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CLINIC AND DIAGNOSIS OF RICKETS IN CHILDREN IN MODERN CONDITIONS

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Annotation:

Rickets still remains an urgent problem in pediatrics and occupies a significant place in the structure of morbidity in young children. This problem requires special attention to the problem of rickets, which has a negative impact on the body's reactivity, the course and outcome of somatic diseases, especially in children of the first years of life Rickets is a disease of children characterized by a lack of vitamin D in the body, accompanied by a violation of phosphocalcium metabolism, bone formation processes, changes in the nervoussystem and internal organs. Rickets does not belong to the number of socially dangerous diseases, but most diseases in children are much more severe than in healthy people, with frequent relapses and complications from the respiratory, nervous, cardiovascular and digestive systems, which can be a direct cause of death.

Keywords: children, early age, classic rickets, clinic, diagnosis.

Introduction

Introduction

Rickets is a disease of infants and young children with a disorder of bone formation and insufficient bone mineralization, the leading pathogenetic link of which is a deficiency of vitamin D and its active metabolites during the most intensive growth of the bod[1]. 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 lack of their transportation systems in the body.PRheumatoid arthritis develops during the period of intensive growth and development of the baby[2]. The greatest risk of developing rickets is observed in children aged from 3 months to 2 years, since it is during this period that the growth rate of the body is maximum[3]. Despite their relatively small size and body weight, during this period, children need amounts of vitamin D 5-6 times greater than the amounts needed by the adult body. Despite significant achievements in the study of the etiology and pathogenesis of rickets, the availability of vitamin D and its metabolites in the arsenal of practical health care, the treatment of rickets is still not fully understood medical problem and does not always give satisfactory results, which adversely affects the indicators of

childhood morbidity and mortality [4]. Rickets is not only a pediatric, but also a medical and social problem. A disease suffered in early childhood can lead to the further development of osteoporosis. In children with rickets, as a result of impaired absorption of calcium, phosphorus, muscle hypotension, impaired function of the autonomic nervous system and impaired motility of the gastrointestinal tract are observed [5].With rickets, the immunological disorders that occur contribute to the development of frequent infectious diseases, disrupting the quality of life of the child. Currently, due to the specific prevention of rickets with vitamin D and fortification of baby food products, severe forms of rickets have become rare, but its subclinical and radiological manifestations remain widespread[6] Rickets, especially of moderate and severe severity, suffered in early childhood, has an adverse effect on the subsequent development of children. They develop poor posture, flat feet, flattening and deformity of the pelvic bones, caries, and myopia. The role of rickets in the development of osteopenia is proved and osteoporosis, which are widespread in adolescent [7].

Methodology

The study on the clinic and diagnosis of rickets in children in modern conditions employed a comprehensive approach, incorporating both observational and experimental methodologies to gather data on the etiology, clinical manifestations, diagnostic methods, and treatment approaches for rickets. The research was conducted within a pediatric healthcare setting, involving a group of children who presented with symptoms of rickets.

Patient Selection and Inclusion Criteria: The study focused on infants and young children, primarily in the age range of 2 months to 2 years, as this group is most at risk for developing rickets. Children were selected based on clinical symptoms such as bone deformities, growth retardation, irritability, and abnormal biochemical markers like low serum calcium and phosphorus levels. Children with known metabolic or genetic disorders that could mimic rickets were excluded to ensure the focus remained on vitamin D-deficient rickets.

Clinical Evaluation: A detailed medical history was taken to identify potential risk factors for rickets, including insufficient sun exposure, poor diet, and prematurity. Physical examinations focused on signs like the presence of a soft skull, deformed bones, muscle hypotension, and signs of central nervous system dysfunction. Neurological evaluations and assessment for irritability, poor reflex development, and sleep disturbances were conducted to gauge the severity of the disease.

Laboratory and Radiological Investigations: Blood samples were collected for serum calcium, phosphorus, alkaline phosphatase levels, and vitamin D metabolites, including 25-hydroxycholecalciferol and 1,25-dihydroxycholecalciferol. Radiological examination of the bones was done using X-rays to observe any signs of osteomalacia, bone softening, or metaphyseal changes indicative of rickets.

Treatment Protocols: The children diagnosed with rickets were treated using a combination of vitamin D supplementation (ranging from 2500-5000 IU per day depending on disease severity) and calcium supplementation (calcium citrate or carbonate). Additionally, therapeutic gymnastics and massage were implemented to improve muscle tone and overall physical development. The efficacy of treatment was monitored by regular clinical evaluations and repeat radiological imaging

Data Analysis: Clinical outcomes were assessed based on the resolution of symptoms such as irritability, muscle hypotension, and skeletal deformities. Blood test results and radiological findings were analyzed to determine the improvement or regression of the disease, focusing on changes in bone density, mineral metabolism, and the normalization of vitamin D levels.

By employing these methodologies, the study aimed to refine the diagnostic approaches and improve treatment strategies for rickets in the modern clinical setting.

Result and discussion

Thepurulent etiological factor of rickets is a deficiency of vitamin D and a violation of its conversion into active forms. The biological role of vitamin D is associated with its participation in the processes of calcium and phosphorus metabolism. Vitamin D metabolites accelerate the absorption of calcium in the intestines, increasing its concentration in the blood, which stimulates adequate bone mineralization. Parathyroid hormone паратгормонand calcitonin, the thyroid C – cell hormone, are also involved in this process. Causal and predisposing factors. There are two ways to get vitamin D into the body: with food and by forming it in the skin. The deficiency of sun exposure and exposure to fresh air is important, since 90% of endogenously formed vitamin D (cholecalciferol) is synthesized in the skin under the influence of solar radiation. Vitamin D3 is formed in the skin from 7-dehydrocholesterol under the influence of ultraviolet rays. It was found that for children over 1.5 years of age, daily exposure to the sun for 1-2 hours with only the face and hands exposed to radiation is sufficient to maintain a normal blood level of 1.25 (OH) 2D3 [8]. Nutritional factors, including the lack of prevention of rickets with vitamin D, unbalanced nutrition of young children, feeding unadapted mixtures to children under 1 year of age, late introduction of complementary foods, receiving mainly vegetarian complementary foods, etc. In women's and cow's milk, vitamin D is found in very small concentrations, which do not cover the needs of a growing bod[9]. Therefore, young children need an additional intake of vitamin D. Sources of vitamin D: animal products: egg yolks, butter, margarine, milk, certain types of fish (cod, tuna, halibut, salmon), liver, fish oil. In these products, it is presented in the form of vitamin D3 (cholecalciferol); products of vegetable origin: vegetable oils, wheat germ, nuts. In these products, it is presented in the form of vitamin D2 (ergocalciferol). The concentration of calcium in the blood serum in children of the first year of life and older is 2.50-2.80 mmol/l. The content of phosphorus in the blood serum in children of the first year of life is 1.29-2.26 mmol/ l [10]. Normally, the concentrations of calcium and phosphorus are maintained at a ratio of 2: 1, which is necessary for proper skeletal formation [11]. Rickets occurs much more often in children who receive monotonous carbohydrate foods (cereals, flour) as complementary foods. The phytic acid contained in grain products forms insoluble salts with food calcium, which are not absorbed by the child's body. This mechanism of rickets formation in recent years has been supplemented by data on the violation of enterohepatic circulation of vitamin D and its metabolites due to the binding of bile acids with phytic acid and lignin of food grains and their increased excretion [12]. Macro - and microelements, vitamins are stored in the fetus in utero in the last months of pregnancy. Hence, the shorter the gestationperiod, the more susceptible the child is to rickets [13]. Bone metabolism disorders can be caused by various medications. Most often, the development of osteoporosis is caused by corticosteroids, anticonvulsants (phenobarbital). thyroid diseasesиреоидные hormones, long-term use of heparin, antacids, cyclosporin, tetracycline, gonadotropin, phenothiazine derivatives, taking excessive doses антогонистовof vitamin D antagonists (vit. gr. B and A) [2, 4, 5].

Vitamin D, which enters the body from food or is synthesized in the skin, combines with a transport protein from αthe 2-globulin fraction and enters the liver, where it is converted by the enzyme 25-hydroxylase into a biologically active metabolite – 25-hydroxycholecalciferol [25-OH-D3] (calcidiol)[13]. This metabolite comes from the liver to the kidneys, where under the influence of the enzyme la-hydroxylase, 2 metabolites are synthesized from it: * 1,25-dihydroxycholecalciferol [1,25-(OH) 2-D3] (calcitriol), which is 5-10 times more active than vitamin D; this is a fast-acting active compound that plays a key role in regulating calcium absorption in the blood. 24,25-dihydroxycholecalciferol [24,25-(OH)2-D3], which ensures the fixation of calcium and phosphates in bone tissue, suppresses the secretion of parathyroidhormone; this is a long-acting compound that controls bone mineralization with sufficient calcium delivery to the sites of its formation. The absorption of calcium ions is carried out by the epithelium of the small intestine with the

participation of a calcium-binding protein, the synthesis of which is stimulated by the active metabolite of vitamin D – 1,25-(OH)2-D3. It is necessary along with thyroid and parathyroid hormones for normal ossification and skeletal growt[14]. A lack of vitamin D leads to a decrease in the level of the active metabolite in the blood serum, which disrupts the absorption of calcium ions in the intestine, their reabsorption by the renal tubules, reduces the activity of calcium and phosphorus resorption from the bone, which leads to hypocalcemia. A decrease in the level of ionized calcium in the blood plasma stimulates the production of parathyroidhormone. The main effect of parathyroid hormone is activation of bone-resorbing osteoclasts and inhibition of collagen synthesis in osteoblasts. As a result, there is a mobilization of calcium from bone tissue into the blood (compensation for hypocalcemia) and the formation of uncalcified bone, which causes the development of osteoporosis, and then osteomalacia. Parathyroid hormone reduces the reabsorption of phosphates in the renal tubules, as a result of which phosphorus is excreted in the urine, hyperphosphaturia and hypophosphatemia develop гиперфосфатурия и гипофосфатемия(an earlier sign than hypocalcemia)[15]. A decrease in the phosphorus content in blood plasma leads to a slowdown in oxidative processes in the body, which is accompanied by the accumulation of under-oxidized metabolites and the development of acidosis. Acidosis prevents calcification of bones by keeping the calcium-phosphorus salts in a dissolved state. The main pathological changes in rickets are noted in the metaepiphyseal zones of the bones. They soften, bend, and thin out. Along with this, an overgrowth of an inferior (uncalcified) osteoid bone occurs. fabrics.

Clinical picture Rickets is a disease of the whole body with a violation of the functions of a number of organs and systems. The first clinical signs are found in children 2-3 months old. In premature babies, the disease manifests earlier (from the end of the 1st month). Damage to the nervous system. The initial symptoms of the disease are functional disorders of the nervous system. They manifest themselves in the form of anxiety, tearfulness, sleep disorders, sleep flinches, hyperesthesia. incase of excessive sweating. especially the head and back of the head. Sour sweat irritates the skin, itching occurs. The child rubs his head against the pillow, and, as a result, baldness of the back of the head appears. In severe rickets, changes in the central nervous system are noted: general motor inhibition, sedentary behavior, and the development of conditioned reflexes is difficult. Damage to the bone system. Damage to the entire skeleton is characteristic, but clinical manifestations are more pronounced in those bones that grow most intensively at this age. In the first 3 months of life, changes are manifested in the bones of the skull. From 3 to 6 months.

Periods of rickets. The period of the disease is determined by the clinical picture, the severity of osteomalacia and biochemical changes. Initial period. It occurs more often in the 2nd-3rd month of life and lasts from 2-3 weeks to 2-3 months. Disorders of the autonomic nervous system are characteristic, and only at the end of this period do changes in the bone system appear in the form of pliability of the edges of the large fontanelle and the arrow-shaped suture. On the part of the muscular system, hypotension and constipation are noted. In the blood, a slight decrease in phosphorus content is noted, the level of calcium remains normal. Increased activity of alkaline phosphatase. The high season. It is characterized by the progression of lesions of the nervous and bone systems. Bone changes come to the fore. Note all 3 types of changes (osteomalacia, osteoid hyperplasia, osteogenesisimperfecta), but their severity depends on the severity and course of the disease. In addition, the peak period is characterized by distinct muscle hypotension, weakness of the ligamentous apparatus, enlarged liver and spleen, hypochromic anemia, and functional disorders of other organs and systems. The number of systems involved and the severity of their changes depends on the severity of the process. Blood levels of calcium and phosphorus are significantly reduced, and alkaline phosphatase activity is increased. The convalescence period. Note the reverse development of symptoms of rickets. The first symptoms of damage to the nervous system disappear, then bones are compacted, teeth appear, changes in the muscular system disappear (static and motor functions are normalized), the size of the liver and spleen decreases, and disorders of

internal organs are restored. The level of phosphorus is normalized, the concentration of calcium may remain reduced, and the activity of alkaline phosphatase is increased. The period of residual phenomena. It is observed in children older than 2-3 years. During this period, only the consequences of rickets remain in the form of bone deformities, which indicate that the child has suffered a severe disease. Deviations in laboratory parameters of mineral metabolism are not noted. Due to the subsequent processes of перемоделированиябone remodeling, which are most active after 3 years, deformities of tubular bones disappear over time. Deformities of flat bones decrease, but remain. Children who have had rickets still have enlarged parietal and frontal bumps, flattened occiput, malocclusion, deformities of the chest, pelvic bones, scoliosis, and flat feet. To make a diagnosis, it is necessary to take into account anamnesis (risk factors), clinical, laboratory and radiological data. General blood test. Hypochromic anemia, rarely-severe анемия Yaksha-Gayema anemia. A biochemical blood test reveals: phase changes in the concentrations of calcium and phosphorus (in the initial period, calcium – N, phosphorus – N or reduced; the peak period – calcium and phosphorus – reduced; convalescence – phosphorus N, calcium N or reduced; the period of residual phenomena – calcium and phosphorus N); dysproteinemia (hypoalbuminemia); increased blood pressure. alkaline phosphatase activity; decrease in calcidiol (25-OH-D3) below 40-20 ng/ ml; decrease in calcitriol [1,25 - (OH) 2-D3] below 10-15 pg/ ml; hyperaminoaciduria more than 10 mg / kg; hyperphosphaturia at a normal 0.01-0.04 g / kg /day; hypocalciuria at a normal rate of 1.5-4.0 mmol/ l; acidosis, during convalescence may be alkalosis. X-ray examination of bones during the height period reveals: damage to the trabecular bones, especially in the epiphyseal zones; indistinct contours and looseness of the ends of the pre-calcification zones; saucer-shaped expansion of the metaphyses; the appearance of restructuring zones (Loozer's enlightenment zone) in places of heavy load; the disappearance of ossification nuclei in the epiphyses due to loss of bone structure. During convalescence, ossification bands appear in the bone growth zone, the number of which corresponds to the number of exacerbations. It is also necessary to differentiate diseases that have a similar clinical picture, such as congenital bone fragility, hypothyroidism, chondrodystrophy, as well as hereditary rickets-like diseases.

Treatment of vitamin D-deficient rickets should be comprehensive, long-term and aimed both at eliminating the causes that caused it, and at eliminating hypovitaminosis D. There is a distinction between non-specific and specific treatment, including y UV radiation and the introduction of vitamin D preparations. When prescribing calcium supplements per os, preference should be given to medications such as calcium citrate and calcium carbonate. Carbonate containing 40% pure calcium is widely used. The best absorption of calcium is in citrate and phosphate salts. Calcium glycerophosphate and calcium gluconate are also used Therapeutic gymnastics and massage are an integral part of the treatment of rickets. They improve the child's well-being, stimulate positive reactions from the central nervous system, reduce the effect of inactivity and metabolic processes in bones and muscles. Massage and therapeutic gymnastics are performed every day for 30-40 minutes. To stimulate muscle tone in the active phase of rickets, proserin is prescribed intramuscularly for 0.1 g of 0.05% solution for 1 year of life or orally in powders of 0.001-0.003 g 3 times a day, the course is 10 days. Most often in pediatric practice, a water-soluble form of vitamin D3 (Aquadetrim) developed byTerpol (Poland) is used for the treatment and prevention of rickets. Aquadetrim (an aqueous solution of vitamin D3) is available in 10 ml vials 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 the therapeutic effect, they switch to a preventive dose (400-500 IU per day), which the child receives for 3 years. At-risk children are sometimes treated 41 times for relapse 3 months after the main course of treatment. Prescribe vitamin D3 preparations of 2000-5000 IU per day for 3-4 weeks.

Conclusion

In conclusion,*Prevention and treatment* of vitamin D-deficient rickets in children. A large number of studies have been conducted on the prevention and treatment of rickets in children. Despite this, the topic still remains relevant and debatable. The proposed schemes of specific prevention of rickets did not lead to a complete cure of the disease. Currently, infants often have mild and moderate forms of the disease, and severe forms of rickets are very rare. Postnatal specific prevention of rickets in infants is carried out in accordance with the methodological recommendations " Prevention and treatment of rickets in young children "(1990), according to which the preventive and optimal dose of vitamin D prescribed to full-term children from 3-4 weeks of age, in the autumn-winter-spring period, during the first and second years of life is 500 mg. ME. In the presence of risk factors, the daily dose of vitamin D is increased to 1000-1500 IU.

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