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EVALUATION OF PATIENTS WITH OCCUPATIONAL ASTHMA

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ANNOTATION: Asthma is a chronic lung disease affecting people of all ages. It is caused by inflammation and muscle tightening around the airways, which makes it harder to breathe. Symptoms can include coughing, wheezing, shortness of breath and chest tightness. These symptoms can be mild or severe and can come and go over time. People with under-treated asthma can suffer sleep disturbance, tiredness during the day, and poor concentration. Asthma sufferers and their families may miss school and work, with financial impact on the family and wider community. If symptoms are severe, people with asthma may need to receive emergency health care and they may be admitted to hospital for treatment and monitoring. In the most severe cases, asthma can lead to death.

Key words: Asthma, occupational asthma, irritant induced asthma, immunologically mediated asthma, nonimmunologic – irritant induced athma, occupational history, exposures.

Main part:

The label "asthma in the workplace" encompasses several entities: (1) asthma exacerbated at work by various environmental conditions; (2) occupational asthma; and (3) variants (eg, eosinophilic bronchitis) [1]. Occupational asthma is a type of work-related asthma that is caused by immunologic (identified or presumed) and nonimmunologic stimuli present in the workplace [2].

Types of OA:

Two main types of occupational asthma have been recognized [1,3]:

●Immunologically-mediated. This type includes immunoglobulin E (IgE) and non-IgE-mediated responses following chronic exposure and respiratory sensitization to high or low molecular weight agents.

●Nonimmunologic, irritant-mediated, also called irritant-induced asthma. This type includes reactive airways dysfunction syndrome (RADS) caused by a single high-level exposure to an irritant, irritant-induced asthma caused by multiple high-level exposures to an irritant, and possibly asthma caused by chronic lower level of exposure, although the latter is controversial.

Some occupational agents, such as diisocyanates, may induce asthma through more than one mechanism. As an example, asthma may develop in some subjects after an accidental high-level diisocyanate exposure (eg, spill) by causing acute airway injury resulting in reactive airways dysfunction syndrome (RADS). In others, respiratory sensitization to diisocyanate develops with lower levels of exposure. Exposure to high levels of diisocyanates causing irritant-induced occupational asthma can also promote the development of immunologic occupational asthma caused by diisocyanates.

The various types of occupational asthma may include different phenotypes or endotypes [4]. As an example, in an analysis of 187 patients with diisocyanate asthma (DA), different clusters were populated, similar to common asthma, with the largest cluster including the youngest patients with

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the shortest duration of exposure to the sensitizers, while another cluster included older male patients with worse lung function and longer occupational exposure [5]. This suggests that occupational asthma (as probably many others) is phenotypically heterogeneous. There are several phenotypes (clinical presentations) and endotypes (mechanistic pathways) of occupational asthma that are characterized by their clinical features, their pathogenesis and inflammatory profile [6]. However, more research is needed to characterize these phenotypes/endotypes.

OA should be suspected in every adult with new onset asthma, as OA accounts for approximately 10 to 16 percent of adult onset asthma with some between study variability [12]. Patients with reappearance of childhood asthma and deteriorating asthma control on stable therapy should also be screened for OA [5]. For all patients, the evaluation begins with an occupational history, focused on known or potential sensitizing agents. Our approach to testing combines complementary tests, as individual tests have limitations when viewed in isolation.

Occupational history — The onset of symptoms relative to exposure varies and a work-related association may not be spontaneously reported by the patient. Thus, the clinical history in patients with possible OA should include detailed questioning about the job description and potential exposures to causal agents, in addition to the routine evaluation of adult-onset asthma [12]. The clinical history, while important, is not sufficient to confirm or exclude the diagnosis of OA [15].

Exposures — All adults with asthma should be questioned not only about their current occupation and exposures, but also about their previous occupations and exposures. Once the patient's occupation is ascertained, the potential associated exposures can be evaluated in greater detail. Asking about exposure to vapors, gas, fumes, or dust may help the patient's recall [8].

As examples, the job description of "engineer" or "clerk" does not yield a full account of the actual exposures in the workplace. Further questioning may reveal indirect exposure to a sensitizing agent depending upon the particular work environment (eg, a work station adjacent to the paint booth in an auto body shop, desk near the site of the flood in an office building, work space adjacent to packaging area in epoxy paint factory). Janitorial and healthcare work may involve use of amines or bleach in cleaning. Exposure to cigarette smoke (active or passive) can occur in many occupations and may trigger symptoms on an irritant basis.

Time course — The natural history of immune-mediated OA is characterized by the following progression.

- Onset of exposure
- Sensitization
- Onset of upper and lower airway inflammation
- Clinical disease
- Cessation or persistence of exposure
- Cure, improvement, or persistence of asthma

The latency period between the onset of exposure and the onset of symptoms is highly variable in OA, ranging from months to years. The latency period may vary according to the type of agent, being shorter with exposure to low molecular weight (LMW) agents, such as diisocyanates and plicatic acid (Western red cedar), than with HMW agents.

As an example, among 1179 OA patients, those exposed to HMW agents developed symptoms after a median of eight years of exposure compared with 5.1 years for those exposed to LMW agents

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[3]. The latency period also varies between HMW agents; sensitization to laboratory animals occurs more commonly and rapidly than sensitization to flour [4].

Atopy — The possibility of pre-existing asthma and atopy (genetic predilection to produce specific immunoglobulin E (IgE) following exposure to allergens) should be explored in all patients with suspected occupational asthma. Thus, we ask patients about prior symptoms suggestive of asthma, allergic rhinitis, and atopic dermatitis. This information can help determine whether the patient has new onset disease or a pre-existing process that has worsened.

Skin and immunologic testing — Skin tests and immunoassays for serum specific IgE (ssIgE) have several roles in the evaluation of OA: they can identify sensitization to potential culprit allergens (when available); they can be used to identify sensitivity to common (nonoccupational) aeroallergens that can contribute to symptoms; and newer molecular methods may eventually improve the ability of testing to relate specific epitope sensitivity to the likelihood of disease.

The presence of immediate skin test reactivity or ssIgE reflects specific sensitization, but a positive result can be found in some patients without symptoms of asthma or rhinoconjunctivitis. Therefore, it is important to document objective evidence of physiological changes (eg, airflow obstruction, airway hyperresponsiveness, and/or increased sputum eosinophils) related to exposure, in addition to skin test reactivity or positive testing for specific IgE.

Pulmonary function tests — Baseline spirometry before and after bronchodilator should be obtained in virtually all patients with suspected OA. If airflow limitation is not present, the next step is either nonspecific bronchoprovocation testing or serial peak flow or spirometry at work and at home. For patients who are no longer working, nonspecific bronchoprovocation is used to document the presence or absence of airway hyperresponsiveness.

Spirometry before and after bronchodilator — Baseline spirometry before and after bronchodilator is used to determine the presence and severity of any airflow limitation [15]. The results of spirometry determine the most appropriate next test and are useful as a baseline for future assessments.

Imaging — A chest radiograph is often obtained in adults who present with new onset or work-related dyspnea to exclude causes of dyspnea and cough other than OA. The chest radiograph in OA may be normal or may show hyperinflation. High-resolution computed tomography (HRCT) is usually not needed for the evaluation of a patient with suspected OA, unless an unexplained abnormality is noted on the plain chest radiograph.

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