# MODERN CONCEPTS OF BLOOD COAGULATION MECHANISMS

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The process of intravascular blood coagulation, or haemocoagulation, occurs constantly throughout human life. At the same time, its intensity varies. Disturbances in the intensity of intravascular coagulation lead to the development of such pathological manifestations as haemorrhages, thromboses and DIC, sometimes sometimes referred to as thrombo-haemorrhagic syndrome [10,11,12].

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Haemocoagulation within the vascular bed is carried out by the interaction of the procoagulant system, which forms fibrin, platelets, which often initiate the 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 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. At the same time, they acquire a spherical shape. This stage is referred to by the term ‘platelet adhesion’. After 30-60 seconds, adhered platelets release ADP, serotonin, adrenaline, fibrinogen, platelet factor 4 and a number of other biologically active substances into the environment. This stimulates platelet aggregation, which means their adhesion to each other [7,8,9].

The release of biologically active substances from platelets increases. This phenomenon is referred to as the «release reaction». This results in the 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 an essential role in this. Platelets themselves contribute to the activation of factor XP, the formation of active factor X and the appearance of tissue factor. When platelet membranes are destroyed, conditions are created for the aggregation of platelet aggregates and compaction of the resulting clot. This phenomenon is called blood clot retraction. Simultaneously with platelets, procoagulants - a group of proteins and calcium ions - participate in the process of haemocoagulation, which in the process of their interaction lead to the formation of fibrin [4,5,6].

Fibrin is the basis of both haemostatic and thrombotic phenomena. 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 referred to as Fletcher factor, and 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 in the sequential interaction of all factors with each other. At the same time, some of them - pro enzymes are transformed into active enzymes, and some of them serve only to ensure the interaction of enzyme and substrate. For a long time, the theory of two pathways of activation of plasma haemostasis and fibrin formation prevailed [1,2,3].

The internal pathway of fibrin formation assumed 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 pro-thrombinase (factor Xa + factor Va), the conversion of prothrombin to thrombin, and the formation of a fibrin clot. Further studies have shown that the leading role in the initiation of blood coagulation belongs to the tissue factor and the external pathway of fibrin formation.

In detail, the actual process of fibrin formation can be represented as follows: initially, activators of factor XI and factor VII are formed. This is carried out through a chain of interactions of Hageman factor (HF), pre kallikrein and high molecular weight kininogen. The next step is the activation of factors XI and VII. The fact of combining the internal and external pathways of factor X activation seems to be new in the modern hypothesis of fibrin formation, which is justified by the ability of kallikrein to simultaneously influence the formation of active factor VII [13,14,15,16,17,18].

Activation of factor IX by factor XIa leads to the possibility of complex

formation: Ca++, phosphopolypids, factor IXa, factor VIII. Complex with phospholipid and calcium ions forms also factor VIIa. These complexes activate factor X, which is key in the formation of thrombin. The complex of factor Xa with phospholipids, calcium and factor V affects prothrombin and leads to the formation of thrombin. Thrombin itself acts already on the fibrinogen molecule, which leads to the formation of a fibrin network. At the same time, low molecular weight peptides are initially detached from the fibrinogen molecule - fibrinopeptide A, detached from the alpha chain of fibrinogen, and fibinopeptide B, detached from the beta chain.

The fibrin monomers that are formed join together to form a fibrin network (polymerisation process). After the action of the factor XSa, it strengthens, stabilises and acquires a complete form. Thrombin generation is the key reaction of haemocoagulation. It is carried out in two stages. In the first stage, in the initiation phase, a small amount of thrombin is formed, which catalyses the activation of clotting factors. In the second stage, in the propagation phase, the so-called «thrombin explosion» occurs, which leads to the formation of a large amount of fibrin, which determines the size of the thrombus. The intensity of intravascular coagulation is controlled by anti-coagulation proteins, which include antithrombin, heparin II cofactor, a2 macroglobulin, a1-protease and C1-complement inhibitors, as well as protein C (Ci), protein S, thrombomodulin and tissue factor pathway inhibitor (TFPI). Protein C (C) activated by thrombin-thrombomodulin complex together with protein S inhibit activated cofactors (Va and VIIIa), and all others inhibit the action of activated factors themselves.

Tissue factor pathway inhibitor (TFPI) limits the activity of factor Xa and the complex of tissue factor with factor VIIa. The size of the thrombus also depends on the blood fibrinolytic system, consisting of plasminogen, its activators and inhibitors, which determines the level of plasmin produced. In the process of activation of coagulation and formation of a fibrin clot, molecules appear in the blood that indicate that this process is taking place. They are considered to be markers of the ongoing process of microclotting within the vascular bed, which may have varying degrees of intensity. One of the most important markers of this process is D-dimer. It provides information about the formation of fibrin in the bloodstream, strengthened by the action of fibrin-stabilising factor (factor XIII). This fibrin has already undergone the action of plasmin and cleaved with the formation of D-dimers. The level of D-dimer in blood plasma allows to judge the intensity of intravascular haemocoagulation. The pre-analytical stage is essential for the quality of laboratory tests, which includes the selection and administration of the test, preparation of the patient, receipt, storage and transport of biological material, direct preparation of material for the study. The material for coagulological studies is plasma containing all components of the coagulation system. Strict adherence to the rules of the pre-analytical stage is very

important for the reliability of haemostasis studies.

Thus, the process of obtaining blood and its initial processing is of particular importance. This is due to the fact that trauma to the vascular wall and contact of blood with air and the surface of the tube triggers a chain of biochemical reactions of the coagulation cascade and activates platelets. Therefore, it is necessary to ensure that the vein puncture procedure is as gentle as possible and that the blood sample is mixed rapidly with the anticoagulant.

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