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Hyperhydration in Burn Patients: the Current State of the Issue

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ABSTRACT The article presents the current state of hyperhydration issue in burn patients as a result of excess volume of infusion therapy during the acute period of burn disease. We report the data on the pathogenesis of burn disease and standard formulas for determining the volume of infusion therapy in case of burn shock, mechanisms and clinical manifestations of hyperhydration in burn patients and modern methods of its management. Since the danger of hyperhydration persists even after a shock in an acute period of burn disease, a method is proposed for determining the baseline volume of infusion therapy in the post-shock period. The developed algorithm is effective for prevention of hyperhydration in burn patients and covered by an invention patent.

Keywords: systemic inflammatory response, high vascular permeability, infusion therapy for burn shock, hyperhydration

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ITT – infusion-transfusion therapy
SIRS – systemic inflammatory response syndrom

INTRODUCTION

It is difficult to overestimate the role of infusion-transfusion therapy (ITT) in the complex treatment of victims with a burn injury. However, in recent years, a significant number of publications, mainly foreign ones, have evidenced severe complications of excessive infusion therapy as a result of overhydration in burn patients [1]. As the burn shock is accompanied by high lethality, many formulas have been proposed for calculating ITT since the 50s of the last century, and the volumes and composition of ITT recommended by various authors during the first 24–48 hours after injury significantly differed [2]. It should be emphasized that in all the proposed formulas we are talking only about the stage of burn shock.

However, the acute period of a burn disease, beginning with a burn shock, lasts as long as clinical scores (2–4 points) of *systemic inflammatory response syndrome* (SIRS) are recorded, which depends on the severity of a burn injury, the timing of the recovery of the skin and the development of complications [3]. Throughout the SIRS, the increased permeability of the microvascular bed and the danger of overhydration in burn patients persist, and its symptoms appear after a patient recovers from shock against the background of continuing excessive infusion therapy. So far, there is no ITT standard for the treatment of burn patients in the post-shock period. The analysis of the pathogenetic mechanisms of hyperhydration in case of burn injury, its clinical manifestations and our own experience in treating the victims allowed us to develop a method for determining the basic volume of infusion therapy in the post-shock period of burn disease.

The high permeability of the capillary bed is the most important pathogenetic mechanism of burn disease, caused by SIRS.

Currently, it is well known that burn damage to the skin triggers the body of the affected SIRS, which is a complex pathophysiological process. However, some authors believe that burn injury should be considered a model of SIRS [4, 5]. SIRS develops not only in burns, but also injuries, infections, pancreatitis, and other pathologies [6–8].

The systemic inflammatory process has all the classic signs of inflammation. One of the brightest pathological manifestations of burn disease is a sharp increase in capillary permeability, stimulated by pro-inflammatory mediators, which leads to the release of plasma from the vascular bed, plasmorrhea through burn wounds, transcapillary transfer of fluid into the interstitial space with the formation of tissue edema. In foreign literature, this is denoted by the term *"fluid creep"* [9, 10]. The increase in capillary permeability is most significant in the first two or three days from the time of the burn injury. This manifests as hypovolemic shock and requires immediate and adequate ITT. Increased capillary permeability is present throughout the clinical manifestations of SIRS and requires a long-term correction of homeostasis disorders until the skin is completely restored. It is a burn wound that

supports the course of SIRS, leads not only to prolonged loss of fluid, but also proteins, electrolytes, microelements. The larger the size of the skin defect is, the greater the loss is, the longer the wound is, the longer the loss is [11].

INFUSION-TRANSFUSION THERAPY FOR BURN SHOCK

For rehydration of burn patients in a state of burn shock, most burn centers use *Parkland* formulas: 2–4 ml x body weight (kg) x burn area (% of body surface); Evans *(Evans)* or Brook *(Brooke):* 2 ml x body weight (kg) x area of burn injury (% of body ssurface) per day [12]. Obviously, according to the Parkland formula, the volume of injected crystalloids on the first day with a burn of 30% body surface and body weight 70 kg may be more than 8,000 ml. Numerous authors point to excessive infusion therapy with crystalloids, including the prehospital stagewhich is, according to various authors, 6 ml / kg /% burn and more. It is reported that with extensive burns, the victim may receive up to 25,000 ml (!) of fluids intravenously within the first 48 hours, which is 300–400% of the volume of blood flow, and which should be avoided [9, 10, 13]. The rules for intensive burn shock treatment are based on intravenous administration of crystalloid, colloid, hyperosmolar solutions or plasma. All the original formulas *Cope* and *Moore*, *Evans*, *Brooke* suggested the simultaneous use of a solution of crystalloid and colloid in burn shock, because the contents of burn blisters revealed the same concentrations of sodium ions as in plasma and about half of the plasma protein concentration [2].

When treating shock, the authors recommend that you comply with the following requirements. In the first 24 hours, the daily ITT is administered unevenly: during the first 8 hours — ½ of the calculated volume, in the next 16 hours — the second half. The maximum amount of ITT in the first day of a burn shock should not exceed 10–12% of the victim's body weight, and the maximum burn area taken into account should be no more than 50% of the body surface. After 24 hours on the 2nd day of shock, the daily ITT volume is reduced by one-half or one-third. In elderly patients, the amount of ITT should not exceed the calculated one, moreover, it should be reduced [13]. Modern authors draw attention to the fact that formulas for intensive care for burn shock patients should serve only as a guideline [14], given that patients already on the first day may drink in the absence of nausea and vomiting.

Z. Bak et al. evaluated fluid therapy for burn shock using the Parkland formula (2–4 ml / kg /% burn) in 10 patients with a burn of more than 20% of the body surface who underwent a transesophageal echocardiography, pulmonary artery catheterization and transpulmonary thermodilution. The authors found that the indicators of oxygen transport, heart rate, mean arterial pressure, left ventricular ejection fraction during infusion therapy did not change significantly. It was noted that intrathoracic blood volume and extravasal fluid of the lungs increased within 12 hours after the burn. Studies have shown that there was no need to increase the total volume of fluid within 36 hours after the burn. Early (12 h) signs of central circulatory hypovolemia confirmed the rule of more rapid fluid administration at the beginning of treatment [15].

MECHANISMS THAT FORM HYPERHYDRATION IN BURN PATIENTS

The basis of hyperhydration in burn patients is the increased permeability of the microvascular bed caused by SIRS, which is aggravated with an excessive amount of ITT. In case of burn disease, SIRS is undulent, its scoring increases to maximum values during episodes of bacteremia and antigenemia, after surgical interventions and extensive dressings have been performed, with the development of sepsis and other septic complications. Each episode of the score growth is accompanied by increased levels of proinflammatory mediators with increased vascular permeability, changes in cellular and humoral immunity, homeostasis indicators, and typical generalized morphological changes in organs and tissues leading to multiorgan dysfunction/failure [16]. Forming local and generalized tissue edema leads to the deepening of burn wounds disrupting the supply of nutrients to the cells.

Generalized edema of damaged and intact tissues and organs are sustained in the shock period by a decrease in plasma oncotic pressure (due to massive loss of proteins) and acute renal failure [17].

Excessive infusion therapy in the period of shock after 3 days leads to the development of acute circulatory failure, pulmonary and brain edema, intra-abdominal hypertension or the polycomplex syndrome. It is the most common cause of acute respiratory failure and pneumonia [18–20]. In fact, overhydration is an iatrogenic complication leading to multiple organ dysfunctions. Positive fluid balance is one of the leading causes of serious complications and prognostic factors of death [10, 21].

CARDIOVASCULAR INSUFFICIENCY AND PULMONARY EDEMA

The burn trauma has a long-term systemic effect on the heart and blood circulation, which increases the length of stay of patients in the hospital and mortality [22]. At the end of the last century, the authors wrote about the activation of the hypothalamic-pituitary-adrenal system with an increase in the blood levels of adrenocorticotropin, cortisol, adrenaline in the burn patients [23-26]. Later it was shown that an increase in the level of catecholamines and severe tachycardia are one of the central effects of exposure to pro-inflammatory interleukins in SIRS [27]. It was also revealed that there is a correlation between markers of systemic inflammation and cardiac arrhythmogenesis (atrial fibrillation and ventricular tachycardia) [28].

Acute cardiomyopathy with multifactorial pathogenesis develops in SIRS and sepsis. Inflammatory mediators, including cytokines, lead to depression of myocardial contractility [29]. Endotoxin, tumor necrosis factor alpha and nitric oxide have a negative inotropic effect [30]. Diastolic dysfunction of the left ventricle of the heart is an independent prognostic marker of mortality in critical burn patients. Transthoracic echocardiography and the study of inflammatory cytokines (tumor necrosis factor alpha and interleukin 6) revealed that the diastolic function of the left ventricle significantly improved with a decrease in the level of cytokines. The result can be used in the treatment

of severe burns by modulation of SIRS [31].

Other mechanisms of damage to the heart include exposure to endogenous intoxication products and microorganisms, electrolyte and metabolic disorders, hypoproteinemia and anemia [32]. Tachycardias play an important role in insufficient blood supply to the heart itself, and the negative effect of a significant increase in heart rate on the functional state of the heart is associated with a shortening of diastole [33].

The risk factors and clinical manifestations of myocardial damage in patients with severe burns were retrospectively analyzed. It has been shown that the incidence and prevalence of myocardial damage in such patients is statistically significantly dependent on the area of the burn lesion and the period since the moment of injury [34]. It has been shown that significantly lower baseline levels of the systolic output and cardiac index with a similar volume of infusion therapy are indicators of a poor prognosis [35].

In the last decade, separate works of authors from the USA, Germany and Australia have appeared on the use of transesophageal echocardiography in the treatment of burn patients. The advantage of the method is the ability to assess the adequacy of infusion therapy, fluid overload, pericardial effusion, pulmonary hypertension, vegetations on the valves, myocardial contractility and heart failure. According to authors, this safe method is able to solve many issues of diagnosis and treatment [36].

There is no doubt that excessive volume of infusion therapy plays a negative role, contributing to the development of heart failure, hydrothorax, hydropericardium, ascites and pulmonary edema. In burn patients, edema of the airways and/or lungs (even in the absence of an inhalation injury) can develop quickly and unpredictably. Despite the inclusion of colloids into the treatment, the incidence of pulmonary edema and deaths is increasing. Reducing systemic and local edema contributes to the continuous introduction of vitamin *C* [14, 37]. *J.C. Manelli* (1996) found that transfusion of colloids in the first 24 hours after a burn injury causes fluid retention in the lungs, and transfusion of albumin after 24 hours contributes to the resorption of edema [38]. Other authors have reported that the inclusion of albumin in the first 24 hours in patients with a burn area of 20% of body surface and more leads to lower doses of vasopressor amines, reduces the duration of ventilation and mortality [39]. In sepsis, an increase in the permeability of the pulmonary capillaries also causes an increase in the extravascular volume of the lung fluid [40], which indicates the danger of overhydration of the lung tissue in both uncomplicated and complicated burn disease.

BRAIN EDEMA

A systemic increase in vascular permeability in burn patients with SIRS impairs all physiological barriers, including the blood-brain barrier, while pro-inflammatory interleukins and phagocytes easily penetrate into the brain and activate sedentary macrophages of the central nervous system. In turn, these cells secrete copious amounts of reactive types of oxygen and nitrogen, proteases, cytokines/chemokines, leading to additional neuronal damage and life-threatening brain edema in patients with severe burns [41].

ABDOMINAL COMPARTMENT SYNDROME

Recently, a significant number of publications has been devoted to intra-abdominal hypertension and abdominal compartment syndrome in patients with sever burns.

Generalized increase in capillary permeability forms intraperitoneal fluid accumulation. The edema of the intestinal wall and the translocation of fluid lead to an increase in intra-abdominal hypertension, which lowers with decreasing capillary permeability. Intra-abdominal hypertension was detected in 64.7–74.5% of patients with burns of more than 30% of the body surface. Abdominal compartment syndrome developed in 4.1–16.6% of patients, while the mortality rate was 74.8–88%. Prevention of the syndrome is a difficult task, but the authors are unanimous in their opinion that the reason is the excess amount of crystalloid fluids. These complications can be prevented by decreasing the volume of infusion therapy in shock [13, 19, 42].

In a burn injury, intra-abdominal hypertension is secondary in contrast to the primary one, which is associated with diseases or injuries of the abdominal organs. In turn, by interfering with respiratory mechanics, it can lead to non-cardiogenic pulmonary edema [43].

The use of plasma or albumin, solutions of starch, hypertonic salt solutions, as well as the use of other markers to assess the effectiveness of intensive therapy (hematocrit, mean arterial pressure), in addition to diuresis, reduce the incidence of complications and compartment syndrome caused by systemic edema during burns [44–46]. The risk of developing abdominal compartment syndrome can be reduced by monitoring intra-abdominal pressure, limiting the volume of infusion therapy, using Ringer's lactate solution, performing necroectomy for anterior abdominal wall burn and percutaneous drainage of the abdominal cavity. A compulsory measure of treatment is decompression laparotomy. But although surgical decompression is effective, the prognosis remains poor [47–50].

HYPERNITRAEMIA

One of the most severe complications of the immediate post-shock period is a violation of the level of serum sodium. The causes of hypernatremia are multifactorial and not fully understood. Most authors associate hypernatremia (more than 146 mmol/l) with systemic dehydration, but it can also occur when using hypertonic solutions of sodium chloride [2, 51, 52]. At the same time, other authors report that in patients with severe burns, hypernatremia is a frequently observed electrolyte balance disorder [53, 54]. In our opinion, hypernatremia may develop as a result of violations of mineralocorticoid adrenal function because SIRS is accompanied by generalized self-destructive changes in all organs [55], contributed by hyperhydration.

Electrolyte disturbances may provoke an increase in the volume and speed of infusion therapy in shock, which is difficult to avoid even in specialized burn centers. *T. Namdar et al.* reported that most cases of hypernatremia evolved on day 5±1.4 in elderly patients with burns over 10% of the body surface, with a history of renal concentration capacity disorders [56]. A retrospective analysis of other authors showed that hypernatremia was more common than hyponatremia, while the mortality rate was 33.5% and 13.8%, respectively, while the mortality rate of patients with burns without disturbing the sodium content was significantly lower and was 4.3. % The obtained data allowed the authors to assert that serum hypernatremia is an independent predictor of mortality [57].

The study of case histories of 105 burn patients with sepsis revealed that hypernatremia developed in 36 of them (34%) with a larger lesion area and a severe degree of inhalation injury. It is noted that patients who underwent early necrotomy were less likely to have hypernatremia. The authors suggest close correlation between the onset of sepsis and the onset of hypernatraemia (over 155 mmol/l) as a marker or diagnostic criterion in the innovative diagnostic scale of sepsis in burn patiens [58, 59].

There are very few publications on the treatment of hypernatremia by hemodialysis as a method of choice. Some studies have reported hemodialysis in victims with extensive burns (without systemic use of heparin to prevent fatal bleeding). Authors consider the method as effective and safe [60].

INFUSION MANAGEMENT AND CONTROL

Studies with a decrease in the amount of infusion of crystalloids ("permissive hypovolemia") and the use of colloids against a background of shock showed that such tactics are safe, well tolerated by patients, effective in reducing organ dysfunction, induced by the accumulation of edematous fluid and insufficient oxygen utilization of tissues [61, 62]. *Z.N. Chen et al.* who treated 34 patients with burns over 80% of the body surface according to a modified formula for calculating the volume of infusion (1 ml x 1% pt x 1 kg of body weight per day), which gives significantly less results compared to other conventional formulas, reported a decrease in the frequency of complications associated with excessive infusion therapy in shock [63].

More than 20 years ago, *A. Bortolani et al.* conducted a comparative study of two randomized groups of patients with burns over 30% of the body surface, in which patients of the same group received Ringer's lactate for 4 hours after the burn trauma, and patients of the other group received is hypertonic lactated saline. It was found that in the hypertonic lactated saline group, the results were better: less tissue swelling, less frequent complications, and lower mortality [64].

At the Army Institute of Surgical Studies (Texas, USA), a computer algorithm for infusion therapy was developed and implemented for the first 48 hours after the injury, with a burn area of more than 20% of the body surface, taking into account the body weight of the patient. The introduction of a computer support system for intensive care in patients with severe burns has reduced the volume of crystalloids and improved the results of treatment [65].

Currently, there are works whose authors recommend using invasive methods of monitoring central and pulmonary hemodynamics in the most severe patients, because, unfortunately, neither the rate of diuresis nor the monitoring of arterial or central venous pressure makes it possible to assess the adequacy of ITT and the danger of overhydration in the stage of burn shock [66, 67].

CONCLUSION

Thus, in case of a burn disease against the background of the development of a systemic inflammatory response with an increase in the permeability of the microvascular bed, pathogenetic mechanisms are formed that promote the release of the liquid blood fraction into the perivasal and interstitial spaces, body cavities, soft tissues and increasing with excessive infusion therapy. With deep and extensive superficial burns, the victims need infusion-transfusion therapy until the skin is completely restored. There is no doubt that the search for measures to eliminate the complications of overhydration in burn patients is important. However, given that iatrogeny is the cause of overhydration, it is necessary to develop methods for effective prevention. In most cases, excessive infusion-transfusion therapy in the stage of burn shock continues in the post-shock period of a burn disease, which contributes to the development of complications of overhydration.

So far, neither in domestic nor in foreign literature there are recommendations for infusion-transfusion therapy after the burn shock. The accumulated experience in treating the victims allowed us to develop and propose an algorithm for infusion-transfusion therapy during the post-acute acute period of burn disease covered by the patent for invention No. 2626689 "Method for determining the basic volume of infusion therapy in the after-shock period of burn disease" (date of state registration in the State Register of Inventions in Russian Federation on July 31, 2017) [68]. The invention relates to the intensive care of burn patients. To determine the basic volume of infusion therapy, the following indicators are used: total burn area, deep burn area and body weight with correction factors for the patient's age and weight. The method allows to standardize infusion therapy throughout the acute period of burn disease. Its peculiarity is that a different volume of infusion therapy is used for 1% surface (0.5 ml) and 1% deep (1.5 ml) burn, which allows to reduce the total daily volume over time as epithelization of the surface burn occurs. The treatment of acute thermal lesions in burn patients in the Department of Acute Thermal Lesions of the N.V. Sklifosovsky Research Institute for Emergency Medicine in accordance with the developed algorithm, showed its high efficiency in preventing overhydration the need toadopt this method in burn departments and centers.

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