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Principles of Rational Treatment of Rickets in Children

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Abstract: Rickets is a disease of a rapidly growing organism, characterized by a violation of mineral metabolism and bone formation. Rickets is manifested by numerous changes in the musculoskeletal system (softening of the flat bones of the skull, flattening of the back of the head, deformation of the chest, curvature of the tubular bones and spine, muscle hypotonia, etc.), the nervous system and internal organs. The diagnosis is made on the basis of laboratory and radiological signs of rickets. Specific therapy for rickets includes the use of vitamin D in combination with therapeutic baths, massage, gymnastics and ultraviolet radiation.

Keywords: Pathogenesis of rickets, Causes of rickets, Classification of rickets, Symptoms of rickets, Diagnosis of rickets, Treatment of rickets, Prognosis and prevention

Introduction

Rickets is a polyetiological metabolic disease, which is based on the imbalance between the child's body's need for minerals (phosphorus, calcium, etc.) and their transport and metabolism. Since rickets mainly affects children aged 2 months to 3 years, in pediatrics it is often called "a disease of the growing organism." In older children and adults, the terms osteomalacia and osteoporosis are used to describe this condition.

The prevalence of rickets (including its mild forms) is 54-66% among full-term infants and 80% among premature infants. Most children aged 3-4 months have 2-3 mild symptoms of rickets, so some pediatricians suggest considering this condition as a paraphysiological, borderline (diathesis-like - constitutional anomaly), which is eliminated spontaneously with the maturation of the body.

Research methods and materials

The decisive role in the development of rickets belongs to exogenous or endogenous deficiency of vitamin D: insufficient formation of cholecalciferol in the skin, insufficient intake of vitamin D with food and impaired metabolism, which leads to impaired phosphorus-calcium metabolism in the liver and kidneys. In addition, other metabolic disorders contribute to the development of rickets - impaired protein and microelement metabolism (magnesium, iron, zinc, copper, cobalt, etc.), activation of lipid peroxidation, polyvitamin deficiency (lack of vitamins A, B1, B5, B6, E), etc.

The main physiological functions of vitamin D (more precisely, its active metabolites 25-hydroxycholecalciferol and 1,25-dihydroxycholecalciferol) in the body are: increasing the absorption of calcium (Ca) and phosphorus (P) salts in the intestine; preventing their excretion in the urine by increasing the reabsorption of Ca and P in the renal tubules; mineralization of bone tissue; stimulation of the formation of red blood cells, etc. With hypovitaminosis D and rickets, all of the above processes are slowed down, which leads to hypophosphatemia and hypocalcemia (low levels of P and Ca in the blood).

Secondary hyperparathyroidism develops as a result of hypocalcemia according to the feedback principle. Increased production of parathyroid hormone leads to the release of Ca from the bones and the maintenance of its sufficiently high level in the blood.

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A shift in the acid-base balance to acidosis prevents the deposition of P and Ca compounds in the bones, which is accompanied by a violation of the calcification of growing bones, their softening and susceptibility to deformation. Instead of full-fledged bone tissue, osteoid non-calcified tissue is formed in the growth zones, which grows in the form of thickenings, tubercles, etc.

In addition to mineral metabolism, rickets also disrupts other types of metabolism (carbohydrate, protein, fat), and disorders of the nervous system and internal organs develop.

Causes of rickets

The development of rickets is mainly associated not with an exogenous deficiency of vitamin D, but with insufficient endogenous synthesis of it. It is known that more than 90% of vitamin D is formed in the skin as a result of insolation (UV radiation), and only 10% comes from the outside with food. Just 10 minutes of local irradiation of the face or hands provides the synthesis of vitamin D necessary for the body, which is why rickets is more common in children born in autumn and winter, when solar activity is very low. In addition, rickets is often common among children living in areas with a cold climate, insufficient natural insolation, frequent fog and cloudiness, and unfavorable environmental conditions (smog).

At the same time, hypovitaminosis D is the leading, but not the only cause of rickets. In young children, a deficiency of calcium salts, phosphates and other osteotropic micro- and macroelements, vitamins can occur due to many ricketogenic factors. Since the greatest increase in the supply of Ca and P to the fetus is observed in the last months of pregnancy, premature babies are more susceptible to the development of rickets.

The development of rickets is associated with an increased physiological need for minerals in conditions of intensive growth. The lack of vitamins and minerals in the child's body can be the result of malnutrition of a pregnant or lactating woman or a baby. Immaturity or pathology of the enzyme systems of the gastrointestinal tract, liver, kidneys, thyroid and parathyroid glands (gastritis , dysbacteriosis , malabsorption syndrome , intestinal infections , hepatitis , biliary atresia , chronic renal failure, etc.).

Children with an unfavorable perinatal history are at risk of developing rickets. Unfavorable factors on the part of the mother include gestosis of pregnant women; physical inactivity during pregnancy; operative, induced or rapid delivery; maternal age less than 18 years and more than 36 years; extragenital pathology.

A certain role in the development of rickets can be played by a child's large weight (more than 4 kg) at birth, excessive weight gain or hypotrophy; early transition to artificial or mixed feeding; restriction of the child's motor activity (too tight swaddling, lack of baby massage and gymnastics, the need for prolonged immobilization in case of hip dysplasia), taking certain medications (phenobarbital, glucocorticoids, heparin, etc.). The role of gender and hereditary factors has been proven: for example, boys, children with black skin and blood group II (A) are more prone to the development of rickets; Rickets is less common in children with blood group I (0).

Research results: Etiological classification suggests identifying the following forms of rickets and rickets-like diseases:

Vitamin D deficiency rickets (calciumpenic, phosphoropenic variant)

Vitamin D-dependent (pseudodeficiency) rickets with a genetic defect in the synthesis of 1,25-dihydroxycholecalciferol in the kidneys (type 1) and genetic resistance of target organ receptors to 1,25-dihydroxycholecalciferol (type 2).

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Vitamin D-resistant rickets (congenital hypophosphatemic rickets, Debre-de-Toni-Fanconi disease, hypophosphatasia, renal tubular acidosis).

Secondary rickets caused by diseases of the gastrointestinal tract, kidneys, metabolism, or drugs.

The clinical course of rickets can be acute, subacute and recurrent; the severity is mild (I), moderate (II) and severe (III). The following periods are distinguished in the development of the disease: the initial stage, the peak of the disease, recovery and residual effects.

Symptoms of rickets

The initial period of rickets occurs at 2-3 months of life, and in premature babies - in the middle of the 1st month of life. The first signs of rickets include changes in the nervous system: tearfulness, timidity, anxiety, excessive excitability, shallow, restless sleep, frequent tossing and turning during sleep. The child's sweating increases, especially on the scalp and back of the head. Sticky, sour-smelling sweat irritates the skin, causing persistent diaper rash. If you rub your head against the pillow, bald spots appear on the back of your head. Characteristic features from the musculoskeletal system are the appearance of muscle hypotonia (instead of physiological muscle hypertonia), sagging of the cranial sutures and edges of the fontanel, and thickening of the ribs ("rachitic beads"). The duration of the initial period of rickets is 1-3 months.

At the peak of rickets, which usually occurs at 5-6 months of life, the development of the osteomalacia process is observed. The consequences of acute rickets can be softening of the cranial bones (craniobases) and unilateral flattening of the back of the head; deformation of the chest with depression ("chest") or bulging of the sternum (keeled chest); formation of kyphosis ("rickety hump"), possibly lordosis , scoliosis ; O-shaped curvature of the tubular bones , flat feet ; formation of a flat rachitic narrow pelvis . In addition to bone deformation, rickets is accompanied by an enlargement of the liver and spleen, severe anemia, muscle hypotonia ("frog" belly), and loose joints.

In the subacute stage of rickets, there is hypertrophy of the frontal and parietal tubercles, thickening of the interphalangeal joints of the fingers ("string of pearls") and wrists ("bracelets"), costochondral joints ("rachitic beads").

Changes in internal organs with rickets occur due to acidosis, hypophosphatemia, impaired microcirculation and may include shortness of breath, tachycardia, decreased appetite, unstable stools (diarrhea and constipation), pseudoascites.

During the recovery period, sleep normalizes, sweating decreases, static functions, laboratory and radiological data improve. The period of residual effects of rickets (2-3 years) is characterized by residual skeletal deformities and muscle hypotonia.

In most children, rickets occurs in a mild form and is not diagnosed in childhood. Children with rickets often suffer from acute respiratory viral infections, pneumonia, bronchitis, urinary tract infections, and atopic dermatitis. A close relationship has been noted between rickets and spasmophilia (infantile tetany). In the future, children with rickets often experience delayed timing and sequence of tooth eruption, malocclusion, and enamel hypoplasia.

Rickets diagnosis

The diagnosis of rickets is made on the basis of clinical signs confirmed by laboratory and radiological data. To determine the degree of mineral metabolism disorders, a biochemical study of blood and urine is performed. The most important laboratory signs that allow us to think about rickets are hypocalcemia and hypophosphatemia; increased alkaline phosphatase activity; decreased levels of citric acid, calcidiol and calcitriol. When examining the acid-base balance of the blood, acidosis is

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detected. Changes in the urine test are characterized by hyperaminoaciduria, hyperphosphaturia and hypocalciuria. Sulkovich's rickets test is negative.

X-ray of the tubular bones reveals changes characteristic of rickets: cup-shaped expansion of the metaphyses, clear boundaries between the metaphysis and epiphysis, thinning of the cortical layer of the diaphysis, unclear visualization of the ossification nuclei, osteoporosis. Densitometry and CT of the tubular bones can be used to assess the condition of the bone tissue. X-rays of the spine, ribs and skull are not recommended due to the severity and specificity of clinical changes in them.

Differential diagnosis of rickets is carried out with rickets-like diseases (D-resistant rickets, vitamin D-dependent rickets, de Toni-Debre-Fanconi disease and renal tubular acidosis, etc.), hydrocephalus, cerebral palsy, congenital hip dislocation, chondrodystrophy and osteogenesis imperfecta.

Conclusion: Comprehensive medical care for a child with rickets consists of organizing the right daily routine, rational nutrition, drug and non-drug therapy. Children with rickets should spend 2-3 hours outdoors every day, get enough sunlight, introduce complementary foods early, and undergo hardening procedures (air baths, rubbing). Proper nutrition of a nursing mother with the intake of vitamin and mineral complexes is of great importance.

Specific therapy for rickets requires the use of vitamin D in therapeutic doses, depending on the severity of the disease: in stage I. - 1000-1500 IU per day (course 30 days), in II - 2000-2500 IU (course 30 days); in III - 3000-4000 IU (course - 45 days). After completing the main course, vitamin D is prescribed in a prophylactic dose (100-200 IU per day). Treatment of rickets should be carried out under the control of the Sulkovich test and biochemical markers to exclude the development of hypervitaminosis D. Since polyhypovitaminosis is often observed with rickets, children are recommended to take multivitamin complexes, calcium and phosphorus preparations.

Nonspecific treatment of rickets includes exercise therapy, general ultraviolet irradiation, balneotherapy (pine and sodium chloride baths), massage with elements of paraffin and the use of therapeutic mud.

Prognosis and prevention

The initial stages of rickets respond well to treatment; After adequate therapy, long-term consequences do not develop. Severe forms of rickets can lead to severe deformation of the skeleton and a slowdown in the physical and neuropsychic development of the child. Monitoring of children with rickets is carried out quarterly for at least 3 years. Rickets is not a contraindication for preventive vaccination of children: vaccinations can be carried out 2-3 weeks after the start of special therapy.

Prevention of rickets is divided into antenatal and postnatal. Prenatal prevention involves the pregnant woman taking special complexes of microelements, spending enough time in the fresh air and eating a balanced diet. After birth, it is necessary to continue taking vitamins and minerals, breastfeeding, adhering to a strict daily regimen and giving the child preventive massage. During daily walks, the child's face should be left open so that the sun's rays can reach the skin. Specific prevention of rickets in newborns who are breastfed is carried out in the autumn-winter-spring period with the help of vitamin D and ultraviolet radiation.

Tibia fractures in children account for more than half of all lower limb fractures. They occur as a result of falls (including from a height), road traffic accidents, direct blows, or twisted legs. Clinical manifestations are determined by the degree of fracture. When the upper metaepiphysis of the symphysis is damaged

Acute rhinitis is most severe in newborns (especially premature babies) and infants, which is associated with the predominance of general symptoms and frequent complications. Due to the

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narrowing of the nasal passages and the small vertical size of the nasal cavity, even with a slight swelling of the nasal mucosa, nasal breathing becomes sharply difficult or stops.

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