3. HEMOSTASIS DISORDERS IN CARDIOVASCULAR PATHOLOGIES AND CIRCULATORY MARKERS

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Introduction. Hemostasis as an important compartment of general homeostasis plays a crucial role in regulation of blood fluidity and vascular wall defect closing and recovery. However, hemostasis disorders accompanied with prothrombotic activity boosting lead to a risk of thrombus formation.

Aim of study. To reveal the main mechanisms of hemostasis disorders in diverse cardiovascular pathologies, and proper circulating markers.

Methods and materials. Was analyzed a material reflected in 22 recent articles found in Google.

Results. Conceptually hemostasis consists of 3 systems: pro-coagulant, anticoagulant and fibrinolytic. Installed prothrombotic statement is a result of primary hemostasis triggering by platelets followed by secondary hemostasis activation due to increased ratio of pro/anticoagulant involvement. White thrombus is characteristic for artery bed leading to a risk of acute myocardial infarction and stroke, but red thrombus is formed in veins being a trigger of deep vein thrombosis and pulmonary thromboembolism. Common for them is endothelial injury due to atherosclerosis, dyslipidemia, inflammation, oxidative stress. Likewise, antiphospholipid syndrome (eg, systemic lupus), decreased activity of anticoagulant and fibrinolytic systems, and pro-coagulant boosting are involved. The pathogenesis of arterial thrombus embraces a special mechanism linked to von Willebrand factor (vWF) released by damaged endotheliocytes which being a pentamer can open its sites for platelet receptors and collagen subendothelial fibers only in a rapid and turbulent blood flow inherent to arteries. For hemostasis disorders assessment are used indices reflecting these 3 systems of hemostasis. Thus, for pro-coagulant activation it's characteristic increased plasma level of vWF, fibrinogen, prothrombin, factor V, anti-phospholipid autoantibodies and monomers of fibrin (the last marker underlines the intensity of fibrinogen depolymerization under the action of thrombin). Anticoagulant decline is represented by decrease of antithrombin III, thrombomodulin, protein C and its cofactor protein S. The anticoagulant property of protein C is linked to endothelium expression of its specific receptors (family of annexins) which might be inhibited by anti-phospholipid autoantibodies. Fibrinolytic incompetence is assayed by diminution of plasmin level.

Conclusion. The main tool of hemostasis disorders induced circulatory dyshomeostasis is based on pro-thrombotic setting and the used markers are referring to activation of the coagulant system as well diminution of anticoagulant and fibrinolytic systems.