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# Peculiarities of the Development of Rickets in Children

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**Abstract:** Infantile 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, hypovitaminosis 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:** hypovitaminosis D, children, rickets, osteomalacia, climatic zone, hypovitaminosis.

According to Professor G.N. Speransky, despite the huge number of works devoted to the history, etiology, pathogenesis, clinic, long-term consequences of this disease, interest in it does not weaken, many aspects of it have not yet been finally identified.

In addition, rickets suffered in early childhood can adversely affect the subsequent development of children. Osteopenia and osteomalacia, observed in infantile rickets, lead to the development of posture disorders, flat feet, flattening and deformation of the pelvic bones, and caries in older children. The consequences of violations of the absorption of calcium, phosphorus and magnesium in them may be muscle hypotension, autonomic dysfunction, dysmotility of the gastrointestinal tract. 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 the development of rickets is necessary to optimize the prevention and treatment of the disease and prevent its long-term consequences.

The first mention of rickets is found in the writings of Soranus of Ephesus (98-138), who observed the deformation of the lower extremities and spine in children and explained it by the early onset of walking. Galen (131-201) was the first to describe rachitic changes in the skeletal system, including deformity of the chest.

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 insolation in this climatic zone. In the 12th century, during the Industrial Revolution, children born in large cities had to grow up in cramped, sunlight-deprived neighborhoods. This contributed to the development of a disease in them with a violation of the bone skeleton in the form of an increase in the epiphyses of long bones, curvature of the lower extremities and spine, deformity of the chest, muscle weakness. It was at this time (1650) that the English orthopedist Francis Glisson made a complete clinical and pathoanatomical description of rickets in De rachitide sive morbo puerile, qui vulgo. The rickets, dicitur, tractatus." In his opinion, the main risk factors for the development of rickets in children are aggravated heredity and maternal malnutrition. The English name "rickets" comes from the Old English "wrickken", which means "to bend", and F. Glisson changed it to the Greek "rachitis" (disease of the spine), since rickets significantly deforms the spine.

In Russia, rickets began to be intensively studied in the 19th century. In 1847, in the book Pediatrics, Professor S.F. Khotovitsky described not only the damage to the skeletal system in rickets, but also

changes in the gastrointestinal tract, autonomic disorders, and muscle hypotension. In the treatment of rickets, he paid the main attention to hygienic measures and the organization of proper nutrition. In 1891 N.F. Filatov noted that rickets is a common disease of the body, although it manifests itself mainly as a kind of change in the bones. He was fully supported by A.A. Kissel. The works of I.A. are devoted to rickets. Shabady, V.P. Zhukovsky, G.N. Speransky, A.F. Tura, E.M. Lepsky, M.N. Bessonova, K.A. Svyatkina and others.

In 1822, J. Sniadecki, based on the fact that children born in Warsaw are more likely to suffer from rickets than infants living in the countryside, suggested that sunlight may play a role in preventing this pathology. In 1980 T.A. Palm published the results of a study indicating a significantly higher incidence of rickets in children living in the large industrial centers of the British Isles compared to children living in poor cities in China, Japan and India. This allowed him to conclude that it is necessary to systematically use sunbathing for the prevention and treatment of rickets [3]. In 1919, K. Huldschinsky first showed that this disease is cured under the influence of the rays of the "artificial mountain sun" (quartz lamp). Around the same time, E. Mellanby discovered the anti-rachitic effect of fish oil, suggesting that it is due to the presence of some kind of vitamin in it. Later, this vitamin was named vitamin D. In 1924, A. Hess was the first to obtain vitamin D from vegetable oils, and in 1937, A. Windaus synthesized it from 7-dehydrocholesterol.

Since then, many researchers began to equate the concepts of "rickets" and "hypovitaminosis D", but as new data accumulated, many researchers began to point out that rickets and hypovitaminosis D are not unambiguous concepts [10].

According to modern concepts, rickets is a disease characterized by a temporary discrepancy between the needs of a growing organism for phosphorus and calcium and the insufficiency of systems that ensure their delivery to the body [8]. This is a disease of a growing organism, caused by a metabolic disorder (primarily phosphorus-calcium metabolism), the main clinical syndrome of which is damage to the skeletal system (impaired formation, proper growth and mineralization of bones), in which the pathological process is localized mainly in the area of bone metaepiphyses . Since the growth and rate of bone remodeling are highest in early childhood, lesions of the skeletal system are most pronounced in children of the first 2-3 years of life. Rickets is a multifactorial metabolic disease, in the diagnosis, prevention and treatment of which all factors of pathogenesis should be taken into account: insufficiency and imbalance of calcium and phosphorus intake with food, immaturity of the child's endocrine system, concomitant diseases, etc. In addition to the pathology of phosphorus-calcium metabolism, disturbances in protein metabolism and trace elements (magnesium, copper, iron, etc.), multivitamin deficiency, activation of lipid peroxidation.

It is now important to understand rickets as a metabolic disorder, and not just as a D-deficient condition. According to ICD-10, rickets does not belong to the section of hypo- and avitaminosis, but to the section of diseases of the endocrine system and metabolism (ICD-10 code - E55.0. Active rickets).

Rickets occurs in all countries, but is especially common among northern peoples who live in conditions of lack of sunlight. Children born in autumn and winter suffer from rickets more often and more severely. The disease is most severe in premature infants [11].

In countries, including ours, where specific prevention of rickets with vitamin D and fortification of baby food products have been introduced, severe forms of rickets have become rare, but its subclinical and radiological manifestations remain widespread. Therefore, rickets still occupies a significant place in the structure of morbidity in young children. The high frequency of the disease, despite a fairly active prevention, requires a revision of existing views on the etiology, pathogenesis of rickets and methods of its treatment.

The main etiological factor of rickets is vitamin D deficiency and a violation of its conversion into active forms.

Vitamin D enters the human body in two ways: with food and as a result of synthesis in the skin under the influence of ultraviolet radiation.

Vitamin D3 (cholecalciferol) is found in the greatest amount in cod liver, tuna and fish oil, to a lesser extent in butter, egg yolk, milk, cottage cheese. Plant products (vegetable oils, wheat germ) contain its analogue - ergocalciferol (vitamin D2). Absorption of vitamin D occurs mainly in the duodenum and jejunum in the presence of bile acids.

Photosynthesis of vitamin D3 in the skin comes from 7-dehydrocholesterol (provitamin D3). This process is tightly regulated, due to which the development of hypervitaminosis D as a result of prolonged exposure to sunlight is impossible. All cholecalciferol, which is formed in the skin and does not enter the systemic circulation, is transformed into inactive compounds with further irradiation.

It has been shown that for the synthesis of the required level of vitamin D in the skin, it is enough for a child to be undressed in the sun for 30 minutes a week.

The effectiveness of vitamin D synthesis in human skin is significantly affected by the geographic latitude of the area, the level of air pollution, and the degree of skin pigmentation.

The formation of vitamin D is significantly lower in regions located north of 35 latitude in the northern hemisphere from November to March and south of 32 latitude in the southern hemisphere from May to August, because. at this time, the angle of incidence of solar radiation is such that it does not provide a sufficient content of UV-B photons in the spectrum of sunlight [14].

Dark-skinned people require longer UV exposure than white-skinned people to synthesize the same amount of vitamin D, since melanin competes with 7-dehydrocholesterol for UV-B photons [6,14]. Under conditions of calcium and phosphorus deficiency in the body, under the influence of the a-1-hydroxylase enzyme, 1,25-dihydroxycholecalciferol (calcitriol) is formed from 25-hydroxycholecalciferol. It is the hormonally active form of vitamin D. It increases serum calcium concentration by enhancing its absorption from the intestine and reabsorption in the kidneys, as well as through the resorption of calcium from the bones. Renal production of calcitriol is stimulated by parathyroid hormone and by low serum calcium and phosphorus levels. Its production decreases with an increase in the serum concentration of the metabolite itself by a negative feedback mechanism.

At normal or elevated concentrations of calcium and phosphorus in the blood serum, the activity of the enzyme 24-hydroxylase increases, under the action of which 24,25-dihydroxycholecalciferol is formed from 25-hydroxycholecalciferol. This vitamin D metabolite provides fixation of calcium and phosphates in bone tissue, promoting bone mineralization, and suppresses the secretion of parathyroid hormone [12].

The biological role of vitamin D is associated with its participation in the processes of calcium and phosphorus metabolism [7]. The concentration of calcium in the blood serum is a constant value and is 2.25-2.7 mmol / l. Normally, the concentration of calcium and phosphorus is maintained in a ratio of 2: 1, which is necessary for the proper formation of the skeleton. Calcium in the blood is in two forms - ionized and protein-bound.

Vitamin D is the most important regulator of phosphorus-calcium metabolism. Calcitriol promotes calcium absorption in the intestine by increasing the production of calcium-binding protein, and stimulates the reabsorption of calcium and phosphorus in the kidneys, which leads to an increase in the content of these elements in the serum to a level that provides adequate osteoid mineralization. In the bones under the action of calcitriol, there are two, on the one hand, multidirectional, and on the other hand, interconnected processes. Calcitriol activates the corresponding osteoblast genes, which leads to an increase in the synthesis of osteocalcin, osteopontin, collagen, which are necessary for the mineralization and functioning of the newly formed bone. At the same time, osteoblasts stimulate osteoclast genesis. Osteoclasts carry out bone resorption, providing an increase in serum levels of calcium and phosphorus.

Along with calcitriol, the main regulators of calcium and phosphorus metabolism in the body are parathyroid hormone (PTH), produced by the parathyroid glands, and calcitonin, a C-cell hormone of the thyroid gland [5].

The third major regulator of phosphorus-calcium metabolism is calcitonin. The secretion of calcitonin increases with an increase in the concentration of calcium in the blood and decreases with its decrease. Calcitonin is a powerful parathyroid hormone antagonist. Its action is aimed at eliminating hypercalcemia. It enhances the deposition of calcium in the bones, preventing the development of osteomalacia and osteoporosis, and also reduces the reabsorption of calcium in the renal tubules.

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