Medical Laboratories Techniques Department

Lecture 17



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Platelets (thrombocytes)

Platelets

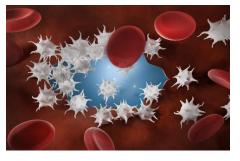
Platelets are small blood cells with several physiological purposes; through their clotting activity and activation of the coagulation cascade, they are crucial to maintaining adequate blood volume in those with vascular injury. The initiation of this activity begins with tissue injury and results in the release and binding of several glycoproteins, growth factors, and clotting factors. The complexity of these processes allows for many pharmacologic targets.

Development

Mature megakaryocytes form platelets. Megakaryocytes are large blood cells whose principal function is the production of platelets. When a megakaryocyte becomes mature, pseudomembrane, platelets, once formed, have an average **lifespan of 7 to 10 days**, at which point they are removed from the bloodstream.

The normal concentration of platelets in the blood is between **150,000 and 300,000 per \muL**.

Platelets have many functional characteristics of whole cells, even though **they do not have nuclei and cannot reproduce**. In their cytoplasm are:



(1) Actin and myosin molecules, which are contractile

proteins similar to those found in muscle cells, and still another contractile protein, **thrombosthenin**, that can cause the platelets to contract.

(2) Residuals of both the endoplasmic reticulum and the Golgi apparatus that synthesize various enzymes and store large quantities of calcium ions.

(3) Mitochondria and enzyme systems that are capable of forming adenosine triphosphate (ATP).

(4) Enzyme systems that synthesize **prostaglandins**, which are local hormones that cause many vascular and other local tissue reactions.

(5) An important protein called **fibrin-stabilizing factor**.

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Function

Platelets maintain **hemostasis** by adhering to the vascular endothelium, aggregating with other platelets, and initiating the coagulation cascade, leading to the production of a **fibrin mesh**, which effectively **prevents significant blood loss**. Platelets are also crucial in inflammation, tissue growth, and immune response. These processes are under the mediation of the release of compounds from the alpha and dense granules, which include numerous growth factors as well as IgG and components of the complement system.

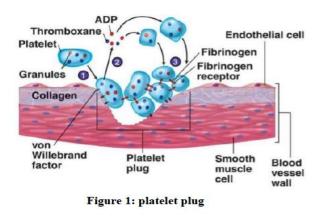
Mechanisms of prevention of blood loss

Whenever a vessel is severed or ruptured, hemostasis is achieved by several mechanisms: (1) vascular constriction, (2) formation of a platelet plug, (3) formation of a blood clot as a result of blood coagulation, and (4) eventual growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.

Active platelets become sticky so that they adhere to collagen in the tissues and to a protein called **von Willebrand factor** that leaks into the broken tissue from the plasma. Platelets secrete large quantities of ADP and the enzyme thromboxane A2.

The ADP and thromboxane in turn act on nearby platelets to activate them as well,

and the stickiness of these additional platelets causes them to adhere to the original activated platelets. Therefore, at the site of a puncture in a blood vessel wall, the damaged vascular wall activates successively increasing numbers of platelets that attract more and more additional platelets, thus forming a **platelet plug**. Then, during the subsequent process of blood coagulation, **fibrin threads** form. These **threads** attach tightly to the platelets, thus constructing an unyielding plug.



References:

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