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HEMORRHAGIC DIATHESES CAUSED BY PATHOLOGY OF THROMBOCYTIC LINK OF HEMOSTASIS

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Abstract:Hemorrhagic diathesis caused by pathology of platelet link of hemostasis is a group of diseases characterized by increased tendency to bleeding due to quantitative or qualitative disorders of platelets.These pathologies include thrombocytopenias (decreased platelet count) and thrombocytopathies (functional defects of platelets), which may be inherited or acquired. The article discusses the main mechanisms of pathogenesis, clinical manifestations, diagnostic methods and modern approaches to the treatment of these conditions. Special attention is paid to the distinguishing features of platelet hemorrhagic diatheses, their differential diagnosis with coagulopathies, as well as relevant methods of laboratory research.Therapeutic strategies including the use of hemostatic drugs, immunomodulatory therapy, platelet transfusions and innovative treatment methods such as target therapy and genetic engineering are considered. The relevance of the topic is due to the high prevalence of platelet disorders and their potential danger to the life of patients. Modern research in the field of hematology is aimed at developing new diagnostic methods and personalized approaches to the treatment of these pathologies.

Keywords: Hemorrhagic diathesis, Thrombocytopenia, Thrombocytopathy, Thrombocytic link of hemostasis, Primary hemostasis, Immune thrombocytopenia, Idiopathic thrombocytopenic purpura, Glanzmann's disease, Bleeding disorders, Coagulopathy, Hemostasiology, Platelet receptors, Hypersplenism, Aplastic anemia, Myelodysplastic syndrome, Platelet sequestration, DIC.

Hemorrhagic diathesis (HD) is a group of pathological conditions characterized by an increased propensity to bleeding due to disorders of the hemostasis system. A special role in the pathogenesis of these conditions plays a platelet link, which provides primary hemostasis. Violations may be associated with a decrease in the number of platelets (thrombocytopenia) or with their functional defect (thrombocytopathies).

Platelets are blood cell elements involved in stopping bleeding. Their functions include:

-adhesion to damaged endothelium (via GPIb-V-IX and von Willebrand factor receptors),

-aggregation with each other (mediated by GPIIb/IIIa and fibrinogen),

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-secretion of coagulation factors and biologically active substances.

Violation of any of these functions leads to pathologic bleeding.

The main cause of bleeding in this type of hemorrhagic diathesis is thrombocytopenia. The cause of increased platelet destruction in this disease is the formation of antiplatelet antibodies related to Ig G. Contribute to the development of the disease viral infections, taking medications such as sulfonamides, butadione, quinine, dopegit, etc. The life span of platelets is shortened to a few hours instead of 7-10 days.

The clinical picture of the disease is manifested at a platelet level of less than 150-109 /L. The course of the disease is chronic, recurrent, but can be acute. The first manifestations, as a rule, are not associated with any previous disease. Appear spotty-petechial bruise hemorrhages, bleeding from mucous membranes. In some patients, an enlarged spleen is detected.Heteroimmune thrombocytopenia is distinguished. In this form of the disease, a viral infection or certain drugs play the role of a hapten associated with the platelet. The AT formed causes platelet destruction and increased bleeding. The type and severity of bleeding, established during the examination, greatly facilitates the diagnostic search.

I. Hematomic with painful tense hemorrhages both in the

soft tissues and joints - typical of hemophilia A and B;

II. petechial-staining (bruising) - characteristic of thrombocytopenia, hemophilia A and B.

thrombocytopenia, thrombocytopathies and some disorders of blood coagulation

blood - hypo- and dysfibrinogenemia, hereditary deficiency of factors X and II, sometimes VII;

III. mixed bruising-hematoma - characterized by a combination of

petechial-stained bleeding with the appearance of individual large

hematomas (retroperitoneal, in the wall of the intestine, etc.) in the absence of lesion

joints and bones (unlike the hematoma type) or with isolated hemorrhages in the joints and bones (unlike the hematoma type).

hemorrhages into joints: bruises may be extensive and painful. This type of bleeding is observed in severe deficiency of prothrombin complex factors and factor XIII, Willebrand's disease, DIC, overdose of anticoagulants and thrombolytics, with the appearance of immune inhibitors of factors VIII or IX in the blood;

IV. vasculitic-purpuric type is characterized by hemorrhages in the form of

symmetrical small-pointed rash, possible accession of inflammation of the kidney and intestinal bleeding; observed in infectious and immune

vasculitis.

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V. angiomatous type is observed in telangiectasis, Randu-

Osler's disease, angiomas, arteriovenous shunts; it is characterized by strictly

localized and tied to local vascular pathology.

hemorrhages.

Hemophilia is a group of diseases in which deficiency of clotting factors leads to the development of a characteristic hemorrhagic syndrome: bleeding, hemorrhages in soft tissues, joints, CNS.

Hemophilia is classified according to the deficiency of antihemophilic globulins.

Hemophilia is a congenital coagulopathy characterized by deficiency of factors VIII (hemophilia A); factor IX (hemophilia B, Christmases disease); factor XI (hemophilia C). The incidence of hemophilia is 1 case per 50000

newborns. Hemophilia A is the most common form of hereditary coagulopathy. Hemophilia A - in 1:5000-10000 newborn boys, hemophilia B - in 1:30000 boys. Of all hemophilias, hemophilia A occurs in 80%, hemophilia B - 19% and hemophilia C - in 1% of cases. Hemophilia A is caused by a deficiency of factor VIII. It is found in plasma or fixed on platelets. The hemophilia A gene is linked to the X chromosome, inherited by recessive type. All daughters of a hemophilia patient are carriers of the gene, and all sons are healthy. In women-conducers of this type of hemophilia, half of the sons may have hemophilia. A woman may be sick if she has a sick father and a gene-carrier mother. Hereditary genesis in hemophilia is established in 70-90% of cases, spontaneous mutations are possible.

Hemorrhagic vasculitis (Schenlein-Genoch disease). The disease was first described by Schoenlein in 1837 and Genoch in 1868. Kidney damage in this disease was described by Johnson in 1852. The essence of the pathological process is multiple microthrombovasculitis affecting the vessels of the skin and internal organs. The disease is more common in childhood and adolescence. By its nature it belongs to immunocomplexes, in particular, it is caused by the damaging effect of low-molecular-weight IRs. Low-molecular complexes and complement activated by them cause microthrombovasculitis with fibrinoid necrosis, perivascular edema, blockade of microcirculation, hemorrhages and deep dystrophic changes. The direct cause of accumulation and development of damaging effect can be transferred viral or bacterial infection, vaccinations, some medications, parasitic infestations and even cold.

Clinical picture. According to the clinical course, a distinction is made:

skin or simple form - purpura simplex

joint form - purpura reumatica

abdominal form - purpura abdominalis

renal form - purpura renalis

fast-flowing form - purpura fulminans.

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There may be a combination of different forms

Skin lesions are characterized by small-pointed symmetrically located petechiae, mainly on the lower extremities, buttocks. The rashes are monomorphic, initially with a distinct inflammatory basis, in severe cases - complicated by central necroses, which are subsequently covered with crusts, leaving pigmentation for a long time. Not accompanied by itching. In severe cases, petechiae are complicated by necrosis. More often the intense rash lasts 4-5 days, then gradually subsides and disappears altogether after which, may remain a small pigmentation. As a rule, the skin form ends with full recovery. Affection of the joints is manifested by sharp soreness, swelling, violation of their function. The site of joint damage is the synovial membrane. Joint damage is completely reversible.

Abdominal vasculitis is manifested by hemorrhages in the mucosa.

mucosa of the stomach, intestine, mesentery. In this form, there are severe abdominal pain, sometimes simulating the picture of acute abdomen. There may be an increase in body temperature, sometimes there is vomiting. Blood is detected in the feces. In most cases, abdominal manifestations are short-term and within 2-3 days pass. Recurrences are also possible. When they are combined with cutaneous petechial rashes, diagnosis is not very difficult. In the absence of cutaneous manifestations of the disease diagnosis is difficult. It should take into account the transferred viral infection, the presence of rashes on the skin, preceding the appearance of abdominal pain. Tests for capillary resistance are used (Nesterov and Konchalovsky tests). Most attention should be paid to the renal form, proceeding as an acute or chronic nephritis, sometimes taking a protracted course with the development of CKD.

subsequent development of CKD.Nephrotic syndrome is possible. Renal damage, as a rule, does not occur immediately, but 1-4 weeks after the onset of the disease Renal damage is a dangerous manifestation of hemorrhagic vasculitis. In the presence of hemorrhagic vasculitis, it is advisable to pay attention to urine composition and renal function throughout the course of the disease. The fast-flowing or cerebral form develops when there is hemorrhage into the cerebral membranes or vital areas.

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