Allergy

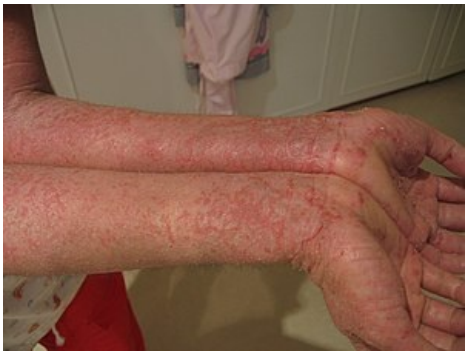
Allergy is a pathological (inappropriate) reaction of the immune system to external noxa – allergens, which are a common part of our environment (pollen, mold, animals, food, insect stings, etc.). Allergens are mostly substances of protein nature. Simple low molecular weight substances are only partial antigens (haptens), they become a complete antigen only in the organism after binding to a protein. The time before an allergic reaction develops is called *the refractory period*. *The reaction time* is the time it takes for a reaction to develop after exposure to the allergen. *Shock tissue* is the location of the allergic reaction. In the case of contact allergy, the shock tissue is the epidermis. Drug allergies take place in the dermis.

Allergic reactions can be differentiated according to the disorder of the immune mechanisms that caused the allergy.

Immunopathological reaction type I

Immunopathological reaction of type I (anaphylactic, atopic type) is a humoral reaction, based on antibodies of the **IgE** class. The **most common** type of allergy is associated with the formation of IgE against some exoantigens:

- penicillin causes allergies - the reactive cyclic lactam group reacts with the amino acids of various serum and cellular proteins and covalently modifies them. Antibodies, including IgE, are formed in some individuals against these newly formed epitopes and an atopic reaction occurs.
- components of pollen grains,
- antigens of dust mites,
- food antigens,
- animal fur, saliva and epithelium.



Clinical picture of atopic eczema

It is also the only immunopathological reaction that **does not occur in autoimmune diseases**. Individuals with a predisposition *to react to harmless antigens by producing IgE are called atopics*. That is why this type of hypersensitivity is sometimes called **atopy**.

Some forms of atopy correlate with certain types of MHC polymorphism, IL-4, subunits β high-affinity IgE receptor (**FceR-I**). The influence of the environment is also important:

- rate of *allergen exposure*
- **climate factors**
- **diets in infancy** (breast milk and its substitutes),
- **infection**: repeated respiratory viral infections lead to bronchial hyperreactivity,
- the absence of *"intestinal parasites"* in developed countries during childhood leads to susceptibility to IgE reaction against harmless antigens.

According to the speed of onset, we divide this type of allergy into "early" and "late" hypersensitivity.

Early-type hypersensitivity

The reaction occurs very quickly after contact with the allergen (minutes). At the first encounter with the antigen, the patient is '*sensitization*. This reaction is similar to those with which the immune system physiologically responds to multicellular parasites. Differentiation of specific clones of TH2-lymphocytes is stimulated, followed by B-lymphocytes. They secrete antibodies of the type IgE under the influence of cytokines (IL-4, IL-5). IgE antibodies bind to high-affinity IgE-receptors of mast cells and basophils. After a repeated encounter with an allergen, IgE molecules are bridged, receptors aggregate on the surface of these cells and their granules are immediately released (phase 1 of the allergic reaction):

- histamine
- heparin

This is followed by **synthesis of signals and release of metabolites**, such as arachidonic acid (phase 2 of the allergic reaction).

Progress

An allergic reaction takes place depending on the input of the allergen:

- **locally**: allergic rhinitis (hay fever), conjunctivitis, asthma bronchiale, atopic dermatitis, exogenous allergic alveolitis,
- **systemically**: anaphylactic shock, Quincke's edema.

🔎 *For more information see Anaphylactic Shock.*

Treatment

- **Prophylaxis**: avoiding the allergen.
- Use of *pharmaceuticals* blocking histamine receptors (antihistamines), inhibiting histamine synthesis, anti-inflammatory (corticoids), inhibiting degranulation (cromoglycate).
- **Hyposensitization**: partially successful, gradually increasing doses of the allergen are administered, with appropriate application a partial shift from the *TH2 to TH1* lymphocyte subset can be achieved, which inhibits the production of allergenic IgE.

🔎 *For more information see Laboratory determination of specific IgE antibodies.*

Immunopathological reaction type II

Immunopathological reaction type II

Immunopathological reaction type III

Immunopathological reaction type III

Immunopathological reaction type IV

Immunopathological reaction type IV

Links

Related Articles

- Food Allergy • Cow milk protein allergy • Epidemiology of food allergies
- Allergy treatment
- Anaphylactic shock
- Allergens • Antibodies • Mast cells • Specific immunity
- Bronchial asthma

External Links

- Allergy (english wikipedia)

References

- HOŘEJŠÍ, Václav – BARTŮŇKOVÁ, Jiřina. *Základy imunologie.* 3. edition. Praha : Triton, 2008. 280 pp. ISBN 80-7254-686-4.