

The State of Anticoagulant and Fibrinolytic Systems in Burn Shock

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Abstract: Modern studies indicate a close relationship between disorders in the hemostasis system and the development of a systemic inflammatory response in burn injury. Activated neutrophils and platelets, interacting with the damaged endothelium, contribute to the formation of a vicious circle that exacerbates microcirculatory disorders and tissue hypoxia. This determines the need for an integrated approach to the study of the mechanisms of development of hemostatic disorders in burn shock and the search for new therapeutic targets.

Keywords: DVS-syndrome, hemostasis, fibrinolysis, burn toxemia, burn shock.

Introduction. Burn injury remains one of the most serious problems of modern medicine, due to the high frequency of occurrence, severity of the course and a significant percentage of adverse outcomes. Burn shock, which develops with extensive thermal damage, is a complex pathophysiological process affecting all body systems and accompanied by profound disorders of microcirculation, water-electrolyte balance and metabolism.

A special place in the pathogenesis of burn shock is occupied by disorders in the hemostasis system, which largely determine the severity of the patient's condition and the risk of life-threatening complications. Anticoagulant and fibrinolytic systems play a key role in maintaining normal blood flow and preventing pathological thrombosis. In case of burn shock, there is a significant imbalance between pro- and anticoagulant mechanisms, which can lead to the development of DIC syndrome and multiple organ failure.

Despite significant advances in the study of the pathogenesis of burn shock, many aspects of the functioning of anticoagulant and fibrinolytic systems in conditions of thermal injury remain insufficiently investigated. Understanding the mechanisms of their disorders is important for the development of effective methods for the diagnosis, prevention and treatment of complications of burn shock. This determines the relevance of a detailed study of the state of these systems in the dynamics of the development of burn shock and their role in the formation of complications of thermal trauma.

In recent years, the understanding of the mechanisms of hemostasis disorders in burn injury has significantly expanded. It has been established that thermal damage triggers a cascade of pathophysiological reactions, including massive release of pro-inflammatory mediators, activation of the complement system, and development of endothelial dysfunction. These processes directly affect the state of the anticoagulant and fibrinolytic systems, leading to depletion of natural anticoagulants such as antithrombin III, protein C and protein S, as well as impaired function of the tissue plasminogen activator and its inhibitors.

Of particular importance is the study of the relationship between the degree of disorders in the anticoagulant and fibrinolytic systems and the severity of the clinical course of burn shock. An imbalance in these systems can serve as an early marker of the development of complications and an unfavorable prognosis. At the same time, existing methods of diagnosis and monitoring of the hemostasis system do not always allow timely detection of initial disorders and assessment of the risk of thrombotic complications.

The issue of developing personalized approaches to the correction of disorders of the anticoagulant and fibrinolytic systems remains relevant, taking into account the individual characteristics of the patient

and the stage of the pathological process. A detailed study of the molecular mechanisms of these disorders may open up new perspectives in the prevention and treatment of burn shock complications, which is important for improving treatment outcomes in patients with severe thermal trauma.

Goal. Assessment of the condition of coagulation, anticoagulation and fibrinolytic systems in the case of burn shock, acute burn toxemia and septicotoxemia.

Material and methods. The study was conducted among patients with deep burns in the Samarkand branch Rntsemp. Patients were divided into two groups:

Group I (control) – no blood loss prophylaxis in 105 patients;

Group II (Primary) – 112 patients had homeostasis correction and blood loss prophylaxis. The two groups were similar in age and burn area.

Results and discussion. The degree of burn shock during hospitalization was assessed on a deficiency in circulating blood volume. In the majority of patients (51.6%), circulating blood volume deficiency is at 20-30% levels, corresponding to Level II of shock. Grade I shock was found in 30.5% and Grade III shock in 17.9%. Also studied was the Alcover index, which depends on plasma loss levels.

AT III-antithrombin III; RFMK – soluble fibrin-monomer complexes.

In severe plasma losses, the Alcover index was noted to increase from 0.54 to 1.45. These data indicate that severe burns shock has significant changes in the homeostatic system. In patients with deep burns, the total volume of circulating blood decreased from 67.5 ± 0.4 ml/kg between Grade I and Grade III shock to 53.9 ± 0.4 ml/kg, giving a circulating blood volume loss of $32.9 \pm 0.7\%$ at Level III.

A hemostasis study conducted in the acute toxemia phase of 59 patients noted thrombocytopenia and hypofibrinemia, as well as increased blood clotting and decreased AT-III levels. Clinical data indicate the initial development of DVS-syndrome in Burnout disease, including increased levels of thrombocytopenia and soluble fibrin-monomer complexes.

Conclusion. In deep burns, hemostasis and fibrinolytic activity disorders are observed, which are associated with the degree of burn shock and stages of burn disease, which can lead to the development of DVS-syndrome.

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