

Disorders of the hemostasis system in the stage of burn shock in patients with burn injuries

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Abstract

Burn injury is a serious medical, social and economic problem. In case of burn injuries, the burn consists in the study of disorders in the hemostasis system in the shock phase. The object of the study was patients admitted to the Burn department RSCUMA in the period from 2016 to 2019. In the phase of burn shock with deep burns from 10 to 35% of the body surface 95 (out of 112 main groups). The age of the patients ranged from 18 to 75 years, the average age was 54.3 ± 0.7 . There were 142 (65.43%) men and 75 (34.56%) women. There was a poorly developed increase in the coagulation system (tolerance to heparin 253.4 ± 21.3 seconds, thrombotest - 4.06 ± 0.2 degrees), PTI decreased to $87.6 \pm 3.1\%$ ($R < 0.05$). there was a constant decrease in fibrinolytic activity. However, a sharp decrease in fibrinolytic activity was observed, amounting to $6.58 \pm 0.69\%$ ($P < 0.05$). When all patients are hospitalized, comprehensive anti-shock measures should be taken to improve the water-salt balance, acid-base balance and disruption of homeostasis.

Keywords: Disseminated vascular transfusion (DCS) - syndrome, circulating blood volume (CBV), recovery time, anti-thrombin, prothrombin index, burn injury.

Introduction

Burns are complex traumatic injuries, and much of the focus of research and clinical treatment has been on the acute trauma, appropriate surgical intervention and survival with reduced scarring [1]. Any injury triggers the process of blood clotting, but thermal injuries, represented by shock, lead to abrupt changes in all coagulation systems. [2-4]. Any damage to the vascular wall, "blood damage", on the one hand, leads to different levels of plasma loss, on the other - can be considered physiological, and then pathological changes in the hemostasis system can lead to spontaneous death of the organism [5,6]. Legitimate severe and common complications of massive injury include acute disseminated vascular hardening syndrome. Hemostasis disorder in patients with severe burns is manifested by QDTQ-syndrome. [7-12]. Disseminated vascular hemorrhage (DV) - the syndrome is not completely detectable, or is detected at the stage of giving a clear clinical picture, such as hemorrhage and / or organ dysfunction. There are several forms of QDTQ-syndrome: severe, acute, acute, chronic, recurrent, latent. The acute form is characterized by an acute period of burns. The aim of the study was to study the disturbances in the hemostasis system during the burn shock phase in patients with burn injuries.

Materials and methods

The research is based on the analysis of the results of treatment of 35 patients (37 women and 68 men) aged 18 to 75 years, treated in the burn department of the Samarkand branch of the Republican Scientific Center for Emergency Care. The mean age of the patients was 39 years. In most of the observations, the cause of the injury was a flame, burning with boiling water, and contact burns were also observed. In patients, the total area of injury ranged from 20 to 40% of body surface area (mainly burns of the extremities, as well as chest, neck, and face), and deep burns of grade III B- IV accounted for up to 20%. All patients underwent necrectomy into healthy tissue with simultaneous autodermoplasty with capillary bleeding. Hemostasis was performed with hemostatic powder obtained from cellulose derivatives with the drug "Geprotzel". The drug was applied 10 mg of powder once a day on the surface of the wound for 3 days. The clinical evaluation included the following criteria: quantity and nature of discharge; bleeding wounds; time of epithelialization of donor sites, transplanted autodermotransplants; completeness of epithelialization; the severity of the wound pain. Surgical interventions in 105 patients with deep burns included the use of a hemostatic drug to stop necrotic skin and subcutaneous

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structures after excision.

Results

At the time of hospitalization of patients with AHH deficiency, we differentiated 3 levels of burn shock. The majority of patients (49; 51.6%) had a baseline AQH deficiency (due to plasma loss) of 20 to 30%, which corresponded to grade II burn shock. In contrast, 29 (30.5%) patients were diagnosed with grade I and 17 (17.9%) with grade III. We studied the Algov index, which is directly related to the level of plasma loss in addition to AQH deficiency at the time of hospitalization (Fig. 1). Based on the data obtained, the Algov index for severe plasma loss was characterized by an increase in the patient from 0.54 in grade 1 to 1.45 in grade 3 plasma loss. Hence, these data suggest that there are deeper changes in the homeostasis system in patients with severe burn shock. At the same time, the data on AQH were analyzed in order to accurately reflect on the real changes and the possibilities of correction therapy during the hospitalization of the patient in the phase of burn shock. The results were evaluated from a comparative perspective with the control group indicators. A study of the condition of AQH and its components has shown that the extent to which changes in it develop is directly proportional to the degree of burn shock. In hospitalized patients with deep burns, a clear statistical decrease in total AQH values from 67.5 ± 0.4 ml / kg to 53.9 ± 0.4 ml / kg, respectively, was found in I- and III-degree shock. The AQH deficit ranged from $22.0 \pm 0.3\%$ in grade I, $32.9 \pm 0.7\%$ in grade II, and $32.9 \pm 0.7\%$ in grade III, respectively, according to the severity of

the burn shock. AQH structural indicators from control group indicators did not differ significantly in patients with grade I shock, although there was a statistical difference between them. These changes are most pronounced in patients with group II-III degree of shock. This is evidenced by a decrease in circulating plasma volume (APH) and globular blood volume (QGH) from 28.4 ± 0.2 ml / kg and 26.5 ± 0.3 ml / kg, respectively, in patients with Sh-level shock. However, a decrease in circulating protein volume (AOH) was observed from 3.3 ± 0.1 g / l to 2.28 ± 0.04 g / l ($R < 0.05$) (Table 1).

We studied changes in the blood coagulation system and fibrinolytic activity in 95 patients depending on the severity of the burn shock (Table 2). It should be noted that treatment tactics aimed at the correction of homeostasis disorders and a significant improvement in AQH indicators after anti-shock therapy were observed. Such a positive trend is more characteristic of patients with grade II and III burn shock. Decreases in AHH deficiency in patients with grade I- and II were observed from $4.3 \pm 0.1\%$ and $10.2 \pm 0.3\%$ ($R < 0.05$), respectively. The positive trend was mainly observed in the increase in APH and GH values, at a mild level of 38.3 ± 0.2 ml / kg and 33.7 ± 0.3 ml / kg, respectively, and at a moderate level of shock at 37.9 ± 0.1 ml / kg. and 32.1 ± 0.4 ml / kg. However, despite the positive dynamics of the indicators, in patients in the severe group of shock, even after the conducted treatment measures, the AQH showed poor indicators. Therefore, AQH deficiency was $12.7 \pm 0.3\%$ in the shock phase in this contingent of patients. However, APH (34.7 ± 0.2 md / kg, $r < 0.05$), AGH (8.9 ± 0.04 g / l, $R < 0.05$), and GH (31.5 ± 0), 2 ml / kg, $R < 0.05$) maintained statistically low values.

Table 1: AHH deficiency.

AQH gradations	Shock levels after necrectomy and ADP				Shock levels after necrectomy and ADP			
	control	I-д.	II-д.	III-д.	control	I-д.	II-д.	III-д.
AQH, ml / kg	74,1±0,3	67,5±0,4*	61,5±0,5*	53,9±0,4*	77,1±0,3	72,0±0,2	70,4±0,42*	66,2±0,4*
AQH deficit,%	5,2±0,1	10,9±0,4*	22,0±0,3*	32,9±0,7*	4,4±0,5	4,3±0,1	10,2±0,3*	12,7±0,3*
APH, ml / kg	40,4±0,5	36,2±0,2*	33,1±0,2*	28,4±0,2*	42,3±0,3	38,3±0,2	37,9±0,1*	34,7±0,2*
GH, ml / kg	33,7±0,2	31,3±0,3*	28,4±0,2*	26,5±0,3*	34,8±0,2	33,7±0,3*	32,1±0,4*	31,5±0,2*
AOH, g / l	3,3±0,1	2,83±0,16*	2,54±0,02*	2,28±0,04*	3,7±0,05	3,0±0,01*	2,92±0,2*	2,79±0,01*
AGH, g / l	10,4±0,2	9,8±0,1*	8,3±0,1*	6,9±0,1*	12,3±0,1	10,1±0,09*	9,7±0,05*	8,9±0,04*

Note: * - the degree of accuracy of the results is $R < 0.05$

Table 1: Changes in the blood coagulation system and fibrinolytic activity in 95 patients depending on the severity of the burn shock.

Coagulogram indicators	Shock Levels			Control Group
	I Level	II Level	III Level	
PTI,%	92,3±1,5	94,6±3,8*	87,6±3,1*	90,2±1,0
Fibrinogen, g / l	2,68±0,3*	2,92±0,29*	2,15±0,25*	3,4±0,2
Trombotest, st.	4,34±0,14*	4,4±0,2*	4,06±0,2*	5,0±0,1
Hematocrit%	49,9±1,1	53,1±1,4*	60,4±2,1*	44,0±0,8
Recoverytime, sec.	82,1±2,9*	87,4±3,8*	89,4±3,2*	74,0±3,2
Tolerancetoheparin, sec.	283,8±8,8	256,3±11,8*	253,4±21,3*	290±7,4
Fibrinolysis,%	10,7±0,27*	8,09±0,42*	6,58±0,69*	15,4±0,6

Note: * - the degree of accuracy of the results is $R < 0.05$.

It should be noted that treatment tactics aimed at the correction of homeostasis disorders and a significant improvement in AQH indicators after anti-shock therapy were observed. Such a positive trend is more characteristic of patients with grade II and III burn shock. Decreases in AHH deficiency in patients with grade I- and II were observed from $4.3 \pm 0.1\%$ and $10.2 \pm 0.3\%$ ($R < 0.05$), respectively. The positive trend was mainly observed in the increase in APH and GH values, at a mild level of 38.3 ± 0.2 ml / kg and 33.7 ± 0.3 ml / kg, respectively, and at a moderate level of shock at 37.9 ± 0.1 ml / kg. and 32.1 ± 0.4 ml / kg. However, despite the positive dynamics of the indicators, in patients in the severe group of shock, even after the conducted treatment measures, the AQH showed poor indicators. Therefore, AQH deficiency was $12.7 \pm 0.3\%$ in the shock phase in this contingent of patients. However, APH (34.7 ± 0.2 md / kg, $r < 0.05$), AGH (8.9 ± 0.04 g / l, $R < 0.05$), and GH (31.5 ± 0), 2 ml / kg, $R < 0.05$) maintained statistically low values. It should be noted that treatment tactics aimed at the correction of homeostasis disorders and a significant improvement in AQH indicators after anti-shock therapy were observed. Such a positive trend is more characteristic of patients with grade II and III burn shock. Decreases in AHH deficiency in patients with grade I- and II were observed from $4.3 \pm 0.1\%$ and $10.2 \pm 0.3\%$ ($R < 0.05$), respectively. The positive trend was mainly observed in the increase in APH and GH values, at a mild level of 38.3 ± 0.2 ml / kg and 33.7 ± 0.3 ml / kg, respectively, and at a moderate level of shock at 37.9 ± 0.1 ml / kg. and 32.1 ± 0.4 ml / kg. However, despite the positive dynamics of the indicators, in patients in the severe group of shock, even after the conducted treatment measures, the AQH showed poor indicators. Therefore, AQH deficiency was $12.7 \pm 0.3\%$ in the shock phase in this contingent of patients. However, APH (34.7 ± 0.2 md / kg, $r < 0.05$), AGH (8.9 ± 0.04 g / l, $R < 0.05$), and GH (31.5 ± 0) 2 ml / kg, $R < 0.05$) maintained statistically low values. Fibrinolysis is statistically undoubtedly reduced to $8.09 \pm 0.42\%$ ($R < 0.05$), while changes in hematocrit to 53.1 ± 1.4 are expected due to intensive loss of blood form elements and hemoconcentration. Similar changes were observed mainly in patients with severe burn shock with deep burns of more than 30% (19 patients) of the body surface. However, the amount of lost plasma was much deeper due to the higher volume. There was a weakly developed increase in the coagulation system (tolerance to heparin was 253.4 ± 21.3 sec, thrombotest - 4.06 ± 0.2 degrees), and PTI decreased to $87.6 \pm 3.1\%$ ($R < 0, 05$). A continuous decrease in fibrinolytic activity was observed. However, a profound decrease in fibrinolytic activity was observed, which was $6.58 \pm 0.69\%$ ($R < 0.05$). A critical increase in hematocrit was observed to $60.4 \pm 2.1\%$ ($R < 0.05$).

Conclusion

In view of the above, comprehensive anti-shock measures should be taken to improve water-salt balance, acid-base balance and homeostasis disorders when all patients are hospitalized

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