WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 4, Issue 2, Feb., 2023

COAGULATION, ANTICOAGULANT AND FIBRINOLYTIC PART OF THE HEMOSTASIS SYSTEM IN BURN SHOCK

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Annotation
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].

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

Introduction

Hemocoagulation is 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. Such patients have accelerated blood coagulation, inhibited fibrinolysis, and often develop a chronic form of DIC syndrome. The tendency to hypercoagulability in the elderly, aggravated 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 start 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]. Thermal injuries are one of the most frequent types of domestic and industrial injuries. According to WHO, they occupy the third place in the general structure of traumatism after transport trauma [1, 2, 3]. Due to the growth of urbanization processes in recent years in different 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. The mechanism of thrombosis is based on the damage to the integrity of the vascular wall. There are distinguished internal and external mechanisms of thrombus formation process [15]. In the internal mechanism, the damage of only the endothelial layer of the vascular wall leads to the fact that the blood flow contacts the subendothelial structures - the basal membrane, in which the main thrombogenic factors are collagen and laminin. Willebrand factor and fibronectin in blood interact with them; a thrombocytic clot is formed, and then - a fibrin clot. It should be noted that thrombi formed under conditions of rapid blood flow (in the arterial system) can 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 integrity of the vessels 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 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].

Research Objective

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

Materials and Methods

To realize the goal and objectives of the study, we implemented data on a total of 50 burn trauma victims treated at the Samarkand Branch of the RRCEMP.

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 Frank's prognostic index (IF) which characterizes the severity of burn injury and is defined in conventional units (1% of superficial burns of degree I-II-IIIA is taken as 1 unit, 1% of deep burns of degree IIIB-IV as 3 units) and taking into account the severity of in halation injury (with IT degree I-II additional 15 units, with IT degree III-IV - 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 that caused activation of intravascular coagulation (removal of necrosis foci that are the source of thromboplastin, elimination of intoxication, hypoxia, acidosis, correction of water-electrolyte disorders, treadment of infectious complications). In the hypercoagulable fase the therapy begins with administration of heparin (400-500 units/hour). The greatest anticoagulant effect of heparin occurs against a background of high levels of antithrombin III. Antithrombin III deficiency is compensat by transfusions of fresh frozen plasma. When treating patients with DIC syndrome preference is given to low molecular weight heparin (fractiparin, clexane), because, unlike unfractionated forms, it does not activate platelet aggregation.

Results and Discussion

Basic laboratory criteria of the state of the coagulation and antiplatelet system in DIC syndrome (K.M. Krylov et al., 2010). To enhance the antithrombotic effect of heparin it is necessary to use disaggregants (curantil, pentoxifylline), proteolysis inhibitors (gordox, contrical). Infusion therapy with crystalloids and colloidal solutions is mandatory. Among colloidal preparetions it is preferable to use amino-starch derivatives, since they have a pronounced disaggregation effect and do not cause hypocoagulation.

When treating DIC in hypocoagulation stage antiproteases (gorex, contrical) and transfusions of fresh frozen plasma up to 1500 ml per day are indicat.

In the acute period of burns diseace there is an initial period of DIC syndrome development: thrombocytopenia, increased level of RFMC amid inhibition of antisclerotic 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-stage plasma therapy was reveal. 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 plathma loss there was also a significant improvement in the indices by the time of discharge. This is evidenc 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 values of PTI, recalcification time and thrombosis testify to the still preserved 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 a 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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