Type IV immunopathological reaction

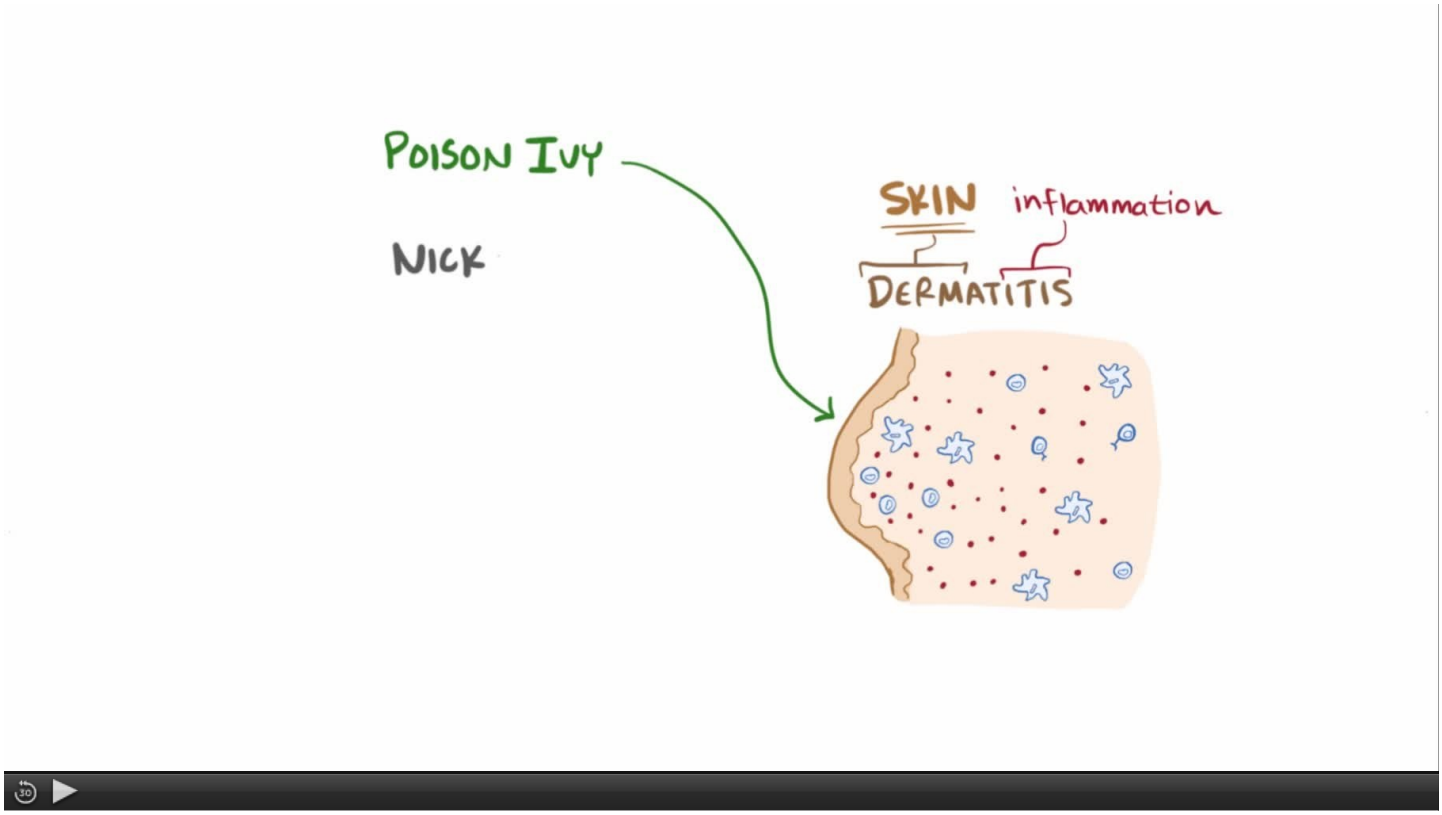
The late type immunopathologic reaction (also referred to as type IV, late type or cell type reaction) occurs 12–48 hours after contact with an allergen. There are no free Ig found in the serum because the reaction is mediated by T-lymphocytes.

Cellular immunopathologic reaction (tuberculin type)

Delayed type hypersensitivity is referred to as **DTH**.

The local reaction is causes by an inflammatory reaction dependent on TH1-lymphocytes, monocytes.

An animal is **exmperimentally** immunized intradermallu with an antigen in a suitable adjuvans. This supports the growth of TH1-lymphocytes. After a few weeks the antigen is again injected intradermally and a characteristic local reaction develops within 24 to 72 hours at the injection site. The time lag is due to the fact that TH1-lymphocytes and macrophages, which stimulate each other, must first migrate to the injection site. Hard edema (*induration*) occurs. This reaction is directed against intracellular parasites, funghi and bacteria under physiological circumstances. Bacteria, fungi, viruses and their products are allergens. The main changes occur in the corium, most often manifested by a papule. During **long-term stimulation**, macrophages can turn into multinucleated syncytia, so-called **giant cells**. The dermatological manifestations are "idic" reactions - mykids, microbides, bacterides, tuberculids. DTH is the basis of the **tuberculin reaction**. through which we determine the state of ones immunity against TBC. Delayed hypersensitivity reactions are responsible for damaging the tissue during **TBC** and **leprosy**. Granulomas often occur and in extreme cases also caseous necrosis (see necrosis). Some autoantigens cause this type of reaction in sarcoidosis or granulomatous vasculitis. Infiltration of Th1 lymphocytes with the production of IFN-γ is characteristic of **demyelinating autoimmune diseases'''.**



Cellular cytotoxic reaction (eczema, epidermal, contact type)

This reaction is similar to **DTH**, but the Th-1 lymphocytes activate other effector components, especially CD8+ T-lymphocytes. Infected or lysed cells are lysed by the effects of cytotoxic T-lymphocytes. It occurs in **viral exanthemas** and in **hepatitis** the main liver damage is caused by the immunopathological action of TC- and TH1-lymphocytes. They attack and destroy infected hepatocytes. They are also utilized in **acute rejection** of transplanted organs and in some forms of autoimmune thyroiditis. The same mechanisms are also responsible for **contact dermatitis** caused by some chemicals (**nickel**, chromium, components of cosmetics and others). The dermatological manifestation has the character of eczema with spongiosis in the dermis and lymphocytic infiltrates.

Reaction to a foreign body

This reaction is similar to DTH, but arises due to non-antigenic foreign material. Hydrophilic materials show better biocompatibility:

- contact lenses;

- joint replacement;
- mammary implants;
- sewing material;
- dialysis membranes;
- vascular replacements...

It depends on biocompatibility. Proteins from blood plasma are **adsorbed** on them. Adsorbed proteins are recognized by receptors of monocytes, macrophages, and platelets. Adhered monocytes are activated, expressing some receptors. Macrophages form IL-1 and TNF producing **syncytia**. These then create local and systemic reactions.

In **silicosis, asbestosis, beryliosis** inhaled particles are stored in the lungs, stimulating alveolar macrophages. Granulomas are formed, fibroblasts are stimulated, which leads to pulmonary fibrosis. In the case of asbestosis, the **carcinogenic effect of asbestos** is added.

Links

Related articles

- Allergy
- Type I immunopathological reaction
- Type II immunopathological reaction
- Type III immunopathological reaction

External links

- Imunopatologická reakce IV. typu – Youtube video (https://www.youtube.com/watch?v=C3E5COZ1XC8)

Used literature

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