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THE STATE OF COAGULATION, ANTICOAGULANT AND FIBRINOLITIC PARTS OF THE HEMOSTASIS SYSTEM IN BURN SHOCK

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Abstract:

The problem of burns is still one of the most urgent and difficult in modern medicine. First of all, it is connected with a significant spread of burns among the population, in connection with what it without exaggeration can be called a modern traumatic epidemic of densely populated cities and industrialized countries, which is caused by the continuing high level of this type of damage, the severity of its medical and social consequences

Keywords:

INTRODUCTION. Thermal injuries are one of the most frequent types of domestic and industrial injuries. According to WHO, they occupy the third place in the total structure of injuries after transport injuries [1, 2, 3].

In connection with the growth of urbanization processes in recent years in various countries of the world there is a tendency to increase the number of fires, accompanied by human casualties [4]. At the same time mortality among severely burned people remains high even in specialized hospitals [5]. Lethality from burn disease varies depending on its stage [6]. The highest percentage (from 65 to 95%) of deaths occurs during toxemia and septic toxemia. The immediate causes of death in burn disease are constant: sepsis, pneumonia, DIC syndrome and on 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 hemostatic system - disseminated intravascular coagulation syndrome (DICS), which in the absence of timely diagnosis and adequate treatment rapidly progresses and leads to death [9, 10].

Hemocoagulation is known to be a complex biochemical process. More than 30 clotting factors (cellular and plasma) are involved in it. The state of hemostasis in patients with burn trauma has been studied by many researchers [11, 12]. It has been established that after a burn, not only platelet aggregation increases, but also erythrocyte aggregation. In such patients blood coagulation is accelerated, fibrinolysis is inhibited, and a chronic form of DIC syndrome often develops. The tendency to hypercoagulability in old age, worsened after the burn, combined with decreased blood flow in the period of shock, significantly increases the risk of thromboembolic complications. Acute cerebral circulatory disorders, thrombosis and embolism of pulmonary arteries, iliac and other arteries of the great circulatory circle are not uncommon complications in these patients [10, 11].

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

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

In the internal mechanism, only damage of the endothelial layer of the vascular wall leads to the fact that the blood flow contacts the subendothelial structures - the basal membrane, where the main thrombogenic factors are collagen and laminin. Willebrand factor and fibronectin in blood interact with them, forming a thrombocytic clot, and then - a fibrin clot.

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

Another mechanism of thrombosis is carried out with the direct participation of Willebrand factor, which significantly increases quantitatively when the vascular integrity is damaged due to the inflow from the Weibol-Pallad endothelial bodies [17].

The most important role in the external mechanism of thrombosis is played by tissue

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thromboplastin, which enters the bloodstream from the interstitial space after the integrity of the vascular wall is broken. It induces thrombosis by activating the blood coagulation system with the participation of factor VII. As the tissue thromboplastin contains phospholipid part, platelets are little involved in this mechanism of thrombosis. It is the appearance of tissue thromboplastin in the bloodstream and its participation in pathological thrombosis and determine the development of acute DIC syndrome [18].

OBJECTIVE. To evaluate coagulation, anticoagulant and fibrinolytic links of the hemostasis system in burn shock, acute burn toxemia and septic toxemia.

MATERIALS AND METHODS OF RESEARCH : In order to realize the goal and objectives of the study, data on a total of 50 burn trauma victims treated at the Samarkand Branch of the RSCEMT were realized.

First, we assessed the informative significance of homeostasis disturbances by severity, and then developed prognostic algorithms and tested their effectiveness.

In patients, the lesion area was estimated according to the generally accepted "rule of nine" proposed by A.B. Wallace (1951), when the area of all body parts is indicated by the number of percentages equal to nine. The depth of the lesion was determined according to the 4-degree classification of A.A. Vishnevsky et al. (1960).

According to the Frank's prognostic index (IF), which characterizes the severity of a burn injury and is defined in conditional units (1% of superficial burns of I-II-IIIA degree is taken as 1 unit (unity), 1% of deep burns of IIIB-IV degree for 3 units) and taking into account the severity of inhalation trauma (for IT of I-II degree additional 15 units were summarized, for IT of III-IV degree - 30 units) the 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 - 11. Burned patients with a favorable prognosis (IF up to 60 units) accounted for 80.18%, and with a doubtful and unfavorable prognosis (IF over 60 units) - 19.82%.

The main principle of prevention and treatment of patients with DIC syndrome is the elimination of factors causing activation of intravascular coagulation (removal of necrosis foci that are the source of thromboplastin, elimination of intoxication, hypoxia, acidosis, correction of water-electrolyte disorders, treatment of infectious complications). In the hypercoagulable phase the therapy begins with administration of heparin (400-500 units/hour). The greatest anticoagulant effect of heparin is manifested against a high content of antithrombin III. Antithrombin III deficiency is compensated by transfusions of fresh frozen plasma. When treating patients with DIC syndrome, preference is given to low molecular weight heparin (Fraxiparin, Clexane), because, unlike unfractionated forms, it does not activate platelet aggregation.

RESULTS AND DISCUSSION. The most informative indices for DIC diagnosis are presented in Table 1.

Table 1
Basic laboratory criteria of the coagulation and
The basic laboratory criteria of the blood coagulation system state in DIC syndrome (K.M. Krylov et al., 2010)

Indicators	Norm (N)	Stages of DIC syndrome		
		Hyper-coagulation	Transitional	Hypoco-agulation
Prothrombin index (%)	80-100	N >	N, <	<
Prothrombin time (s)	14-20	N <	N, <	>
Thrombin time (s	14-16	N, <	N, <	>
BHTV (c)	35-45	N, <	N, <	>
ATS (c)	50-70	N, <	N, <	>
Fibrinogen, g/l	2-4	>	>	N, <
Ethanol test	Negative.	+, -	+, -	+, -
Orthophenanthroline test (mg/L)	35	>	>	>
Erythrocyte content (mln/µl)	3,7-5,1	N, N	N, <	N
Haematocrit (%)	37-53	N, <	N, <	<
Platelet content, thousand/µmol	142-424	N, <	N, <	<
Activity of antithrombin III (%)	85-115	<	<	<
PDF (mg/L)	0-2	>	>	>

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XIIa-dependent fibrinolysis (min)	4-10	>	>	>

N is the normal value, >N is above normal,< N is below the norm.

To enhance the antithrombotic effect of heparin it is necessary to use disaggregants (curantil, pentoxifylline), proteolysis inhibitors (gorex, contrical). Infusion therapy with crystalloids and colloidal solutions is mandatory. Among colloidal preparations it is preferable to use amino-starch derivatives, since they have a pronounced disaggregation effect and do not cause hypocoagulation.

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

In the acute period of burn disease there is an initial period of DIC syndrome development: thrombocytopenia, RFMC level increase against the background of inhibition of anticoagulation mechanisms of hemostasis system.

Due to adequate preoperative therapy in the postoperative period there was a tendency to normalization of all coagulogram parameters in patients with I-degree plasma therapy. At the same time, PTI was statistically reliable 91,6±3,9%, fibrinogen 2,3±0,3 g/l, thrombotest 5,0±0,14 degree (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 a moderate degree of severity of plasma loss there was also a significant improvement in the indices by the time of discharge. 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 clotting system, even after therapeutic measures, by discharge there are still violations of coagulogram. Increased rates of PTI, recalcification time and thrombosis testify to still persisting hypercoagulation with suppressed fibrinolysis (P<0,05).

Patients with threatening burn sepsis had the initial period of DIC syndrome development: thrombocytopenia, increased RFMC level against the background of decreased 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

CONCLUSIONS. Burn shock and acute burn toxemia, especially of severe degree, cause significant disturbances in the blood coagulation system. The state of hypercoagulation observed in the victims during burn shock and toxemia requires appropriate correction to prevent thromboembolic complications.

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