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RICKETS IN CHILDREN, CLINIC AND DIAGNOSTICS, MEETING TIMES

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Annotation*: Infant rickets is a disease that has existed since time immemorial, the most common among young children. In young children, rickets causes skeletal deformities and disrupts all types of metabolism, which significantly worsens the course of other diseases. In preschool and school age, hypovitaminos is D manifests itself in the form of muscle hypotension, insufficient mineralization and softening of tubular bones, and in adults-in the form of osteoporosis.*

Keywords: *rickets, vitamin D, osteoporosis.*

PEarly childhood RCC can have an adverse effect on the subsequent development of children. Osteopenia and osteomalacia observed in infantile rickets lead to the development возрасте of postural disorders, flat feet, flattening and deformity of the pelvic bones, and caries in older children. The consequences of impaired absorption of calcium, phosphorus and magnesium may include muscle hypotension, autonomic dysfunction, and gastrointestinal motility disorders. The role of rickets in the predisposition to frequent infectious diseases as a result of immune dysfunction due to a decrease in the level of interleukins, interferon, and phagocytosis indicators has been proven. Thus, further study of the pathogenetic mechanisms of rickets development is necessary to optimize the prevention and treatment of the disease and prevent its long-term consequences. Galen (131-201) was the first to describe rickety changes in the bone system, including chest deformity. In the middle Ages, rickets was called an English disease, since it was in England that the prevalence of its severe forms was noted, which was associated with insufficient sunlight in this climatic zone. In the twelfth century, during the Industrial Revolution, children born in large cities had to grow up in cramped, sun-deprived neighborhoods. This contributed to the development of a disease with a violation of the bone skeleton in the form of an increase in the epiphysis of long tubular bones, curvature of the lower limbs and spine, chest deformity, muscle weakness. It was at this time (1650) that the English orthopedist Francis, in his opinion, the main risk factors for the development of rickets in children is burdened heredity and improper nutrition of the mother. The English name "rickets" is derived from the Old English "wrickken", which means "to bend", and F. Glisson changed it to the Greek "rachitis" (disease). Spine), since rickets significantly deforms the spine. With rickets, changes in the internal organs may occur. Deformity and violation of the excursion of the chest, insufficient contraction of the diaphragm, hypotension of intercostal muscles reduce pulmonary ventilation, which predisposes to the development of respiratory diseases, and can lead to impaired hemodynamics. Therefore, children suffering from rickets are more likely to suffer from bronchitis and pneumonia. They may have tachycardia, muffled heart tones, and functional systolic murmur. Due to a violation of the secretory activity of the stomach, intestines and pancreas, unstable stools and flatulence may occur. Often there is an increase in the liver and spleen. In some children, hypochromic anemia is detected in the midst of rickets, and in rare cases severe Yaksh-Gayem anemia develops. The initial characteristic radiological symptoms of rickets are: an increase in the distance between the epiphysis and the diaphysis (zones of preliminary calcification) due to the expanding metaphysis, osteoporosis. In the future, osteoporosis increases, the metaphyseal zone expands even more, the calcification zone turns

from convex to horizontal and fringed due to unevenly collapsing bone tissue. The signs of ossification of bones in rickets appear in a timely manner, but are less clearly visible due to pronounced osteoporosis. At the height of the disease, sub-periosteal fractures of the long tubular bones and ribs characteristic of rickets are possible in the "green twig" type – fractures of the cortical layer with a slight longitudinal displacement on the concave side of the curved bone. During the convalescence period метафизах, calcium phosphate deposits in meta physes in the form of a thin, dense strip of calcification. With the recurrent course of rickets, several such calcification strips are formed, the number of which corresponds to the number of exacerbations. The earliest biochemical sign of rickets, detected even before the clinical and even radiological symptoms of this disease, is an increase in the activity of alkaline phosphatase in the blood serum. The level of alkaline phosphatase in the blood serum reflects the activity of the rickets process. There are phase changes in the concentration of calcium and phosphorus in the blood. At the beginning of the disease, as a result of hypovitaminosis D, calcium absorption in the intestine decreases and hypocalcemia develops (the norm is 2.22.8 mmol/l). At the same time, the level of phosphorus in the blood serum remains normal (0.8-1.4 mmol/ l). Hypocalcemia stimulates the production of PTH, which helps restore the level of calcium in the blood, but increases the excretion of phosphates, amino acids, bicarbonates through the kidneys, leading to the development of hyperphosphaturia, hypophosphatemia and acidosis. If vitamin D deficiency persists, the sensitivity of bones to excess PTH decreases, which leads to a decrease in the extraction of calcium from them and the reappearance of hypocalcemia. The body's supply of vitamin D is judged by the content of 25-OH-D3 in the blood, since its level is a total reflection of the endogenous formation of cholecalciferol in the skin and its intake as part of food or vitamin preparations [13]. In addition, the synthesis of this metabolite is not as tightly regulated as the formation of 1, 25-(OH)2-D3, which does not always correlate with the clinical manifestations of vitamin D deficiency. The average content of 25-OH-D3 in the blood serum of healthy people is 50-100 nmol/ l (20-40 ng/ ml) [13]. A decrease in its level below 30 ng/ ml indicates a lack of vitamin D in the body. According to this classification, when making a detailed diagnosis of the disease, its severity (I, II, III degree), period (initial, peak, convalescence, residual phenomena) and the nature of the clinical course (acute, subacute, recurrent) are evaluated (Table 2). Examples of the diagnosis formulation: "Rickets I, initial period, acute current", "Rickets I, peak period, subacute course"," Rickets I, convalescence period, subacute course"," Rickets II, peak period, acute course"," Rickets II, peak period, recurrent course","Rickets III, peak period, acute course". The period of the disease is determined by the clinical picture and biochemical changes. The initial period most often occurs at 2-3 months of life and lasts from 2-3 weeks to 2 months. The earliest clinical signs of rickets are disorders of the autonomic nervous system, and only at the end of this period do changes in the bone system appear in the form of softening of the edges of the large fontanel and pliability of bones along the sutures of the skull, primarily the arrow – shaped suture. Damage to the muscular system is manifested by hypotension of the muscles, constipation (Table 3). During a biochemical blood test, an increase in alkaline phosphatase, a slight decrease in phosphorus content, and a normal level of calcium are noted. There are no radiological changes. High season ("blooming" rickets) It is characterized by progressive damage to the nervous and bone systems. Bone changes come to the fore. All three types of changes are noted (osteomalacia, osteoid hyperplasia, and osteogenesisimperfecta), but their severity depends on the severity and course of the disease. In addition, the peak period is characterized by: distinct muscle hypotension, weakness of the ligamentous apparatus, enlarged liver and spleen, hypochromic anemia, functional disorders of other organs and systems. The number of systems involved and the severity of their changes depends on the severity of the process. Biochemical blood tests show

increased alkaline phosphatase activity and reduced levels of calcium and phosphorus. The severity of biochemical changes depends on the activity of the rickets process, the degree of vitamin D deficiency, and the production of parathyroid hormone. Radiographs show typical rickets bone changes, which were mentioned above. The convalescence period is characterized by the reverse development of rickets symptoms. The first symptoms of damage to the nervous system disappear. Then the bones are compacted, teeth appear, changes in the muscular system disappear (normalize static and motor functions), the size of the liver and spleen decreases, and previously disturbed functions of internal organs are restored. In the blood serum, the activity of alkaline phosphatase remains elevated, the concentration of calcium may remain reduced, and the level of phosphorus increases to normal values. Radiological changes in the bones undergo reverse development. During the period of residual phenomena, only the consequences of rickets in the form of bone deformities remain, which indicate that the child has suffered a severe disease (II or III degree). Deviations in laboratory parameters of mineral metabolism are not noted. Due to the subsequent processes of перемоделированиябone remodeling, which are most active after three years, deformities of tubular bones disappear over time. Deformities of flat bones decrease, but remain. In children who have had rickets, an increase in parietal and frontal tubercles, flattening of the occiput, malocclusion, deformities of the chest, pelvic bones remain.

Rickets of the first degree (mild) is characterized by a slight violation of the general condition and clinical symptoms from the nervous and bone systems involving 1-2 parts of the skeleton. Sometimes there is an unspoken hypotension of the muscles. After rickets of the first degree of residual phenomena are not noted. Rickets of the second degree (moderate) is characterized by a clear violation of the general condition and moderate changes in the nervous, bone, muscle and hematopoietic systems. With this degree of rickets, there are distinct deformities of the skull, chest and limbs, a slight increase in the liver and spleen, and moderate anemia. Grade 2 rickets (severe) is now almost nonexistent. It is manifested by significant changes in the central nervous system: sleep disorders, appetite disorders, lethargy, lagging behind in the development of speech and motor skills. Changes in the bone system are characterized by multiple, clearly pronounced deformities (softening of the bones of the base of the skull, sinking of the bridge of the nose, "Olympic" forehead, gross deformation of the chest, limbs, pelvic bones). Fractures of bones without displacement or with angular displacement are possible. There are pronounced changes in the muscular system (violation of static functions). The liver and spleen are significantly enlarged, there are distinct functional disorders of the cardiovascular system, respiratory organs, gastrointestinal tract, and severe anemia. The acute course of the disease is manifested by a rapid increase in symptoms (neurological changes in muscle hypotension), the predominance of osteomalacia processes over the processes of osteoid hyperplasia. This course of rickets is observed more often in the first half of life in children who are overweight at birth or in infants with a large monthly weight gain who do not receive vitamin D prophylaxis. The subacute course of rickets is characterized by a slower development of the disease. Symptoms of osteoid hyperplasia predominate: frontal and parietal bumps, "rosaries" on the ribs," bracelets","threads of pearls". Kraneotabes not typical. Such a course of the disease is more common after 6 months in children born prematurely or with a delay in intrauterine development, as well as with ineffective prevention dueto insufficient amounts of vitamin D. The recurrent course of the disease is established if periods of improvement in the condition of a child with rickets are replaced by periods of exacerbation of the rickets process. This may be due to the interruption of treatment, the presence of concomitant diseases, poor nutrition, or other newly emerged risk factors. On radiographs of bones, this process is reflected by the formation of ossification bands in the bone growth zone, the number of which corresponds to the number of

exacerbations. There are prenatal and postnatal, specific and non-specific prevention of rickets.

Antenatal prevention of rickets should begin long before delivery. Adequate formation of endogenous reserves of vitamins and minerals at the time of birth of a child can occur only if they are sufficiently absorbed into the mother's body. Therefore, an important task of preventing rickets in an infant is to organize a rational diet and regime of the expectant mother. A pregnant woman should take walks in the fresh air for at least 2-4 hours daily, in any weather, observe the daily routine, and sleep enough day and night. To replenish the body of a pregnant woman with all vitamins and trace elements, a rational diet is recommended. The Department of Medical and Social Problems of Family, Motherhood and Childhood of the Ministry of Health and Social Development of the Russian Federation approved in 2006 the recommended food intake standards developed by the State Research Institute of Nutrition of the Russian Academy of Medical Sciences for pregnant women. Every day a pregnant woman should consume at least 170 g of meat, 70 g of fish, 50 g of cottage cheese, 15 g of cheese, 220 g of bread, 500 g of vegetables, 300 g of fresh fruit, 150 g of juices, 0.5 liters of milk and fermented dairy products, 25 g of butter, 15 g of vegetable oil. Instead of milk, you can use special milk drinks intended for women during pregnancy and lactation ("Dumil mama", "Femilak", etc.). In the absence of these special milk drinks, you can recommend taking multivitamins that include vitamin D during pregnancy and the entire lactation period. The daily requirement of a pregnant woman for vitamin D is 400-500 IU. Pregnant women at risk (with an unfavorable course of pregnancy, chronic extragenital infections, especially liver and kidneys, diabetes mellitus and symptoms of hypocalcemia) in the third trimester of pregnancy may be recommended to take additional vitamin D until the daily dose of 1000 IU is reached, regardless of the time of year. Pregnant women over the age of 35 years in the absence of signs of hypocalcemia Additional vitamin D supplementation is not recommended because of the risk of excessive calcium deposition in the placenta and the development of fetal hypoxia. The increased need for calcium during pregnancy is met by significantly increasing its absorption in the intestines of the expectant mother. But if a woman has been diagnosed with hypocalcemia before pregnancy, she can be recommended an additional intake of 300 mg of calcium daily for the entire duration of pregnancy and lactation. It is recommended to take calcium supplements simultaneously with dairy products. Postnatal non-specific prevention of rickets provides for the rational formation of the child's daily routine and the organization of his proper nutrition. If the infant has malabsorption syndrome, pathology of the biliary tract and kidneys, adequate correction of these disorders is necessary.

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