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VASCULAR COMPONENT OF HEMOSTASIS

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Abstract:

In the process of blood coagulation, initially under the influence of serotonin, the injured blood vessel shrinks and reduces the area of bleeding. Heparin is a direct-acting anticoagulant and has an anti-coagulant effect. Heparin on the surface of the endothelium keeps blood in a liquid state and prevents clotting factors from sticking to the surface of blood vessels.

Keywords:

Damage to the blood vessel wall leads to the release of active thromboplastin. And it is considered the impetus for the beginning of coagulation hemostasis. At the same time, the Willebrand factor released from the blood vessel wall leads to the activation of platelet hemostasis.

In the process of blood coagulation, initially under the influence of serotonin, the injured blood vessel shrinks and reduces the area of bleeding. Heparin is a direct-acting anticoagulant and has an anti-coagulant effect. Heparin on the surface of the endothelium keeps blood in a liquid state and prevents clotting factors from sticking to the surface of blood vessels. Also, antithrombin I, II and III factors in vascular endothelial cells prevent the increase of thrombin and its activity in blood serum.

1. Blood coagulation cell factors

Platelets participate in all phases of the hemostatic process. A platelet is a blood cell that contains more than 60 biologically active substances. 12 of them are factors that directly affect the blood clotting process.

Thrombocytes participate in the following processes:

1. Provides integrity, resistance and permeability of microvessels.
2. It participates in the formation of the primary thrombocytic plug through adhesion and aggregation activity.
3. Brings plasma clotting factors to the site of bleeding.
4. It provides blood clot retraction.

Adhesion, aggregation and retraction activities of platelets are known. Adhesion is the adhesion of platelets to the damaged area of the blood vessel. Willebrand factor released from the damaged vessel wall causes platelet activation and adhesion to the vessel wall. Adhesive platelets also release Willebrand's factor and cause platelet aggregation. Aggregation is the sticking together of platelets. The reduction of the formed thrombus is ensured by platelet retraction. Retraction begins 15-30 minutes after clot formation and lasts from 30 minutes to 3 hours. Leukocytes and erythrocytes also adhere to the formed white blood clot, and a red blood clot is formed. But this thrombus is liquid and flowable and can only stop bleeding from small-caliber vessels. Platelet thromboplastin and plasma thromboplastin combine to form active thromboplastin, which activates coagulation hemostasis.

2. Blood coagulation plasma factors

There are 13 blood clotting factors in blood plasma:

Factors	Composition of factors
I factor	Fibrinogen
II factor	Protrombin
III factor	Thromboplastin in blood serum
IV factor	Calcium ions
V factor	Proacceler
VI factor	Acceler
VII factor	Provonvertin and convertin
VIII factor	Antihemophilic globulin A
IX factor	Antihemophilic globulin V
X factor	Styuart – Prauer's factor
XI factor	Antihemophilic globulin S

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XII factor	Xageman's factor
XIII factor	fibrin strengthening factor

Blood clotting is a complex process, which consists of the following stages:
Stage I - formation of active thromboplastin;
Stage II - conversion of prothrombin to thrombin;
Stage III - conversion of fibrinogen to fibrin;
Stage IV - blood clot retraction:
Stage V - fibrinolysis.
In the blood of a healthy person, serum factors are in an inactive form, and they become active only when there is a danger to the human body, that is, when a blood vessel is damaged, and they ensure the formation of a blood clot.
Redraktozyme - provides reduction, i.e. retraction, of a thrombus formed to stop bleeding at the site of an injured blood vessel.
Fibrinolysin belongs to the group of anticoagulants and dissolves the clot after the blood has stopped.
Under the influence of active thromboplastin, prothrombin turns into thrombin, and thrombin, in turn, turns fibrinogen into fibrin. Fibrin strands strengthen the thrombus.
After the bleeding stops, the blood vessel wall is restored, and the clot is dissolved under the action of fibrinolysin or plasmin.
Hemostasis is conditionally divided into 2:
1. primary vascular-platelet hemostasis.
2. Secondary coagulation hemostasis.
Examination of the hemostasis system
Preparation for inspection.
Blood is taken in the morning on an empty stomach. Prolonged constriction of the blood vessel or excessive movement of the arm during blood collection leads to an increase in plasma factors.
Material for inspection.
Venous blood taken in 3.2% sodium citrate.
Platelet-rich plasma of blood taken in 3.2% sodium citrate for aggregation.
Requirements:
1. The main material for checking hemostasis is venous blood.
Capillary blood can be used in the following cases:
- determining the amount of platelets;
- determination of bleeding time;
- blood clotting time check;
- for express diagnostics in babies.
2. The duration of venous stasis during blood sampling should not exceed 1 minute.
3. Blood is taken from the wrist vein using a wide slotted needle.
4. Blood is collected in a plastic tube with 3.2% sodium citrate. It is not possible to take a bottle into a test tube.
5. The ratio of blood to 3.2% sodium citrate should be 9:1.
6. The collected blood should be delivered to the laboratory within 45 minutes.

METHODS OF CHECKING VASCULAR THROMBOCYTIC HEMOSTASIS.

1. Determination of the amount of platelets.
Normal platelets are 180-320 x109/l. In practice, two different methods are used:
- counting directly in the blood (using a Goryaev camera or analyzer).
- Counting according to 1000 erythrocytes in a blood smear by the Fonio method and multiplying by erythrocytes in 1 l of blood.
2. Determination of bleeding time (according to Duke).
According to Duke, the bleeding time is determined after the integrity of surface blood vessels is broken. Usually 2-4 minutes. Prolonged in thrombocytopenias and thrombocytopathies.
3. Platelet adhesion.
Platelet adhesion is determined by the number of platelets trapped in the fibers after passing a certain volume of blood through glass fibers at a standard speed. Normally platelet adhesion is 20-40%. Decreased platelet adhesion is characteristic of thrombocytopathies.
4. Platelet aggregation.
Platelet aggregation is determined photometrically using an aggregometer. In this case, an aggregate-forming substance (ADF, collagen, adrenaline, ristomycin) is added to platelet-rich plasma, and a curve is formed in the aggregometer. Platelet aggregation is normally 55-145%, and its decrease is characteristic of thrombocytopathies.
5. Blood clot retraction.
Blood without stabilizers is taken in a test tube and poured into a water bath at 37oC, and the presence of blood clot retraction is checked. Normally, the blood clot retracts after 30-60 minutes. Retraction is not observed in severe thrombocytopenia and thrombocytopathies.

COAGULATION HEMOSTASIS TEST METHODS.

1. Blood clotting time (according to Moravits).

Blood coagulation time normally starts in 2-3 minutes and ends in 4-5 minutes. Prolongation of blood clotting time is observed in coagulopathies.

2. Plasma recalcification time (according to Bergerhof and Roka).

The optimal amount of calcium chloride is added to platelet plasma and the clotting time is measured. The norm is 60-120 seconds. Prolongation of recalcification time is associated with deficiency of plasma factors and excess of heparin in plasma.

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3. Tolerance of plasma to heparin.
Effect of heparin on recalcification time of citrated or oxalate plasma. Usually 6-9 minutes. Its shortening indicates antithrombin III deficiency, and its lengthening indicates hypersensitivity to heparin. It is not used in patients with hypocoagulation.

4. Prothrombin time (PT).
Factor VII activity of PT is important in determining the monitoring of treatment with direct anticoagulants. Normally 9-12 seconds. Reduction of PT is characteristic of hypercoagulation, and reduction of PT is characteristic of hypocoagulation.

5. Active partial thromboplastin time.
APTT is important in the control of heparin therapy, in determining the internal factors of blood coagulation. APTT is normally 21-35 seconds. Reduction of APTT is characteristic of thrombosis and thromboembolism. Prolongation of APTT is characteristic of deficiency of plasma factors (VIII - hemophilia A, IX - hemophilia V, XI, XII).

6. Factors VIII, IX and XI - antihemophilic globulinA, V and C. Congenital deficiency of antihemophilic globulin A, V and C is the cause of hemophilia. Factor VIII is normally 70-150%. Factor IX is normally 60-140%.

7. Factor V
Normally 70 - 140%. Congenital deficiency of factor V is the cause of parahemophilia.

8. Thrombin time (TT).
TT assesses the final stage of blood coagulation - the conversion of fibrinogen to fibrin under the influence of thrombin. Normally TT is 15-18 seconds. Prolongation of TT is characteristic of heparin therapy, hypofibrinogenemia (fibrinogen less than 1.0 g/l). TT reduction is characteristic of hyperfibrinogenemia (fibrinogen more than 6.0 g/l) and DVS-syndrome hypercoagulable stage.

9. Fibrinogen.
Fibrinogen is converted into fibrin under the influence of thrombin and factor XIIIa. Normally, fibrinogen is 2.0-4.0 g/l.
Since fibrinogen is an acute phase protein, its amount can exceed 10 g/l in severe bacterial infections, trauma, and thrombosis. At the same time, the amount of fibrinogen is related to kidney diseases (pyelonephritis, glomerulonephritis, hemolytic-uremic syndrome), collagenoses (rheumatoid arthritis, nodular periarteritis), nocturnal paroxysmal hemoglobinuria, tumors, etc. increases. A decrease in the amount of fibrinogen occurs in congenital fibrinogen deficiency, liver failure, DVS-syndrome hypocoagulation stage, acute fibrinolytic conditions, infectious mononucleosis. Drug-induced hypofibrinogenemia can occur when taking sodium valproate, fibrates, phenobarbital, streptokinase, urokinase, L-acnapaginase.

10. Ethanol and protamine sulfate tests.
50% ethanol solution or 1% protamine sulfate solution is added to the plasma. When there are soluble fibrin monomer complexes, a gel is formed and the result is considered positive. A positive result is observed in the stage of DVS-syndrome hypercoagulation, massive thrombosis and thromboembolism.

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