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RICKETIS IN CHILDREN: CAUSES, CLINICAL PICTURE, DIAGNOSIS, TREATMENT

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ABSTRACT

Rickets occurs during a period of intensive growth and development of the child. The main cause of rickets in children is a lack of vitamin D. Diagnosis of rickets is based on anamnesis, clinical picture of the disease, and biochemical blood tests. To assess skeletal deformities, X-rays of long bones are taken. Computed tomography and densitometry provide a more accurate assessment of the condition of bone tissue. Treatment of vitamin rickets should be comprehensive, long-term and aimed both at eliminating the causes that caused it and at eliminating hypovitaminosis D.

KEYWORDS

Rickets, children, clinic, diagnosis, treatment, prevention.

Introduction

Rickets is a common pathology among newborns and children in the first years of life, which is based on a lack of minerals for the formation of bone tissue [1-10]. Rickets occurs during a period of intensive growth and development of the child. This pathology is a growth disease, as it mainly affects children aged from two months to three years [11-16]. The disease is diagnosed in 55-65% of full-term newborns, and among premature infants its number reaches 80%. Rickets is a polyetiological disease of infants and young children associated with insufficient intake of vitamin D in the body, which leads to disruption of various metabolic processes, primarily phosphorus-calcium metabolism, which contributes to damage to many organs and systems, and mainly the bone skeleton. This definition allows us to consider rickets as a nosological entity, which reflects its etiological and pathogenetic aspects.

CAUSES OF THE DISEASE

The main cause of rickets in children is a lack of vitamin D. This substance performs two important functions: it helps the absorption of calcium in the small intestine and reduces its leaching in the kidneys. Despite their relatively small size and body weight, during this period, children need amounts of vitamin D 5-6 times greater than the amounts required by the adult body [17-23].

The causes of hypovitaminosis D are: exogenous and endogenous.

Exogenous causes of hypovitaminosis D are:

1) insufficient intake of vitamin D from food, due to the lack of foods rich in vitamin D in the diet (egg yolk, cod fat, fish and bird liver, caviar, milk, butter, etc.);

2) insufficient insolation, i.e. insufficient exposure of infants to fresh air, which leads to disruption of the formation of vitamin D3 from 7-dehydrocholesterol in the epidermis under the influence of ultraviolet rays.

Endogenous causes of hypovitaminosis D are:

1) disruption of the absorption of vitamin D in the intestine, which is observed in intestinal malabsorption syndrome, biliary obstruction and other pathological conditions;
2) disruption of the processes of hydroxylation of inactive forms of vitamin D into its active forms (vitamin D3) in the liver and kidneys, which may be associated with chronic diseases of these organs, as well as with genetic disorders of the processes of synthesis of vitamin D3;
3) impaired absorption of Ca and P in the intestine, their increased excretion in the urine and impaired utilization of bone tissue;
4) impaired functional activity or absence of receptors for vitamin D3.

RISK GROUPS FOR THE DEVELOPMENT OF VITAMIN D DEFICIENCY RICKETS

- premature babies;
- children born with intrauterine malnutrition;
- born with signs of morphofunctional immaturity;
- children with impaired intestinal absorption syndrome (cystic fibrosis, celiac disease, exudative enteropathy, etc.);
- children with convulsive syndrome receiving treatment with anticonvulsants;
- with reduced motor activity (paresis and paralysis, etc.);
- with chronic pathology of the liver, biliary tract;
- frequently ill children;
- children receiving poor nutrition;
- with a family history of impaired phosphorus-calcium metabolism;
- from twins or from repeated births with short intervals between them.

It is known that vitamin D enters the child’s body in two ways: with food and as a result of synthesis in the skin under the influence of ultraviolet rays.

Vitamin D comes in small quantities from food, but its main share (up to 80%) is produced in the layers of the epidermis under the influence of solar ultraviolet radiation. Vitamin D comes through the intestines in an inactive form, and to participate in calcium metabolism it must be activated by passing through the liver and kidneys.

Most often, signs of rickets are observed in children living in large cities with unfavorable environmental conditions.

In dysfunctional and low-income families, children with rickets are more common than in families that do not experience financial difficulties. In addition, risk factors for development are:

• mother's age: either too young (under 18 years old) or mature (after 40 years old);
• limited maternal exposure during pregnancy;
• insufficient nutrition of the mother during pregnancy, unreasonable dieting, lack of physical activity;
• chronic diseases of the mother;
• third and subsequent pregnancies;
• premature pregnancy;

• birth of twins;
• frequent births (less than two years between births);
• artificial feeding of a child with incomplete absorption of necessary substances from the nutritional mixture;
• incorrect nutritional choices that do not correspond to the child's age;
• underfeeding or overfeeding;
• intrauterine development disorders;
• diseases of the nervous system;
• digestive disorders, insufficient absorption of nutrients in the intestines.

Boys suffer from rickets more often than girls, dark-skinned children more often than light-skinned children. In addition, the child's second blood group is an additional risk factor.

CLINICAL PICTURE

Until 3 months of age, the disease practically does not manifest itself, and quite distinct clinical symptoms do not form earlier than in the second half of the year.

Parents should be wary if their child:

• becomes irritable, frightened by sharp sounds or the flash of a switched-on lamp;
• sweats profusely, and the scalp often becomes moist, 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, he begins to sit, crawl and stand on his feet;
• when palpated, a large fontanel with soft edges is felt.

In severe forms of rickets, the symptoms of the disease in children manifest themselves in the form of deformation of bone tissue. In a sick child:
• legs are bent, knees are turned outward or brought together;
• sunken sternum;
• flattened occiput;
• parietal and frontal tubercles protrude, bald patches appear on the forehead;
• teeth do not erupt for a long time and the fontanel does not close.

The disease affects the activity of internal organs, which manifests itself as:
• digestive dysfunction, stool instability, decreased weight;
• anemia;
• reduced 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.

DIAGNOSIS

Laboratory diagnosis of rickets includes blood and urine tests to determine the level of calcium and phosphorus, as well as an analysis for alkaline phosphatase, a specific enzyme involved in the transfer of calcium and phosphorus. The dynamics and ratio of these indicators make it possible to clarify the period of the disease.

To assess skeletal deformities, X-rays of long bones are taken. Computed tomography and densitometry provide a more accurate assessment of the condition of bone tissue.

TREATMENT

Currently, the treatment of rickets is complex. 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 compensate for the deficiency;
• non-drug therapy – massage, therapeutic exercises, ultraviolet baths, balneotherapy.

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

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

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

- better absorption from the digestive canal (an aqueous solution of vitamin D3 is absorbed 5 times faster, and the concentration in the liver is 7 times higher);
- absorption requires less tension in the intestinal enzyme systems, especially in premature infants, given their immaturity;
- 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 taking D3 and 10–14 days when taking D2);
- convenience and safety of the dosage form.

Aquadetrim (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 severity and the nature of the course of vitamin D-deficient 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.

Nonspecific treatment includes the organization of a protective regime appropriate to the patient's age, with the elimination of loud noise, bright light, and additional irritants. It is necessary to ensure that the child spends a long time in the fresh air during the daytime with stimulation of active movements. Carrying out therapeutic exercises and massage, hygiene procedures (bath, rubdown) are of great importance.

The diet is recommended in accordance with the age and needs of the child and is adjusted 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; earlier, yolk and cottage cheese are prescribed. 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 quickly enough and without long-term consequences, but only if measures were taken in 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, as well as the negative impact of the disease on the nervous system, are not

without consequences. For three years after recovery, the child remains under clinical supervision with quarterly medical examinations.

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