Pathogenesis, Clinic and Principles of Treatment of Burn Shock

Avazov Abdurakhim Abdurakhmanovich
Samarkand State Medical University, Samarkand branch of the Republican Scientific Center for Emergency Medical Aid. Uzbekistan.
abduraxim_avazov79@mail.ru +998913135535

Elmuradov Akhtamkhon
Samarkand State Medical University, Samarkand branch of the Republican Scientific Center for Emergency Medical Aid. Uzbekistan.
elmuradovaxtam@mail.ru +998939960472

Umedov Khushvakt Alisherovich
Samarkand State Medical University, Samarkand branch of the Republican Scientific Center for Emergency Medical Aid. Uzbekistan.
xushvaqtumedov1988@mail.ru +998979155624.,

Zhumanov Khusniddin Alisher ugli
Samarkand State Medical University, Samarkand branch of the Republican Scientific Center for Emergency Medical Aid. Uzbekistan.
xusniddin_jumanov@mail.ru +998916320736.

ABSTRACT

The main questions concerning pathogenesis, clinical diagnostics and principles of treatment of burn shock are consecrated in the article. It is proved that the introduction of the principles of emergency anti-shock therapy burned at the prehospital stage and complex treatment with the use of refortan or stabilazole facilitates the course of shock, reduces mortality in patients with critical and supercritical burns.

Keywords: burn shock, critical and supercritical burns, intensive therapy.

Burn shock is the first stage of burn disease and develops when the surface of the body is affected by an area of 10-15% in superficial and 5-10% in deep burns [6,11,14].

The pathogenesis of burn shock. In the first hours of burns, the severity of the condition of patients is due to pain syndrome and psycho-emotional stress, which serve as a trigger for the neuroendocrine response, manifested by the release of hormones of the adrenal cortex and other biologically active substances of the pituitary gland. Clinically, this is expressed by vasospasm, an increase in total peripheral vascular resistance, and centralization of blood circulation, which leads to tissue hypoxia and acidosis. In addition, a violation of the function of external respiration (a decrease in respiratory volume, vital capacity of the lungs) contributes to a decrease in blood oxygen saturation and tissue oxygenation, the accumulation of under oxidized products, respiratory and metabolic acidosis [2,3,12]. It is noteworthy that in the first hours of a burn injury, the BCC slightly increases (due to the release of deposited erythrocytes), the stroke and minute volumes

of the heart increase, which then, as hypovolemia increases, begin to decrease.

The most important and significant in the pathogenesis of burn shock is hypovolemia, which develops as a result of damage to the vascular wall. As a result of an increase in the permeability of the endothelium, a transition occurs within the vascular fluid into the interstitial space. The leading role in increasing the permeability of the vascular wall belongs to vasoactive amine (histamine), kinin system (bradykinin), complement C3 fraction, oxygen radical, lipid peroxidation, eicosanoids (thromboxane A2, prostacyclin, prostaglandin E2). An increase in osmotic pressure due to an increase in the concentration of sodium ions in the burn area enhances the flow of fluid into the burnt tissues and increases edema [11,14]. The release of protein (mainly albumin) from the vascular bed leads to an increase in oncotic pressure in the interstitium, which contributes to an even more active flow of water from the vessels.

In severe burns due to impaired membrane permeability, sodium ions from the extracellular space pass into the cells and entail water, causing intracellular edema, especially dangerous when it is localized in the brain [1,5,9].

Hypovolemia developing within 5-7 hours becomes the cause of hemodynamic disorders in the form of centralization of blood circulation. Simultaneously increasing rheological (increase in viscosity) and coagulopathy (hypercoagulation) changes in the blood lead to even deeper microcirculation disorders, manifested by necrosis in the burn area, stress ulcers of the gastrointestinal tract, and renal liver failure [1,5,9].

Burn shock clinic. Burn shock is divided into 3 degrees of severity, which correspond to a different severity of clinical signs. Unlike traumatic, in burn shock, blood pressure is not of decisive importance. The leading clinical symptoms of burn shock are oligoanuria, hypothermia, hemoconcentration, and later hypotension. The clinic of burn shock is presented in the table

Table
Burn shock clinic

Clinical signs	Degrees of burn shock		
	I	II	III
Systolic pressure (mm Hg) st	Norm	Moderate hypotension	Hypotension
Heart rate (min)	Up to 100	100-120	Over 120
CVP (cm H2O) st	About 0	Negative	Negative
Diuresis (ml / h)	Short-term delay	30	Less than 30 anuria
Urine color	Normal	Normal or concentrated	Concentrated
Vomiting	No	Rare	Rare
Intestinal paresis	No	Yes	Yes
Temperature, 0C	Subfebrile	Norm	35-36 and less
White spot symptom, sec.	1-2	2-3	Over 3
Consciousness	Clear, excitation	Lethargy	Sopor
Hematocrit (%)	50	60	65-70

Analgesia. For the treatment of pain in patients with burn shock, it is advisable to use analgesics such as stadol (0.08 mg/kg 3 times a day), nubain (0.3 mg/kg 3 times a day), which have a minimal effect on hemodynamic parameters. and breathing.

Principles of treatment. Treatment of those burned in a state of shock is based on pathogenetic prerequisites and is carried out according to the rules of intensive or resuscitation therapy. Manipulations include:

- ensuring the patency of the respiratory tract;
- catheterization of the central vein;
- bladder catheterization;
- holding a gastric tube.

In the anti-shock chamber, it is necessary to provide microclimatic conditions with an air temperature of 37.0-37.5°C [6,7,8].

A complex of therapeutic measures is carried out during the period of burn shock, taking into account the constitutional and age characteristics of patients. Recently, synthetic apioids with agonist properties for copyate receptors (butarphanol tartrate, nalbuphine hydrochloride, norphin) have become widespread.

A distinctive feature of opiate analgesics is their minimal effect on central and peripheral hemodynamics in patients with burn injury. Butarphanol tartrate (stadol, moradol) are prescribed at the rate of 0.08 mg/kg 3 r/day, nalbuphine hydrochloride (nubain) - 0.3 mg/kg 4 r/day. Additionally, tranquilizers are used in small doses, antipsychotics (mainly droperidol), GHB.

If the patient is on a ventilator, the introduction of morphine is acceptable.

Narcotic analgesics should be combined with neuroleptics (droperidol), tranquilizers (diazepam, dormicum), sodium oxybutyrate, central adrenomimetics (clophelin). Clonidine, administered in the first hours of intensive care for burn shock at a dose of 0.4 mg/kg h, along with potentiation of the action of drugs, leads to a decrease in the excessive activity of the sympathetic-adrenal system, a decrease in the functional load on the heart, and reduces oxygen consumption.

Antistress and antihypoxic effects are achieved using synthetic enkephalinadalargin at a dose of 1-2 mg intravenously.

Adaptogen mildronate, used at a dose of 7 mg/kg, eliminates vasospasm caused by adrenaline and angiotensin, stabilizes cell membranes, and has a cardioprotective antiarrhythmic effect.

A good analgesic and sedative effect is exerted by novocaine administered intravenously at a dose of 200-400 ml of a 0.125% solution.

Immediate fluid administration is the next mandatory event for a severely burned person.

It was established that at 1-2 degrees of severity of burn shock, the majority of patients retained the absorption function and peristalsis of the gastrointestinal tract. Therefore, in the absence of infusion media, oral administration of a solution of an alkaline-salt mixture should be started, consisting of 1/2 teaspoon of baking soda and 1 teaspoon of sodium chloride dissolved in 0.5 liters of water. High efficiency was shown by the use of dosed administration of liquids through a gastric tube using a peristaltic pump. It is advantageous to combine this method with infusion therapy.

Treatment of a patient with extensive burns, as well as with limited deep lesions, should be carried out in specialized burn departments (centers). However, anti-shock therapy should be carried out in the nearest hospital to the site of injury. The transfer of a person burned in a state of shock from one medical institution to another is categorically contraindicated even on any, the most equipped, vehicles.

In cases where burn victims cannot be immediately transported to specialized or large hospitals, it is advisable to focus on a standardized infusion therapy regimen developed on the basis of the collective experience of several hospitals; the use of such a scheme turns out to be the only possible and correct one when a large number of victims are received as a result of disasters.

The approximate volume of infusion agents required by a patient with burn shock on the first day is calculated according to the formula proposed in 1952 by Evans: V \u003d 2 ml x burn area (in%) x body weight (in kg) + 2000 ml of 5% glucose solution .

This formula is used for burns less than 50% of the body surface. Depending on the severity of shock, the calculations should take into account the different ratio of colloids and crystalloids. In severe shock, the calculated volume should include 2/3 crystalloids and 1/3 colloids, and in extremely severe shock and burns over 50% of the body surface, crystalloids and colloids are used in a 1: 1 ratio.

In burn patients older than 50 years, the daily volume of infusion agents is reduced by 1-2 times due to the danger of overloading the pulmonary circulation compared to that calculated by the Evans formula. Burn shock can last up to 3 days. Infusion therapy should be carried out all the time without interruption. The rate of fluid infusion on the first day should

be such that at least half of the calculated daily volume is administered within 8 hours of therapy. On the second day, its volume is reduced by 2 times, and on the third day - by 3 times compared with the volume of infusion on the first day.

Evans' formula should be considered as a guideline. In the future, the volume and rate of administration of therapeutic agents is adjusted based on diuresis, hematocrit, hemoglobin, pulse and blood pressure in dynamics.

Which of the infusion preparations is the most effective and indicated for burn shock?

Since a large amount of sodium ions (0.5-0.6 meq /% burn / kg of patient weight) leave the vascular bed in the place with plasma during burns, infusion therapy primarily aims to fill the vascular bed and restore the sodium content in it. For this, physiological saline or Ringer's lactate solution is used [10,13].

The latter is more preferable because its composition is closer to the extracellular fluid.

If infusion therapy is started at low blood pressure (usually several hours after the injury), hemodynamic recovery requires the introduction of more effective large molecular colloidal drugs (polyglucin).

In case of normotonia, the infusion is started with Ringer's solution or isotonic sodium chloride solution. In case of hypotension, we begin therapy with a transfusion of refortan or stabilizol, after stabilization of hemodynamics, we use crystalloids (acesol, disol, etc.).

Refortan for the purpose of hemodilution for 2-3 days, 500 ml daily (84 liters in total) was administered intravenously to 89 patients, and stabizol was also administered intravenously, 500 ml (55 liters) intravenously to 42 patients, under the control of hemoglobin, hematocrit, plasma proteins and blood electrolytes (K+, Na+, Cl, etc.). We did not observe any allergic reactions after the transfusion of Refortan or Stabizol.

Protein solutions (preferably fresh frozen plasma) are administered 8-12 hours after the start of infusion therapy. Albumin solutions (Infezol-40) are used after a decrease in the permeability of the vascular wall and the cessation of the increase in edema in the burn area. Usually, the rate of infusion of protein preparations is 1-2 ml/kg/hour.

After the blood pressure stabilizes, it is advisable to start the introduction of isotonic crystalloids. After 8-10 hours from the start of treatment with stable hemodynamics and sufficient hourly diuresis, the rate of infusion can be gradually reduced.

It is advisable to start the introduction of protein colloidal solutions 12-16 hours after the start of infusion therapy, when there is some balancing of the intra- and extravascular sectors. The greatest effect is provided by native plasma, which has all the protein fractions and affects the osmotic and oncotic properties of the blood. Albumin solutions should be used when the violation of the permeability of the vascular wall decreases and the increase in edema in the burn zone stops.

The rate of infusion of protein preparations is calculated at the rate of 1-2 ml / kg / hour. In order to improve the rheological properties of blood, protein-free medium and low molecular weight colloidal solutions are prescribed in a volume of 400-800 ml at a rate of 2 ml/kg/h.

Currently, in burn shock, blood transfusion is not performed, however, the latter is indicated for large blood loss during necrotomy or with massive hemolysis immediately after the patient is taken out of shock.

In severe and extremely severe shock, with late initiation of therapy, it may be impossible to maintain blood pressure above 90 mm Hg. Art. the introduction of crystalloids and colloids in calculated amounts. In such cases, it is advisable not to increase the volume of injected fluids, since this can lead to an increase in interstitial and intracellular fluid, but to use inotropic drugs (dopamine at a dose of 5-10 mg/kg/min). At this dosage, dopamine improves myocardial contractility and increases cardiac output. At a dosage of 1-3 mg / kg / min, it improves renal perfusion.

During the infusion, it is also necessary to inject 6% solution of vitamin B1 - 1.0; 2.5% solution of vitamin B6 - 1.0; solution of vitamin B12 - 200 micrograms.

The greatest difficulties in the treatment of burned patients arise when skin burns are combined with thermal inhalation damage to the respiratory tract. In such patients, the course of shock is sharply aggravated by the toxic effects on the respiratory tract and the body as a whole of toxic combustion products. A feature of infusion therapy in these patients is the need to "balance" the volume of infusion, since there is always a threat of developing pulmonary edema, and a decrease in the rate and amount of intravenous fluids causes a decrease in renal perfusion, contributes to the preservation and aggravation of hypovolemia. In such cases, you can resort to infusion therapy with hypertonic sodium solution (240 mEq / l). In this case, it is necessary to ensure that the level of sodium in the plasma does not exceed 160 meq / l. It is advisable to limit the introduction of a hypertonic solution to the first 8-10 hours after receiving a burn, that is, the time of the most pronounced violations of the permeability of the vascular wall. In those cases when the phenomenon of respiratory failure develops, patients need to carry out artificial ventilation of the lungs with positive expiratory pressure.

Almost always, burnt patients develop acidosis, more often metabolic, compensated by respiratory function. With thermal inhalation lesions, acidosis becomes mixed and decompensated. Therefore, patients need the introduction of a 4-5% solution of sodium bicarbonate.

Normalization of the rheological properties of blood is carried out by the infusion therapy described above, i.e. due to the correction of hypovolemia, as well as through the use of low doses of heparin (up to 20,000 units / day or more).

Recently, we have managed to reduce the number of a serious complication of burn shock - stress Curling ulcers, which is associated with the introduction of complex organ-protective therapy in combination with full anesthesia, normalization of volemic and rheological parameters and, most significantly, with the appointment of histamine H2-blockers from the first hours of injury. receptors.

Upon admission of victims with extensive burns, the question always arises of the method and place of venipuncture for infusion therapy. Currently, catheterization of the central veins (subclavian or femoral) is widely used. Such methods provide the possibility of adequate infusion in the first days after injury. Our many years of experience have shown that with prolonged use of central veins for infusion, a large number of complications occur, including septic ones, so careful care is needed for the catheter and the puncture site: regular change of dressings, catheter and the use of heparin to prevent thrombosis.

Shock Exit Criteria:

1) An increase in body temperature of at least 1 degree from the norm.
2) Stabilization of hemodynamics.
3) Positive CVP.
4) Stabilization of hourly diuresis.
5) Decreased hemoconcentration.
6) The cessation of dyspeptic disorders and the assimilation of the drunk liquid are indicators of the adequacy of treatment and the patient's exit from the state of burn shock.

From the point of view of eliminating hypovolemia, hydroxyethyl starch (refortan, stabizol) demonstrates the best qualities, which gives advantages in the speed of correction of hemoconcentration and the preservation of the volemic effect, as well as leading to an increase in oxygen delivery, which could result in a faster recovery from shock and improved burn wound trophism.

We have studied the course of burn shock in 2 groups of victims before and after the introduction of the above organizational and medical principles, from the moment of organization and functioning of burn resuscitation of the Samarkand branch of RRCEM. In just 10 years, 87.4% of burn patients were diagnosed with burn shock. In 60 patients with deep burns (50-85% of the body surface), who started anti-shock therapy at the pre-hospital stage, the course and outcomes of burn shock were favorable. Prior to the introduction of these principles, mortality was 28.8%, and after the introduction, 17.8% of victims with extensive deep burns (75-85% of the body surface) died.

Findings. Thus, the introduction of the principles of emergency anti-shock therapy for those burned at the pre-hospital stage and complex treatment with the use of refortan or stabilizol facilitates the course of shock and reduces mortality in patients with critical and supercritical burns.

References:

1. Авазов А. А. и др. ПАТОГЕНЕЗ, КЛИНИКА, ПРИНЦИПЫ ЛЕЧЕНИЯ ОЖОГОВОГО ШОКА //Вестник науки и образования. – 2021. – №. 3-2 (106). – С. 99-102.
2. Avazov A.A. Kompleksnoe lechenie bolnyx s obshirnymi glubokimi ojogami nijnix konechnostey / Shakirov B.M., Avazov A.A., Khursanov Yo.E.// Mejdunarodnyy nauchno-issledovatelskiy zhurnal. «Eurasian Union Uchenyx. Seriya: meditsinskie, biologicheskie i ximicheskie nauki »2021. p. 24-26
3. Avazov A.A. Xirurgicheskaya reabilitatsiya postradavshix ot sandalovyx ojogov / Shakirov BM, Mustafakulov IB, Avazov AA, Khursanov Yo.E. // Problems of biology and medicine. 2021 https://doi.org/10.38096/2181-5674.2021.6.1
4. Alekseev A.A., Lavrov V.A., Dutikov V.N. Ojogovyy shock: pathogenesis, clinic, treatment // Vestnik intensivnoy terapii. - 1995. - №2. - p. 29-37.
5. Arev T.Ya. Termicheskie porazheniya. L., 1966.
6. Vinogradov V.P., Lavrov V.A. Ojogovyy shock: invasive monitoring // Kombustiologiya. - 2003. - №3. - p. 1-9.
7. Vixriev B.S., Burmistrov V.M. Ojogi. L., 1985.
8. Intensive therapy ugrojayushchix sostoyaniy. Pod red. dots. V.A.Koryachkina, prof. V.I.Strashnova. Sankt-Peterburgskoe meditsinskoe izdatelstvo, 2002. - 276 p.
9. Krylov K.M., Shlyk I.V., Pivovarova L.P., Orlova O.V. Ojogovyy shock. In the book: Pathogenesis and treatment of shock of various etiologies. St. Petersburg, 2010. p. 320-360.
10. Kuzin M.I., Sologub V.K., Yudenich V.V. Ojogovaya bolezn. M., 1982.
11. Lavrov V.A., Vinogradov V.P. Ojogovyy shock: pathogenesis, clinic, treatment // Kombustiologiya. - 1999. - №1. - p. 1-10.
12. Murazyan R.I. Clinic and transfusion treatment ojogovogo shock. M., 1973.
13. Paramonov B.A., Porembskiy Ya.O., Yablonskiy V.G. Ojogi. Manual for doctors. St. Petersburg, spetsLit. - 2000. - 488 p.
14. Pekarskiy D.E., Shalimov A.A. Ojogovyy shock. Kiev, 1976.
15. Rozin L.B., Batkin A.A., Katrushenko R.N. Ojogovyy shock. L., 1975.
16. Warden G.D. Burn Shock Resuscitation. World J.Surg.16, 16-23, 1992.
17. Carvajal H.F., Parks D.H. Burns in Children. Chicago-London-BocaRaton, 1988.