

Clinical Assessment of Occupational Asthma and its Differential Diagnosis

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KEYWORDS

- Occupational asthma • Peak expiratory flow monitoring
- Specific inhalation challenges • Diagnosis • Skin tests
- Sputum induction • Questionnaire

Asthma is the most frequent respiratory disease, affecting up to 8% of adult working populations. Population-based studies have estimated that the proportion of adult-onset asthma caused by occupational exposures ranges from 5% to 10% in Europe, 10% to 23% in the United States, to 17% to 29% in Finland. The proportion of persons with asthma who experience worsening symptoms caused by work activities or environments is not well known, ranging between 16% and 31%, depending on the study design and population studied.¹

Work-related asthma refers to asthma that is attributable to, or is worsened by, environmental exposures in the workplace. It can be categorized into occupational asthma (OA) and work-exacerbated asthma (WEA). Several definitions have been given to both terms.¹⁻³ OA is defined as asthma caused by sources and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace.⁴ Two types of OA are distinguished based on their appearance after a latency period or not. The most frequent type, which is usually quoted as OA, appears after a latency period leading to sensitization, either allergic or immunoglobulin E (IgE)-mediated like most high- and certain low-molecular-weight agents or through unknown mechanisms. The other type does not require a latency period and includes irritant-induced asthma or reactive dysfunction syndrome, which may occur after single or multiple exposures to high concentrations of nonspecific irritants.^{5,6} WEA, or work-aggravated asthma, can be defined as the worsening of preexisting or coincident (new-onset) asthma by workplace exposures.

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The purpose of the review is to outline the clinical assessment and differential diagnosis of sensitizer-induced OA.

HOW TO MAKE THE DIAGNOSIS OF OA?

The diagnosis of OA relies on the objective evidence that asthma is triggered by work exposure; it needs to be distinguished from WEA and other conditions that may mimic asthma at work.

The different steps involved in the investigation of work-related asthma are history; pulmonary function tests; immunologic tests; combined results of serial peak expiratory flow (PEF) monitoring, nonallergic bronchial responsiveness (NABR), and sputum induction; and specific bronchial challenges. Although specific inhalation challenges (SIC) are considered the diagnostic reference standard, all steps involved in the investigation have their own value and contribute to establishing the diagnosis. Combining the various elements strengthens the likelihood of a proper diagnosis.

History

The classical history of OA is one of a worker whose asthma is worse at work, improving over weekends or holidays. However, even workers without work-related asthma regularly report improvement of asthma during weekends and holidays (41% and 54% of cases, respectively).⁷ Furthermore, this pattern is often absent because symptoms are also usually present outside the workplace and triggered by exposure to irritants, such as cold air, fumes, or exercise. The concomitant occurrence of rhinoconjunctivitis at work, especially in a worker exposed to high-molecular-weight chemicals who develops asthma, is surely suggestive of OA.⁸ Symptoms may develop after latency periods of only a few weeks or several years; the duration of exposure tends to be shorter for low-molecular-weight chemicals.⁹ A previous history of asthma does not exclude the diagnosis of OA.

Physicians evaluating workers with possible OA should inquire about exposure to potential sensitizers and irritants, either directly or indirectly. Work that generates dust, such as the use of abrasive materials, or devices and work locations in which there are strong odors or where combustion or chemical reactions are taking place are often problematic because they may trigger asthma symptoms through irritant or sensitizing mechanisms. Material safety data sheets may be useful to document the nature of exposure to any sensitizing or irritant agents; but they are not always consistent in their format and content and may not identify sensitizing substances as such, particularly if present at low concentrations in the product or its ingredients (ie, <1%).¹⁰

Even if the history is essential to make a diagnosis of asthma, it is often misleading. Indeed, several studies have shown that asthma is overdiagnosed in the general population because of the lack of objective confirmation of the diagnosis. Aaron and colleagues¹¹ showed that among 492 subjects with physician-diagnosed asthma, nearly one-third did not have asthma when objectively assessed after medication withdrawal and proper follow-up. In occupational settings, the authors have previously shown that a diagnosis of OA based on clinical history has a positive predictive value of only 63%¹²; among those with a very likely or likely diagnosis of OA, 13.5% had even no objective evidence of asthma. Furthermore, among 169 subjects referred for respiratory symptoms suggestive of work-related asthma, Chiry and colleagues¹³ have shown that 69 (40.8%) had no objective evidence of asthma, although the type and severity of their respiratory symptoms were similar to those reported by subjects with confirmed asthma except for wheezing, which was more frequent in the latter group.

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