Occupational asthma

It is caused by the inhalation of harmful substances in the workplace and it only manifests after employment since it is an occupational disease. Occupational asthma represents **2% to 15%** of all cases of asthma, depending on the country. However, it is believed that this number is underestimated by about 5 to 10 times.

Causative agents:

- High molecular weight substances (plant and animal proteins, microbial products)
- Low molecular weight compounds (isocyanates, acid anhydrides, platinum salts)
- Irritant and pharmacological substances (smoke, fumes - Cl_2, NH_3, insecticides)
- Synthetic organic and inorganic chemicals
- Disinfectants

High-risk activities: grain handling, woodwork, paramedics, smoke inhalation.

Etiopathogenesis

Asthma is a chronic inflammatory disease of the respiratory tract. The main cells involved in this process are **eosinophils and mast cells**, and neutrophils and basophils to a lesser extent. Inflammation increases bronchial reactivity, which induces **bronchospasm and manifestations of bronchial obstruction**. If reversible obstruction or bronchial reactivity (or both) is caused by occupational exposure to noxious substances, the condition is termed **occupational bronchial asthma (OA)**.

Two main types of occupational asthma exist:

1. Sensitizer-induced asthma (immunological form)
2. Irritant-induced asthma

1. Immunological occupational asthma

- It occurs only in a small number of cases.
- It occurs after an asymptomatic period.
- The asthmatic response is triggered by substances that the worker has previously tolerated well.
- It involves a specific immunological response to the etiological agents.

High molecular weight substances induce an IgE-mediated response; hence, the detection of **specific IgE antibodies** is possible. How low molecular weight substances can induce this form of asthma is not yet known. Types of **immunological responses** involved: **I, III, or IV**

2. Irritant-induced occupational asthma

The process is not fully understood, but **neurogenic mechanisms and the release of neurotransmitters** probably play a role. It occurs without previous sensitization and does not involve a specific response to etiological agents. It manifests after exposure to highly irritating substances (dust, aerosol, etc.).

Asthma caused by short-term but high-intensity exposure is called **RADS** (Reactive Airways Dysfunction Syndrome).

Pathologic changes

The disease leads to remodeling of the wall of the respiratory tract (thickening of the bronchial wall due to smooth muscle hypertrophy). The bronchial mucosa is heavily infiltrated by eosinophils and lymphocytes. Goblet cell hyperplasia results.

Clinical manifestation

Typical symptoms (shortness of breath, wheezing especially during expiration, cough, chest tightness) appear during exposure (at work). Quite often, patients report the occurrence of eye symptoms, rhinitis, and a feeling of nasal blockage. Symptoms diminish during the weekend, holidays, or when the work environment changes. When symptoms become chronic, they can completely lose their connection to the work environment.

Diagnosis

- Spirometry - **obstructive ventilation disorder**
- Histamine non-specific bronchoprovocation test - **decrease in FEV_1** (forced expiratory volume in 1 second) by at least 20%; MEF_{25-75} (mid-expiratory flow) by at least 30%; 100% increase in resistance;
- A specific inhalation bronchoprovocation test using available probable workplace allergens
- Bronchodilation test - evaluation of reversibility of bronchospasm in patients with permanent obstruction

- Peak expiratory rate (PEF) series - peak-flow meter
- Elimination and re-exposure test

Links

Related articles

- Asthma

References

- PELCLOVÁ, Daniela. *Nemoci z povolání a intoxikace.* 2. vydání. Praha: Karolinum, 2006. 207 s. s. 89-93. https://www.wikiskripta.eu/w/Speci%C3%A1ln%C3%AD:Zdroje_knih/80-246-1183-X.