Figure 2. Reconstruction of the sample volume by the software Amira ResolveRT in automatic mode (feature Volume Rendering). The volume structure is built on the contrast of the original image.

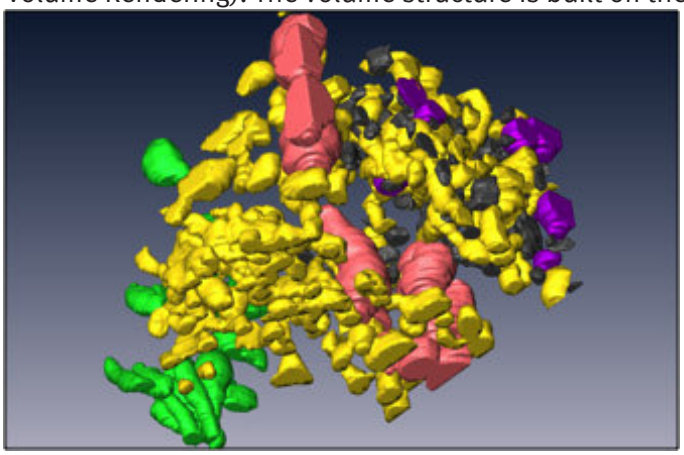

Figure 3. Reconstruction tracheal epithelium portion (a few cells). Yellow (1) marked the mitochondria, Green (2) - bacteria, pink (3) - the inclusion in the cage confining the excretory duct of protein and mucous glands, violet (4) - lysosomes, black (5) - advanced tubules EPR, green - bacteria.

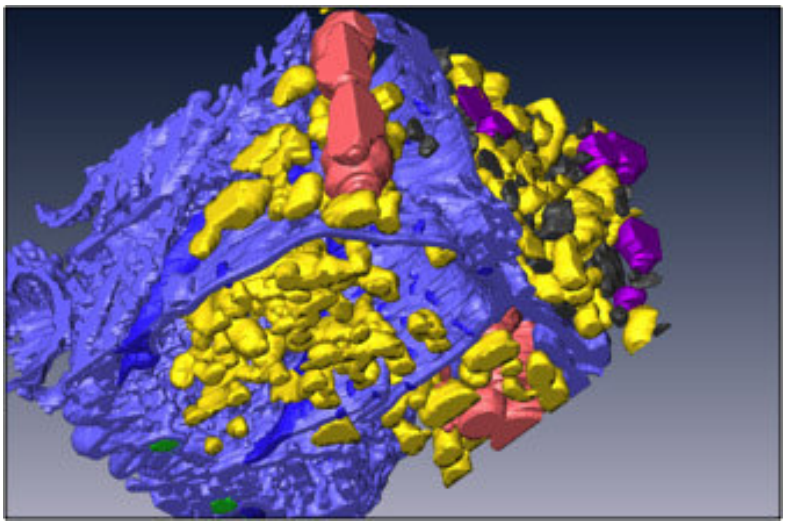

Figure 4. All the same as that in Figure 3 but with the membrane. Blue (1) denotes the membrane blue (2) - a dense cell contact and desmosomes.

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MODERN METHODS OF TREATMENT OF IDIOPATHIC THROMBOCYTOPENIC PURPURA

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Abstract The article describes the experience in the treatment of autoimmune (idiopathic) thrombocytopenia purpura in the hematology department of the Amur regional clinical hospital. Currently available treatments for advanced high AITP, with timely diagnosis of the disease and the appointment of adequate therapy in most cases of the disease prognosis - favorable.

Key words: autoimmune idiopathic thrombocytopenic purpura, treatment.

Thrombocytopenia is a condition in which the peripheral blood platelet count drops below 150 × 109 / L. The autoimmune (idiopathic) thrombocytopenic purpura is a disease characterized by the destruction of platelets in the peripheral blood under the influence of antibodies. According to ZS Barkagan (2005), the incidence of this nosology is 4.5 males and 7.5 females per 100,000 population [1]. There are acute (lasting from 3 to 6 months) is more common in children and chronic forms of autoimmune (idiopathic) thrombocytopenic purpura (AITP) is observed more often in adults [4]. The disease develops often, for no apparent connection with any previous disease. The main clinical symptom is haemorrhage. Severity of hemorrhagic syndrome varies, from individual small bruises and petechiae to massive bleeding from internal organs and bleeding in vital organs and centers. Spontaneous hemorrhagic syndrome in these patients developed platelet counts in

less than 50 × 109 / L, which is an indication for on-treatment values.

Diagnostic criteria AITP: 1) isolated thrombolytic thrombocytopenia (less than 150 × 109 / L) in the absence of other abnormalities in the calculation of the formed elements of the blood; 2) the absence of clinical and laboratory signs of disease in blood; 3) increased number of megakaryocytes in the bone marrow; 4) lack of patients clinical manifestations of other diseases or factors that can cause thrombocytopenia, NIJ (SLE, HIV, acute leukemia, myelodysplastic syndrome, aplastic anemia, certain medications); 5) Detection of anti-platelet anti-bodies; 6) Effects of glucocorticoid therapy. Symptomatic immune thrombocytopenia associated autoimmune and immune complex diseases and syndromes must be excluded.

AITP Treatment includes four stages. The first stage - the appointment glucocorticosteroids, in most cases, prednisolone tablets at a dose of 1 - 2 mg / kg body weight within 1 - 4 months. The second stage - intravenous immunoglobulins (Ig). Indications for on-the value of intravenous Ig are heavy, threatening bleeding, profuse uterine and gastrointestinal bleeding, as well as preparation for splenectomy. Ordered intravenous Ig for 0,4g per 1 kg of body weight per day for 5 days. Intravenous Ig raise platelet level for a short time (2 - 4 weeks), and therefore can not be used as long basic therapy. The third stage - recombinant thrombopoietin. Thrombopoietin (TPO) - glycoprotein hormone regulating division, differentiation megakaryocyte maturation and platelet yield in the peripheral blood. There are 2 forms of TPO products: one of them, called human recombinant TPO (rhTRO), it is a full polypeptide. Another, which is divided into a polypeptide portion comprising only the receptor-binding region, which is chemically modified with polyethylene glycol (PEG), and it is called PEG-conjugated recombinant human megakaryocyte growth factor (PEG-rHuMGDF). The systematic use of TPO platelet count begins to rise after 3-5 days of treatment. This is due to the fact that TPO stimulates the production and maturation of megakaryocytes. After the abolition of TPO thrombocytopenia recurs in most cases. In the absence of the effect of treatment with prednisolone performed splenectomy (fourth stage). The quantity of platelets for a short time can be improved by introducing intravenous Ig or recombinant thrombopoietin. After a splenectomy it is possible to attain remission in 70% of cases. The fifth stage - the appointment of cytostatics, in the absence of the effect of splenectomy. Previously, cytostatic treatment using azathioprine, cyclophosphamide, vincristine, etc. Recently, for the treatment of relapsing forms after splenectomy AITP used a chimeric antibody specific action directed against the CD20 antigen - rituximab (MabThera). CD20 antigen present on the surface of B-lymphocytes. This fact was the basis for the use of rituximab in AITP. The recommended dosing regimen in patients with AITP - 4-6 infusions at a dose of 375 mg / m2 once a week. MabThera is administered intravenously, infusion (slow) through a separate catheter. Before each infusion of MabThera should be carried premedication (analgesic / antipyretic such as acetaminophen, an antihistamine, such as diphenhydramine). If not applicable rituximab in combination with chemotherapy containing corticosteroids in the sedation also includes glucocorticoids. In case of remission, is a maintenance therapy for two years. When using rituximab objective response is seen in 52% of patients with chronic AITP [5]. In applying the drug in patients with refractory AITP treatment effect was registered in 72% of them in 28% of patients have long-term complete remission [6].

Materials and methods. The analysis of 150 outpatients AITP patients, aged 18 to 78 years. Patients were under the supervision of a hematologist in the Amur regional advisory clinics and treated at the hematology department of the Amur Regional Clinical Hospital for 10 years (2006 - 2015). Most AITP diagnosed in women (98 cases) than in men (52 people). In 88 patients AITP diagnosed before the age of 30 years. This study examined the cases it is idiopathic thrombocytopenic purpura. Autoimmune thrombocytopenia amid SLE and other immune diseases [2] were excluded from the study.

The results of the study and discussion. Of the 125 patients with AITP needed treatment - there has been a decrease in platelet count to less 30 × 109 / L (90), or less than 50 × 109 / L if expressed hemorrhagic syndrome (35 patients).

In 20 patients, thrombocytopenia was associated with HIV. In the first place it on the most-significant thrombocytopenia effective treatment for people with HIV - this highly active antiretroviral therapy (HAART) [3]. Antiretroviral drugs are drastically reduced, the level of virus in the blood, and thus do not give HIV to infect megakaryocytes. VA-ART as "soothing" the immune system, that is, makes it less active, which slows down the process of development of the autoantibodies contribute to thrombocytopenia. Thus, HAART - the first method of treatment, which is usually offered for thrombocytopenia. In cases where HAART is ineffective (8 patients) was used prednisolone by the standard technique. Only 2 patients with HIV noted recurrence AITP. Such patients splenectomy was not performed.

Last time often become AITP diagnosed primarily in pregnant women - 24 patients. From them - 18 needs treatment. In accordance with the national guidelines for the treatment of AITP primarily have used intravenous Ig. In the absence of the effect of multiple courses of therapy Ig (10 patients) administered glucocorticoids. We prefer to conduct pulse therapy with intravenous factions, and only in the absence of effect (7 patients) was administered prednisone tableted in different dosages. Terms of delivery in these patients was determined individually, from 34 to 38 weeks. Neonates AITP not mentioned.

Patients aged from 18 to 50-55 years in need of treatment (non-pregnant) as first-line therapy adminis-

tered in a unit dose of prednisolone - 1 - 2 mg per 1 kg weight. In the absence of the effect of receiving glucocorticoids in a dose within 1 - 4 months' (45) made splenectomy. In 37 of their number after splenoctomy complete remission of the disease was achieved and AITP not recurred more. With 8 of them after splenectomy had recurrent disease. Such patients we administered rituximab 4 - 6, infusion of 375 mg / m2 once a week. After that, all was in remission. Within two years he performed with rituximab maintenance therapy - 375 mg / m 2 - 2 of the introduction of 3 - 6 months. After 2 years, all remained in remission and rituximab canceled.

Attempts to use rituximab before (instead of) splenectomy in any case, not result to achieve stable remission. That corresponds to the literature - rituximab is effective only after the removal of the spleen. [4]

Recombinant thrombopoietin not often used, due to their high cost. Revoleyd (eltrombopag) was ordered for three patients. Dosing regimen was administered individually based on the number of platelets, the initial dose - 50 mg 1 time per day. If after 2-3 weeks of initial therapy the platelet count remained below the level required from a clinical point of view (50,000 / ml), the dose was increased to the maximum - 75 mg 1 time per day. Standard dose correction downward or increase was 25 mg per day. At the level of platelet 200000-400000 / l reduced dose. Enpleyt (romiplostim) was administered to patients one to three times a week by subcutaneous injection. Romiplostima initial dose was 1 mg / kg body weight. Romiplostima weekly dose was increased in increments of 1 .mu.g / kg body weight as long as the number of platelets in the patient did not achieve more than 50 × 109 / L. All patients treated with both forms of recombinant TPO in patients receiving drugs platelet count normalized. After its cancellation AITP recurred.

Fisher-Evans syndrome Classic (combination AITP gemolitiche and autoimmune anemia) was diagnosed in two patients. In both cases the remission was achieved after splenectomy.

Deaths from AITP only 5 patients (3%) was diagnosed in 10 years, there has been a brain hemorrhage in all cases.

Conclusion. Thus, there currently are available treatments for advanced high AITP. With timely diagnosis of the disease and the appointment of adequate terapy FDI in the majority of cases of the disease prognosis - favorable.

Literature

1. Barkagan Z.S. Pathology platelet hemostasis // Manual Hematology / ed. A.I. Vorobyov, third edition in 3 m. T. 3. M .: Nyudiamed 2005. P. 29 - 45.

2. Voytsehovsky V.V., Landyshev Y.S, Tseluyko S.S., Lysenko A.V. Leukemoid re-stock and erythrocytosis. Blagoveshchensk .: "Zeya" CRP. 2013. 231 p.

3. Pivnik A.V., Korovushkin V.G., Tuaeva O.A. et al. Thrombocytopenia with HIV // Therapeutic Archives. 2008. №7. Page 75. 80.

4. Rukovitsin O.A. Hematology. National leadership. M .: GEOTAR Media. 2015. 776 p.

5. Cooper N., Feuerstein M., McFarland J. et al. Investigating the mechanism of action of ri-tuximab, an anti-CD20 monoclonal antibody in adults with immune thrombocytopenic purpura // Blood, 2002a; 100: 479a.

6. Cooper N., Stafi R., Feuerstein M., et al. Transient B cell depletion with rituximab, an anti-CD20 monoclonal antibody, resulted in lasting complete responses in 16 of 57 adults with refrac-tory immune thrombocytopenic purpura // Blood, 2002b; 100: 52a.

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RESPIRATORY AND NEURAL SYSTEM DAMAGE CAUSED BY SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract A brief review of the literature devoted to the disorder of the respiratory and neural system in systemic lupus erythematosus (SLE). Characteristics of the most common pathology - pleurisies the are disorder of the diaphragm, upper respiratory tract, pulmonary vessels. Particular attention is given to lupus pneumonitis and pulmonary (alveolar) hemorrhage. As an example, here is the case of the personal experience of the authors. At the young age of the patient at the acute stage of SLE leading clinical manifestation was caused by lung disease, manifested by acute respiratory failure, hemoptysis, anemia, and X-ray picture of bilateral interstitial and alveolar lesions of the lung tissue. Based on the identification of laboratory signs of active SLE the lupus lung damage of hemorrhagic alveolitistype was diagnosed. After treatment with glucocorticoids (pulse therapy and long-term reception of high doses orally), pulse therapy of cyclophosphamide, cascade plasma filtration the regression of lung manifestations of SLE has been achieved.

Key words: systemic lupus erythematosus (SLE), respiratory and neural system.