Hemostasis

Hemostasis is one important homeostatic mechanism of the human body. It prevents loss of blood when a blood vessel is teared. There are 3 mechanisms involved in this process:

1. vasonstriction;
2. formation of a platelet plug;
3. blood coagulation.

Homeostasis overiew:

Homeostasis division (primary and secondary):

Homeostasis:

Vasoconstriction

Once the blood vessel has been cut or ruptured, the smooth muscle of the vessel begins to contract reducing immediately the blood flow. The reduction in blood flow may last for minutes or even hours. The contraction is caused by:

1. Local myogenic contraction (spasm) which is initiated by direct damage to the wall of the blood vessel
2. Local humoral factors from the damaged tissues and the platelets (for example, thromboxane A and serotonin)
3. Nervous reflexes which are initiated by pain or other nerve impulses that originate in the site of the damage or surrounding tissues.

The greater the damage of the blood vessel, the greater the degree of the spasm.

Formation of a Platelet Plug

Many small vascular holes develop during the day throughout the human body. These small holes or any other minor cuts are often sealed by a platelet plug rather than a blood clot.

The whole mechanism of the formation of the platelet plug is based on important functions of the platelets. Once the platelets come in contact with the collagen fibres present in the vascular wall they instantly alter their own characteristics.

1. The platelets start to swell
2. They take irregular forms with many irradiating pseudopodes projecting from their surfaces
3. Their contractile proteins (actin, myosin and thrombosthenin) contract resulting in the release of granules that contain several actice factors
4. The platelets become sticky and therefore are able to adhere to the collaged present in the damaged vascular tissue and to a protein called con Willebrand factor that has leaked into the damaged tissue from the plasma.
5. They secrete large amounts of Adenosine Diphospate (ADP)
6. The enzymes of platelets form thromboxane A
7. ADP and thromboxane A activate nearby platelets and those additional sticky platelets adhere to the original activated platelets.

The damaged blood vessel wall manages to activate successively increasing numbers of platelets and those platelets in turn, attract more additional platelets resulting in the formation of a platelet plug.

Initially, the platelet plug is loose but it is successful in stopping the blood loss from a minor vascular opening. During the blood coagulation process fibrin fibres form which attach tightly to the platelets making in an unyielding plug.

Links

Bibliography

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