WEB OF SCIENTIST: INTERNATIONAL SCIENTIFIC RESEARCH JOURNAL
ISSN: 2776-0979, Volume 5, Issue 01, January, 2024

MAIN CHARACTERISTICS OF RHABDOMYOLYSIS IN CLINICAL PRACTICE

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Abstract
According to modern concepts, multiple organ failure syndrome is defined as insufficiency (dysfunction) of multiple organ systems and represents a serious problem in surgical patients in critical conditions [18]. Until recently, skeletal muscles were not formally included in the organ systems affected by multiple organ failure syndrome.

Keywords: Rhabdomyolysis, clinical picture, diagnosis, treatment.

Introduction

In this case, dysfunctions of the lungs, kidneys, liver, brain, heart, caused by disturbances in the processes of microcirculation and metabolism in the cells and tissues of these organs are well known [4]. However, the participation of skeletal muscles in it has not been studied. Meanwhile, muscles make up about 40% of body weight, and there is reason to think about the possible participation of muscle damage (rhabdomyolysis) as a trigger or complicating factor in the development of multiple organ failure. Until now, rhabdomyolysis was considered a rare complication, although this is most likely due to the difficulty of diagnosis. Interest in muscle damage has increased due to the development of highly accurate methods for the determination of myoglobin (MG) in biological fluids. Today, rhabdomyolysis is a clinical and laboratory syndrome resulting from damage to skeletal muscles with the release of cellular contents of myocytes into the plasma. During rhabdomyolysis, a large amount of intracellular substances (MG, lysosomal and mitochondrial enzymes, histamine, serotonin, oligo- and polypeptides) enters the systemic circulation with the development of endotoxicosis. The entry of muscle destruction products into the general bloodstream leads to the development of systemic complications (acute renal failure), serious disturbances of homeostasis, and multiple organ failure syndrome, often threatening the patient's life. The causes of rhabdomyolysis are divided into traumatic and non-traumatic. The first include long-term compression syndrome, positional ischemia syndrome, convulsions, and

significant physical activity [6]. In traumatic (direct) rhabdomyolysis, destruction of skeletal muscles develops as a result of direct impact on them due to injury, damage or compression. Non-traumatic causes include muscular dystrophies, electrolyte disorders (hypokalemia), inflammatory muscle diseases (dermatomyositis, polymyositis) and various systemic infections (leogynellosis, leptospirosis, influenza, etc.) [14, 21, 3]. The main risk groups are: patients with morbid obesity, patients constantly taking drugs that lower blood lipid levels, patients in the postoperative period [22, 32]. The clinical consequences of rhabdomyolysis are the result of local damage (eg, muscle trauma, muscle swelling, compression) and the systemic effects of numerous biochemical toxins and bioactive substances, as well as hypoxia. A huge number of different diseases can be accompanied by rhabdomyolysis, myoglobinemia and myoglobinuria. This indicates the participation of such a large organ (more than 40% of body weight) as muscle mass in various pathological processes and their undoubted influence on the course and outcome of the underlying disease. Infections are not considered to be common causes of rhabdomyolysis, although reports are rapidly increasing. Rhabdomyolysis has been described in Legionnaires' disease, diphtheria, leptospirosis, influenza, viral hepatitis, staphylococcal infection, HIV, etc. [40, 41]. In this case, direct muscle damage from bacterial toxins is possible, as well as the adverse effects of hyperthermia, as well as circulatory disorders (bacterial shock). Rhabdomyolysis can complicate water and electrolyte disturbances (water intoxication, dehydration, hypokalemia, hypo- and hypercalcemia, as well as fasting) [31, 34]. The morphological substrate of rhabdomyolysis is dystrophic, degenerative and necrotic changes in skeletal muscles [10]. The first morphological signs of damage appear after one hour of ischemia and represent local inflammatory changes. These changes become more pronounced after 3–4 hours. Microscopically, in the damaged muscles, against the background of perimysial edema and inflammatory infiltration, there is a sharp plethora, erythrocyte sludge and fresh blood clots in the microvasculature. Foci of coagulative necrosis, edema, disintegration and homogenization of muscle fibers up to the complete disappearance of fibrillar structures are found everywhere [5]. E.A. Nechaev et al. (1993) describe structural changes in skeletal muscles as liquation necrosis of muscle fibers of the discoid type, manifested by sharp relaxation of the discoid type with lysis of isotropic discs. Clinical examination reveals pain on palpation, hardening and swelling of the damaged muscles.

The basis is the successful treatment of the primary disease, multiple organ failure. Treatment of rhabdomyolysis includes a complex of general and, if necessary, local treatment. General treatment includes preventing and treating hypovolemia with

saline fluid therapy, which improves perfusion in the muscles and kidneys. The cornerstone of successful treatment of rhabdomyolysis remains the preservation of renal function. The key to successful treatment is the early start of infusion therapy. One cannot but agree that maintaining circulating blood volume (CBV) is the most important initial aspect in this direction in patients with rhabdomyolysis complicated by acute renal failure (ARF) [42]. The Guidelines for the Treatment of Crash Syndrome in Disaster Victims states that isotonic saline administration may be a choice for volume correction in crash syndrome complicated by the development of rhabdomyolysis. The rate of intravenous administration of the solution is also proposed here: in the first 2 hours after injury, 1 l/hour and 500 ml/hour for the next 120 minutes with mandatory monitoring to avoid volume overload and to control the acid-base state. Infusion therapy begins within 6 hours of muscle damage, the goal is to achieve a diuresis rate of at least 300 ml/hour. The use of sodium bicarbonate solution to prevent the development of acute renal failure is based on the concept of increased nephrotoxicity of MG in an acidic environment. Therefore, an alkaline environment can reduce the formation of free radicals and the formation of myoglobin casts in the kidneys. This raises the possibility that the administration of bicarbonate will raise the urine pH level above 6.5 and prevent the development of acute renal failure, metabolic acidosis. However, it must be remembered that bicarbonate can cause paradoxical intracellular acidosis, volume overload in patients with respiratory and/or vascular insufficiency as a result of the fact that the bicarbonate buffer system can cause an increase in the concentration of CO_2 in the circulation according to the formula: $HCO_3 + H^+ \rightarrow H_2CO_3 \rightarrow H_2 + CO_2$. Another important area of treatment is the elimination of myolysis products. Water loading and stimulation of diuresis with mannitol accelerates the elimination of toxic products [60]. To date, no consensus has been reached on the use of mannitol and this is due to the side effects of its use - a decrease in the volume of circulating fluid and potentially dangerous azotemia. At the same time, theoretically, there are positive effects from its use in the form of increased diuresis, improved renal perfusion, MG excretion and direct antioxidant effect on the renal parenchyma.

Conclusion

Authors proposing the use of mannitol indicate that mannitol should be prescribed only if infusion therapy does not lead to an increase in diuresis rate above 300 ml/hour [28]. Mannitol should be completely avoided in patients with anuria.

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