MORPHOLOGY OF PATHOLOGICAL FORMS OF PLATELETS

Khalimova Yulduz Salokhiddinovna
Assistant, Department of Clinical Sciences,
Asian International University of Bukhara, Uzbekistan.

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Abstract. *Platelets are small (2-3 microns) nuclear-free, flat, colorless shaped blood cells. They are formed by fragmentation of their megakaryocyte precursors in the bone marrow, with a platelet lifespan of 5-9 days. For a long time, researchers have proposed various classifications of platelet morphological forms based on variations in one parameter or another. They play an important role in the body, performing a number of complex functions, participating in various processes. It is on platelets that the preservation of blood in a liquid state, the dissolution of formed blood clots and the protection of the walls of blood vessels from damage depend.*

Key words: *platelets, megakaryocytes, granulomer, hyalomer, thrombocytopathies.*

МОРФОЛОГИЯ ПАТОЛОГИЧЕСКИХ ФОРМ ТРОМБОЦИТОВ

Аннотация. *Тромбоциты — это небольшие (2–3 мкм) безъядерные, плоские, бесцветные клетки крови. Образуются путем фрагментации своих предшественников мегакариоцитов в костном мозге, продолжительность жизни тромбоцитов составляет 5–9 дней. Исследователи давно предлагают различные классификации морфологических форм тромбоцитов, основанные на вариациях тех или иных параметров. Они играют важную роль в организме, выполняя ряд сложных функций, участвуя в различных процессах. Именно от тромбоцитов зависит сохранение крови в жидком состоянии, растворение образовавшихся тромбов и защита стенок сосудов от повреждений.*

Ключевые слова: *тромбоциты, мегакариоциты, грануломер, гиаломер, тромбоцитопатии.*

Platelets are an important component of the hemostatic system: platelet adhesion to the site of vascular injury, aggregation, secretion of coagulation factors, subsequent clot retraction, spasm of small vessels and the formation of a white platelet thrombus stop bleeding in microcirculatory vessels with a diameter of up to 100 nm. Activation of the coagulation system induces the formation of fibrin on the surface of activated platelets and the formation of a full-fledged thrombus. When platelets are activated by natural stimulants such as thrombin or collagen, which are exposed when the vascular wall is damaged, they are able to eject the contents of their granules containing clotting factors, peroxidase, serotonin, calcium ions – Ca2+, ADP, Willebrand factor, platelet fibrinogen, platelet growth factor, etc. At the highest degree of activation, the platelet surface becomes procoagulant due to the exposure of phosphatidyl serine and stimulates the

formation of a blood clot. Platelets also play an essential role in the healing and regeneration of damaged tissues by releasing growth factors that stimulate cell division and proliferation in the damaged area. Hereditary platelet dysfunction encompasses a diverse group of hemorrhagic diseases caused by congenital defects in platelet morphology and/or function in normal numbers.

Various structures can be damaged and various processes in platelets can be disrupted: membrane receptors, intra-platelet signaling, granules, etc. This leads to various clinical manifestations of bleeding [2-4]. Thrombocytopathies are characterized primarily by the development of spontaneous and post-traumatic mucosal bleeding. The recognition and differentiation of thrombocytopathies is based on the detection of microcirculatory bleeding with impaired functional properties, morphology and biochemical characteristics of platelets. Based on these manifestations, the modern classification of thrombocytopathies is based, which is divided into 2 large groups – hereditary and acquired. Clinical manifestations depend on the characteristics of qualitative and quantitative platelet defects – the severity of hemorrhagic syndrome can vary significantly and does not depend directly on the degree of defect. With mild bleeding, there may be a tendency to bruising with minor and minor injuries, at the site of compression with an elastic band; periodic excessive nosebleeds, familial prolonged menstruation in women, etc. In the case of massive hemorrhagic syndrome, life-threatening blood loss may develop. Let's look at some thrombocytopathies in more detail.

Glanzmann's thrombasthenia is a hereditary disease characterized by hemorrhagic manifestations, in which there is an elongation of bleeding time, as well as a complete absence or a sharp decrease in the intensity of blood clot retraction against the background of a normal platelet count per unit volume of blood [5, 6]. This is the result of a decrease in platelet aggregation capacity. The disease was first described in 1918. Dr. Edward Glanzman. Glanzmann's thrombasthenia gravis is a rare disease and occurs with a frequency of approximately 1 case per 1 million [7]. The manifestation of the disease occurs in early childhood. The main clinical manifestations are cutaneous hemorrhagic syndrome, bleeding from mucous membranes, including gastrointestinal, up to life-threatening. The formation of soft tissue hematomas of various localization is possible. Hemorrhagic syndrome can be either post-traumatic or spontaneous. Carrying out any surgical interventions without hemostatic therapy, including tooth extraction, is accompanied by the development of bleeding. Currently, there are 3 types of Glanzmann's thrombasthenia gravis: type 1 – deficiency of the GPIIb–IIIa complex of surface glycoprotein aggregation receptors < 5% of the norm; type 2 – deficiency of the GPIIb–IIIa complex 5-20% of the norm, type 3 – the GPIIb–IIIa complex is present in normal or almost normal amounts, but is functionally unstable. Nevertheless, there is no correlation between the number of GPIIb–IIIa on the platelet surface and the severity of the clinical manifestations of the

disease [6]. The pathogenesis of this disease is based on a deficiency or dysfunction of platelet membrane proteins, integrin aIIbb3 (GPIIb–IIIa), which form a heterodimer complex on the platelet surface that binds fibrinogen, Willebrand factor, fibronectin and vitronectin. This membrane complex is a necessary component of the final stage of aggregation activated by physiological agonists [8].

Upon activation, the GPIIb–IIIa complex changes its conformation and binds fibrinogen and other soluble adhesive proteins, which, with the participation of Ca2+ ions, mediate the aggregation of adjacent platelets in the forming clot [9-11]. Glanzman's thrombasthenia gravis has an autosomal recessive type of inheritance [6, 12]. Both the aIIb and β3 integrin genes are located on the long arm of chromosome 17q21.32 and are encoded in the ITGA2B and ITGB3 genes, respectively. Gene expression occurs independently of each other [8, 13]. Small deletions and modifications are more common than large rearrangements of genes [14]. Despite the fact that the ITGB3 fragment has a smaller size, due to the presence of a larger number of exons, its mutation occurs with a higher frequency [15]. As a result of the mutation, patients with Glanzmann's thrombasthenia may have a deficiency or disruption of the GPIIb–IIIa structure. The presence of a molecular defect in one or two genes is sufficient for the formation of thrombocytopathy [16-19]. The severity of the clinical picture of Glanzman's thrombasthenia does not depend on the identified mutations and may vary within the same family.

Gray platelet syndrome. CCT (OMIM #139090) was first described by Raccuglia in 1971 [20]. It was characterized as a pathological condition accompanied by thrombocytopenia, rather mild manifestations of bleeding and the presence of agranular platelets in peripheral blood. Biochemical and electron microscopic methods have shown that all organelles, except for α-granules, are unchanged and present in normal amounts [21-23]. It is assumed that the cause of this syndrome is the inability of megakaryocytes to form specific vesicles and fill them with α-granular components [24].

At the same time, the number of megakaryocytes in the bone marrow is usually normal [25]. Microscopically, large, pale-colored platelets are found in the cell. Platelet dysfunction manifests itself in a decrease in aggregation with collagen and/or thrombin. Patients with CST are often characterized by varying degrees of macrothrombocytopenia, bleeding of the mucous membranes, and myelofibrosis and splenomegaly develop over the course of life [26, 27]. Bleeding is usually not life-threatening, however, with surgical interventions or serious injuries, massive blood loss is possible that cannot be stopped by standard methods. In such patients, platelet aggregation tests give very variable results, and no general vector of change in this indicator could be identified [28]. In most families, there is only 1 case of CST or several siblings suffer at the same time. The inheritance pathways and genes linked to this disease are diverse.

There are known cases of X-linked inheritance of a mutation in GATA-1, which leads to a general decrease in the number of granules in platelets and the appearance of defective red blood cells [29]. In 2014, D. Monteferrario et al. [30] reported the identification of a previously unknown nonsense mutation in the gene of transcription factor (repressor) GFI1B in a family with autosomal dominant CT. The NBEAL2 gene is most often damaged in CST, a variety of mutations in which were identified in 2011 by C.A. Albers et al. [31].

Bernard–Soulier syndrome is a hereditary thrombocytopathy caused by a genetic defect or a decrease in the functional activity of the GPIb-IX–V platelet complex. This complex is a Willebrand factor receptor, and is also necessary for thrombin fixation on the platelet surface. From a functional point of view, platelet adhesion to the vascular subendothelial matrix is impaired, which is also characteristic of Willebrand's disease. The main diagnostic criteria for this pathology are macrothrombocytopenia and the absence of Willebrand factor-dependent aggregation with ristocetin, with a normal amount and normal activity of the factor itself. There may also be a decrease in aggregation with thrombin against the background of normal aggregation with other agonists. Deficiency of the GPIb-IX–V surface glycoprotein complex can be confirmed by flow cytofluorometry and by genetic analysis of the GPIBA, GPIBB and GP9 genes.

Bernard–Soulier syndrome is manifested by significant bleeding of microcirculatory and mixed type, which manifests itself immediately after birth. Inheritance is autosomal recessive [32]. Wiskott–Aldrich syndrome. Microthrombocytopenia and impaired platelet aggregation indicate the presence of a qualitative or quantitative defect in the specific WASP protein (Wiskott–Aldrich syndrome protein). The classical form of CBO is characterized by a complex of disorders, which includes increased bleeding, recurrent bacterial, viral and fungal infections, as well as skin eczema.

There is a milder form of the disease – X-linked thrombocytopenia. The disease is characterized by the absence of pronounced signs of immunodeficiency and eczema. In order to verify the diagnosis of this group of patients, bone marrow puncture and myelogram analysis should be performed. A normal number of unchanged megakaryocytes is noted in the myelogram during CBO. Immunological defects in patients with CBO are the result of a violation of lymphocyte homeostasis, manifested in a sharp decrease in the proportion of T and B lymphocytes. When studying functional disorders of platelets in patients with CBO, increased expression of phosphatidylserine and the formation of microparticles in response to a stimulus are detected. A probable mechanism for the development of thrombocytopenia is increased removal of platelets expressing phosphatidylserine by spleen macrophages. To confirm the diagnosis of Wiskott–Aldrich syndrome and X-linked thrombocytopenia, it is necessary to analyze protein expression and determine the WASP gene mutation [33].

The MYH9 group of syndromes. The presence of large basophilic inclusions (Dele bodies) in granulocytes and monocytes in a blood smear during Romanovsky-Giemse staining is a marker of the MYH9 group of syndromes.

This group of syndromes includes the May–Hegglin anomaly, Fechtner, Epstein, and Sebastian syndromes. The May–Hegglin anomaly was described by the German physician R. May (1863-1937), and later by the Swiss physician R.M. Hegglin (1907-1969). The pathology is based on a mutation of the MYH9 gene encoding the non–muscular myosin IIA heavy chain (NMMHC-IIA). It is asymptomatic in most cases, but in some patients it is manifested by increased bleeding. The type of inheritance is autosomal dominant. It is accompanied by thrombocytopenia, kidney damage (nephritis), neuro-sensory hearing loss and cataracts, but the presence of these pathologies is not mandatory, especially in children. Platelet aggregation with collagen is often disrupted in patients with May–Hegglin anomaly during normal aggregation with other agonists, especially with ristocetin. The detection of NMMHC–IIA aggregates in neutrophils by immunofluorescence confirms the diagnosis of this group of syndromes. In order to determine a specific mutation, a genetic analysis is recommended [34].

Storage pool deficiency syndromes. These include the Hermansky–Pudlak and Chediaka–Higashi syndromes. They are inherited in an autosomal recessive way. These syndromes include albinism, frequent infections, pulmonary fibrosis, granulomatous colitis, prolonged bleeding time, and minor blood clotting disorders. The cause of the disease is a deficiency in the contents of dense granules or themselves. Studies of platelet function reveal a violation of aggregation in reaction with ADP, adrenaline, ristocetin and collagen. In Chediak–Higashi syndrome, dense granules detected by electron microscopy are larger than normal and similar in size to granules of melanocytes, leukocytes, and fibroblasts [35].

Scott syndrome. Thrombocytopathy, inherited by autosomal recessive type, caused by a defect in phosphatidylserine release during platelet activation and, as a result, a violation of platelet interaction with plasma coagulation factors. In this case, defective complexes of coagulation factors Va–X and VIII–IXa are formed on the membrane. Defects in the binding of these complexes lead to incomplete activation of factor X and prothrombin, as well as to impaired activity of platelet factor 3 [36].

Thus, the recognition and differential diagnosis of thrombocytopathies should be based on a comprehensive study of hemostasis, the study of platelet morphology by light and electron microscopy, the assessment of functional activity by flow cytofluorometry, as well as genetic analysis to identify mutations correlating with various types of thrombocytopathies.

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