ISSN 3060-4567

MODERN CONCEPTS OF BLOOD COAGULATION MECHANISMS

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The study of blood coagulation mechanisms and their regulation is impossible without the use of laboratory tests. To date, virtually all factors involved in blood coagulation are known. Their primary, secondary and tertiary structures have been discovered. Genes responsible for the formation of these factors have been discovered and deciphered. At the same time, in everyday practice, the study of haemocoagulation in the human body is carried out using laboratory methods, which are not always perfect.

Keywords: *blood coagulation, haemocoagulation, DIC, thrombosis, fibrinogen;*

The process of intravascular blood coagulation, or haemocoagulation, occurs constantly throughout human life. At the same time, its intensity varies. Violations of the intensity of intravascular coagulation lead to the development of such pathological manifestations as haemorrhages, thromboses and DIC, which is sometimes called thrombo-haemorrhagic syndrome.

Haemocoagulation within the vascular bed is carried out by the interaction of the procoagulant system, which forms fibrin, platelets, which often initiate clotting processes, and the fibrinolysis system, which regulates the size of the forming blood clot. Activated platelets and membranes of damaged cells participate in the formation of specific complexes consisting mainly of proteins - procoagulants, which provide the

ISSN 3060-4567

phenomenon of blood coagulation itself. Modern ideas about the mechanism of functioning of the platelet component of haemocoagulation can be presented as follows.

Normal platelets are disc-shaped and move in the circulating blood in isolation from each other without interacting with the vascular endothelium. When the vascular wall is damaged, platelets with the help of Willebrand factor adhere to subendothelial structures - collagen fibres, myofibrils, myocytes. In this case, they acquire a spherical shape. This stage is denoted by the term 'platelet adhesion'. After 30-60 seconds, adhered platelets release into the environment ADP, serotonin, adrenaline, fibrinogen, platelet factor 4 and a number of other biologically active substances.

There is a stimulation of platelet aggregation, which means their adhesion to each other. In this case, the release of biologically active substances from platelets increases. This phenomenon is referred to as the 'release reaction'. As a result, there is a rapid formation of a loose platelet plug, which provides primary haemostasis, but is unstable and can be destroyed. In this regard, this phase is usually called reversible platelet aggregation.

Due to the avalanche-like increase in the concentration of aggregates, the reversible phase of platelet aggregation turns into irreversible. Thrombin, formed as a result of activation of plasma clotting factors, plays a significant role in this. Platelets themselves contribute to the activation of factor CP, the formation of active factor X and the appearance of tissue factor. When platelet membranes are destroyed, conditions are created for the unification of platelet aggregates and compaction of the resulting clot. This phenomenon is called blood clot retraction. Simultaneously with platelets in the process of haemocoagulation participate procoagulants - a group of proteins and calcium ions, which in the process of their interaction lead to the formation of fibrin. It is fibrin that is the basis of both haemostatic and thrombotic phenomena.

Procoagulants are designated by Roman numerals: I - Fibrinogen, II - Prothrombin, III - Tissue factor, IV - Calcium ions, V-VI - Proaccelerin-Accelerin, VII - Proconvertin, VIII - Antihaemophilic globulin, IX - Christmas factor, X - Stuart factor, XI - Plasma thromboplastin precursor, XII - Hageman factor, XIII - Fibrin-

ISSN 3060-4567

stabilising factor. It is now generally accepted to use the numerical designation of factors other than tissue factor and calcium ions, sometimes fibrinogen and prothrombin. In addition to these factors, prekallikrein, also known as Fletcher factor, and a high molecular weight kininogen, called Fitzgerald factor, are involved in the process of fibrin formation. It is assumed that the process of fibrin formation consists of the sequential interaction of all factors with each other.

At the same time, some of them - proenzymes are converted into active enzymes, and some serve only to ensure the interaction of the enzyme and substrate. For a long time, the theory of the presence of two pathways of activation of plasma haemostasis and fibrin formation prevailed. The internal pathway of fibrin formation assumed the initial activation of factor XII, which with the participation of prekallikrein and high molecular weight kininogen activates factor XI, then factors IX and VIII are activated and include active factor X in the process.

The external pathway began with the formation of a complex of factor VIIa and tissue factor, which activated factor X. This was followed by the formation of prothrombinase (factor Xa + factor Va), the transition of prothrombin to thrombin and the formation of fibrin clot. Further studies showed that the leading role in the initiation of blood coagulation belongs to the tissue factor and the external pathway of fibrin formation.

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