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Various Occupational Allergies and their Clinical Symptoms

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Abstract

In general, it is important to make an accurate diagnosis of occupational asthma (OA). The disease not only has a significant impact on the health of affected workers, but also has significant socioeconomic impacts on workers, their employers and society at large. Failure to diagnose OA can lead to continued exposure to the pathogen and exacerbation of the disease. Conversely, the absence of a diagnosis of OA can lead to inappropriate exclusion from exposure, with unnecessary economic and social consequences. The most accurate assessment is a specific inhalation challenge at an experienced center, but this is a scarce resource and often relies on other tests. Based on available evidence, a practical diagnostic algorithm is proposed that can be adapted to the suspected pathogen, diagnostic objectives, and available resources. For better or worse, many of the techniques and their interpretation are only available in specialized centers, and when in doubt, referral to such centers is probably recommended. The establishment or development of such specialized centers should greatly improve the diagnostic assessment of work-related asthma.

Keywords: Diagnosis; Occupational asthma; Inhalation; Work-related

Introduction

The work environment can lead to the development of different phenotypes of work-related asthma [1-3]. The term "work-related asthma" now generally includes both asthma that is attributed to work (i.e., occupational asthma) and pre-existing or concomitant asthma exacerbated by non-specific stimuli at work [4]. Occupational asthma (OA) is defined as immunologically mediated sensitization (i.e., "immunological OA" or "sensitization") to certain substances in the workplace. It may result from either substance-induced OA - or through exposure to high levels of irritating compounds (i.e., "irritant asthma"), most commonly represented by reactive airway dysfunction syndrome, [5] of plant and animal origin, and low molecular weight (LMW) chemical IgE-related hypersensitivity mechanisms [6], whereas in most LMW agents (isocyanates, persulfates, aldehydes, and other wood flours), the immunological mechanisms are not yet fully elucidated. In most cases, OA remains a diagnostic challenge for clinicians due to the lack of a simple test that can diagnose the disease with a sufficiently high degree of confidence. Instead, the diagnostic process consisted of combining various steps in iterative processes [1-3]. Quantitative evidence using repeated testing regimens or combinations of diagnostic tests to guide a stepwise diagnostic approach. In addition, these documents recommended immunological tests to detect her IgE sensitization to occupational pathogens, but did not include the results of these tests in the diagnostic decision-making process. The purpose of this document is to critically analyze the available information on the effectiveness and practicality of diagnostic procedures, in order to provide practical guidance to physicians facing the management of work-related asthma symptoms.

Classification of occupational asthma

Work-Related Asthma (WRA) comprises two major entities, OA, defined as a type of asthma "caused" by the workplace and work-exacerbated asthma (WEA), which refers to asthma triggered by various work-related factors (e.g., aeroallergens, irritants, or exercise) in workers who are known to have pre-existing or concurrent asthma, (e.g., asthma that is occurring at the same time but is not caused by workplace exposure [7, 8].

Allergic, induced by sensitizers, this appears after a latency period necessary for the worker to acquire sensitization to the causal agent. It encompasses OA caused by most high (HMW) and some low-molecular-weight (LMW) agents for which an IgE-mediated mechanism has proven, and OA induced by some specific LMW agents in which the allergic mechanisms responsible have not yet been fully characterized. Allergic OA is the most common type of OA, accounting for more than 90% of cases.

Irritant-induced OA (IIOA) (or "non-immunologic/non-allergic OA") is a form of OA characterized by the development of asthma (or the reactivation of quiescent asthma) caused by exposure to irritant substances at the workplace that are capable of inducing an inflammatory reaction of the airways and non-specific bronchial hyperresponsiveness (NSBH) through non-sensitizing mechanisms. There are two subtypes; the "acute irritant-induced OA", or "Reactive Airways Dysfunction Syndrome" (RADS) [9] characterized by the onset of asthma symptoms within 24 hours after a single, most often accidental, high-level exposure to a wide variety of irritant substances in subjects without pre-existing asthma; and "sub-acute irritant-induced OA" [10] in this case asthma develops, often more insidiously, in subjects with a history of multiple symptomatic high-level/moderate exposures. A causal relationship between occupational exposure to irritants and the development of asthma can be established with a reasonable level of confidence for asthma resulting from single high-level or multiple exposures to irritants.

Bronchitis

It is a condition associated with chronic cough associated with occupational exposure, characterized by asthma-like sputum

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eosinophilia, but unlike asthma, patients may experience a variety of airway obstructions and bronchial hyperactivity. There is no evidence of it can be classified in the spectrum of work-related respiratory illnesses [11].

Diagnosis of OA

Diagnosis of OA should be made using objective methods [12, 13, 14]. Complete cessation of exposure to the causative agent, which usually means removing the affected individual from work, is the mainstay of OA treatment. Proper management of patients with suspected OA therefore depends on establishing a definite diagnosis. It is important that workers are not advised to leave the workplace until a proper investigation has been conducted. Diagnosis can be very difficult in patients who have left work and cannot return, so early referral and medical evaluation are necessary. Several aspects should be considered when examining a patient with suspected osteoarthritis. OA caused by occupational allergens must be distinguished from incidental episodes of non-work-related asthma. In addition, other causes of asthma-like symptoms should be included in the differential diagnosis. Work-related exacerbation of latent or pre-existing asthma due to exposure to known asthma triggers at work is one of the most common and conflicting differential diagnoses with true her OA. Work-related changes in airway diameter can occur in work-related asthma, but bronchial responsiveness to methacholine is not usually reduced after exposure [15]. Diagnosis of OA requires the use of a stepwise combination of different functional, environmental, and immunological methods. Methods to assess airway inflammation in the clinical setting have recently been introduced into OA research [16]. In most cases of OA, the perpetrator can be identified. Identifying the environmental and specific occupational causes of OA has the potential to profoundly alter the clinical outcome of affected patients. Therefore, appropriate clinical and immunological investigations should be performed for diagnosis to identify the disease and the causative agent.

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Clinical symptoms

Medical history is an important component of OA assessment and usually provides important information for diagnosing asthma and work-related possibilities. Moreover, medical history provides a rational basis for the selection and timing of additional diagnostic procedures. The medical history should include several key questions such as incidence of asthma symptoms, asthma severity and persistence, temporal relationship between occupational exposure and disease exacerbation, clinical course of asthma, and known triggers and co-morbid factors. OA should be suspected in him in adults with new-onset asthma, whereas work-related asthma should be considered in patients with pre-existing asthma symptoms. Suspicion is heightened when patients report that their asthma symptoms are worse on weekdays than on weekends and holidays. This exacerbation can occur within minutes (immediate asthmatic reaction) or hours after the onset of workplace exposure. Symptoms may appear in the evening or at night after leaving work (a delayed asthmatic reaction). Patients may also exhibit immediate and delayed (biphasic or biphasic) asthmatic reactions. Medical history was found to be highly sensitive (87%) but less specific (22%) for diagnosing OA caused by multiple pathogens [17]. Similar results have been obtained in the diagnosis of latex-induced asthma [18]. It is usually assumed that OA patients are symptomatic at work and noticeably better on weekends and holidays. However, this distinct pattern occurs primarily during disease onset. After prolonged exposure, patients' illness tends to show an insidious course. In this situation, the patient presents with mainly nocturnal symptoms and may react not only to the work material, but also to non-specific irritants found outside the workplace, and in the final stage, the patient may be affected outside the workplace can lose reversible patterns in work. In that case, suspicion of work-related asthma can be much more difficult. A common misconception is that asthma is not work-related if asthma symptoms do not improve outside of work should be considered good reason to investigate its potential impact on asthma symptoms. The medical history should also identify risk factors. Atopic patients are at increased risk of developing her OA due to HMW substances (such as animal and plant allergens). Therefore, atopic status should be assessed in all individuals with suspected OA by performing skin prick testing with a panel of common aeroallergens. In addition, atopic patients with OA may also experience asthma exacerbations when exposed to common aeroallergens outside of work. The patient's smoking history is also important. Cigarette smokers are at increased risk of sensitization and asthma through IgE-mediated mechanisms. Tobacco smoke acts synergistically with atopy and increases the risk of disease.

Physical examination findings in people with osteoarthritis are often unremarkable. If present, they are no different from other types of asthma and are diagnostic. History and physical examination should also note the presence of associated allergic disorders such as rhinitis, sinusitis, conjunctivitis, urticaria, and dermatitis. Nasal and ocular allergic symptoms usually precede and accompany immune OA. Certain pathogens that cause osteoarthritis can cause food allergies after ingestion by some patients, so specific questions should be asked about possible symptoms of food allergy (e.g. eggs), crustacean protein, soybean meal, etc).

Potential exposures should be listed for regular and occasional work. Occupational and environmental history should assess the intensity (duration and concentration), peak concentration, and frequency of exposure. Symptoms of mucosal or skin irritation and odor perception are useful for quantitative exposure assessment. The list of substances found in the workplace can be compared with the extensive list of known pathogens of occupational asthma [19-21]. However, the absence of a suspect substance on such a list does not preclude a diagnosis of OA. This is because new occupational allergens are listed each year. It is also worth obtaining previous occupational health records whenever possible. Data on previous spirometry, problems with methacholine inhalation, blood tests, and chest x-rays may be particularly helpful in assessing the course and severity of the condition. In some patients, her OA may develop after sensitization to a single environmental factor, while in others; sensitization to various inhaled allergens may lead to respiratory disease. An example of a complex environment with many potential allergens is a bakery or confectionery (grain flour, soy flour, fungal enzymes, storage mites, mold, egg protein, etc.). Some potential allergens (such as natural rubber latex) can also induce and induce an allergic inflammatory response in the airways within certain exposures. Diagnosis of irritant-induced OA relies entirely on clinical and occupational/environmental history, with documented reduction in airway diameter and objective evidence of nonspecific bronchial hyperactivity [22].

Some immunological assessment methods for OA

Many drugs that cause OA are allergenic. Immunological tests that

measure IgE responses are valuable when combined with other data. A major limitation of immunological evaluation is that the detection of her IgE antibodies to a specific allergen is indicative of sensitization, whereas sensitization may occur in exposed individuals who do not have asthma or other allergic symptoms can occur.

High-molecular weight agents

Most HMW agents are complex mixtures of polypeptides that act as perfect antigens and stimulate IgE synthesis (animal allergens, flour, latex, etc.). In some cases relatively pure proteins or peptides (such as enzymes) and recently purified recombinant allergens are available as clinical tools. Skin prick testing (SPT) is commonly used to identify sensitization to HMW allergens because it is safer, more specific, and more sensitive than most in vitro assays. However, a positive skin test response only confirms exposure and sensitization. Some individuals may respond positively to SPT and not exhibit allergen-induced asthma. Few occupational allergen extracts are commercially available for skin testing, most are not standardized, and some of the available extracts may not be sufficiently potent and reliable [23].

Combining a positive skin prick test with nonspecific bronchial hypersensitivity is an effective method of documenting IgE-dependent osteoarthritis, at least for some HMW allergens. In vitro tests such as radioallergosorbent test (RAST) and enzyme-linked immunosorbent assay (ELISA) for detection of allergen-specific IgE antibodies are also available. However, skin tests are usually more sensitive than in vitro tests for early detection of sensitization. Under certain circumstances, the specificity of IgE measurements to working substances can be compromised by allergic cross-reactivity with common aeroallergens or panallergens such as profilin. HMW allergens are also suitable for more detailed immunological studies such as: B. Electrophoresis in combination with immunoblotting, cross-radiation immunoelectrophoresis, and other methods that allow identification and characterization of allergen components. Identification of the specific causative allergen for OA may allow determination of different allergen components from the same allergen source under different exposure conditions (eg, soybean meal-induced OA and soybean-induced epidemic asthma). Immunological inhibition studies may also be used to detect allergic cross-reactivity between different antigens, which may or may not be clinically relevant.

Low-molecular weight agents

Some LMW chemicals, such as anhydrides, platinum salts, persulfates, and reactive dyes, act as haptens and stimulate IgE production by binding endogenous proteins to form hapten-protein complexes increase. Many low-molecular-weight substances, such as isocyanates, plicatic acid, and glutaraldehyde, can induce her OA, but only a minority of affected workers induce specific her IgE antibodies. In other cases, specific his IgE antibodies were not detected (such as acrylates). Skin tests cannot adequately assess responses to LMW antigens. Skin testing is therefore of little use for most small molecules, but is a diagnostic tool for workers sensitized to platinum complexes and, in some cases, patients exposed to other metal salts and persulfates. It is excellent as although protein conjugates of some reactive chemicals such as diisocyanates and anhydrides are readily available, the optimal ligand-to-protein conjugation ratio has not been defined. Serological testing has limited utility in diagnosing isocyanate-induced asthma, but is preferred to skin testing in diagnosing his OA due to acid anhydrides. Cell-mediated hypersensitivity may be directly involved in mediating airway inflammatory responses leading to OA induced by certain LMW agents [24]. However, the use of assays for allergen-specific cellular immune responses has been investigated to a very limited extent and has no clinical application to date.

Conclusion

A cornerstone of the diagnosis of occupational asthma is establishing a causal relationship between exposure to the causative agent and both clinical symptoms and physiological changes. The medical history should include detailed questions about the onset and severity of asthma symptoms, the temporal pattern between occupational exposure and disease exacerbation, and the circumstances surrounding the course of the disease. However, medical history is more reliable to rule out than to confirm a diagnosis of OA. Occupational exposure assessment is an important step in assessing the contribution of the workplace to a patient's asthma. Information from the site can be obtained by walking around and referring to industrial hygiene data and material safety data sheets. In special circumstances, qualified personnel may perform air sampling monitoring using regional or personal sampling pumps.

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