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THROMBOCYTOPENIC PURPLE

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Abstract:

Thrombocytopenic purpura (Werlhof's disease, benign thrombocytopenia) is a hematological pathology characterized by a quantitative deficiency of platelets in the blood, accompanied by a tendency to bleeding, the development of hemorrhagic syndrome. With thrombocytopenic purpura, the level of platelets in the peripheral blood drops significantly below the physiological level - 150x109 / l with a normal or slightly increased number of megakaryocytes in the bone marrow. In terms of the frequency of occurrence, thrombocytopenic purpura ranks first among another hemorrhagic diathesis.

Keywords: (TP) thrombocytopenias, Isoimmune, Transimmune, angiotrophic

Etiopathogenesis In the overwhelming majority of cases, thrombocytopenic syndrome is acquired, however, there is a small group of hereditary thrombocytopenias (TP) associated with structural platelet inferiority, leading to a shortening of their life span. Acquired TPPs, in turn, are subdivided into immune (the most common, accounting for 4/5 of all TPPs) and non-immune. Non-immune TPP can be caused by insufficient formation of platelets (with AA, OL), their increased consumption (disseminated intravascular coagulation, hemangiomas), increased destruction (with mechanical trauma, splenomegaly). Immune TPP can be symptomatic, but in most cases it is primary TPP is an independent disease. In the latest international classification of diseases, the name idiopathic thrombocytopenic purpura (ITP) has been approved, which replaces a number of widely used terms: Werlhof's disease, essential TPP. However, in children, unlike adults, autoimmune thrombocytopenias, which include most forms of ITP, are relatively rare. Depending on the pathogenesis, i.e., the mechanism of production of antiplatelet antibodies in children, the following forms of immune TPP are distinguished:

1. Isoimmune (alloimmune) form. Its etiopathogenesis is largely identical to hemolytic disease of newborns, but incompatibility and immunological conflict relate to platelet aneshgens received by the child from the father and absent from the mother. Usually the mother does not have the platelet antigen PLAI (in the population of such people, 2-5%), but the child does. In the sensitized body of the mother, antiplatelet antibodies (AT) appear, which penetrate the mother's placenta and cause thrombocytolysis in the fetus. The isoimmune form occurs in 1 in 5,000-10,000 newborns. At an older age, this form of TPP may develop during blood transfusions.
2. Transimmune form, in which the anti-thrombotic autoantibodies of the mother with ITP cross the placenta to the fetus, causing him TP. This form of the disease occurs in 30-50% of newborns born to mothers with ITP.
3. The heteroimmune form is associated with the formation of antibodies in response to a change in the antigenic structure of platelets as a hapten under the influence of factors damaging the platelet. This form of TPP is more common in children.
4. Autoimmune form, in which antibodies are produced against their own unchanged platelets. The initial triggers for the appearance of AT remain unclear. Diseases and external influences immediately preceding the onset of ITP in a child can be different: infections, more often viral, less often bacterial, preventive vaccinations, administration of gammaglobulin, intake medications, overheating in the sun, hypothermia. Time elapsed after them before the development of purpura, on average, is 2 weeks. In 1/3 of patients with ITP starts for no apparent reason.

METHODS

In the development of ITP, the immunopathological process is of decisive importance, in particular the synthesis of antiplatelet antibodies. Platelets under the influence AT are dying. With ITP, platelet lifespan is reduced from 7 to 10 days to several hours, their production in the bone marrow remains normal or even increases (the so-called hyperregenerative TP. AT can be directed against megakaryocytes, then this sprout appears to be devastated \ hyporegenerative TP). Currently, the

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immune genesis of ITP is generally recognized, it is confirmed by the detection of platelet antibodies, a high level of Ig G on the surface of platelets, and the detection of lymphocytes sensitized to autoplatelets in the blood of patients.

The spleen plays a role in the development of ITP. It happens increased destruction of platelets, in addition, the spleen is a site production of antiplatelet antibodies (formed mainly by the splenic pool of lymphocytes). In the pathogenesis of bleeding in TPP, thrombocytopenia is the leading one; the participation of platelets in hemostasis and their angiotrophic function. Vascular endothelium, deprived of platelet feeding spontaneous hemorrhages. The incidence of TPP is 1.5-2 per 100,000 child population. The disease can occur at any age, but more often in preschool and school age. After 10 years, a clear predominance of girls is revealed, 2-3 times more often boys get sick.

SYMPTOMS (SIGNS)

The main sign of the appearance of thrombocytopenic purpura is the appearance of a specific rash (hematoma) on the skin and the occurrence of bleeding from the mucous membranes. The acute form of the disease begins abruptly: hemorrhages appear on the skin, blood pressure decreases, body temperature rises, and lymph nodes increase. Along with the rash or after a while, bleeding appears: nasal, uterine, from the respiratory tract, gums or digestive tract. In the chronic form, the body temperature, as a rule, does not rise.

In addition to the listed symptoms, patients may experience:

- dizziness and headaches;
- neurological abnormalities;
- tachycardia (heart palpitations);
- joint pain;
- low hemoglobin in the blood (anemia);
- enlargement of the spleen;
- arrhythmia (violation of the frequency and rhythm of heart contractions).

The most dangerous manifestation of the disease is thrombotic thrombocytopenic purpura. It is characterized by a malignant course and the formation of blood clots in the vessels. Distinguishing features are fever, renal failure, tissue necrosis.

IN CHILDREN

In addition to the appearance of a characteristic rash, children always have an increase in the size and soreness of the lymph nodes. Nose and gum bleeding is common. The most dangerous are hemorrhages in the brain and internal organs. The disease in children, as a rule, is acute.

RESULTS

Treatment begins if there is a wedge, manifestations of the disease. Usually, prednisolone is prescribed at a dose of 1 to 2 mg / kg of body weight. In 60% of cases, there is an increase in the level of platelets to normal, in most patients hemorrhagic syndrome is stopped. In about 10% of cases, glucocorticoid hormone therapy is ineffective in terms of platelet levels. Treatment with prednisolone rarely leads to a permanent cure. Renewal of hemorrhagic syndrome after withdrawal of prednisolone is an indication for splenectomy (see), to-Ruyu it is advisable to perform as soon as possible not earlier than after 6 months. from the moment the disease manifests itself. If large doses of glucocorticoid hormones do not stop the bleeding that threatens the patient's life, the spleen is removed urgently, regardless of the duration of the disease. According to statistics, splenectomy in 75% of cases leads to the final recovery of patients, in 10-15% of patients after surgery, bleeding either sharply decreases or stops completely, despite persisting thrombocytopenia).

CONCLUSION

In the absence of the effect of splenectomy and the resumption of severe hemorrhagic syndrome, treatment with cytostatic immunosuppressants, often in combination with corticosteroid hormones, is carried out. The effect of therapy is manifested after 1 - 2 months, after which glucocorticoid hormones are canceled. The following drugs are used: vincristine, 1.4 mg / m2 once a week, course duration 1-2 months; imuran (azathioprine) 2-3 mg / kg per day, course duration up to 3-5 months; cyclophosphamide (cyclophosphamide) 200 mg per day (usually 400 mg every other day) for a course of approx. 6-8 g. Prescribing cytostatic drugs before surgery is unacceptable, except for cases in which splenectomy cannot be performed due to the presence of severe intercurrent pathology. It is necessary to avoid their use in children in the absence of vital indications.

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