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Hemostasis Systems in the Formation of Destruction in Tuberculosis

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Annotation: Infectious inflammation caused by the pathogen triggers the uncontrolled spread of proinflammatory mediators, followed by their activation of macrophages, neutrophils, lymphocytes and a number of other cells.With the development of tuberculosis against the background of pronounced immunodeficiency and the predominance of the exudative nature of inflammation with damage to the microhemocirculatory bed of the lungs, a significant deposition of fibrin in the inflammatory zone becomes possible.

Keywords: hemostasis, pulmonary tuberculosis, fibrin.

Relevance. Taking into account the individual characteristics of patients' metabolism, it is necessary to individually select the dose of the drug and regularly monitor its effectiveness using laboratory methods, since excessive and insufficient doses of the drug can equally lead to the development of complications [3,5]. According to modern concepts, the biological role of the hemostasis system in conditions of pathology consists in the formation of fibrin for the removal of inflammatory, dystrophic, necrotic, tumor lesions of organs and their subsequent restoration [1,7,11]. The deposition of fibrin in fresh inflammatory processes is considered an urgent, "emergency" reaction of the tissue to the occurrence of a lesion. The output of fibrin occurs from the vascular bed and is directly related to the severity of the exudative component of inflammation and vascular damage. The latter, in turn, are determined by the degree of disorders in the immune system [5,13]. At the same time, fibrin is not a permanent structure of the body and after performing its biological function must be removed using the fibrinolysis system [1,21,6]. With the development of tuberculosis against the background of pronounced immunodeficiency and the predominance of the exudative nature of inflammation with damage to the microhemocirculatory bed of the lungs, a significant deposition of fibrin in the inflammatory zone becomes possible. Vascular endothelial damage leads to activation of "contact" factors, activation and adhesion of platelets and promotes the release of tissue thromboplastin, which plays the role of a catalyst in relation to the beginning processes of local secretion and fibrin coagulation. Alveolar macrophages with the ability to produce procoagulant factors, in particular tissue thromboplastin and factor V, can play an important role in the pathogenesis of fibrin loss and the formation of the indurative nature of the inflammatory tissue reaction in tuberculosis [13, 12, 10]. The appearance of local local fibrin deposits can be accompanied by the same local release of plasminogen activators without any noticeable changes in the blood coagulation system and its fibrinolytic activity. Obviously, this explains the slight differences in indicators that we found in groups of patients with various forms of pulmonary tuberculosis with moderate severity of the process. With an increase in the immunodeficiency state of the body, the severity of exudative disorders, coagulation disorders at the local level in the lung tissue become more pronounced and are already able to influence the blood hemostasis system. At the same time, a "vicious circle" is formed that supports the progression of the pathological process, when the state of hypercoagulation favors the formation of fibrin, and the

latter is localized where stasis and vascular lesions become decisive factors. We believe that the deployment of pathological reactions in the focus of tuberculous inflammation can activate the blood clotting system and affect the local distribution of fibrin, which, together with the coagulating effect on the protein substances of the alveolar exudate of immune complexes and the released lipid components of the mycobacterial cell, leads to the formation of a caseous fibrin clot. The following lysis of fibrin as a result of activation of the plasmin system in tissues and blood contributes to the melting of this clot and the formation of destruction of lung tissue. At the same time, the extent of destructive phenomena in the lungs obviously depends on the initial functional state of the body (especially the immune system), which affects both the volume of caseous tissue degeneration and the activity of coagulation processes and the amount of fibrin deposits. The absence of noticeable abnormalities in the blood clotting system of patients with moderate activity of the inflammatory process is obviously explained by the rapid development of obliterating thrombangiitis, blockade of the release of active substances into the peripheral blood and the prevalence of local pathological reactions. With a high degree of inflammatory process with extensive damage to lung tissue, it is possible to change the coagulating potential of the blood, the degree of which may be a marker of the severity of the tuberculosis process. Our conclusions are consistent with the research of Astrup T. [13, 14], who formulated the theory of "integration", which represents the process of tissue repair through the local formation of fibrin and its subsequent dissolution. The author considered fibrin deposits as a matrix for migrating fibroblasts during the formation of reparative connective tissue. This function is especially pronounced in cases of tissue lesions accompanied by the formation of a pre-fibrin-containing exudate without the presence of shaped blood elements [8]. Consequently, the deposition of fibrin is a natural phenomenon and a necessary prerequisite for the normal recovery of affected tissues. At the same time, the final resorption and removal of fibrin is a necessary condition for successful repair and depends on the fibrinolysis system. Obviously, the occurrence of destruction in tuberculosis pneumonia largely depends on the ratio of coagulation and anticoagulation systems at the local tissue level. Lysis of the caseous-fibrin clot is caused by the release of the plasminogen activator from the endothelium of the vascular, mainly venous, bed, as well as an increase in the activity of alveolar macrophages, which are also able to activate fibrinolysis and phagocytize fibrin and its decay products [17,19].

Materials and methods. We assume that the processes leading to the lysis of fibrin deposited in the areas of caseous necrosis are the basis for the melting of caseous masses, followed by their rejection and the formation of destructive phenomena in the lung tissue. Indirectly, this is confirmed by the peculiarities of the influence of hemostasis factors on the development of tuberculosis in the case of species differences in the body's resistance to infection. Thus, in the lung tissue of rats characterized by natural resistance to tuberculosis, a high content of plasminogen activator and low thromboplastic activity were registered. In guinea pigs, which are highly sensitive to tuberculosis infection, on the contrary, the activator content is extremely low, and the number of antiproteases is increased.

Results. When rats are infected with virulent MBT cultures, destructive changes are rarely found in the lungs , which is probably due to the effective inhibition of the activation of coagulating factors in the lung tissue and the absence of significant local fibrin deposits. Adverse reactions occur in guinea pigs, resulting in the formation of extensive areas of tuberculous lesions and multiple cavities. It is obvious that the primary pathogenetic link at the local level underlying the development of destructive phenomena in tuberculosis is the activation of coagulating factors of lung tissue. Based on the data obtained, it is possible to outline the main directions in the tactics of treatment of patients with pulmonary tuberculosis. The main determining factors should be an assessment of the state of the immune system and, accordingly, the degree of activity of the inflammatory process, as well as, to a lesser extent, the ratio of the coagulating and anticoagulation systems of the blood. The significance of the latter in the system of laboratory diagnostics is determined not by the contribution of these systems to the process of destruction formation in the lung tissue, but by the insufficient information content of indicators determined

in peripheral blood. Therefore, it is desirable to develop techniques that allow a clearer understanding of the processes of formation and lysis of fibrin directly in the lung tissue. Thus, in pulmonary tuberculosis, the hemostasis system can play one of the main roles in the pathogenesis of destructive phenomena in the lung tissue, causing the deployment of a cascade of interdependent pathogenetic reactions with the formation of a fibrin-caseous clot, its subsequent lysis, melting of caseous masses, their rejection and the formation of decay cavities. Correction of the revealed disorders in the hemostasis system will increase the effectiveness of treatment of destructive tuberculosis, while the criteria for the appointment of appropriate measures need further development to determine clear indications for the appointment of pro or anticoagulant therapy, taking into account the stage of development of the pathological process. It is also necessary to develop techniques that allow to get an idea of the processes of formation and lysis of fibrin directly in the lung tissue.

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