Allergic asthmas caused by the inhalation of substances consistently recognised as causing allergies and inherent to the type of work

Definition of causal agent

Occupational asthma is a disease characterised by airway inflammation, reversible variable airflow limitation, and airway hyper-responsiveness due to causes and conditions attributable to a particular occupational environment. Specifically, it is induced by workplace exposure to an airborne dust, gas, vapour or fume. Allergic occupational asthma is characterised by a latent period (see exposure criteria); and once established can be provoked by exposure to minimal concentrations of the inducing agent in the workplace.

Main occupational uses and sources of exposure:
Either high-molecular mass agents (usually glycoproteins) of biological origin or chemical substances of low molecular mass. The most common reported causes of occupational allergic asthma are listed below. The list is an open one.

Diagnostic criteria

The diagnosis of asthma is established by the association of episodic dyspnoea with one or more of the following:

- bronchial obstruction significantly reduced by inhaled bronchodilator medication
- non-specific bronchial hyperreactivity
- increased diurnal variability in lung function.

The diagnosis of occupational asthma requires demonstration of a clear relationship between exposure to the causal agent and both clinical and physiological changes. Lung function and bronchial reactivity may become normal after cessation of occupational exposure. Occupational asthma arising from high molecular mass and some chemical agents is associated with the production of specific IgE antibodies.

History:

- occupational exposure to a substance known to induce occupational asthma
- a sequence of symptoms in direct relation to the work schedule. Attacks may begin several minutes or several hours (depending on the allergen) after exposure
- recurrence of symptoms and signs following re-exposure to the same agent

Examination:

There may be no clinical findings on examination but rhonchi can be detected on auscultation of the chest during an asthmatic attack.

Investigations:

For agents that provoke specific lgE antibodies skin prick or serological testing can be used to assess sensitization. Numerous occupational allergens are not standardised and information on the sensitivity and specificity of skin-prick or serological tests is not always available.

Supportive evidence may be provided by:

- serial monitoring of peak expiratory flow or spirometry during periods at and away from work
- specific inhalation challenge (bronchial provocation test). This test is not necessary for disease recognition, however it may be indicated when the diagnosis remains in doubt, in determining the precise causative agent, and in the investigation of new causes of occupational asthma. Testing should be done only in specialised centres with appropriate facilities.

Exposure criteria:

Minimum intensity of exposure: not specified, as there is insufficient evidence for exposure thresholds in occupational asthma and variation in individual susceptibility.

Minimum duration of exposure: occupational allergic asthma requires a sensitisation period usually ranging from a few weeks to years. In exceptional cases it may be as short as a few days.

Maximum latent period: between allergen exposure and the manifestation of clinical symptoms in a sensitized individual - no more than 48 hours. The onset of sensitisation and occupational asthma occur only during employment that involves exposure to the initiating agent.

Induction period: Few weeks to several months. In exceptional cases it may be as short as a few days

Most common reported causes of occupational allergic asthma

Many workplace substances have been identified as capable of inducing occupational asthma. Those that have been well established include:

<u>High molecular mass substances:</u>

- animal-derived allergens (e.g. laboratory animal antigens, cow dander)
- arthropods (e.g. grain mites)
- plant derived allergens (e.g. wheat, rye and soya flour, natural rubber latex)
- enzymes (e.g. protease, amylase)

<u>Low molecular mass substances:</u>

- diisocyanates (e.g. toluene diisocyanate, diphenylmethane diisocyanate)
- acid anhydrides (e.g. phthalic anhydride, trimellitic anhydride)
- amines (e.g. ethylene diamine, paraphenylene diamine)
- fluxes (e.g. colophony)
- components of some wood dusts (e.g. western red cedar)
- metals (e.g. platinum salts)
- drugs (e.g.spiramycin, penicillins, psyllium)
- biocides (e.g. glutaraldehyde, chloramine T)
- plastics (e.g. acrylates)

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This list is not exhaustive.