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Opinion Article

Importance of Platelets and Its Activation for Healing Process

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DESCRIPTION

Platelets play an important role in controlling bleeding from blood vessels in the human body. Platelet activation is a key event in hemostasis and thrombosis. Upon injury to the vessel wall, platelets amplify the initial stimulation and aggregate to form a platelet plug, from which the fibrin component of the thrombus arises. When activated, platelets change its shape majorly, become stickier, and secrete various proteins and chemicals. Platelets are also involved in inflammation, angiogenesis, innate immunity, and other non-hemostatic physiological processes. Platelet activity is primarily associated with initiation of the coagulation cascade. Platelet adhesion to the extracellular matrix is the first step in primary hemostasis. Primary hemostasis is the response to vascular injury that produces a platelet clot at the site of damage in the body. For primary hemostasis to occur the blood get attached to the damaged tissue and become activated. This activation act as an initiation step in which more platelets can be "recruited" to form a platelet "plug" and helps in stopping the blood loss from the injured area. Platelets dysfunction can be examined using flow cytometer. It uses a laser to identify proteins on the surface of platelets and how they change when platelets are activated. Flow cytometer can be used to measure platelet activation and assess the degree of platelet sensitivity to added agonists. Classically, platelet activation is induced by collagen or soluble platelet agonists binding to G protein-coupled receptors, leading to activation of platelet adhesion receptors, primarily integrin αIIbβ3, which mediates platelet adhesion and aggregation. Disorders of platelets can lead to the symptoms like bleeding. Discovering the defects in the platelets before time using appropriate experimental methods paves the way for effective management and treatment of platelet diseases. When faced with an order for platelet function tests, choosing the most appropriate approach to detect platelet disease can be a challenge for researchers and clinical physicians. Common platelet agonists used in flow cytometer are arachidonic acid, epinephrine, collagen, thrombin, thromboxane A2 analogs, and Thrombin Receptor Activating Peptide (TRAP). The four main analytical methods for platelet counts are; manual counting using phase-contrast microscopy, impedance analysis, light scattering/fluorescence analysis using various commercially available analyzers and immune-platelet counting by flow cytometer. Platelet counts can be performed manually using commercially available dilution systems, hemocytometers, and microscopy. These counts are less accurate than automated counts because it can be difficult to distinguish between platelets and debris. Platelet aggregation also reduces platelet counts on hemocytometers. During an injury, platelets get collected and form clusters on the site of the wound by a method known as clotting. It seals the blood vessels tightly and at the same time stops the flowing of extra blood from the body. The activation of platelets may be defined in 3 steps: adhesion of platelets to adjoining platelets, collagen fibrils of the subendothelium or synthetic surfaces; spreading and aggregation of platelets through autocatalytic signaling; activation of platelets and formation of a thrombus clot. Further, extra solid thrombus is shaped with the aid of using a fibrin mesh binding to the platelet mixture. Platelets are activated to alternate shapes right into a pseudopodal shape upon the adhesion to the injured place as a way to spark off the collagen receptors on their floor membrane, named GpIIbIIIa, to go through launch reactions. Platelets are cells that assist blood clot, and that they incorporate unique proteins referred to as increase elements. Growth factors play essential role in the restoration and recuperation of the body by bringing greater collagen, oxygen, and different factors to broken tissue. Platelet activation is done by using certain platelet secretion process and neighborhood prothrombotic elements consisting of tissue factor. Multiple pathways can cause platelet activation. In the resting, unstimulated state, P-selectin is found on the surface of platelets. However, after platelet (or endothelial cell) activation, fusion of the cell membrane with the granule membrane results in rapid expression of P-selectin on the cell surface. PAF is produced by endothelial cells, macrophages, neutrophils, eosinophil, monocytes and mast cells and activates many inflammatory cells. ATP then initiates platelet activation through the P2X1 receptor and inhibits ADP-mediated activation when acting as an antagonist at the P2Y receptor. Lab specialists examine how platelets diffuse in the liquid portion of blood (plasma) and whether platelets aggregate after the addition of certain chemicals or drugs. When platelets clump together, the

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blood sample becomes more transparent. Platelets interact with several clotting factors, and the clotting product thrombin is a potent platelet-activating agonist. Platelet activation is stimulated by bound platelet secretory products and local prothrombotic factors such as tissue factor. Several pathways may lead to platelet activation. Platelet-Activating Factor (PAF) is a potent phospholipid mediator first described for its ability to induce platelet aggregation and vasodilation. It is now also known as a potent mediator of inflammation, allergic reactions, and shock. With a lifespan of approximately 8-10 days, platelets are continuously produced from bone marrow megakaryocytes, which release platelets into the blood to maintain levels of 150,000-400,000 per microliter of blood. This is main reason of human body for using platelets to heal injury.