The State of Coagulation, Anticoagulant and Fibrinolitic Parts of the Hemostasis System in Burn Shock

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ABSTRACT

The problem of burns still remains one of the most urgent and complex in modern medicine. First of all, this is due to the significant spread of burns among the population, in connection with which it can be called without exaggeration a modern traumatic epidemic in densely populated cities and industrialized countries of the world, which is due to the continuing high level of this type of injury, the severity of its medical and social consequences. Burn shock and acute burn toxemia, especially severe, causes significant disturbances in the blood coagulation system. The state of hypercoagulability observed in the victims during the period of burn shock and toxemia requires appropriate correction for the prevention of thromboembolic complications.

Keywords: Burn shock, burn toxemia, prothrombin index (PTI), disseminated intravascular syndrome (DIC), coagulation system, anticoagulation system

Introduction. Thermal injury is one of the most common types of domestic and industrial injuries. According to the WHO, they rank third in the overall structure of injuries after traffic injuries [1, 2, 3].

In connection with the growth of urbanization processes in recent years in various countries of the world, there has been a tendency to increase the number of fires, accompanied by human casualties [4]. At the same time, the lethality among the heavily burned patients remains high even in specialized hospitals [5].

Mortality from burn disease varies depending on its stage [6]. The largest percentage (from 65 to 95%) of deaths occurs during periods of toxemia and septicotoxemia. The immediate causes of death in burn disease are constant: sepsis, pneumonia, DIC, and against their background, developing multiple organ failure [7, 8].

Burn disease is one of the most severe pathological processes, often accompanied by a serious complication of the hemostasis system - disseminated intravascular coagulation syndrome (DIC), which, in the absence of timely diagnosis and adequate treatment, rapidly progresses and leads to death [9, 10].

It is known that hemocoagulation is a complex biochemical process. In which more than 30 coagulation factors (cellular and plasma) are involved. The state of hemostasis in patients with burn injury has been studied by many researchers [11, 12]. At the same time, it was found that after receiving a burn, not only platelet aggregation, but also erythrocyte

aggregation increases. In such patients, blood clotting is accelerated, fibrinolysis is inhibited, and a chronic form of DIC often develops. The tendency to hypercoagulation in the elderly, aggravated after a burn, in combination with a decrease in blood flow during the period of shock, significantly increases the risk of thromboembolic complications. Acute disorders of cerebral circulation, thrombosis and embolism of the pulmonary arteries, iliac and other arteries of the systemic circulation are a frequent complication in these patients [10, 11].

With extensive deep burns, immediately after the injury, various pathological processes are triggered, forming the pathogenesis of burn disease. One of the first to destabilize is the hemostasis system in the form of DIC with thrombosis and bleeding [13, 14].

The mechanism of thrombus formation is based on damage to the integrity of the vascular wall. At the same time, internal and external mechanisms of the process of thrombus formation are distinguished [15].

With an internal mechanism, damage to only the endothelial layer of the vascular wall leads to the fact that the blood flow comes into contact with the structures of the subendothelium - with the basement membrane, in which the main thrombogenic factors are collagen and laminin. They interact with the von Willebrand factor and fibronectin in the blood; a platelet thrombus is formed, and then a fibrin clot.

It should be noted that thrombi that form under conditions of fast blood flow (in the arterial system) can exist practically only with the participation of the von Willebrand factor. On the contrary, both the von Willebrand factor and fibrinogen, fibronectin, and thrombospondin are involved in the formation of thrombi at relatively low blood flow rates (in the microvasculature, venous system) [5, 11].

Another mechanism of thrombus formation is carried out with the direct participation of the von Willebrand factor, which, when the integrity of the vessels is damaged, increases significantly in quantitative terms due to the supply of endothelium from Weibol-Pallad bodies [17].

The most important role in the external mechanism of thrombosis is played by tissue thromboplastin, which enters the bloodstream from the interstitial space after a rupture of the integrity of the vascular wall. It induces thrombus formation by activating the blood coagulation system with the participation of factor VII . Since tissue thromboplastin contains a phospholipid part, platelets participate little in this mechanism of thrombosis. It is the appearance of tissue thromboplastin in the bloodstream and its participation in pathological thrombosis that determine the development of acute DIC [18].

Target. Assess the state of the coagulation, anticoagulant and fibrinolytic components of the hemostasis system in burn shock, acute burn toxemia and septicotoxemia .

Materials and methods. To achieve the goals and objectives of the study, data were collected on a total of 50 victims with burn injuries who were treated at the Samarkand branch of the RRCEM.

First, an assessment was made of the informative significance of homeostasis disturbance in terms of severity, and then the development of predictive algorithms and testing of their effectiveness.

In patients, the area of the lesion was assessed according to the generally accepted rule of nines proposed by A . b . Wallace (1951), when the area of all parts of the body is indicated by the number of percent equal to nine. The depth of the lesion was determined in accordance with the 4-degree classification of A.A. Vishnevsky et al. (1960).

In accordance with the prognostic Frank index (IF), which characterizes the severity of a burn injury and is determined in arbitrary units (1% of the superficial burn I - II - IIIA degree is taken as 1 unit (unit), 1% deep burn III B - IV Art. for 3 units) and taking into account the severity of inhalation injury (with IT I - II degree additionally summarized 15 units, with IT III - IV degree - 30 units) patients were divided into 4 groups: Group I - IF < 30 units. – 13 patients. II - IF 30-60 units. - 13, III - IF 61-90 units. – 13, IV – IF > 90 units. - eleven. Burned patients with a favorable prognosis (FI

up to 60 units) accounted for 80.18%, with doubtful and unfavorable prognosis (FI over 60 units) - 19.82%.

The main principle of prevention and treatment of patients with DIC is the elimination of factors that caused the activation of intravascular coagulation (removal of foci of necrosis, which are the source of thromboplastin, elimination of intoxication, hypoxia, acidosis, correction of water and electrolyte disorders, treatment of infectious complications). In the hypercoagulable phase, therapy begins with the introduction of heparin (400-500 IU/h). The greatest anticoagulant effect of heparin is manifested against the background of a high content of antithrombin III. Deficiency of antithrombin III is replenished by transfusions of fresh frozen plasma. In the treatment of patients with DIC, preference is given to low molecular weight heparin (fraxiparin, clexane), since, unlike non-fractionated forms, it does not activate platelet aggregation.

Results and discussion. The most informative indicators for the diagnosis of DIC are presented in Table. 1.

Table 1

The main laboratory criteria for the state of the blood coagulation and anticoagulation system in DIC (K.M. Krylov., et al., 2010)

Indicators	Norma (N)	Stages of DIC		
		Hyper coagulation	Transitional	Hypocoagulation
Prothrombin index (%)	80-100	H, >	H, <	<
Prothrombin time (s)	14-20	H, <	H, <	>
Thrombin time (s)	14-16	H, <	H, <	>
APTT (s)	35-45	H, <	H, <	>
AVR (s)	50-70	H, <	H, <	>
Fibrinogen, g/l	2-4	>	>	H, <
Ethanol test	negative	+, -	+, -	+, -
Orthophenanthroline test (mg/l)	35	>	>	>
Content of erythrocytes (million/μl)	3.7-5.1	N, N	H, <	H
Hematocrit (%)	37-53	H, <	H, <	<
The content of platelets, thousand/μmol	142-424	H, <	H, <	<
Antithrombin III activity (%)	85-115	<	<	<
PDF (mg/l)	0-2	>	>	>
XII a-dependent fibrinolysis (min)	4-10	>	>	>

H - normal value, > H - above the norm, < H - below the norm.

To enhance the antithrombotic effect of heparin, it is necessary to use antiplatelet agents (chimes, pentoxifylline), proteolysis inhibitors (gordox, contrykal). It is mandatory to carry out infusion therapy with crystalloids and colloid solutions. Of the colloidal preparations, it is preferable to use amino starch derivatives, because. they have a pronounced disaggregation effect and do not cause the development of hypocoagulation.

In the treatment of DIC in the stage of hypocoagulation, antiproteases (gordox, contrykal) and transfusions of fresh frozen plasma up to 1500 ml per day are indicated.

In the acute period of burn disease, the initial period of the development of DIC is observed: thrombocytopenia, an increase in the level of RFMK against the background of inhibition of the anticoagulant mechanisms of the hemostasis system.

Due to adequate preoperative therapy in the postoperative period, a tendency to normalization of all coagulogram parameters in patients with I-degree plasma therapy was revealed. At the same time, PTI statistically significantly amounted to 91.6±3.9%, fibrinogen 2.3±0.3 g/l, thrombotest 5.0±0.14 degrees (P<0.05). However, a slight inhibition of fibrinolytic activity persisted even when patients were discharged from the hospital, amounting to 20.1±0.45% (P<0.05). With moderate severity of plasma loss, there was also a significant improvement in discharge indicators. This is evidenced by the normalization of PTI, fibrinogen and blood hematocrit.

In contrast to the indicators in patients with I- and II-degree plasma loss, with a severe degree of plasma loss in the blood coagulation system, even after therapeutic measures, violations of the coagulogram parameters persist until discharge. Increased rates of PTI, recalcification time and thrombotest indicate still persistent hypercoagulability with suppressed fibrinolysis (P<0.05).

In patients with the threat of burn sepsis, the initial period of the development of DIC was observed: thrombocytopenia, an increase in the level of RFMK against the background of a decrease in the activity of physiological anticoagulants. Timely detection of this life-threatening complication, adequate and early correction of the hemostasis system is the key to a favorable outcome of the disease.

Conclusions. Burn shock and acute burn toxemia, especially severe, causes significant disturbances in the blood coagulation system. The state of hypercoagulability observed in the victims during the period of burn shock and toxemia requires appropriate correction for the prevention of thromboembolic complications.

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