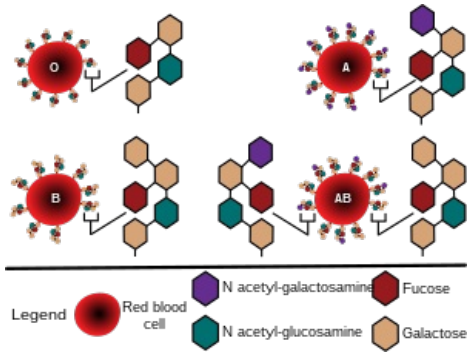
Type II immunopathological reaction

Type II immunopathological reaction (cytotoxic type) is a humoral reaction, based on IgG and IgM antibodies and subsequent activation of **cytotoxic leukocytes** by lysis of antibody-labeled cells**. Importantly, this type of response is elicited by antibodies to antigens located on cell surfaces.**. The mechanism caused by the gradual activation of complement with the formation of the membranolytic complex of the terminal part of complement (C5b–C9) is also utilized. In some immunopathological conditions, antibodies do not lead to cell death, but to a functional disorder by occupying receptors (followed by receptor stimulation or blockade).

Cytotoxic antibodies

Antibodies of class IgG a IgM have the ability to activate complement and cause antibody dependent cytotoxicity. Phagocytes and NK-cells express **Fc-receptors** on their surface. These can bind Fc portions of antibody of class **IgG**. Leukocytes are activated and target cells are killed by cytotoxic mechanisms. At other times, a direct effect on complement activation with the formation of the C5b-C9 membranolytic complex is exerted without cellular involvement.



Blood groups

Transfusion reaction

The reason for this is the existence of antibodies against *allelic forms* of various surface antigens of red blood cells, platelets and leukocytes. These can occur after the first incorrect transfusion, after a different type of sensibilization etc. Natural **IgM** and antibodies produced by various microbial polysaccharide antigens (especially intestinal microflora) are found in large amounts in the blood. These IgM antibodeis bind various saccharide structures including those, which are **similar to blood group A and B substances**. The body does not normally allow the formation of antibodies that react with antigens on the surface of its own cells. In addition to antigens A, B and 0, there are a number of other more or less polymorphic erythrocyte antigens (e.g. Rh system). Repeated transfusion of inappropriate red blood cells can lead to damage mediated by the complement or phagocytes. TThis also applies to neutrophils and platelet alloantigens. Antibodies against allelic forms of Fc-receptors (CD16) of neutrophils are involved in, neonatal neutropenia. Antibodies against platelet aloantigens cause neonatal thrombocytopenia in children of mothers who have given birth several times, or received blood transfusions.

Example

When blood cells A are transfused into recipient B, antibodies bind and the classic complement pathway is activated. This leads to lysis of the „foreign“ cells.

Hemolytic disease of newborns

Is caused by antibodies against RhD antigens, if the mother is RhD- and the fetus is RhD+ and the mother has been previously **immunized against RhD**. IgG antibodies cross the placenta and hemolysis of fetal erythrocytes occurs. At the same time **neonatal jaundice** develops, which can lead to the so-called kernikterus.

Autoimmune diseases

In autoimmune diseases, cytotoxic antibodies are used in the so-called **organ specific autoimmune diseases**, in which the autoimmune reaction is directed against the autoantigens specific for cell lineage or tissue. Erythrocytes, granulocytes, platelets, membranes of glomeruli, and components of skin are damaged most.

Blocking or stimulating antibodies

A condition in which autoantibodies do not directly destroy the target structure, but block or stimulate its function. Antibodies against membrane receptors can stimulate the function of a natural ligand (so called **stimulatory effect**), or conversely, compete for binding with a particular ligand and prevent its binding (**blocking effect**). The inhibitory effect is not only applicable in cells but also in the case of soluble proteins (enzymes). This means that the autoantibody inhibits physiologic functions of the corresponding protein. An example of an **stimulatory antibody** is Graves-Basedow's disease against the TSH receptor (thyroid stimulating hormone). An example of **blocking antibodies** is myasthenia gravis. Due to binding of the autoantibody to the acetylcholine receptor the neuromuscular transmission is blocked.

Other examples:

- antibodies against the intrinsic factor block the absorption of vitamin B12 → pernicious anemia,
- antibodies against hormones of the thyroid gland → hypothyroidism,
- antibodies against the receptors for insulin → different forms of DM,
- antibodies against various phospholipids (cardiolipin) – affect the process of blood coagulation → antiphospholipid syndrome, phlebotrombosis,

- antibodies against caoagulation faktor VIII → rare form hemophilia,
- antibodies against cytoplasmatic antigens of neutrophils (ANCA) → pathogenesis of some types of vascultitis. These stimulate oxidative metabolism of granulocytes and inhibit their mikrobicidal activity, antibodies against sperm cells, oocytes etc. → problems with fertility.

Links

Related articles

- Allergy
- Type I immunopathological reaction
- Type III immunopathological reaction
- Type IV immunopathological reaction
- AB0 system
- Rh system

External links

- Imunopatologická reakce II. typu – Youtube video (https://www.youtube.com/watch?v=kLaUz58CBMc)

Used literature

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