Features Of Rickets In Children: Diagnosis, Causes, Treatment

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ABSTRACT

The article outlines modern views on the frequency and causes of rickets in young children. Clinical, radiological and laboratory signs of the disease, classification of rickets, as well as its main forms - calcium deficiency, phosphate deficiency and vitamin D deficiency are presented. Diagnostic and differential diagnostic criteria are given. Particular attention is paid to issues of nonspecific and specific prevention, as well as treatment.

Keywords: young children, the role of vitamin D, phosphorus-calcium metabolism disorders, rickets, prevention, treatment.

Introduction

Bone tissue is a dynamic system in which, throughout a person's life, the processes of destruction of old bone and formation of new one simultaneously occur, which constitutes the cycle of bone tissue remodeling.

In childhood, bone undergoes particularly intense remodeling. In the first months and years of life, along with the rapid growth of the bone skeleton, multiple restructuring of the bone tissue structure occurs - from a coarse fibrous structure to lamellar bone with secondary Haversian structures.

Materials And Methods

Phosphates, in addition to participating in the formation of bone tissue, also play an important role in many parts of cellular metabolism. In accordance with the nutritional standards adopted in our country (SanPiN 2.3.2.2401-08), the recommended amount of phosphate is: up to 300 mg/day at 1–3 months, up to 500 mg/day at 7–12 months, for children from 2 to 3 years old - 800 mg/day, from 4 to 17 years old - 1450–1800 mg/day, for adult men and women - 1200 mg/day, for pregnant and nursing mothers - 1500 mg/day.

The main form of vitamin D circulating in the blood is its intermediate metabolic product, 25-hydroxycholecalciferol (25-OH D3), which is formed in the liver [1]. Then this metabolite in the proximal tubules of the kidneys under the action of hydroxylases is transformed into final products, the main of which are 1,25- and 24,25-dioxycholecalciferol. Both metabolites - 1,25-(OH)2D3 and 24,25-(OH)2 D3 - activate the processes of differentiation and proliferation of chondrocytes and osteoblasts, as well as the production of osteocalcin, the main non-collagenous bone protein. It is synthesized by osteoblasts and is considered a sensitive indicator of bone formation. It is 1,25-(OH)2 D3, together with parathyroid hormone and thyrocalcitonin, that ensure calcium phosphorus homeostasis, the processes of mineralization and bone growth. And it is possible that it is not so much an exogenous deficiency as congenital and acquired dysfunctions of the intestine (absorption), liver and kidneys (metabolism) that make a

significant contribution to the development of endogenous hypovitaminosis D.

Results And Discussion

Rickets is a disorder of the mineralization of growing bone, caused by a temporary discrepancy between the needs of the growing body for phosphates and calcium and the insufficiency of the systems that ensure their delivery to the child's body [2]. In this case, the differentiation of chondrocytes and the mineralization of the growth zone are disrupted [3], the child's skeletal system develops signs of osteomalacia or osteoid hyperplasia, the typical X-ray picture of rickets is mainly a violation of the mineralization of the metaphyses (metaphyseal rickets).

The clinical picture of the so-called classic rickets cannot be unambiguously considered a manifestation of exogenous hypovitaminosis D, as was previously accepted. Rickets and hypovitaminosis D are ambiguous concepts, and the development of bone signs of rickets in young children is due to rapid growth rates, high rates of skeletal modeling and a deficiency of phosphates and calcium in the growing body with imperfect ways of their transport, metabolism and utilization (heterochrony of maturation) [4]. Therefore, it is no coincidence that rickets is now often classified as a borderline, transient condition in young children. Sometimes, as in the past, it continues to be called nutritional [3, 5] or infantile rickets [5].

Factors contributing to the development of rickets in children are:

1. High rates of growth and development of children at an early age and an increased need for mineral components, especially in premature infants.
2. Deficiency of calcium and phosphates in food associated with nutritional defects.

The exchange of calcium and phosphates is of particular importance in the antenatal period of life, if a woman does not consume dairy products for various reasons (vegetarianism, allergy to milk proteins, lactase deficiency, etc.), with dietary restrictions on meat, fish, eggs, with an excess of fiber, fat in the diet, or taking enterosorbents. In breast milk, the calcium content ranges from 15 to 40 mg/dL, and children in the first months of life receive from 180 to 350 mg of calcium per day [2]. At the same time, the required amount of calcium for children during the first 6 months is at least 400 mg per day. From the age of 2, according to our data, only half of children 2–3 years old regularly receive cottage cheese and whole milk in their diet (51% and 44%, respectively). The phosphate content in breast milk ranges from 5 to 15 mg/dl, and children in the first months of life receive from 50 to 180 mg of phosphates per day. For children in the first half of the year, the required amount of phosphates should be at least 300 mg. A deficiency of calcium and phosphates in the diet and a violation of their ratio are possible if the principles of rational nutrition are not observed in children who are bottle-fed, or if natural feeding is prolonged [4].

The causes of calcium deficiency and the development of a calcium deficiency state are, first of all, a lack of calcium in food, especially with long-term feeding of children with breast milk, and impaired calcium absorption due to malabsorption syndrome.

Phosphate deficiency rickets can develop not only due to insufficient intake of phosphates as the main cause of osteopenia in premature infants, but also with an increased need for phosphates in conditions of rapid growth, impaired absorption in the intestine, and also in children on total parenteral nutrition. Currently, disorders of phosphate metabolism are largely associated with changes in fibroblast growth factor 23 (FGF23), extracellular matrix phosphoglycoprotein and other metabolites known as phosphatonins [2]. It is the deficiency of phosphates, and not calcium or vitamin D, that correlates with the degree of rachitic bone changes and the degree of radiological signs of rickets [3]. According to P. Lapatsanis et al. [4], rickets, caused primarily by vitamin D deficiency, has minimal bone manifestations, in contrast to hypophosphatemic rickets.

Exo- or endogenous vitamin D deficiency as a modulator of phosphate and calcium metabolism also leads to rickets. In case of vitamin D deficiency, secondary disorders of

calcium and phosphate metabolism are possible, which determine the nature of clinical, biochemical and radiological manifestations.

For effective treatment of rickets, a set of measures is prescribed to normalize the child's lifestyle, with sufficient insolation, and provide him with a nutritious, balanced diet in accordance with his age. When artificially feeding, the use of adapted formulas containing larger amounts of phosphates and calcium compared to breast milk makes it possible to compensate for the deficiency of phosphates and calcium in the diet of such children. Vitamin D therapy is prescribed, which, by influencing target organs, helps to optimize phosphate-calcium metabolism. The selection of doses of vitamin D is carried out in accordance with the characteristics of the clinical picture of rickets, the degree of its severity and the dynamics of the disease. To treat rickets, various vitamin D preparations are used, most often Aquadetrim.

Conclusion

If there is no effect from the indicated therapeutic doses of vitamin D, the diagnosis should be clarified through a more in-depth examination in a hospital, since we can talk about hereditary forms of rickets, called rickets-like diseases, genetically determined osteopathies or vitamin D-resisting diseases. stent forms of rickets. These are hereditary hypophosphatemic vitamin D-resistant rickets, renal tubular acidosis, Fanconi-Debreu-de-Toni disease, vitamin D-dependent rickets, etc.

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