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RHABDOMYOLYSIS AS A CONSEQUENCE OF EXCESSIVE PHYSICAL ACTIVITY IN CHILDREN AND ADOLESCENTS

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Abstract. Rhabdomyolysis remains a major diagnostic and therapeutic and preventive problem for clinicians, especially when working with athletes of child and adolescent age. A diverse etiology, the absence of specific symptoms, late diagnosis, the development of systemic complications worsen the prognosis of the disease and complicate treatment. Rhabdomyolysis associated with physical activity in children and adolescents is non-traumatic in origin, is often caused by hereditary causes and is provoked against the background of infectious diseases and metabolic disorders. Timely implementation of correct infusion therapy remains the cornerstone of the treatment of rhabdomyolysis. A clinical case of successful treatment of a teenager with rhabdomyolysis complicated by acute kidney injury is presented.

Key words: *non-traumatic rhabdomyolysis; children and adolescents; acute kidney injury; literature review; clinical case.*

РАБДОМИОЛИЗ КАК СЛЕДСТВИЕ ЧРЕЗМЕРНОЙ ФИЗИЧЕСКОЙ НАГРУЗКИ У ДЕТЕЙ И ПОДРОСТКОВ

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Резюме. Рабдомиолиз остается большой диагностической и лечебно-профилактической проблемой для клиницистов, особенно при работе со спортсменами детского и подросткового возраста. Разнообразная этиология, отсутствие специфических симптомов, поздняя диагностика, развитие системных осложнений ухудшают прогноз заболевания и затрудняют лечение. Рабдомиолиз, связанный с физической нагрузкой, у детей и подростков по своему происхождению относится к нетравматическому, часто обусловлен наслед-

ственными причинами и провоцируется на фоне инфекционных заболеваний и нарушений метаболизма. Своевременное проведение корректной инфузионной терапии остается краеугольным камнем лечения рабдомиолиза. Представлен клинический случай успешного лечения подростка с рабдомиолизом, осложненным острым поражение почек.

Ключевые слова: *нетравматический рабдомиолиз; дети и подростки; острое повреждение почек; обзор литературы; клинический случай.*

INTRODUCTION

Rhabdomyolysis (ICD-10 code: M60–M63 Muscle diseases) is a clinical and laboratory syndrome of damage and destruction of myocytes of striated muscles, manifested by myalgia, muscle weakness, swelling of the affected muscles, a decrease in volume and the appearance of dark brown urine due to the release and entry into the systemic circulation of myolysis products, primarily myoglobin (MG), as well as creatine phosphokinase (CPK), lysosomal and mitochondrial enzymes, histamine, serotonin, oligo- and polypeptides and others, with the subsequent manifestation of endogenous intoxication. There are traumatic and non-traumatic rhabdomyolysis [1, 2]. In general medical practice, rhabdomyolysis is more often associated with traumatic damage to muscle tissue [3]. The lack of alertness of pediatricians regarding the possibility of developing non-traumatic rhabdomyolysis in children and adolescents can lead to diagnostic errors and an increase in adverse outcomes [4]. Among the many causes of non-traumatic rhabdomyolysis, one should remember the reality of the threat of muscle tissue damage due to high-intensity physical activity [5, 6].

In severe cases, rhabdomyolysis is an extreme degree of myopathy with the subsequent development of acute kidney injury (AKI), severe disturbances of homeostasis, disseminated intravascular coagulation syndrome, multiple organ failure with a threat to the patient's life [7, 8]. An Acute pigmented nephropathy, or myoglobinuria-induced AKI, myoglobinuric nephrosis, myorenal syndrome arethe most common systemic complications of rhabdomyolysis.

The occurrence of non-traumatic rhabdomyolysis in adult and pediatric population has not been precisely established, but the frequency of AKI in this pathology is known and, according to various sources, ranges from 10 to 55%, which indicates the relevance of studying this problem [9]. Among the causes of AKI, the share of rhabdomyolysis reaches 25%, and the mortality rate in patients with rhabdomyolysis complicated by AKI exceeds 10% [3, 10].

ETIOLOGY OF RHABDOMYOLYSIS

The incidence of non-traumatic rhabdomyolysis depends on the etiology (sporadic and recurrent forms) [4, 11, 12]. In childhood, adolescence and young adulthood, the development of non-traumatic rhabdomyolysis is facilitated by hereditary metabolic disorders, and the disease is characterized by a relapsing course [13–15]. An autosomal recessively inherited disease from the group of genetic disorders of fatty acids transport, namely the myopathic form of carnitine palmitoyltransferase II deficiency (CPT II), is one of the common causes of myoglobinuria [16, 17]. Other hereditary causes of rhabdomyolysis may include very long chain acyl-CoA dehydrogenase deficiency, congenital muscular dystrophy, and idiopathic paroxysmal rhabdomyolysis (Meyer–Betz syndrome) [12, 14, 18].

Triggers of non-traumatic rhabdomyolysis in childhood and adolescence, in addition to prolonged heavy physical activity, are infectious diseases of viral (influenza, COVID-19, enteroviruses and other agents) [19–21] and bacterial etiology (rickettsiosis, legionellosis, salmonellosis, tularemia, malaria, etc) [22], convulsions, hypothermia, overheating, stressful situations, metabolic disorders (water intoxication, dehydration, hypokalemia, hypo- and hypercalcemia, fasting) [23, 24], some drugs [4, 5, 25, 26], as well as idiopathic inflammatory myopathies (dermatomyositis, polymyositis or antisynthetase syndrome) [27].

PATHOGENESIS OF RHABDOMYOLYSIS

In *traumatic rhabdomyolysis* destruction of skeletal muscles occurs as a result of direct impact on them, damage or compression (compression syndrome), ischemia of the limbs due to disturbances in the main blood flow and increased tissue pressure inside the fascial spaces. The Disruption of arterial blood flow lasting more than 4 hours leads to muscle changes, the entry of myolysis products into the systemic circulation and the occurrence of multiple organ disorders. A type of long-term compression syndrome is positional ischemia syndrome, in which muscle compression is carried out

by the weight of one's own body while the patient is unconscious [28]. A Long-term immobilization of a limb with a plaster cast can also cause an increase in MG levels and CPK activity in the blood serum [29]. It should be noted that some authors incorrectly classify rhabdomyolysis due to excessive physical activity as traumatic [30].

The pathophysiological mechanisms of the development of non-traumatic rhabdomyolysis are different and in most cases are assumed [29]. Rhabdomyolysis during very strenuous exercise is caused by insufficient oxygenation of the skeletal muscles, as well as dehydration due to excessive sweating.

As a result of muscle breakdown, ATP synthesis switches from the aerobic to anaerobic pathway, in which ATP reserves are quickly depleted; a subsequent accumulation of lactic acid leads to intracellular acidosis. Due to hypoxia and a decrease in pH, the functioning of K^+-Na^+-ATPase is disrupted, while K^+ ions leave the cells into the vascular bed, and Ca^{2+} and Na^+ ions move in the opposite direction, which contributes to an increase in osmotic pressure in the myocyte, edema and disruption of its integrity, increased lipid peroxidation in cell membranes with the formation of peroxyl radicals. The peroxyl radicals, during interaction with the structural units of cell membranes, cause fragmentation of proteins, damage to DNA and lipids, which inhibits bioenergetic processes in myocytes, leading to their death, this is accompanied by an increase in the content of CPK and MG in the blood. The MG as an oxygen transporter ensures oxygenation of muscles during their contraction. A hypermyoglobinemia is a consequence of muscle damage and a marker of endotoxemia, since there is a direct relationship between serum MG levels and the severity of tissue hypoxia. An oxidative stress is a leading factor in the development of AKI in rhabdomyolysis [31]. The CPK is a component of muscle cells and one of the key enzymes of energy metabolism. The level of CPK, as a rule, correlates with the content of MG, but its peak content is reached much later. The MG is able to penetrate the glomerular basement membrane, bind to the Tamm–Horsfall protein and cause tubular obstruction, forming a sediment in the lumen of the distal tubules in the form of pigmented cylinders. Against the background of existing hypovolemia, the MG causes renal vasoconstriction and additional activation of the renin-angiotensin-aldosterone system. The pathogenesis of AKI due to rhabdomyolysis includes a hypovolemia, decreased renal perfusion, obstruction of the renal tubules by myoglobin casts, and myoglobinuric nephrosis [32]. At the same time, if rhabdomyolysis is immediately identified and adequately treated with ample hydration, the condition of most patients soon improves and no complications are observed [6].

CLINICAL PICTURE OF RHABDOMYOLYSIS

Clinical manifestations of exercise-related rhabdomyolysis are characterized by a triad of symptoms: muscle pain, weakness, change in urine color (reddish-brown, brown). Proximal muscle groups (shoulders, hips, lower back, calves) are most often affected. In severe cases nausea, vomiting, disturbances of consciousness of varying severity and decreased diuresis up to anuria are observed [5, 33]. The subclinical form of rhabdomyolysis is characterized by "tenderness" of certain muscle groups and mild pain during movement, felt 1-2 days after exercise, which is called delayed onset muscle soreness syndrome. Sometimes patients do not present any complaints; in these cases the disease is diagnosed by changes in biochemical blood parameters [34].

LABORATORY METHODS FOR DIAGNOSTICS OF RHABDOMYOLYSIS

Biochemical blood test. The rhabdomyolysis (myoglobinuria) is diagnosed when the serum myoglobin level exceeds 80 ng/ml and the creatine kinase level increases 5 times the upper limit of normal, exceeding 1000 U/L [6, 7]. The concentration of MG reaches a maximum on the 1st day of the disease, CPK — on the 3rd–5th day, which is explained by the low rate of elimination of CPK from the bloodstream. The increase in MG in the blood outpaces the increase in the activity of the enzymes lactate dehydrogenase (LDH), aspartate aminotransferase (AST) and alanine aminotransferase (ALT). CPK indicators are different depend on the gender, age, race, and level of physical development of patients. The level of CPK increases during periods of intensive growth in children [35]. Characteristic changes for this pathology are acidosis, increased creatinine, uric acid, C-reactive protein, hyperkalemia, hyperphosphatemia, hypocalcemia [5].

Assessment of diuresis. A decrease in urine output by 8–10% indicates the onset of AKI development. With further development of the pathological process, the diuresis decreases by 25% or more; an anuria may develop. During the recovery period, the daily diuresis increases significantly.

Urine analysis. The most typical changes in urine color from dark brown to black, impaired urine density, proteinuria, acetonuria, and the presence of pigmented casts [5, 16, 27]. The hematuria may occur [6].

Genetic research. Molecular genetic studies are usually carried out (next generation sequencing (NGS), fluorescence *in situ* hybridization (FISH test), polymerase chain reaction (PCR), Sanger sequencing, etc.) to detect hereditary causes of rhabdomyolysis, these studies are aimed at identification of gene polymorphisms affecting the activity of enzymes regulating lipid metabolism [5, 13, 15].

With the advent of tandem mass spectrometry (TMS), early diagnosis of a large number of hereditary metabolic diseases, the age of manifestation and the range of clinical manifestations of which is very variable, has become possible [36]. Certain variants of defects in mitochondrial β-oxidation of fatty acids determine the development of non-traumatic rhabdomyolysis. Thus, myopathy, manifested by exercise intolerance, is caused by a deficiency of carnitine palmitoyltransferase type 2 (CPT 2). This disease typically begins in adolescence or adulthood. Episodes of rhabdomyolysis may occur with an associated risk of developing AKI. The TMS method detects an increase in long-chain acylcarnitines (C16, C18), and determines a mutation in the *CPT2* gene, which is mapped to 1p32.3.

The another inherited cause of rhabdomyolysis may be very long-chain fatty acid acyl-CoA dehydrogenase (VLCAD) deficiency. Complete deficiency manifests itself in the neonatal period with severe cardiomyopathy and is often fatal. A partial failure begins in adolescence or adulthood with hypoketotic hypoglycemia, myopathy, and rhabdomyolysis. The *ACADVL* gene is located on 17p13.1 [37]. During TMS, an increase in acylcarnitine (C14) in the blood is detected.

Mitochondrial trifunctional protein (TFP) deficiency and long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency have similar clinical manifestations, but TFP is more severe. Patients have cardiomyopathy, hypoketotic hypoglycemia, liver dysfunction (Reye's syndrome), cholestasis, and rhabdomyolysis. The half of patients have pigmentary retinopathy. In LCHAD a mutation in the *HADHA* gene (2p23.3) is detected [37], in TFP — *HADHA* (2p23.3), *HADHB* (2p23.3). When performing TMS, patients with TFP show an increase in C16OH acylcarnitine, and in patients with LCHAD, an increase in C16OH and C18OH acylcarnitines [37]. To accurately verify the diagnosis after TMS, a molecular genetic study is performed to detect pathogenic nucleotide variants, structural rearrangements and variations in the copy number of relevant genes [36].

INSTRUMENTAL METHODS FOR DIAGNOSTICS OF RHABDOMYOLYSIS

Ultrasound. In the early stages of skeletal muscle necrosis, ultrasound signs of edema and destruction of muscle tissue are detected: a diffuse increase or decrease in the echogenicity of the muscles and their thickening are determined. There is a decrease in structure and erasure of the muscle pattern, arch-like bulges of the muscle fascia, and the appearance of intermuscular fluid inclusions are detected. The formation of calcifications indicates a late stage of the disease. However, changes detected in the gray scale mode are not specific to rhabdomyolysis and can be observed in other pathological muscle conditions. A Skeletal muscle ultrasound is more often used when the patient's condition is severe and magnetic resonance imaging is unavailable [38].

Compression sonoelastography and shear wave sonoelastography. These methods are used for a more accurate assessment of structural changes in muscles, since they allow the assessment of not only qualitative, but also quantitative indicators of muscle density [32, 39].

Magnetic resonance imaging (MRI). The MRI is a highly informative and sensitive method for diagnosing rhabdomyolysis, especially in cases of atypical distribution of damage, involvement of small muscle groups and a high probability of compressive effects on the neurovascular bundles. According to the MRI, muscles affected by rhabdomyolysis have increased signal intensity in T2-weighted and STIR modes, as well as reduced intensity in T1-weighted modes. The T2-weighted examination allows to identify areas of muscle necrosis. An improved muscle MRI technique with quantitative assessment of T2 relaxation time has high sensitivity and specificity (94 and 82%, respectively) in the diagnosis of rhabdomyolysis [40, 41].

Morphological diagnosis of skeletal muscle necrosis. In some cases, if rhabdomyolysis is suspected, a biopsy and histomorphological examination of the muscle tissue involved in the pathological process are performed [41].

In forensic medical expert practice, there are cases when differential diagnosis of rhabdomyolysis of a traumatic and non-traumatic nature is

necessary, which is achieved based on the results of a histological examination of autopsy material of skeletal muscles [28].

DIFFERENTIAL DIAGNOSTICS

The clinical picture of non-traumatic rhabdomyolysis is quite nonspecific, and requires additional research, including the exclusion of glomerulonephritis and a number of infectious diseases (viral hepatitis, leptospirosis, etc.).

TREATMENT OF RHABDOMYOLYSIS

Patients with rhabdomyolysis need the earliest possible appointment of adequate rehydration infusion therapy, prescribed in the first 6 hours from the moment of muscle damage, correction of electrolyte disturbances (hypo- and hyperkalemia, hyperphosphatemia). A diet limited in protein and potassium-containing foods is prescribed. The volume of drug therapy depends on the severity of the patient's condition and is aimed at preventing the development of AKI. Extracorporeal detoxification methods, eliminating myolysis products from the systemic circulation, are the most effective methods for treating complications of rhabdomyolysis [1, 4, 8, 32].

PROGNOSIS

The prognosis of rhabdomyolysis is determined by the risk of developing life-threatening conditions. The AKI, hyperkalemia accompanied by severe arrhythmias, hypovolemic shock and disseminated intravascular coagulation syndrome can be the causes of death in patients.

Severe rhabdomyolysis is accompanied by the development of a secondary immunodeficiency state, which promotes the activation of an opportunistic microbiome with a high risk of developing the manifestation of sepsis [8, 19, 28].

To illustrate the above, we present one of the clinical cases of typical non-traumatic rhabdomyolysis in a teenager, caused by intense physical activity and complicated by impaired renal function in the acute period of the disease.

CLINICAL CASE

Patient M., 14 years old, was urgently admitted by ambulance to the emergency department of St. Petersburg State Budgetary Institution Children's City Hospital № 2 of St. Mary Magdalene, accompanied by his mother, with a diagnosis of closed injury of the lumbar spine dated 08.11. Macrohematuria? Myositis of the back and thighs.

At the time of admission to the hospital, there were complaints of severe back pain, change in the color of urine (the color of "dark beer").

Anamnesis morbi. On November 7th, during classes in the volleyball section, the patient performed the same type of exercises on the hyperextension trainer for a long time. He limited his water load during classes. By the evening a pain appeared in the back and calf muscles, the muscles of the back of the thigh. Over the next two days the pain increased without any effect on oral Nurofen. On November 9th severe weakness appeared, the urine darkened to the color of "dark beer". The body temperature increased to 37.3 °C.

Anamnesis vitae. He grew and developed without signs of lag. The vaccination history corresponds to the vaccination calendar by age. He is not registered at the dispensary for diseases. There is no allergic history. There were no injuries or operations. A heredity is not burdened. An Epidemiological history is unremarkable. Periodically over the last year, a myalgia has been observed after prolonged physical exertion, which resolves within two days.

Objective examination data upon admission to the hospital. The general condition is moderate. A consciousness is clear. The food is satisfactory. Height — 182 cm (7 points; high physical development). Weight — 65 kg (4 points; harmonious development). BMI — 19.7 (within normal values). Heart rate — 66 per minute. Blood pressure — 120/80–110/70 mm Hg. The body temperature is 37.0 °C. A consciousness is clear. The physique is correct. The skin is pink, without rash. Visible mucous membranes are pink. The tongue is clean and moist. The boundaries of relative cardiac dullness are not expanded. Heart sounds are clear and rhythmic. The percussion sound above the lungs is clear. The breathing is vesicular, carried out in all fields, without wheezes. The abdomen is symmetrical, participates in the act of breathing, is accessible to deep palpation, is not swollen, soft, painless, pathological formations are not palpable. Symptoms of peritoneal irritation are negative. The liver is not enlarged. The spleen is not palpable. The chair is decorated. The urination is not difficult. A urine is the color of «dark-beer».

Local status. A percussion of the spinous processes of the spine is painless. The back muscles are swollen paravertebrally, their palpation is sharply painful; the muscles of the back of the thighs are painful on palpation.

Data from laboratory research methods. Blood test: 10.11 — hemoglobin — 142 g/l; erythro-

cytes — 4.84×10^{12}/l; hematocrit — 0.39; average hemoglobin concentration — 366 g/l; average hemoglobin content — 29.9 pg; platelets — 238.1×10^{9}/l; leukocytes — 7.7×10^{9}/l (11.14 — 5.3×10^{9}/l; 24.11 — 5.0×10^{9}/l); neutrophils — 70.7%; neutrophils abs. quantity — 5.47×10^{9}/l (24.11 — 2.29×10^{9}/l); eosinophils — 3.8%; basophils — 0.38%; lymphocytes 19% (11.11 — 27.1%); lymphocytes abs. quantity — 1.47×109/l; monocytes — 10% (24.11 — 9.1%); ESR — 5 mm/h. Changes in the blood test on 10.11 indicated the presence of indirect signs of hypoxia (increased average hemoglobin concentration; norm 346–354 g/l) [42] and inflammatory manifestations in the form of neutrophilia (relative and absolute), lymphocytopenia (relative and absolute), monocytosis. In the dynamics of observation (11.11, 14.11, 24.11) the appearance of leukopenia, neutropenia, and persistence of monocytosis was noted, which, along with low-grade fever, did not exclude the course of an infectious disease.

Biochemical blood test: 10.11 — increased ALT — 267 units/l (24.11 — 39 units/l); AST — 1218 units/l (11.11 — 1316 units/l; 24.11 — 23 units/l); LDH — 2156 units/l (24.11 — 436 units/l); CPK — 107,990 units/l (24.11 — 238 units/l); CPK-MB — 3762 units/l; myoglobin — 1801.1 μg/l; total bilirubin — 31.3 μmol/l; direct bilirubin — 12.1 μmol/l; glucose — 5.8 mmol/l (24.11 — 4.9 mmol/l); low-density lipoprotein cholesterol — 1.9 mmol/l; phosphorus — 1.48 mmol/l; chlorine — 109 mmol/l (24.11 — 105 mmol/l) with normal troponin levels — 0.02 ng/ml; C-reactive protein — 2 mg/l; creatinine — 86 mmol/l; urea — 3.9 mmol/l; rheumatoid factor — 3 IU/ml; gamma-glutamate transferase — 18.2 units/l; total protein — 66 g/l; albumin — 44 g/l; total cholesterol — 3.5 mmol/l; high-density lipoprotein cholesterol — 1.21 mmol/l; uric acid — 293 mmol/l; total calcium — 2.45 mmol/l; potassium — 4.1 mmol/l; sodium — 146 mmol/l; iron — 24.6 μmol/ml. A glomerular filtration rate (GFR) according to the Schwartz formula at admission was 83 ml/min/1.73 m^2, at discharge — 130.4 ml/min/1.73 m^2. The values and dynamics of AST, ALT, LDH, CPK, CPK-MB, myoglobin, electrolytes (phosphorus, chlorine), GFR indicated rhabdomyolysis with AKI without a decrease in nitrogen excretory function, toxic hepatitis and the presence of cholestasis. A dyslipidemia reflected the patient's underlying lipid metabolism disorder.

A study of humoral immunity factors in the blood revealed a decrease in IgG (10.11 — 621 mg/dl; 24.11 — 663 mg/dl) with normal levels of IgA and IgM — 117 and 76 mg/dl, respectively, complement components C3 — 117 mg/dl and C4 — 29 mg/dl.

Urine examination: 09.11 — quantity — 40 ml (24.11 — 80 ml); color brown (24.11 — straw yellow); transparency — slightly turbid; relative density — 1.030 (24.11 — 1.018); the reaction is slightly acidic; protein — 0 g/l; glucose — 0 g/l; hemoglobin 10.11 — 3+. A microscopy of urine sediment: 09.11 — squamous epithelium — 0–1 in the field of view (n/z); leukocytes — 3–5 in p/zr; unchanged erythrocytes — 1–2 in p/zr (10.11 — 15–20 in p/zr); changed erythrocytes — 1–3 in p/zr; mucus — 2–3; oxalate salts — 1–2; urate salts — 0–1. A daily diuresis 11.11 — 950 ml; daily protein — 0.170 g. An urine culture is sterile. A study of urine test data revealed signs characteristic of rhabdomyolysis: discoloration, hematuria, proteinuria [6].

Coagulogram parameters are within normal limits. A blood test for RNGA with salmonellosis, pseudotuberculosis, dysentery (Sonne, Flexner, Newcastle), yersinia (O3 and O9) diagnosticums is negative. Nasopharyngeal swab — rapid antigen test for COVID-19 — negative. A serum antistreptolysin-O was slightly elevated — 211 IU/ml. The study of antibodies to helminthiasis (ascariasis, toxocariasis, anisakiasis) in the blood is negative. The study of IgG and IgM to cytomegalovirus, IgG to herpes virus type 6, IgM to capsid, nuclear and early antigens of the Epstein-Barr virus — negative, IgG to herpes viruses types 1 and 2 — titer 1:1600 (positive) with normal IgM values. The data obtained excluded the presence of current infectious diseases and helminthiasis in the patient.

Results of instrumental research methods. An ECG: heart rate — 65 beats/min; ectopic atrial rhythm, incomplete blockade of the right bundle branch, impaired repolarization (the detected changes were regarded as functional). An X-ray of the lumbar spine in two projections: a lumbar lordosis is preserved, the height of the intervertebral discs is not changed; the ratio of the posterior parts of the vertebrae and the shape of the vertebral bodies are not disturbed; At the lower contour of the spinous process of LIII, an additional shadow of bone density measuring 5×3 mm with clear contours and a homogeneous structure (unfused ossification core) is determined. An ultrasound of the kidneys revealed hyperechogenicity of the cortical layer, characteristic of kidney disease. A bladder data without pathology.

For the first two days, the patient received treatment in the intensive care unit, where intravenous infusion of glucose-saline solutions was carried out, then he was transferred to the nephrology department, where detoxification therapy continued. Discharged on the 14th day of hospitalization with improvement in clinical and laboratory parameters under the supervision of a local pediatrician and nephrologist, genetic consultation was recommended, planned hospitalization in the nephrology department in a year, exemption from physical education for a month, then classes are possible after monitoring kidney function and permission from a sports doctor .

This example demonstrates a typical clinical picture of rhabdomyolysis, complicated by AKI, in a teenager of high physical development, which arose as a result of excessive physical activity during sports activities. Considering the anamnestic data on previously observed long-term myalgia after heavy physical activity, hereditary causes of the disease cannot be excluded. The triggers for rhabdomyolysis could be the asymptomatic course of the infectious disease and the presence of dyslipidemia. A feature of the developed AKI was the absence of a decrease in nitrogen excretion function and the rapid recovery of GFR against the background of detoxification and rehydration therapy. Laboratory manifestations of hypoxia, toxic hepatitis and cholestasis reflected the severity of endotoxemia caused by rhabdomyolysis.

CONCLUSION

Rhabdomyolysis associated with physical activity (post-exercise) in children and adolescents is non-traumatic in origin, often due to hereditary causes and provoked against the background of infectious diseases and metabolic disorders. The acute kidney damage is a typical potentially reversible complication of rhabdomyolysis and requires differential diagnosis, timely adequate treatment and subsequent follow-up of patients to prevent the development of chronic kidney pathology [43].

ADDITIONAL INFORMATION

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

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