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PATHOGENETIC TREATMENT OF MECHANICAL BURN SHOCK CAUSED BY EXTENSIVE BURN INJURY AND LONG-TERM COMPRESSION SYNDROME (LITERATURE REVIEW)

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Abstract. Mechanical burn shock caused by extensive burn injury and prolonged compartment syndrome is a complex and multifaceted problem. A number of pathological processes, such as severe pain, plasma loss, hypovolemia, disseminated intravascular coagulation syndrome, systemic hypoxia, electrolyte imbalance, reperfusion tissue damage, endotoxicosis, and metabolic disorders determine the severity of the pathology in patients with combined trauma. Despite the variety of methods of pathogenetic treatment of mechanical burn shock, mortality among this group of patients still remains at a high level. In accordance with this, the question of a more detailed study of the pathogenesis and improvement of methods of pathogenetic treatment of mechanical burn shock remains relevant.

Keywords: mechanical burn shock, skin burns, prolonged compression syndrome, pathogenesis of shock

ПАТОГЕНЕТИЧЕСКОЕ ЛЕЧЕНИЕ МЕХАНО-ОЖОГОВОГО ШОКА, ВЫЗВАННОГО ОБШИРНОЙ ОЖОГОВОЙ ТРАВМОЙ И СИНДРОМОМ ДЛИТЕЛЬНОГО СДАВЛЕНИЯ (ОБЗОР ЛИТЕРАТУРЫ)

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Резюме. Механо-ожоговый шок, обусловленный обширной ожоговой травмой и синдромом длительного сдавления, является сложной и многогранной проблемой. Ряд патологических процессов, таких как выраженный

болевой синдром, плазмопотеря, гиповолемия, синдром диссеминированного внутрисосудистого свертывания, системная гипоксия, расстройство электролитного баланса, реперфузионное повреждение тканей, эндотоксикоз, обменные нарушения, обусловливают тяжесть течения патологии у пациентов с комбинированной травмой. Несмотря на разнообразие способов патогенетического лечения механо-ожогового шока, летальность среди этой группы больных по-прежнему остается на высоком уровне. В соответствии с этим актуальным остается вопрос о более детальном изучении патогенеза и усовершенствовании методов патогенетического лечения механо-ожогового шока.

Ключевые слова: механо-ожоговый шок, ожоги кожи, синдром длительного сдавления, патогенез шока

INTRODUCTION

Skin burns occupy the third place among all traumas in the Russian Federation [21]. Despite the high level of health care development, even in some European Union countries the incidence of burn injuries reaches 295 cases per 100,000 population [39]. Annual reports of the Ministry of Health of the Russian Federation state that burn lesions were detected in 220–240 victims in the period 2021–2022, among whom the mortality rate was 6.9–7.3% [33]. Over the last two decades (from 2003 to 2020), the main causes of burns are household injuries — 92%, while industrial injuries account for 8% [13]. Such indicators are primarily associated with the active industrialization of society and an increase in the standard of living.

The main causes of mass burn traumatism are natural and man-made disasters, hostilities, and industrial emergencies [5]. All of the above events increase the risk of not only temperature exposure but also mechanical exposure, which, in turn, may be the cause of the development of long compression syndrome (LSS) [46]. Prolonged compression syndrome is a life-threatening condition resulting from prolonged compression of a body part and its subsequent release, causing a state of shock [37]. Long-term compression syndrome was first described by E. Bywaters in 1941 during World War II. The author analyzed the experience of treating patients affected by massive bombardment [43]. In the following decades, no emergency event in large cities passed without the syndrome of prolonged compression.

The pathogenesis of prolonged compression syndrome is based on ischemic intoxication that develops during decompression. Toxins and products of cytolysis enter the organism from the long-term compressed (ischemic) tissues in the mode of normo- or hypoperfusion [42]. Toxins, in turn, increase the permeability of microcirculatory blood vessel walls, causing plasma effusion into the intercellular space, increasing edema of the injured limb and worsening blood rheology. In addition, an important link in the pathogenesis is pain, especially in the case of limb injury, rich in a large number of nerve endings in the skin, skeletal muscle and periosteum. As a result of complex effects of pain syndrome, toxic lesion, psychological and emotional state, traumatic shock may develop [34].

Due to the steadily increasing process of urbanization and industrialization of society, burn injuries and prolonged compression syndrome are always present in any natural and man-made disasters, and they are closely intertwined with each other. The search for methods to improve the provision of medical care in these conditions is an urgent issue of modern health care.

To optimize the treatment of patients with long compression syndrome in extensive burns, it is important to consider the pathophysiological aspects of this problem. The main cause of severe course and/or lethal outcome due to long compression syndrome with burns is shock. Shock is a state of the organism characterized by a complex of pathological shifts leading to hypoperfusion of organs with the development of subsequent cellular dysfunction [1]. The main pathogenetic factors of shock in these conditions include intoxication, metabolic disorders, hypovolemia, plasma loss, severe pain, cardiac and central nervous system dysfunction [1, 2].

Specialized literature devoted whole sections to the peculiarities of the course and causes of mechanical and burn shock on the background of long compression syndrome [19]. The main cause of unfavorable outcomes in combined lesions is the rapid depletion of homeostasis mechanisms [14]. The pathogenesis of long compression syndrome is characterized by endotoxemia by products of ischemia, tissue decay and reperfusion, the consequence of which is the development of toxic damage to the kidneys, heart and liver.

When a patient suffers from the syndrome of prolonged compression and extensive burns at the same time, a general reaction of the body is formed, which is accompanied by a peculiar clinical picture of mechanical-burn shock [31]. The mechanism of development of such shock is primarily associated with the loss of plasma, including proteins and electrolytes. These patients have systemic disorders due

to hypovolemia, hemoconcentration, increase in peripheral resistance and rheologic disorders in the first hours after injury [45]. These pathological processes are accompanied by a characteristic clinical picture in the form of increased heart rate, decreased stroke volume, and coronary blood flow disorders [8, 45]. In addition to toxic kidney damage by myoglobin due to the development of long compression syndrome, hypocirculation and hypercoagulability with subsequent oligo- or anuria develop under the influence of hemolysis products and tissue necrosis [40].

Generalized hypoxia is one of the leading causes of irreversibility of mechanical and traumatic shock [11]. The term "hypoxia" is understood as oxygen starvation arising from insufficient supply of oxygen to the tissues of the body or disruption of its utilization in the processes of biological oxidation. Hypoxia is a secondary phenomenon arising in the long compression syndrome and often has a significant impact on the outcome of burns, as it increases the existing oxygen deficiency [30].

Mechanical traumatic shock is caused by the syndrome of prolonged compression and extensive deep burns, the concentration of catecholomines in the blood increases 5–7 times, which leads to centralization of blood circulation and redistribution of blood to internal organs [17]. As a result, there is even greater ischemia of the burn area, which, in turn, leads to the formation of zones of secondary necrosis, even in areas where the initial burn was borderline [24].

Thus, a whole range of severe pathological processes such as pronounced pain syndrome, plasma loss, hypovolemia, disseminated intravascular coagulation syndrome, systemic hypoxia, electrolyte balance disorder, reperfusion tissue damage, intoxication, metabolic disorders are realized in the development of mechanical burn shock against the background of burn injury and long compression syndrome. All these pathological processes inevitably lead to multiorgan failure and septic course of traumatic disease in this category of patients.

The main cause of lethal outcomes in this type of shock is the development of multiorgan failure. Therefore, it is necessary to improve the methods of infusion therapy of extensive burns combined with the syndrome of prolonged compression. There are many scientific works in the modern literature, which describe the features of infusion therapy in shock, aimed at different links of the pathological process.

Based on well-known literature data, the conventional boundary of mechanical and burn shock reversibility is widespread blood stasis in metabolic capillaries. Its prevention is the main goal of antishock therapy [9]. The task of infusion therapy in mechanical and burn shock is to maintain the volume of circulating plasma and blood [44]. Infusion and transfusion therapy with crystalloid solutions is used to replenish the volume of circulating blood in shocks of various etiologies. The main preparations of these groups include: physiologic solution 0.9%, Ringer's solution, ringer-lactate, lactosol, sterofundin, etc. [18]. The infusion rate is also an important parameter. It can be: fast and slow replenishing the deficit of circulating blood volume [28]. When choosing the infusion rate in the acute period of mechanical burn shock, three main parameters serve as a reference point: arterial pressure, central venous pressure and hourly diuresis [28].

One of the most important hemodynamic parameters is oncotic blood pressure. In the phase of circulatory decentralization, special attention should be paid to this parameter, because the increase in oncotic blood pressure will promote the transfer of fluid from the intercellular space into the vascular bed, thus restoring the reduced volume of circulating blood [16]. Native or synthetic colloidal preparations are used for this purpose. Their volume can amount up to 75% of the total infusion volume [10].

The use of infusion therapy with crystalloid solutions without the use of blood products and synthetic colloids can lead to serious complication in the form of hyperhydration with subsequent interstitial pulmonary edema [26]. There is debate in the scientific literature, but most sources cite the opinion that a state of hyperhydration in combined burn injury is less preferable than mild hypohydration [20].

There was performed a comparative evaluation of the efficacy of antishock therapy schemes using synthetic and native colloids in different ratios. It was found that the value of cardiac output in patients who underwent infusion with colloids amounting to more than 50% was significantly higher in the first day after trauma [28].

The use of hydroxyethyl starch solutions with a molar mass of 130 kDa was also quite effective. A group of authors found that the use of this solution in combined burn injuries provides rapid recovery of circulating blood volume, cardiac output, parameters of oxygen delivery and consumption [12].

Sodium lactate is equally important in the treatment of mechanical-burn shock, since severe hypoxia disrupts the processes of lactic acid absorption in the Krebs cycle [38]. In addition to sodium lactate it is reasonable to use such preparations as lactasol and quintasol, where lactate is replaced by acetate [38].

As described above, both systemic and local hypoxia influence the development of mechano-burn shock. As a result, energy deficiency develops at the cellular level, leading to the disruption of a number of important energy-dependent processes in the cell [36]. Accordingly, it is reasonable to add antihypoxic agents [35]. The effect of these drugs is achieved mainly by reducing tissue oxygen demand

and energy potential, blocking calcium channels, inhibiting arachidonic acid metabolism and lipid peroxidation. Such group of drugs includes: amtisol, gutimin, cytochrome C, pyridoxine, sodium oxybutyrate [41]. It has been proved that antihypoxants in crystalloid solutions significantly increase the therapeutic efficacy of antishock infusion therapy when treating mechanical and burn shock [23].

The next group of drugs that improve the efficacy of infusion therapy in burn shock are antioxidants. Their necessity is caused by pathophysiological processes associated with the inevitable development of oxidative stress in burn shock and reperfusion when blood flow is restored in the area of tissue compression [29]. The problem of chain oxidation of cell membrane lipids has been known for a long time and was demonstrated back in the 50–60s of the last century by a group of scientists led by N.N. Semenov. Since then, a vast experience of fundamental and experimental data has been accumulated, confirming the key role of free radicals in physiological and pathological processes in the human body [27]. All antioxidants can be divided into natural and artificial ones. The first group includes vitamin E, which is a classic phenolic antioxidant, vitamin A, carotenoids, ascorbic acid, vitamins of the K group, ubiquinone (coenzyme Q10), flavonoids, melatonin, and estrogens. The second group includes selenium preparations (ebselen), ionol, phenozan, probucol (fenbutol), emoxipin, mexidol, idebenone, neurostrol, thiotriazolin, oliphen, amtisol, dimexide, etc. [27].

It is possible to use ascorbic acid and tocopherol acetate from this wide list of antioxidants in case of patients with burn shock [32]. Their joint administration allows to stabilize cell membranes and prevent excessive vascular permeability, plasma loss, hemolysis [15]. There are also single works on the use of superoxide dismutase and catalase in extensive burns, which allowed to reduce the vascular reaction, the severity of tissue edema and prevent hypoproteinemia [22].

Another pathologic process accompanying mechanical burn shock is the pain syndrome. The problem of pain control has not been completely solved to date [17]. A pain impulse that develops in mechanical-burn shock has a twofold significance. Firstly, it activates excessive defense mechanisms, which often causes energetically disadvantageous strengthening of the most important life-support systems. Secondly, a pain impulse is one of the mechanisms regulating the emergence of inflammatory reaction, which always accompanies traumatic injury and itself can be a source of a pathological impulse, aggravating the patient's condition [4].

To reduce pain syndrome, anesthetics, narcotic and non-narcotic analgesics, hypnotics, tranquilizers, and sedatives are used both as independent therapy and in combination [3]. However, it should be noted that general anesthesia methods cause an additional load on the body and depress many vital systems [6]. Accordingly, they cannot be considered as a specific method of burn shock treatment. Similar problems have been described with the use of narcotic analgesics, which depress the respiratory center, cause hypotension and reduce cardiac output [25]. Currently, modern narcotic analgesics such as buprenorphine and Prosidol are more preferable since they have fewer of the above-described side effect [7].

Thus, mechanical burn shock caused by extensive burns in combination with long compression syndrome is a complex and multifaceted problem. The development of a number of pathological processes, such as pronounced pain syndrome, plasma loss, hypovolemia, DIC, systemic hypoxia, electrolyte balance disorder, reperfusion tissue damage, endotoxemia and metabolic disorders, determine the severity of the pathology in this category of victims. Nowadays there are many variants of solutions, preparations and treatment schemes for infusion therapy of mechanical-burn shock, but lethality still remains high. Accordingly, it is urgent to thoroughly study the possibilities of increasing the effectiveness of infusion therapy in mechanical-burn shock caused by extensive burns in combination with the syndrome of prolonged compression.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

Competing interests. The authors declare that they have no competing interests.

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