

Type I immunopathological reaction

Type I immunopathologic reaction (anaphylactic, atopic type) is a humoral reaction, based on antibodies of class **IgE**. **Most common** type of allergies are linked with the production of IgE against some exoantigens:

- Penicilin can cause allergy – reactive cyclic lactam group reacts with amino acids of various serum and cell proteins and modifies them covalently. In some individuals antibodies including IgE are formed against these newly formed epitops leading to an atopic reaction.
- components of pollen grains,
- dust mite antigens,
- food antigens,
- animal fur, saliva a 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 produce IgE and are called **atopics**. This is the reason why this type of hypersensitivity is sometimes referred to as **atopy**.

Some forms of atopy correlate with certain types of polymorphism MHC, IL-4, subunits β high-affinity IgE receptoru (**Fc ϵ R-I**). The effect of the environment is also important:

- degree of **exposure to allergens**,
- **climatic factors**,
- **diets during breastfeeding age** (maternal milk and its replacements),
- **infections**: recurrent viral respiratory infections lead to bronchial hyperreactivity,
- absence of **intestinal parasites** in developed countries during childhood leads to subsceptibility to IgE reaction against harmless antigenum.

According to the rate of onset, this type of allergy can be divided to **early** and **late** hypersensitivity.

Early type hypersensitivity

The reaction occurs very quickly after contact with the allergen (minutes). During the first encounter with the antigen, the patient is **sensitized**. This response is similar to those, in which the immune system responds physiologically to multicellular parasites. The differentiation of specific clones of TH2-lymphocytes, subsequently B-lymphocyty. These secrete IgE antibodies under the influence of cytokines (IL-4, IL-5). IgE antibodies bind to high-affinity IgE-receptors on mast cells and basophils. After **repeated encounters** with the allergen, IgE molecules are bridged, **receptors** on the surface of these cells **aggregate** leading to their immediate **release of granules** (phase 1 of the allergic reaction):

- histamin,
- heparin.

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

The course

The course of the allergic reactions is dependent on the entry pathway of the allergen:

- **local**: allergic rhinitis (hay fever), conjunctivitis, asthma bronchiale, atopic dermatitis, exogenous allergic alveolitis,
- **systemic**: anaphylaktic shock, Quincke's edema.

Treatment

- **Prophylaxis**: avoid allergens.
- Usage of **pharmaceuticals** blocking receptors for histamine (antihistaminics), inhibiting synthesis of histamine, anti-inflammatory (corticosteroids), inhibiting degranulation (cromoglycate)
- **Hyposensitization**: partially successful, gradually increasing doses of allergen are administered. With suitable application it is possible to achieve the transfer from **TH2 to TH1** lymphocytic subset, which inhibits the formation of allergenic IgE

Links

Related articles

- Allergy
- Type II immunopathological reaction
- Type III immunopathological reaction
- Type IV immunopathological reaction
- Atopic eczema

External links

- Imunopatologická reakce I. typu – Youtube video (https://www.youtube.com/watch?v=2tmw9x2Ot_Q)

Used literature

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