International Journal of Pediatrics and Genetics
Vol. 3 No.1 (2025)
ISSN: 2995-5483

Platelet Aggregation Condition in Patients With Parodontitis

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Relevance of the study. Vascular walls and platelets are closely related functionally and morphologically, which creates a system called vascular-platelet or primary hemostasis. The vascular wall and normal blood circulation depend on this system. Failures in this system can lead to serious problems. Failures in primary hemostasis are responsible for 80% of cases of bleeding and up to 95% of cases of thrombosis. Platelets and vascular walls work together to prevent bleeding and respond quickly to damage. Platelets are activated immediately upon violation of the integrity of the vascular wall, adhere to the damage and begin to form a platelet aggregate. This unit temporarily closes the wound and prevents further blood loss. This process consists of platelet adhesion to exposed collagen fibers and their subsequent aggregation, which triggers a sequence of blood clotting reactions. Bleeding, which can be acute or chronic, can occur from various organs and tissues as a result of damage to the vascular wall or insufficient platelet function [2.4.6.8.10].

Results and analyzes. Platelet activation plays a key role in the development of ED. Although the main function of platelets is to ensure blood clotting, all blood cells are involved in the process of thrombosis. The number and activity of platelets determine the effectiveness of platelet hemostasis. In healthy people, the platelet count is 170-350 x 10-9 / L. There is a risk of thrombosis if their concentration is above 800x10x9/ l, and if it is below 80x10x9/L. Nevertheless, not only the quantitative but also the qualitative composition of platelets is crucial, as well as the presence of inhibitors that interfere with the performance of their functions, as well as the state of the plasma hemostasis system. Platelets ensure the integrity of the vascular system and hemostasis by performing a number of tasks. They promote the restoration of endothelial cells by secreting growth factors, and also support the structure and function of microvascular walls. In addition, platelets secrete vasoactive substances that promote spasm of damaged vessels in order to regulate vascular tone. When the integrity of the vascular wall is disrupted, platelets attach to the subendothelium and aggregate to form a platelet plug. This is the first step in stopping the bleeding. The growth factor secreted by platelets plays a key role in repairing damaged vascular walls. The effectiveness of primary hemostasis, that is, the process of formation of a primary platelet plug, depends on several factors, including the number of platelets, the level of PV, and the presence of glycoproteins responsible for platelet adhesion and aggregation [1.3.5.7.9.11].

Platelets are activated when the vascular endothelium is damaged. PV asserts that components of the subendothelial layer of the vascular wall, such as collagen, elastin, and proteoglycans, are essential for platelet adhesion. However, inactive platelets support and repair the vascular endothelium, performing an important trophic function under normal conditions. Various factors such as hypoxia, metabolic disorders of the vascular wall and exposure to toxic substances can lead to endothelial dysfunction and its transformation from an antithrombotic surface to a thrombogenic one. This is accompanied by damage to endothelial cells and an increase in the synthesis of procoagulant factors such as IL, ET, tissue thromboplastin, PV and factor V, which promotes thrombosis. Various physiological agonists are also involved in this process, which differ

in their chemical structure and ways of influencing platelets. Analysis of platelet aggregation activity under the influence of various inducers, such as adenosine diphosphate (ADP) in different concentrations, adrenaline, collagen and ristomycin, is an important diagnostic tool for detecting acquired and hereditary platelet dysfunction, as well as for evaluating the effectiveness of antiplatelet therapy. Adrenaline and ADP are important inducers of platelet aggregation, which play a key role in the blood clotting process. However, the mechanisms of their action differ, which is reflected in the shape of the aggregatogram, a curve that records the process of platelet aggregation over time. When epinephrine is exposed to platelets, it interacts with α2-adrenoreceptors on their surface. This leads to the inhibition of adenylate cyclase, the enzyme responsible for cAMP synthesis. A decrease in the level of cAMP in platelets causes a change in the permeability of the cell membrane to calcium ions, which is the trigger for the first wave of aggregation. The second wave of aggregation, observed under the action of adrenaline, is caused by more complex processes. It is associated with the release of platelet aggregation agonists such as ADP and serotonin, as well as with the synthesis of thromboxane A2, a powerful aggregation stimulator and vasoconstrictor. Thus, adrenaline causes two-wave platelet aggregation, which is reflected on the aggregatogram in the form of two consecutive peaks.

ADP is also a powerful inducer of platelet aggregation. However, unlike epinephrine, it acts directly on specific receptors on the platelet surface called P2Y1 and P2Y12. Activation of these receptors leads to an increase in calcium levels inside platelets, which triggers a cascade of reactions leading to aggregation. When exposed to low concentrations of ADP (for example, 1*10^-7 M), two-wave aggregation is observed on the aggregatogram. The first wave is caused directly by the binding of exogenous ADP to platelet receptors, and the second wave is caused by the release of their own agonists, such as ADP and thromboxane A2. Thus, the second wave of aggregation is the result of increased action of ADP due to the release of endogenous agonists. When exposed to high concentrations of ADP (for example, 1*10^-5 M), both waves of aggregation merge into one powerful and long-lasting wave. This is due to the fact that a high concentration of ADP causes maximum platelet activation and the release of a large number of endogenous agonists, which leads to increased and prolonged aggregation [12.13.14].

ADP and collagen are powerful platelet activators, but their mechanisms of action vary. When ADP is exposed to platelets, intracellular calcium levels increase. This triggers a cascade of reactions, the key link of which is the activation of calcium-dependent phospholipase A2. This enzyme cleaves arachidonic acid from membrane phospholipids. Further, arachidonic acid, under the action of cyclooxygenase and thromboxane synthetase, is converted into thromboxane A2, a powerful aggregation stimulator and vasoconstrictor. Thus, ADP indirectly, through an increase in calcium levels and activation of phospholipase A2, stimulates the synthesis of thromboxane A2, which leads to platelet aggregation. Ristocetin (ristomycin) is a glycopeptide antibiotic that was originally developed to treat bacterial infections. However, shortly after its introduction into clinical practice, it was discovered that it causes thrombocytopenia – a decrease in the level of platelets in the blood. As a result, ristocetin was withdrawn from the market as a medicinal product, but found application in the laboratory diagnosis of blood clotting disorders. In laboratory conditions, ristocetin is used at a concentration of 1.5% in combination with buffer solutions and stabilizers. It does not have a direct effect on platelet metabolism, that is, it does not cause their activation and degranulation, as do other aggregation inducers such as ADP or collagen. However, ristocetin has a unique ability to cause platelet aggregation indirectly, through interaction with PV.

Ristocetin binds to PV and changes its conformation, which leads to the exposure of

Vol. 3 No.1 (2025)
ISSN: 2995-5483

additional binding sites for the GPIb receptor on the platelet surface. As a result, bridges are formed between PV molecules and GPIb receptors, which leads to platelet aggregation. This phenomenon is used in laboratory diagnostics to assess the functional activity of PV and identify disorders of its structure or function. For example, in von Willebrand's disease, a hereditary disease associated with a deficiency or defect of PV, platelet aggregation in response to ristocetin is reduced or absent. In addition, ristocetin-induced platelet aggregation is used to evaluate the function of the GPIb receptor. For example, in Bernard-Soulier syndrome, a hereditary disease characterized by a defect in the GPIb receptor, platelet aggregation in response to ristocetin is also disrupted.

In the laboratory, thrombocetin is used to diagnose and investigate platelet aggregation disorders, especially if von Willebrand's disease or other thrombocytopathies are suspected. Ritocetin detects aggregation defects caused by insufficient activity or abnormalities of PV. This is important for making a correct diagnosis and choosing effective treatment. PV is a highly accurate marker of ED. This multicomponent glycoprotein, synthesized by megakaryocytes and endothelial cells, plays an important role in blood clotting and vascular-platelet hemostasis. It provides the transport of blood coagulation factor VIII to the site of damage to the vessel and promotes the attachment of platelets to the collagen of the vascular wall. The synthesis of PV occurs in the endothelium and platelets in several stages. Normally, PV is in a free state in the blood plasma, and is also stored in endothelial cells and platelet granules. When blood vessels are damaged, PV is released from these depots and participates in the formation of a blood clot. Most of the PV is stored in Weibel bodies, a cascade of endothelial cells that are similar to alpha-granules of platelets [11.13.14].

PV circulating in the blood in combination with coagulation factor VIII plays a key role in the process of platelet adhesion to damaged areas of blood vessels, especially in conditions of high shear rates characteristic of arterial blood flow. Hemodynamic factors, such as the speed and direction of blood flow, have a significant impact on the effectiveness of this process. Platelet adhesion mediated by PV occurs most intensively in the arterial system. PV is vital for arterial circulation, where effective hemostasis mechanisms are necessary for rapid blood flow. Willebrand's disease, which is characterized by blood clotting disorders and can lead to increased bleeding, can be caused either by a lack of PV in plasma or by its biological inactivity [12.14].

Elevated PV levels in blood plasma are an important indicator of vascular wall damage and may indicate the presence of thrombosis or a predisposition to it. High concentrations of PV indicate activation of the endothelium and an increased risk of blood clots. The unique ability of PV to cause bonding of formaldehyde-treated and lyophilized platelets in the presence of the antibiotic ristocetin underlies the method for determining its activity.

Conclusion. Thus, an increased level of PV in the blood plasma not only indicates damage to the vascular wall, but is also an indicator of the progression of atherosclerosis and thrombosis. In addition, PV is a reliable prognostic marker for patients with peripheral vascular diseases. In acute and chronic ischemic disorders of cerebral circulation caused by damage to the main arteries, there is an increase in the level of PV in blood plasma and an increase in both spontaneous and induced platelet aggregation. These results confirm the function of PV in the development of thrombotic and ischemic conditions and emphasize its importance as a diagnostic and prognostic tool in clinical practice.

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