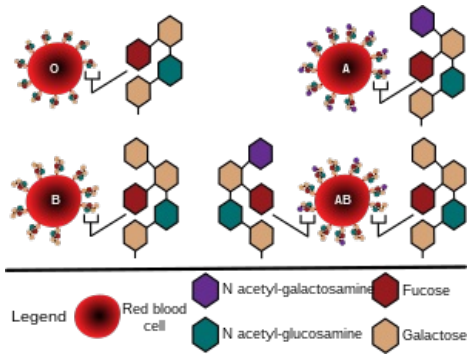
Immunopathological reaction II. type

Immunopathological reaction II. type **(cytotoxic type)** is a **humoral reaction, based on antibodies of type IgG and IgM and subsequent activation of** cytotoxic leukocytes **with lysis of antibody-labeled cells.**

***Importantly, this type of reaction is triggered by antibodies against 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 applied. In some immunopathological conditions, antibodies do not lead to the death of the cell, but to a functional disorder by occupying the receptor (with subsequent stimulation or blocking of the receptor).*

Cytotoxic antibodies

Antibodies of the IgG and IgM class have the ability to activate complement and cause an antibody-dependent cytotoxicity response. Phagocytes and NK-cells express **Fc-receptors** on their surface. They can bind Fc parts of antibodies class **IgG**. There is activation of leukocytes and destruction of target cells cytotoxic mechanisms. At other times, a direct effect on the activation of complement with the formation of the membranolytic complex C5b-C9 is applied without cellular participation.



Blood groups

Transfusion reaction

The cause is the existence of antibodies against the *allelic forms* of some surface antigens of red blood cells, platelets and leukocytes. These can arise after the first wrong transfusion, after another type of sensitization, etc. In the blood, there are large amounts of natural **IgM'** and antibodies formed after encountering various microbial polysaccharide antigens (mainly intestinal microflora) . These IgMs bind a variety of carbohydrate structures including those that are **similar to substances of blood groups A and B**. Normally, the body is not allowed to form antibodies that would 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 (for example Rh system). When inappropriate blood cells are repeatedly transfused, damage by complement or phagocytes may occur. This also applies to neutrophil and platelet alloantigens. Antibodies against allelic forms of Fc-receptors (CD16) of neutrophils are involved in neonatal neutropenia, antibodies against platelet alloantigens cause neonatal thrombocytopenia in children of mothers who have given birth multiple times or received blood transfusion.

Example

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

🔎 *For more information see AB0 system, Rh system.*

Hemolytic disease of the newborn

It is caused by antibodies against the RhD antigen if the mother is RhD- and the fetus is RhD+ and the mother has previously been **immunized against RhD.** *There is passage of IgG antibodies across the placenta and hemolysis of fetal erythrocytes.* ***During this,*** *newborn jaundice'* develops, which can lead to the so-called kernicterus.

🔎 *For more information see Rh System.*

Autoimmune diseases

In autoimmune diseases, cytotoxic antibodies are used in so-called *organ-specific autoimmune diseases*, in which the autoimmune reaction is directed against autoantigens specific to a certain cell line or tissue. Erythrocytes, granulocytes, thrombocytes, membranes of glomeruli, components of skin are mostly damaged.

Blocking or stimulating antibodies

A condition where autoantibodies do not directly destroy the target structure, but block or stimulate its function. Antibodies against the membrane receptor can stimulate the function of the natural ligand (the so-called *stimulatory effect*), or, on the contrary, compete for binding with a certain ligand and prevent its binding (the **blocking effect***). The inhibitory effect applies not only to cells, but also to soluble proteins (enzymes). This means that the autoantibody inhibits the physiological functions of the respective protein. An example of* ***stimulating antibodies*** *is Graves-Basedow disease against the TSH (thyroid stimulating hormone) receptor.*

An example of **blocking antibodies** is myasthenia gravis. As a result of autoantibody binding to the acetylcholine receptor, neuromuscular transmission is blocked.

Other examples:

- antibodies against intrinsic factor block absorption of vitamin B12 → pernicious anemia,
- antibodies against hormones thyroid glands → hypothyroidism,
- antibodies against the insulin receptor → forms of DM,
- antibodies against certain phospholipids (cardiolipin) – interfere with the blood clotting process → antiphospholipid syndrome, phlebothrombosis,
- antibodies against coagulation factor VIII → a rare form of hemophilia,
- antibodies against neutrophil cytoplasmic antigens (ANCA) → pathogenesis of some types of vasculitis. They stimulate the oxidative metabolism of granulocytes and inhibit their microbicidal activity,
- antibodies against sperm, oocytes, etc. → fertility disorders.

Links

Related Articles

- Allergy
- Immunopathological reaction type I
- Immunopathological reaction III. type
- Immunopathological reaction IV. type
- AB0 system
- Rh system

External links

- Immunopathological reaction II. type - Youtube video (https://www.youtube.com/watch?v=kLaUz58CBMc)

References

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