PATHOGENETIC ASPECTS OF THE FLOW OF BURN INJURY IN CHILDREN

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ABSTRACT — The article discusses the changes occurring in a child's body in the acute phase of burn injuries, which affect a number of organ systems including the gastrointestinal tract. An important role in the pathogenesis of burn disease belongs to a disorder of the gastrointestinal tract with the syndrome of bacterial translocation, which requires a search for rational methods of treatment and prevention. There is still a possibility of constant learning and improving of approaches to the treatment of purulent-septic complications, which are the main causes of mortality in the stage of acute burn toxemia.

KEYWORDS — children, burn injury, purulent-septic complications

Burn injury occupies one of the leading places among the injuries in peacetime. According to the WHO and UN Children's Fund (UNICEF), mortality in children takes 3rd place (1). In Russia 200000 patients with burn injury are hospitalized annually, 40% of whom are children. Mortality from burn injuries reaches from 0.4% to 1.4% in different age groups. The main causes of mortality in the stage of acute burn toxemia are purulent-septic complications in combination with endogenous intoxication of metabolic and microbial origin, they account for 25–30% of cases of mortality in burns. The risk of early sepsis (4–5 days after injury) increased to 75–90% with deep burns over 10–15% of the body surface area. Due to anatomical and physiological characteristics, the younger the child, those with relatively smaller area of the lesions developing burn shock. So the critical values for the children of the first year are 5%, from age 1 to 5 years — 10%, 6–15 years 20% of body surface (1, 2).

Changes occurring in the body during the acute period of burn disease, affect many organ systems including the gastrointestinal tract. Gastrointestinal blood circulation gets an average of 15–20% of total cardiac output, the bulk of which receives the small intestine. In critical states the centralization of blood flow occurs to ensure the functioning of the organs with a constant high metabolic rate of the brain, heart and lungs. The gastrointestinal tract is experiencing ischemia, to which the most sensitive stomach and small intestine that has been proven in animal experiments. Ischemia is exacerbated by the release into the bloodstream of a large number of vasopressor substances — epinephrine, angiotensin, vasopressin, which operate on a duodenum gut and skinny due to the prevalence in their structure of α-receptors (3, 6).

Rapidly progressive disturbance of microcirculation in the form of primary change to the spasm of blood vessels in congestive hyperemia due to the expansion of precapillary sphincters and preservation of the high tone post-capillary venules lead to damage of the mucous membrane, extending from the submucosa to the intestinal lumen. The development of hypoxia leads to an increase in the permeability of cellular and lysosomal membranes for enzymes. Activated proteolytic enzymes (pepsin, trypsin) and lysosomal hydrolases (acid phosphatase, β-glucuronidase) destroy the mucous membrane, the resistance of which is reduced due to circulatory disorders, oppression of synthesis and destruction of mucin, which is compounded by the pathogenic action of proteolytic enzymes of bacteria (6).

Normal intestinal microflora performs a nonspecific protective function in the organism (antagonist, enzymatic, vitaminoterapiya, stimulation of the immunological reactivity of the organism, partly the organization and maturation of the reticuloendothelial system), but with the development of pathological conditions in which changes in composition and properties, develops goiter. Reduces the total number

of typical E. coli, an increasing number of putrefactive, pyogenic, spore and other pathogens (6, 7).

It has been experimentally proven changes in the composition of microbiocenosis of large intestine in burn injury in children: only 10% of patients qualitative and quantitative composition of microbiota was normal in 1–3 days after injury. In other cases, on the background of quantitative changes in the intestinal microflora occurred and its significant qualitative change. In half of the cases the species composition of the microflora were consistent with 2–3 degrees of dysbiosis. As a result of damage of the intestinal mucosa, increasing its permeability, changes in the number and pathogenicity of bacteria contained in the lumen of the gastrointestinal tract, the conditions for the penetration of bacteria, their metabolic products and other toxic substances through the mucous barrier of the intestinal wall into the systemic circulation (bacterial translocation) (4, 5, 7).

Describes the cases of insolvency of the intestinal barrier under the influ-ence of massive bacterial invasion (stress factor in the form of high dose of staph introduced subcutaneously, a massive breakthrough of fecal streptococcus from the intestine to the lymph nodes, blood and internal organs. In animal experiments established that the barrier function of the small intestine is damaged after 5 minutes after receiving the burn injury, reaching a maximum after 4 hours and remains high for a long time. Noted that the violation of the barrier function of the intestinal mucosa with the development of dysbiosis in burn injury may lead to hematogenous and lymphatic spread of intestinal microflora in the body, the development of sensitization to conditionally pathogenic microorganisms of the intestine, further leading to autoimmune reactions (2). As the elimination of burn shock and restore microcirculatory blood flow in the systemic circulation receives a great number of toxic products from lesions undergoing ischemia during shock.

The bulk of the toxic products formed by exposure on the cells and the destruction of the high molecular compounds, various reactive oxygen species (nitric oxide, hypochlorite, peroxyl radical, etc.) produced by neutrophilic granulocytes and macrophages as a manifestation of the nonspecific response of the body in response to injury. Many researchers call this phenomenon the "oxygen metabolic burst of leucocytes", others – "respiratory explosion". Under the influence of cytokines neutrophil leukocytes is a "respiratory burst", consisting in a sharp 10–15 times amplification of oxidative processes with the formation of nitric oxide, which have a cytotoxic effect (4).

In severe and persistent inflammatory process in the body is excessive accumulation of biologically active substances. As a result of their actions long flowing hyperactivity gives way to hyporeactivity and then areactivity, which is accompanied by increasing privatedata development in severe cases, multiple organ failure (6). The disposal of toxic substances (products of tissue decay, medium molecular peptide and other toxic substances) occurs in different ways: 1) secretion through the kidneys, gastrointestinal tract, skin; 2) deactivation in the liver; 3) mononuclear-macrophage system of the lungs. However, it should be noted that the greater depth and area of the burn, the sooner you can come decompensation protective means of the body, leading to accumulation of toxic substances in the body in high concentrations, causing disruption of their properties and functions, with the development of multiple organ failure (4, 5).

According to the literature of burn trauma in children occupies a leading position among all injuries in peacetime that require constant learning and improving treatment approaches. Along with microcirculatory disorders, the action of cytokines, proteolytic enzymes and toxins, an important role in the pathogenesis of burn disease belongs to violations of the gastrointestinal tract with the syndrome of bacterial translocation, which requires a search for rational methods of treatment and prevention.

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