
Review Article

Critical Aspects of the History of Occupational Asthma

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Abstract

The medical history is the gateway to the diagnosis of occupational asthma. The medical history should indicate whether a patient's asthma began during a work period and whether the asthma worsens during work periods or improves on days when the patient is off work or on holidays. A suspicion of sensitizer-induced occupational asthma will increase if the patient was exposed to a recognized respiratory sensitizer in the workplace at the time of the onset of symptoms or if the patient had associated symptoms of allergic rhinitis and conjunctivitis. A history of accidental high respiratory irritant exposure shortly before the initial onset of symptoms would raise the possibility of irritant-induced occupational asthma. Although such features of the history are sensitive indicators of occupational asthma, they are not specific and should therefore be followed by further investigations to confirm the diagnosis of asthma and its relation to the workplace exposure. The earlier the diagnosis is suspected and investigated, the better the outcome is likely to be for the patient.

Occupational asthma (OA) is defined as asthma that is due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace.¹ It has been estimated to account for 10 to 15% of all adult-onset asthma.² It is most often caused by sensitization to a workplace substance (via immunoglobulin E [IgE] antibody responses or other immunologic mechanisms) but can also be due to high-level irritant exposure in about 5% of all cases (irritant-induced asthma,³ of which the clearest example is reactive airways dysfunction syndrome [RADS]⁴). This article will not discuss work-aggravated asthma, which is the aggravation of underlying asthma by factors in the workplace such as exertion, cold air, dusts, smoke, or fumes.

The best medical outcome for those with OA related to a sensitizer is associated with removal from further exposure to the sensitizer, especially early after the onset of symptoms and when the asthma is relatively mild.⁵ Nevertheless, the diagnosis can result in very significant socioeconomic effects, even with appropriate workers' compensation support.⁶ An accurate and early diagnosis is therefore of great importance, to allow a better medical outcome and to facilitate early relocation within the same company or retraining to give the worker the best chance for reentry at another workplace. Unfortunately, there are often delays in the diagnosis of OA, and the diagnosis may not be reached until several years after the onset of symptoms.⁷ A recent chart review at an occupational lung clinic in Ontario showed a mean time to diagnosis of over 3 years. Delayed suspicion of work-related asthma by the primary care physician was related to the patient's fear of losing work and to a lack of inquiry by the physician about the relationship of the asthma to the patient's work.⁷ Lower income and education levels were also associated with a longer time to diagnosis.

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OA will not be suspected without a careful history. The symptoms of OA are identical to the symptoms of nonoccupation-related asthma—episodic wheeze, dry cough, shortness of breath, and chest tightness—and may occur singly or in combination. However, the physician should ask at least the following key questions when assessing any patient with asthma that starts or worsens during the patient's working life:

- Was there an unusual workplace exposure within 24 hours before the onset of initial asthma symptoms?
- Is there a difference in asthma symptoms during weekends or other days away from work?
- Is there any difference in asthma symptoms on holidays away from work?

These questions have implications as explained further.

An unusual exposure at work—a spill or some other high-level exposure to a potentially irritant chemical, especially within 24 hours before the onset of initial asthma symptoms and especially if symptoms were severe enough to lead the worker to seek emergency treatment at that time—would raise the suspicion of irritant-induced asthma or RADS. RADS is the most definitive form of irritant-induced asthma and has the following features (most of which are based on history, as described by Brooks and colleagues⁸):

- Onset within 24 hours of a high-level exposure to a respiratory irritant
- Symptoms typical of asthma
- No previous history of lung disease in the patient
- Symptoms of asthma that persist for at least 3 months after onset
- Pulmonary function findings that show airflow limitation with a significant bronchodilator response or that show a positive methacholine or histamine challenge response

Although these criteria (for RADS) are the most stringent criteria for irritant-induced asthma, some reports have described the onset of irritant-

induced asthma at a later point, after a high-level irritant exposure (up to a week later in some reports). Some authors have reported this syndrome to occur with nonmassive irritant exposures although the certainty of its being caused by the patient's work becomes less as the criteria become less stringent. A positive response to the above-mentioned screening question should therefore be followed by further questioning to determine the conditions of exposure, the time of the onset of asthma symptoms after the exposure, the duration of the symptoms, and a history of likely previous lung disease or previous allergic symptoms. The potential diagnosis is then further investigated by objectively confirming a diagnosis of asthma; objectively assessing the accidental exposure conditions when possible; and by reviewing previous medical records to exclude prior lung disease when possible.

Two of the key questions listed above—Are asthma symptoms better on weekends away from work, and are asthma symptoms better on holidays?—are not specific to work-related asthma (patients with asthma can feel better when relaxing on a beach or elsewhere on weekends or on holidays, compared with when they are working). Nevertheless, a response that indicates improvement when away from work is a sensitive indicator for OA related to a work sensitizer and also for work-aggravated asthma. Among patients who were being objectively assessed for OA in a study from Québec, 88% of those who were objectively confirmed to have OA gave a positive response to the question about improvement in symptoms on holiday, as opposed to 76% of those who did not have OA (but who still may have had work-aggravated asthma).⁹ Therefore, although the question appears to be a sensitive one, further information is needed for a diagnosis of OA.

Similar responses to this question were identified among Ontario Workers' Compensation claimants.¹⁰ Among those whose claims of OA were accepted, 70% reported improvement during weekends off work and 88% reported improvement when on holidays, as opposed to 41% and 54%, respectively, of those who were deemed to have asthma unrelated to work. Thus, a relatively high

percentage of those with a final medical diagnosis or compensation decision of unrelated asthma had a positive history even though it may have been expected that such a history might have increased the likelihood of a positive compensation decision.

In a more recent study from Belgium and Québec, Vandénplas and colleagues found that (1) wheezing reported from history to be related to work was the most specific symptom for OA, occurring in 88% of those with OA; (2) an associated history of work-related eye and nasal symptoms had a relatively good predictive value for OA; and (3) additional associated itching of the nose and eyes in OA was related to high-molecular-weight sensitizers.¹¹ In contrast, loss of the voice, as part of the work-attributed symptoms, was negatively associated with OA. Vandénplas and colleagues reported a 74% correct prediction of the diagnosis in the study when using a predictive model from patient history as opposed to specific challenge tests (their “gold standard”).

A detailed occupational history can also help to better assess likely exposure to a known workplace respiratory sensitizer. It should focus particularly on exposures at the time the asthma started or worsened at work. Workers whose occupations are commonly associated with sensitizer-induced OA include the following:

- Workers exposed to diisocyanates (eg, while making polyurethane foam, using spray paints, or using these compounds in moulds in foundries or in the manufacture of particleboard)
- Workers exposed to animals (eg, veterinarians, farmers, animal laboratory workers)
- Bakers, workers exposed to grain (farmers, grain handlers), and workers exposed to airborne food proteins
- Workers exposed to wood dusts (eg, carpenters, construction workers)
- Metal workers exposed to complex platinum salts, soldering flux, or metal salts

Commonly reported causes of irritant-induced OA include accidental spills or other high-level exposures to diisocyanates, acids, chlorine or chlorones, irritant alkaline dusts, and smoke.

The patient’s job title may not give a clear indication of his or her exposure, and the patient also may be exposed to dusts or fumes from products used by nearby workers. Therefore, it is important to question the patient about his or her job description and that of others in the same work environment. However, the patient may not know what he or she is exposed to at work, and it can be very helpful to ask the patient to obtain copies of Material Safety Data Sheets (MSDSs) from the workplace for review. Workers are entitled to receive copies of these sheets, which list the known hazardous ingredients in products used at the workplace. In Canada, if a recognized respiratory sensitizer constitutes $\geq 0.1\%$ of a chemical product, it must be listed as a hazardous ingredient (the US requirement is $\geq 1\%$). Providing the patient with a note asking the patient’s supervisor or workplace Health and Safety Committee to provide MSDSs for products used by the patient and by co-workers in the area can assist in obtaining these. However, MSDSs are designed to list toxic effects rather than hypersensitivity responses and may not list “natural products” that contain potentially sensitizing proteins, so it may be necessary to request additional information from the product manufacturer or from the workplace (with the patient’s permission). In addition, there are over 250 reported workplace sensitizers, and OA can occur in multiple workplace settings. (Many of these sensitizers are listed on-line at <www.asmanet.com>.) However, the absence of an identified exposure to a sensitizer does not exclude OA.

There are other aspects of the history that are very useful. Patients with OA from exposure to high-molecular-weight sensitizers that induce OA through an IgE-antibody-mediated mechanism commonly have associated symptoms of allergic rhinitis and conjunctivitis when exposed to the sensitizer at work.¹² These symptoms may start prior to or concurrently with the onset of asthma.¹³

The initial onset of asthma symptoms that are related to a sensitizer (an actual or presumed immune response) occurs after a latent period of exposure, which can range from weeks to years. This is in contrast to the onset of irritant-induced asthma, which most typically begins within 24 hours after a very high irritant exposure. Once sen-

sensitizer-induced OA is present, asthma symptoms related to work exposures can range from immediate responses (ie, within minutes after further exposure to the sensitizer) to late responses (typically 4–6 hours after exposure, more commonly as an isolated response when the sensitizer is a low-molecular-weight agent) or can be a dual asthmatic response (an immediate response followed by a late response). The association with work may be less obvious to the patient if an isolated late response occurs, because the worsening of symptoms may begin or progress after the work shift. In addition, improvement away from work may not be evident for a few days away from exposure and therefore may be noted only at the end of a weekend away from work or during a holiday period.

Improvement away from work has been considered a sensitive marker of OA from history, but studies of workers with OA who have been removed from further exposure at work have identified a subgroup of workers whose asthma does not improve or whose asthma even worsens by the time of follow-up assessment. Lack of improvement has been seen more commonly when there has been a longer period before diagnosis and more severe asthma at the time of diagnosis, emphasizing the need for physicians to think of OA and to ask asthmatic patients early on about a work relationship.

Although the history is clearly an essential part of making the diagnosis, the incorrect prediction in 26% from history alone in the recent study¹¹ emphasizes the need for further assessment of those with a positive response (as detailed in recent guidelines and reviews^{14,15}), to avoid unnecessary job loss. The history is critical to the diagnosis of OA; without an appropriate history, the patient is unlikely to undergo the objective tests needed to make the diagnosis^{14,15} and allow appropriate workplace environmental changes.

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