EVOLUTION OF HEMOSTASIS METHODS IN DAMAGE PARENCHYMATOUS ORGANS

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Annotation: In this article we can learn evolution of hemostasis methods in damage parenchymatous organs. Hemostasis is the physiological process of stopping bleeding (hemorrhage). It protects the body from blood loss and exsanguination and restores blood circulation within the blood vessels. In general, it includes the conversion of liquid blood into solid (gel) form which plugs the ruptured blood vessel and prevents from blood loss. The body will then heal the ruptured vessel and repair the vessel for closed circulation.

Key words: primary hemostasis, secondary hemostasis, process, collagens, Clotting factors, mechanism.

Hemostasis can broadly be classified into two stages; (i) primary hemostasis, and (ii) secondary hemostasis. The primary hemostasis is the process of formation of the soft platelet plugs around the ruptured point. It is the first strategy adopted by the body to prevent blood loss. In this process, collagens are activated and accumulated in and around the area of the ruptured blood vessel. These accumulated collagens attract platelets and make them stick together to form a seal of the platelet. Besides, the platelets will induce vasoconstriction and release of factors which will attract more platelets in that ruptured site.

The secondary hemostasis is the process of formation of fibrin fiber which will solidify the soft platelet plug into a hard and strong fibrin clot. It is induced after the

primary hemostasis and involves a complex cascade which is described below in detail. Clotting factors are the plasma proteins that help in the formation of blood clots. These are the primary elements that form and maintain blood clots. These proteins are found freely floating in blood plasma mostly in the inactivated form. Once the body detects hemorrhage, these factors are activated and incorporated into the hemostasis cascade to form the blood clot. The major clotting factors are fibrinogen and prothrombin which forms fibrin and thrombin proteins respectively after activation and forms blood clots. However, there are more than 30 clotting factors that are involved in the complex process of hemostasis. The thirteen primary clotting factors are designated by the Roman numeral I to XIII while the other remaining factors and cofactors are named differently.

The primary clotting factors involved in hemostasis with their major functions are tabulated below. Other factors like Fibronectin, von Willebrand factor, Antithrombin III, High molecular weight (HMW) kininogen, Heparin cofactor II, Plasminogen, etc. are also involved in hemostasis.

Mechanism of Hemostasis

As soon as the blood vessel rupture exposing the components inside the vessel wall into the blood and blood leaks outside the vessels, the hemostasis mechanism is triggered. It can be completed within seconds (around 15 seconds) or may take several hours based on the physiological and biochemical status of the person and the site and the degree of blood vessel rupture. It is a complex process that can be described into three major steps viz. vascular spasm, platelet plug formation, coagulation, and the last step of fibrinolysis.

1. Vascular Spasm

Vascular spasm, also known as vasoconstriction, is the initial response that falls in primary hemostasis. As the endothelium cells are damaged during the vascular rupture, endothelin-1 (a vasoconstrictor) is released which mediates the vasoconstriction. The damaged endothelium of the vessel exposes other chemical components like sub-endothelial collagen, ATP (adenosine triphosphate), von

Willebrand factor, and inflammatory mediators into the circulation. All of these promote vasoconstriction. The sub-endothelial collagens and von Willebrand factors promote platelet accumulation and adhesion in that ruptured site. The attached platelets rupture and release serotonin, ADP (adenosine diphosphate), and thromboxane A2. All these components of the platelets further increase vasoconstriction. During the rupture of the blood vessel, local pain receptors initiate reflexes which further promote the vascular spasm. The effect of vascular spasm is more promising in smaller vessels with minor rupture. The effect may last for 30 minutes to several hours.

2. Platelet Plug Formation

The freely floating platelets in the circulation begin to clump together forming spiked and sticky platelet clumps and attaching over the exposed vesicular lining and collagen. Under the influence of von Willebrand factors, the platelet plug stabilizes and further accumulates over the exposed endothelial tissue. These attached platelets release their contents, mainly the ADP, further attracting other platelets toward that site. These platelets bound with collagen and endothelial lining form a temporary seal called the platelet plug. The platelet plug temporarily seals the vessel and prevents or slows down the rate of blood loss. This process results in primary hemostasis. Once the platelet plug is formed, the clotting factors are activated and the secondary hemostasis (blood coagulation process) begins.

3. Coagulation (Clotting)

Blood coagulation also called blood clotting, is the process of solidification of the blood due to the formation of fibrin fiber-associated blood clots. It is the secondary hemostasis stage and results in a stable solid blood clot. The overall process completes three major tasks; first, the activation of clotting factors, second the conversion of prothrombin into thrombin, and finally the conversion of fibrinogen into fibrin fiber. Coagulation Pathways The coagulation process, also called coagulation cascade, is initiated through two different pathways; the extrinsic pathway and the intrinsic pathway, leading to a final common pathway of activating and stabilizing fibrin. The

fibrin traps the platelets and blood cells forming a stable, gelatinous, and robust clot that ceases the hemorrhage.

It is one of the initial cascades leading to the activation of factor X. It is a very fast and explosive pathway that begins immediately after the exposure of tissue factor (factor III) on circulation, and is completed within about 15 seconds of the exposure. It is induced by the tissue factor, hence the pathway is also known as the tissue factor pathway.

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