

RICKETIS IN CHILDREN: CLINICAL PICTURE, DIAGNOSIS AND TREATMENT

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ABSTRACT

Rickets is a disease that has been known to mankind for many centuries. It is known that vitamin D plays a significant role in the regulation of calcium and phosphorus homeostasis and, consequently, in the development of rickets. The main etiological factor of rickets is considered to be insufficient exposure of children to ultraviolet sunlight. This article presents the clinical picture, diagnostics and treatment of rickets.

KEYWORDS: rickets, children, calcium, bone deformities, diagnosis, treatment.

INTRODUCTION

Rickets was first described in 1645 by Whistler (England), and in detail by the English orthopedist Gleason in 1650, although this disease was mentioned in the works of Galen (131-211 AD). In 1918, Melanby [English] demonstrated in an experiment on dogs that cod oil acts as an anti-rachitic agent due to the presence of a special vitamin in it. For some time, it was believed that the anti-rachitic activity of cod oil depended on vitamin A, which was already known at that time. McCollum, passing a stream of oxygen through cod oil and activating vitamin A with it, discovered that the anti-rachitic effect of the fat was preserved after this. During further searches, another vitamin with a strong anti-rachitic effect was found in the unsaponifiable part of cod oil - vitamin D [1-4].

It was finally established that food substances have the property of preventing and curing rickets mainly depending on the greater or lesser content of vitamin D in them. In 1919, K. Galchinsky (German: Kurt Huldshinsky) discovered the effective action of a mercury-quartz lamp (artificial "mountain sun") in the treatment of children with rickets. Since that time, the main etiological factor of rickets is considered to be insufficient irradiation of children with ultraviolet sunlight.

CLINICAL PICTURE OF RICKETIS

Up to 3 months of age, the disease practically does not manifest itself, and quite distinct clinical symptoms are formed no earlier than in the second half of the year [1-5].

Parents should be wary if the child:

- becomes irritable, is frightened by sharp sounds or a flash of a switched-on lamp;
- sweats profusely, and the scalp often gets wet, sticky sweat with a sour smell irritates the skin and causes itching, the child often scratches his head;
- muscle tone is reduced, so the child is delayed in physical development - later than peers, begins to sit, crawl and stand on his feet;
- when palpating, a large fontanelle with soft edges is felt.

In severe rickets, symptoms of the disease in children are manifested in the form of deformation of bone tissue. In a sick child:

- crooked legs, knees are turned outward or brought together;
- sunken sternum;

- flattened occiput;
 - protruding parietal and frontal tubercles, bald spots appear on the forehead;
 - teeth do not erupt for a long time and the fontanelle does not close.
- The disease affects the functioning of internal organs, which manifests itself as:
- digestive dysfunction, stool instability, decreased weight;
 - anemia;
 - decreased immunity, frequent colds and respiratory infections.

The listed symptoms may indicate not only rickets, but also a number of other diseases, so laboratory tests are necessary to confirm the diagnosis [7-14].

Most clinical manifestations in children with rickets are due to the effect of vitamin D deficiency on the process of mineralization of growing bones or on calcium homeostasis. D. Fraser et al. [5] described three stages of progression of vitamin D deficiency. Stage I is characterized by hypocalcemia with clinical features related to the presence of hypocalcemia, in stage II abnormal bone mineralization becomes evident, and in stage III features of both hypocalcemia and severe rickets are present. This division of the progression of rickets and vitamin D deficiency is conceptually useful, but there is considerable clinical overlap between the different stages. Characteristic clinical manifestations of rickets - bone complications of the disease, which are mandatory for diagnosis:

- protrusion of the frontal tubercles,
- craniotabes (softening of the skull bones, usually detected by palpation of the frontal sutures in the first 3 months of life),
- late closure of the large fontanelle (normally closes by 2 years),
- late eruption of teeth,
- deformations of the lower limbs (O-shaped / X-shaped curvature of the legs),
- rachitic rosary,
- widening, flattening or concavity, saucer-shaped deformation, roughness of the surface and trabecularity of the metaphyses,
- deformations of the pelvis, including narrowing of the outlet of the small pelvis,

In severe rickets, a lag in weight gain and growth, delayed formation of motor skills and muscle hypotonia, anxiety and irritability may be observed. The detection of symptoms of dysfunction of the autonomic nervous system (sweating, anxiety, irritability) is currently not a basis for making a diagnosis, as was previously accepted [6-10].

COMPLICATIONS OF RICKETIS

Extraosseous complications of rickets

Hypotonia, decreased motor activity, and protuberant abdomen are characteristic of progressive rickets in infants and young children. Delayed tooth eruption is a feature of rickets in the young child, and enamel hypoplasia may occur if rickets develops before enamel formation is complete. The latter has been noted in primary teething children born to vitamin D-deficient mothers and in secondary teething children who suffered from rickets in early childhood [11-15].

Immune complications of rickets

Infants and young children with rickets are prone to an increased number and severity of various infections, especially acute respiratory viral infections and influenza [15,23]. The increase in respiratory infections can be explained by chest wall abnormalities (rib softening, enlarged costochondral junctions, and decreased respiratory movements due to muscle

weakness), but various studies have shown a direct role for vitamin D in modulating immune function [16,17,19,26]. Impaired phagocytosis and neutrophil motility have been described in children with vitamin D-deficient rickets [18]. There is growing evidence that vitamin D may play an important role in modifying the risk of developing various autoimmune diseases, such as type 1 diabetes, multiple sclerosis, and rheumatoid arthritis, etc. [20-25].

DIAGNOSIS OF RICKETIS

Laboratory diagnostics of rickets includes blood and urine tests to determine calcium and phosphorus levels, as well as an analysis of alkaline phosphatase, a specific enzyme involved in the transfer of calcium and phosphorus. The dynamics and ratio of these indicators allow us to clarify the period of the disease. X-rays of tubular bones are taken to assess skeletal deformities. A more accurate assessment of bone tissue condition is provided by computed tomography and densitometry.

In a biochemical blood test for alimentary rickets, a decrease in PTH, phosphorus and calcium in the blood serum, a decrease in calcium and an increase in phosphorus in the urine can also be detected.

Of all the available biochemical indicators that can be changed in rickets, alkaline phosphatase has been most often used as a screening test.

TREATMENT OF RICKETIS

Currently, treatment of rickets is comprehensive. It includes:

- organizing a daily routine with daily long walks in the fresh air;
- providing adequate nutrition that satisfies the child's need for calcium and other minerals;
- drug therapy - taking vitamin D in a dosage sufficient to replenish the deficiency;
- non-drug therapy - massage, therapeutic exercises, ultraviolet baths, balneotherapy.

Treatment of vitamin rickets should be comprehensive, long-term and aimed at both eliminating the causes that caused it and eliminating hypovitaminosis D.

Most often in pediatric practice for the treatment and prevention of rickets, a water-soluble form of vitamin D3 (Akvadtrim), developed by the company "Terpol" (Poland) is used.

The advantages of an aqueous solution of vitamin D3 (Akvadtrim) are (N.A. Korovina et al., 2003):

- better absorption from the digestive tract (an aqueous solution of vitamin D3 is absorbed 5 times faster, and the concentration in the liver is 7 times higher);
- less strain on the intestinal enzyme systems is required for absorption, especially in premature babies given their immaturity;
- a longer-lasting effect when using an aqueous solution (lasts up to 3 months, and an oil solution - up to 4-6 weeks);
- higher activity;
- rapid onset of clinical effect (5-7 days after the appointment of D3 and 10-14 days when taking D2);
- convenience and safety of the dosage form.

Akvadtrim (an aqueous solution of vitamin D3) is available in 10 ml bottles with a special pipette and contains 500 IU in 1 drop. Taking into account the severity of the pathological process, its degree of severity and the nature of the course of vitamin D-deficiency rickets, vitamin D3 is prescribed at 2500-5000 IU per day for 30-45 days. After achieving a



therapeutic effect, they switch to a prophylactic dose (400-500 IU per day), which the child receives for 3 years. Children at risk are sometimes treated against relapse of the disease 3 months after the main course of treatment.

Non-specific treatment includes organizing a protective regimen corresponding to the patient's age, eliminating loud noise, bright light, and additional irritants. It is necessary to ensure that the child spends a long time outdoors during the daytime with stimulation of active movements. Conducting therapeutic exercises and massage, hygienic procedures (bath, rubbing) are of great importance.

The diet is recommended in accordance with the age, needs of the child and is corrected taking into account existing deficiencies. For this purpose, a 3-4 month old child who is breastfed is given vegetable and fruit decoctions and juices instead of drinking, and yolk and cottage cheese are prescribed earlier. With mixed and artificial feeding, vegetable complementary foods should be prescribed at an early age, and the amount of milk, kefir and porridge should be limited.

If clinical recommendations are followed, rickets is cured fairly quickly and without long-term consequences, but only if measures were taken at the initial stages of the disease.

In severe forms of the disease, it is not always possible to overcome skeletal deformation, delays in physical development, and the negative impact of the disease on the nervous system are not without consequences. For three years after recovery, the child remains under dispensary observation with quarterly medical examinations.

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