education

All workers exposed to asthmagens should be warned about relevant symptoms potentially attributable to exposure, how agents in the workplace can affect health, and how best to avoid problems. Workers should be informed what to do, and in particular to whom they should report, if they develop relevant symptoms, particularly if these occur between health surveillance visits.

Educational programmes should be aimed at employers and healthcare professionals, including nurses and doctors (based in industry, primary and secondary care), occupational hygienists, and workers.

Occupational lung disease

Diagnostic process

Health practitioners who suspect a worker of having OA should make an early referral to a physician with expertise in OA.

All those involved in the potential identification of OA have an obligation to minimise delays.

Medical history

The latest evidence supports the importance of nasal symptoms in addition to asthma symptoms. Specifically, rhino-conjunctivitis may precede or coincide with the onset of OA, and the risk of OA development is highest in the year following the onset of rhino-conjunctivitis.

Occupational history

Hairdressers have been added to the list of workers with OA most commonly reported to schemes. Data from recent population studies also identify that cooks, healthcare workers, woodworkers and mechanics are also at greater risk of reporting asthma. The most frequently reported agents causing OA have been expanded to include adhesives, metals, resins in addition to isocyanates, flour and grain dust, colophony and fluxes, latex, animals, aldehydes and wood dust.

The full list of most commonly reported agents, workers and jobs from population-based studies with elevated OA risks are given in table 2 in the online appendix.

Investigations

Lung function

All suspected cases of OA should undergo FEV₁ and FVC measurement according to agreed criteria, and the results compared with a predicted value and previous results if available.

Pre-shift to post-shift changes in lung function have high specificity but only low sensitivity for OA. If these changes are present, they may support a diagnosis, but they are frequently absent in people subsequently confirmed to have OA. It is recommended that pre-shift and post-shift FEV₁ changes are only used in conjunction with other diagnostic approaches.

There is a considerable evidence base for the use of serial peak expiratory flow (PEF) measurements to investigate workers when OA is suspected. With appropriate training and explanation, it is possible to achieve high-quality recordings in these workers. While these tests may be susceptible to falsification and transcription errors, they offer the best and easiest first-line approach to assessing physiological response to asthmagens at work. High-quality recordings can be obtained for over 70% of patients.

Serial PEF should be recorded at least four times a day for at least three continuous weeks. Recordings for shorter durations are of lower diagnostic value. It is best to aim for readings every 2 h, so that practically at least four good measures per day will be achieved. Suitable record forms can be downloaded from <http://www.occupationalasthma.com> or <http://www.scottishshield.org>. Ideally, inhaled steroids should be withheld until the series is completed or required doses kept constant and as low as possible.

If the person is currently not exposed at work, serial PEF can be measured during a 2-week 'run in' period, followed by a return to work. All recordings should be entered onto a computer for analysis using suitable software. Computer-based analyses of PEF may be helpful in the diagnosis of OA. At least one software program calculates a work-effect index from discriminant analysis based on pattern recognition. Such anal-

ysis allows charts to be graded positive, equivocal or negative for a diagnosis of OA. A positive chart has a quoted sensitivity of approximately 75% and a specificity of 95% for a diagnosis of OA, although these estimates are quality dependent, and pooled estimates suggest 64% sensitivity (95% CI 43% to 80%) and a specificity of 77% (95% CI 67% to 85%).⁵ It is important to note that PEF charts do not confirm a specific cause, nor do they distinguish OA from work-aggravated asthma. Any problems with interpretation of serial PEF charts should be referred to a specialist centre.

Immunological testing

New evidence supports skin prick and serological testing as sensitive for detecting specific IgE caused by most high molecular weight agents, but these tests are not specific for diagnosing asthma or OA. Skin prick and serological tests are less sensitive for detecting specific IgE and OA caused by low molecular weight agents and while specificity may be higher they are not specific for diagnosing OA.

Non-specific bronchial responsiveness

While assessment of non-specific bronchial responsiveness is a useful diagnostic investigation, single and serial measures have only moderate specificity and sensitivity for the validation of OA.

Specific broncho-provocation testing

Comments relating to specific broncho-provocation testing remain unchanged. These tests should be performed only in specialised (tertiary) centres. A positive test identifies the cause of OA, provided exposures received are equivalent to those in the workplace. Negative tests do not necessarily exclude OA, as the challenge may not adequately reproduce exposures at work. Exposure received during SBPT should be measured if practical.

Alternatively, workplace challenge may be used (a variation of serial PEF or serial FEV₁ measurements). This usually involves frequent monitoring of FEV₁ or PEF on multiple days of work, during and between periods of exposure to the suspected agents. One way of approaching this type of challenge is to take data from non-exposed days to calculate a mean and 95% CI of the 'expected' FEV₁ at each time point. These are compared with FEV₁ values measured on exposed days.

Other tests

Newer techniques are available to investigate potential cases of OA. The role of fractional exhaled nitric oxide measurements in the diagnosis of OA has not been established. A normal value does not exclude a diagnosis of OA.

With regard to sputum eosinophilia for the diagnosis of OA, such measurements may be helpful in the diagnosis of OA, although the absence of sputum eosinophilia does not exclude a diagnosis of OA.

Management

Medical management

The pharmacological management of OA does not differ from the management of asthma that is not work related (summarised by the BTS at <http://www.brit-thoracic.org.uk/>). Once a diagnosis of OA is confirmed, the patient should be advised (preferably verbally and in writing) that the prognosis is improved by early and complete removal from exposure.

Symptoms and functional impairment associated with OA may persist for many years after avoidance of further exposure to the causative agent. Evidence supports the view that OA may become a chronic condition, similar to non-OA, and may require similar prolonged medical management.

Patients with confirmed or possible OA should be followed up at a specialist centre while risks of continuing exposure to allergen remain. The recommended follow-up is every 3 months for 1 year, and then every 6 months thereafter.

Patients with confirmed OA who have left work, or who have no ongoing asthmagen exposure risk, should be followed up for a minimum of 3 years at a specialist centre.

Communicating with the workplace is useful, but requires the patient's written consent. Patients should be informed of the possible adverse health effects of continuing exposure to themselves and to co-workers should they not permit necessary workplace investigations.

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