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**HYPOVITAMINOSIS D: LABORATORY DIAGNOSIS AND
CORRECTION**

(monograph)

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The problem of rickets is currently given very little attention. Many scientific studies on the specifics of bone metabolism and mineral metabolism are mainly devoted to the problem of osteoporosis. Some pediatricians continue to perceive rickets as a physiological condition that does not require correction.

The monograph covers one of the most common problems in childhood, rickets. Rickets is one of the most common diseases in young children. The monograph considers such aspects as the pathogenesis, classification, clinic and diagnosis of rickets. The article presents facts and rules based on evidence about rickets, as well as its own point of view based on the results of modern research methods. Special attention is paid to modern methods of rickets research today. The proposed monograph presents the results of long-term observation of children with signs of rickets. The analysis presented provides a reasonable diagnostic algorithm for the examination and treatment of rickets in young children and the prevention of the consequences of rickets.

The monograph is intended for students of medical universities, masters, clinical residents, postgraduate cadets and pediatricians.

The monograph was discussed and approved at a meeting of the Scientific Council of Samarkand State Medical University and recommended for publication.

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TABLE OF CONTENTS

Introduction	4
Chapter I. In young children rickets disease to develop impact provider frequency and etiological factors.....	6
In young children rickets Pathogenetic aspects of the formation of	12
Rickets classification	18
prevention and treatment of rickets in young children.....	20
Chapter II. Under investigation children's features	28
Research methods	30
Chapter III. Vitamin D Deficiency in Children Risk Factor Assessment	33
Nerve perinatal damage to the nervous system within children rickets of signs features.....	38
Under investigation 25(OH)D3 levels in children and biochemical indicators comparative taxonomy	42
Blood Serum 25(OH)D3 levels looking at danger Importance of factors	48
Chapter IV. Rickets traditional prevention during children blood Serum 25(OH)D3 microdose change features	53
Effect of modified rickets prevention on serum 25(OH)D3 levels in children.....	55
Treatment of rickets in children, depending on its severity and serum 25(OH)D3 levels	57
Rickets in children traditional prevention assessing the effectiveness of	61
Conclusion	65
Abbreviations list	79

INTRODUCTION

OVERVIEW

A disruption in the body's calcium and phosphorus metabolism leads to rickets, a condition that primarily affects young infants. A baby's first few years of life go by swiftly. Vitamin D controls how calcium and phosphorus are absorbed from the intestines and transported to bones and other tissues for healthy bone formation. Vitamin D deficiency causes rickets. The majority of vitamin D is produced in the skin under the effect of sunshine, and it is only found in trace amounts in diet. This issue is still significant today even though rickets has been known for a long time.

Low food intake or inadequate vitamin D production in growing children's skin are the main etiological factors for rickets development. This issue has been discussed for a long time, but neither developing nor developed countries have been able to reduce the incidence of rickets despite our country's abundance of sunlight. Considering the current environmental and ethnic factors, rickets affects 27% of one-year-old children in Uzbekistan, which calls for the development and improvement of prevention strategies.

In light of this, it is critical to evaluate the risk factors for rickets development in young children within the primary healthcare system and to create modified rickets prevention to significantly lower the incidence of hypocalcemic seizures.

There are several etiological and predisposing variables for primary vitamin D deficiency rickets, according to the literature. These include consuming foods that are not suitable for infants, introducing complementary foods incorrectly, eating repetitive vegetarian cuisine, and not getting enough sunlight or fresh air. However, perinatal factors are equally significant: vitamin D inadequacy is frequently caused by premature delivery, intrauterine growth retardation, placental insufficiency, etc.

In recent decades, fundamentally new information has been obtained about the metabolism and functions of vitamin D, which has significantly changed the views on the etiology, pathogenesis of rickets, and methods of its prevention and treatment. Endogenous vitamin D insufficiency and rickets process to develop predisposed internal mechanisms essential roles plays. Life first in the years children and especially early born babies bone tissue fast growth and active again build processes due to vitamin D and phosphorus-calcium to the salts this minerals to the bone delivery to give and their assimilation to do mechanisms relative maturity with high need appearance will be. According to our information, children's risk of developing rickets increases during the spring, when they are growing quickly.

In Uzbekistan, rickets is caused by a number of factors, including poor maternal health, the unique characteristics of baby-rearing, and parents' lack of concern for their children, particularly in rural areas. Consequently, the literature claims that a high incidence of rickets and a propensity for hyperexcitability are seen in the republic as a result of an imbalance in children's calcium and phosphorus metabolism. In newborns, this is particularly crucial because low levels of this metabolite are a risk factor for the development of muscular atonia, spasmophilia, and hyperexcitability of the central and peripheral nervous systems, all of which lower infants' quality of life.

CHAPTER I

1.1. Frequency and etiological factors influencing the development of rickets in young children

When the body's phosphorus-calcium metabolism is disrupted, vitamin D levels fall, causing rickets [from the Greek word *rhachis*, which means "spine"]. Children who are artificially fed or premature babies are at high risk of contracting rickets. Rickets can result from a child's body not getting enough vitamin D or their skin not producing enough vitamin D because of a lack of UV rays if they are underweight, poorly cared for, or do not enjoy the outdoors and sunlight. Rickets is also brought on by the child's recurrent illnesses and dietary violations by the mother during pregnancy.

Rickets causes metabolic problems and interferes with the way different organs and systems work. The metabolism of mineral salts like calcium and phosphorus is particularly disrupted by this condition. In addition to causing bone fragility and softening of its tissues, altered intestinal absorption of calcium and its subsequent deposition in the bones also interfere with the nervous system's and internal organs' ability to operate. The patient's neurological system changes in the early stages of the disease: the youngster becomes shy, agitated, erratic, or agitated; he perspires a lot, and when he feeds, his face and back are drenched in perspiration.

The child's back swells as a result of rubbing his head against the pillow because he is itchy. The muscles become weaker and twisted as the disease worsens; the child walks later than a healthy child, his eyes swell, his stomach frequently gets hard or sucks, and eventually the bone system changes: the head and shoulder blades get bigger, the forehead and crown protrude, the forehead gets convex, and the bones in the skull and occipital area soften. The head's big frenulum doesn't ossify in due time. The ribs close to the chest often get thicker.

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frequently gets hard or sucks, and eventually the bone system changes: the head and shoulder blades get bigger, the forehead and crown protrude, the forehead gets convex, and the bones in the skull and occipital area soften. The head's big frenulum doesn't ossify in due time. The ribs close to the chest often get thicker. The child has crooked legs that form an X or an O when he/she first starts to walk.

Additionally, the chest wall's form varies, either retracting or protruding forward.

Rickets is as old as the globe, and research has been done on its pathophysiology, treatment, and management. It would appear that there is nothing to consider and that everything is obvious. However, there are still a lot of unknowns around this issue. Soran of Ephesus (98–138 AD) was the first person to mention rickets in scholarly literature. He observed that children with the condition had deformed lower bodies and spines. The English anatomist and orthopedist Glisson published a comprehensive clinical categorization of rickets in 1650, and the corresponding illness was dubbed "English disease" or "slum disease."

A number of pediatricians made their contribution to the study of rickets: Filatov N.F., Kisel A.A., Speransky G.N., Tur A.F., Lukyanova Ye.M. and others. Vitamin D was first discovered and isolated by McCollum in 1922, after which it became possible to study its specific effects on bones, muscles, intestines and kidney tubules. Due to the lack of continuity in the management of patients, terminological variances in the definition of rickets lead to some differences in approaches to the treatment and prevention of the condition. This issue is still significant today even though rickets has been known for a long time.

Rickets was eradicated in several nations when the discovery of vitamin D and its function in the first half of the 20th century made prevention strategies obvious.

The fact that it is so common in young children is notable. Every country has rickets, but the Nordic nations, which receive little sunlight, are more susceptible. Rickets is more common and more severe in children born in the fall and winter.

Regarding the incidence of rickets, during the first year of life, 25% to 55% of children are diagnosed with moderate rickets and 100% with mild rickets.

Rickets is more common than vitamin D insufficiency in Russia, where it is found in 50–70% of infants during the first year of life. The use of vitamin D prophylaxis decreased the incidence of rickets in children aged 3-6 months from 24% to 4%, according to statistics from Turkish literature published in the 1990s. Rickets incidence has been on the rise in the United States, Japan, and Europe in recent years. In Bulgaria, rickets affects 20% of children under one year old, even though the country has plenty of sunshine days all year round.

The Russian Federation's State Statistics Committee reports that between 1992 and 1998, there was a 23.2% increase in the incidence of rickets of the II–III degree. According to data from local pediatricians, the prevalence of rickets in Moscow infants is currently below 30%. However, because rickets is only diagnosed in relatively severe instances and its mild versions are not statistically considered, this number is at least twice as high as it should be. In preschool-aged youngsters (ages 6-7), residual symptoms and incomplete rickets are identified.

The experience of nations with an adequate number of sunny days per year demonstrates the necessity of providing vitamin D all year round for preventive purposes during a child's first and second years of life, while "bone tissue" is still growing. Researchers in Samarkand have found that 30% of babies suffer rickets. Vitamin D insufficiency has been linked in recent years to the development of neurological disorders, type 2 diabetes, blood pressure problems, malignancy, and some types of cancer, as well as weakened immunity. Even though Uzbekistan receives a lot of sunlight, rickets is still diagnosed, which is thought to make children's respiratory and diarrheal illnesses worse.

In the last ten years, rickets has become less common in youngsters, its course has changed, and its mild versions are more common. However, the body becomes less resistant to infections, particularly acute respiratory conditions, even in moderate cases of rickets. When rickets is present, pneumonia is chronic or

recurrent. Children who suffer rickets develop neurologically and physically more slowly.

Changes in the skeletal, muscular, and neurovegetative systems are hallmarks of rickets. The respiratory and cardiovascular systems also deteriorate. Air saturation in the lungs is compromised, respiratory morbidity rises, and bronchoobstructive syndrome develops and worsens as a result of illnesses of the chest deformities and muscular hypotension.

As a result, rickets has resurfaced in both affluent and developing nations in recent years, making respiratory and diarrheal illnesses more severe. This is linked to a decline in the body's total immunological responsiveness, which exacerbates the progression and results of several illnesses and raises the morbidity and mortality rate among children. As a result, all pediatricians should be knowledgeable about rickets and promptly implement sensible preventive measures.

According to M.C. Maslov (1961), "in order to correctly understand the etiology and pathogenesis of rickets, it is necessary to clearly distinguish between predisposing and direct causative factors."

The review papers on this issue reveal the critical role that dietary variables play in the etiopathogenesis of rickets. The most common causes of vitamin D deficiency and, consequently, the development of rickets today include the use of vitamin D antagonist medications, early mixed and artificial feeding, an inadequate diet that lacks nutrients for the child's age, and malabsorption syndrome (when breastfeeding, 70% of calcium salts and 50% of phosphorus are absorbed, while when feeding with cow's milk, only 30% of calcium is absorbed). 20% phosphorus.

Premature kids are at a higher risk of developing rickets because their bodies contain less vitamin D, calcium, and phosphorus, and their growing bodies require these nutrients more than full-term babies do. As a result, infants need 50–55 mg/kg of sodium per day, full-term newborns need 0.75 mg/kg, preterm babies need 100–225 mg/kg, and full-term newborns need 40 mg/kg of phosphorus per

day, while premature babies need 100–150 mg/kg. Rickets can also occur as a result of a child's inadequate motor activity (lack of vigorous walking, massage, and gymnastics), deterioration of the blood supply to the bones, and a decrease in the electric field's potential.

The investigation of anamnesis also produced the following findings: the current occurrence of rickets in the first year of life is associated with unfavorable factors, such as a lack of physical education components in the family, a child's limited outdoor walking, a number of comorbid conditions, body weights under 2500 g or over 3500 g, and high or low growth rates. In addition, the mother's occupation, living and housing conditions, and other socio-hygienic factors have a significant impact on the onset and progression of rickets.

Although this issue has not yet been investigated in Uzbekistan, the country's natural climatic, geographic, and ethnic features may have an impact on the incidence and progression of rickets in infants during the first year of life. Premature babies (77.4%), babies weighing up to 3000 g (71.4%), mixed (64.9%), and artificially fed babies (70.8%) had the highest rate of rickets. According to a study on socio-hygienic living situations, rickets is more prevalent in families with poor family budgets (60.4%), those with the least pleasant housing conditions (68.4%), student parents (81.8%), and those with partial secondary school (62.6%).

Perinatal risk factors have become more important in the development of rickets in recent years. According to the authors' examination of risk factors for baby rickets development, 27% of the children with mild and moderate types of rickets who were evaluated were born between the third and fifth trimesters. Premature labor, either induced or surgical, was found in 73% of moms. A problematic pregnancy and delivery course was noted in 63% of the women. Eight percent of mothers were between the ages of 17 and 18 at the time of delivery. 10% of prematurely born newborns with an average weight of 32–34 g (minimum: 1880 g, maximum: 3110 g) had rickets.

Only 7.9% of children were breastfed at the time of the study, and 23.8% of children who were artificially fed and exhibited clinical signs of rickets received

diluted and undiluted cow's milk, kefir, and unadapted milk mixtures. Of the full-term patients, 46% were overweight (on average 13.4%), and 6.9% had weight loss (on average 12.6%). It should be noted that all children with rickets and hypotrophy had signs of perinatal encephalopathy. Among children with rickets, 79.3% had recurrent pulmonary bronchitis, 27% had UTIs, 15.9% had atopic dermatitis, 7.9% had iron deficiency anemia, and 6.3% had hypotrophy. Seizure syndrome was found in 6.3% of the children who were examined.

The following factors are the most common causes of rickets in the first year of life: low body mass index, premature birth, calendar date of birth, early complementary feeding, low birth weight, low growth rate, high (53.9%) or low (36.2%), extragenital (24.5%) and obstetric pathologies in mothers, lack of physical education in the family (81.2%), and little outdoor exercise (72.2%). Of the youngsters, only 18.8% used natural preventive factors, 81.2% did not receive massage and gymnastics, 68.7% received specialized prophylaxis, and 72.2% spent at least three hours outdoors.

As a result, it is essential to increase parental accountability for their children's health and create radically new strategies for encouraging a healthy lifestyle.

A youngster who consumes 400–500 IU of vitamin D per day gets 40–70 IU from one liter of breast milk and 5–40 IU from one liter of cow's milk. The high phosphate content of cow's milk inhibits the production of parathyroid hormone and the intestinal breakdown of calcium, which is caused by the extensive use of phosphate fertilizers. Cases of hypocalcemia and poor bone mineralization in children born with extragenital disease and challenging pregnancies are documented in the literature.

In addition to healthy pregnant women with calcium deficiency and non-specific symptoms of vitamin D and phosphorus-calcium metabolism, mothers of children with rheumatism, chronic inflammatory diseases of the kidneys, liver, or late toxicosis in the final months of pregnancy are at extremely high risk of giving birth to children with rickets. The primary issue is children with allergic disorders;

in the majority of these situations, the elimination protective diet recommends calcium-containing goods in addition to those high in vitamin D. However, seizure syndrome—metabolic seizures brought on by a calcium deficiency—occurs outside of a child's first or second year of life and may be a sign that the parathyroid glands are functioning properly.

As a result, rickets is still common today even though nursing is very common. There are many different etiological elements that contribute to its creation, making it challenging to identify one in particular. They are distinct to every climate and geographic area, necessitating a more thorough investigation that takes into consideration our region's unique features.

1.2. Pathogenetic aspects of the formation of rickets in early childhood

Insufficient exogenous consumption or compromised endogenous generation of skin sterols under UV radiation might result in vitamin D insufficiency. Insufficient insolation during the winter months raises the chance of contracting rickets.

The primary cause of bone demineralization is a reduction in calcium and phosphorus absorption brought on by a vitamin D deficit.

The development of rickets involves several organs, including the liver, spleen, adrenal glands, and thymus, all of which have interrelated functions. Rickets starts with liver dysfunction, which is caused by a disruption in the metabolism of cholesterol, which is the source of the formation of the metabolically active form of vitamin D. Rickets is a metabolic disorder that occurs in early childhood, when metabolism is extremely stressful and the child's body is functionally immature due to age-related changes.

Rickets is a metabolic disorder in early childhood. At this age, metabolism is extremely stressful, and the child's body is functionally immature due to its age-related changes. A number of organs are involved in the development of rickets - the liver, spleen, adrenal glands, thymus. The functions of these organs are interrelated. Rickets begins with liver dysfunction, in which the metabolism of

cholesterol, which is the source of the formation of the metabolically active form of vitamin D, is disrupted.

D-hypovitaminosis plays the primary role in the pathological process at the beginning, when vitamin D levels fall and blood alkaline phosphatase activity rises. Later, the pathological process may involve other organs and systems, and in particular, functional insufficiency of the adrenal glands, impaired metabolism of vitamins, enzymes, and microelements, and violation of the mineralocorticoid function of the adrenal glands is manifested in phosphorus deficiency, which arises due to impaired reabsorption and increased excretion in the urine. Hypophosphatemia is one of the key pathogenetic mechanisms of rickets, and it is not accompanied by a drop in calcium levels.

The link between Ca and P is disrupted when the normal constant, which is computed for the absorption of Ca by the bones, varies. Thymus insufficiency is identified when the bone tissue is weakening and deforming. The spleen alters and enlarges to its maximum when the thymus is not functioning properly. The primary criterion for determining the severity of rickets, which forms the foundation for differentiating between three levels of severity, is splenomegaly. The hepatosuprarenal-thymolytic condition is what we might call rickets.

It is interesting to notice that when the bones are unable to absorb calcium, the excess is eliminated by the intestines, but hypocalcemia does not develop. Presumably, the thymus handles this function; the parathyroid glands, which primarily control phosphorus-calcium metabolism, are not implicated. It is evident that rickets is a condition about which there is still much to learn. The pathophysiology of vitamin D deficiency rickets has been the subject of several investigations; however, these studies do not provide a comprehensive picture of all the processes of defective phosphorus-calcium metabolism, which significantly influence the disease's clinical presentation.

Reducing the incidence of rickets in first-year children is made possible by the focused repair of this pathological state made possible by the clarification of the primary connections in the pathogenesis of rickets.

The development of active metabolites of vitamin D in the liver and kidneys, insufficient dietary intake of vitamin D, and poor cholecalciferol synthesis in the skin are the most significant pathways in the pathophysiology of rickets. As previously stated, vitamin D deficiency and, consequently, abnormalities in phosphorus-calcium metabolism are the primary cause of rickets. Vitamin D₂ (ergocalciferol) and vitamin D₃ (cholecalciferol) are the two primary members of the group D vitamins (calciferols).

Under the impact of UV radiation with a wavelength of 280–310 nm, the skin synthesizes 90% of the vitamin D₃ from 7-dehydrocholesterol. The primary form of vitamin D that circulates in the blood, 25-(OH)D, or calcidiol, is created in the liver by 25-hydroxylase after vitamin D₂ generated in the skin and vitamin D₂ absorbed from the intestines are carried there attached to globulin. The vitamin D-binding protein transcalferrin then transports 25-hydroxycalciferol to the kidneys, where it undergoes recurrent hydroxylation. The most active metabolites, 1,25-dihydroxycalciferol (also known as calcitriol) and 24,25-dihydroxycalciferol, are produced by the kidneys at the mitochondrial level.

The kidney enzyme alpha-hydroxylase is involved in the production of the primary metabolite, calcitriol. The liver, muscles, and adipose tissue serve as vitamin D depots. Bile and urine are used by the body to remove decay products. The production of metallothioneins, namely calcium-binding protein, which facilitates calcium transport across the intestinal wall, declines with vitamin D insufficiency, lowering blood calcium levels. The synthesis of parathyroid hormone, which is primarily responsible for maintaining a steady level of calcium in the blood, rises when hypocalcemia promotes the parathyroid glands' activity.

The kidneys and bone tissue are the organs that this hormone targets. As a result, it promotes osteoclast activity in the bones, increases the leaching of inorganic calcium from the bones, and enhances the reabsorption of calcium in the kidneys. Phosphates in the urine rise as a result of decreased phosphorus reabsorption in the renal tubules caused by increased parathyroid hormone release. Acidosis happens, hypophosphatemia sets in, and the blood's alkaline reserve

drops. Phosphorus-calcium salts do not build up in the osteoid tissue in such circumstances. Because calcium salts seep out of the bones during calcification, the bones become pliable and prone to deformation.

Incomplete osteoid tissue development takes place in the growth zones concurrently. Internal organs and the central nervous system both malfunction as a result of acidosis. The immune system of the body weakens. The information provided on the metabolism and mode of action of vitamin D enables us to draw a conclusion that is crucial to a proper comprehension of the pathophysiology and etiology of rickets. The primary idea behind this finding is that the production of active forms of vitamin D and their reliance on transport, enzyme, and structural protein systems are crucial for the maintenance of calcium homeostasis inside the body.

It is thought that vitamin D₂ molecules primarily influence calcium metabolism in bone tissue, whereas the metabolites associated with the biliary chain vitamin D₃ primarily function in the intestinal absorption and transport of calcium ions. By activating osteoblasts and alkaline phosphatase-dependent receptors, the metabolite 24,25(OH)₂D₃ facilitates the development and mineralization of bone tissue. The transfer of calcium into the blood and the production of calcium-binding protein in the enterocytoma of the interstitial fluid from the gastrointestinal tract are 1,25(OH)₂D₃'s most noticeable homeostatic effects.

The mechanism of action of calcitonin, another thyroid hormone that helps maintain calcium-phosphate balance, is linked to an increase in osteoblast activity and a decrease in osteoclast activity and quantity. It eradicates all forms of osteoporosis by increasing the amount of calcium deposited in bone tissue.

The relationship of vitamin D with insulin

Insulin secretion is delayed in people with vitamin D insufficiency. Since calcium stimulates the pancreatic synthesis of insulin, this direct or indirect action is linked to an increase in calcium. It has been determined that vitamin

1,25(OH)₂D₃ and insulin secretion are related; insulin has the ability to increase the hydroxylation of vitamin D₃'s 1-position, which is linked to parathyroid hormone, and the ratio of active vitamin D₃ to diabetes. 1,25(OH)₂D₃ suppresses the growth of myelogenous leukemia cells while also preferentially differentiating and inducing cells of the monocyte and macrophage systems.

Immunomodulatory effect of vitamin D

Vitamin D is known to have immunomodulatory effects; interleukin production is altered. Osteoblast activity is decreased by the stimulation of interleukin-3 synthesis, whereas lymphocyte activation results from the delay of interleukin-2 generation. As a result, a wealth of fresh information has emerged in the research of rickets pathogenesis, broadening our understanding of the fundamentals of the disease process. There are a lot of unknowns, though, that need more investigation.

Numerous illnesses and clinical disorders are signs of calcium-phosphate metabolism violations. Specifically, rickets is directly linked to spasmophilia-convulsions or a propensity to twitch, as both disorders are linked to a disruption in the fundamental mineral-calcium metabolism. A calcium deficit causes muscles to become more excitable, a condition known as spasmophilia (sometimes termed tetany). This frequently results in the development of chronic seizures, particularly in newborns with high birth weights who are fed a "rachitogenic diet" throughout the first six months of their lives. Although the link between spasmophilia and rickets has long been known, it was established in the 1970s when all children with spasmophilia who were evaluated had low blood levels of 25-hydroxycholecalciferol.

In children older than 1-2 years, D-dependent and D-resistant rickets are very uncommon. An autosomal recessive condition known as phosphate diabetes (also known as vitamin D-resistant rickets or X-linked hypophosphatemia) is brought on by a mutation in the gene encoding the vitamin D receptor, which hinders the absorption of vitamin D. The disease's initial symptoms, which are

progressive, often show up around the second year of life. The condition may emerge early (before the end of the first year of life) or late (4–10 years). The conversion of the inactive metabolite of vitamin D into the active state (calcitriol) and a violation of phosphate reabsorption in the kidneys' proximal tubules constitute the pathogenetic process.

Children who get vitamin D to prevent rickets may develop vitamin D-dependent rickets, also known as pseudovitamin D-deficient rickets, between the ages of three and six months. It is characterized by decreased calcium and phosphate levels while alkaline phosphatase activity remains at normal levels. This is linked to the target organ receptors' hereditary resistance to calcitriol. Those who are married to second-degree relatives are frequently affected by this type of illness.

As a result, the primary metabolic processes that control calcium-phosphate metabolism have been thoroughly investigated. Because these processes are interdependent, pathological states arise when one of them is lacking. Determining the amount of this specific metabolite is a criteria for diagnosing and treating rickets and spasmophilia since the active form of vitamin D is crucial in controlling phosphorus-calcium metabolism.

1.3. Classification of rickets

- Classical vitamin D deficiency-rickets is divided into clinical variants, depending on the nature of the course, severity and periods of the disease.
- 1. Clinical variants of rickets are characterized by changes in the concentration of calcium and phosphorus in the blood serum. Distinguish the variants:
 - * calcipenic;
 - * phosphopenic;
 - * without pronounced changes in the level of calcium and phosphorus.
- 2. Downstream:

- acute - osteomalacia and neurological symptoms predominate. The bones of the skull soften and neurological symptoms appear: anxiety, sleep disorders.
- subacute - osteoid hyperplasia phenomena predominate. Bone tissue grows strongly, which increases the frontal, parietal and occipital tubercles. At the same time, the skull acquires the same square shape. Osteoid hyperplasia occurs when the bones enlarge where the bone section transitions to cartilage, such as in the ribs ("rosary"), wrist region ("bracelets"), and finger interphalangeal joints ("strings of pearls"). Typically, the skull, chest, and legs are malformed in the first three months of life, five to six months, and the second half of life, respectively.
- recurring (wave-like): Radiological, laboratory, and clinical evidence of active rickets in the past are also seen if the kid develops acute rickets.

- 3. *By severity:*

- *The first stage of rickets is represented by I—light. the early stages of a calcium, phosphorus, or vitamin D insufficiency. It is typified by mild symptoms and alterations. There may occasionally be noticeable muscular hypotension.*
- *II: Moderately severe alterations to the internal organs and skeletal system. moderate alterations in the hematopoietic, muscular, and bone systems, as well as a marked decline in overall health. The arms, thorax, and cranium all exhibit distinct deformities.*
- *III—severe—includes the development of rickets complications, serious harm to internal organs and the neurological system, and damage to several skeletal components.*
- *marked by significant harm to the central nervous system, internal organs, and skeletal system. Delays in mental and physical growth, inhibition, decreased appetite, and trouble sleeping are some of the symptoms of this. The cardiovascular system is not functioning properly.*

- 4. *Throughout its cyclical life, the illness progresses through four sequential stages:*

- *-the first time frame;*

the duration of residual phenomena; the convalescence phase; and the peak of the illness.

Secondary rickets often occur:

- * For chronic renal or biliary tract illnesses
- * For metabolic diseases (tyrosinemia, cystinuria, etc.)
- * With malabsorption syndromes
- * Caused by long-term use of glucocorticoids, diuretics, parenteral nutrition, and anticonvulsants (diphenin, phenobarbital).

Vitamin D-dependent rickets:

- Type I: a genetic abnormality in the kidneys' production of 1,25-dihydroxyvitamin D, or 1,25(OH)₂D.

Type II refers to the target organ receptors' hereditary resistance to 1,25(OH)₂D.

Rickets resistant to vitamin D:

Among them are:

- * Hypophosphatasia;
- * De Toni-Debré-Fanconi syndrome;
- * Phosphate diabetes; and
- * Renal tubular acidosis.

1.2. Prevention and treatment of rickets in early childhood

Nonspecific treatment entails setting up a suitable hygienic and sanitary routine that includes regular walks throughout the year, ample exposure to fresh air, and restful sleep, as long as the kid is shielded from too many outside stimuli (light, noise). During the waking hours, the child's mental and physical activities should be encouraged. A healthy diet and extra vitamin C and group B (B1, B2, B6) prescriptions are necessary for a youngster with rickets. Natural-feeding children require 0.25–0.5 g of calcium gluconate preparations or 1 teaspoon of calcium chloride 5–10% 2-3 times per day. Citrate combination is commonly

utilized; for one to one and a half months, one teaspoon is taken three to four times daily. Proserin and dibazol are used at age-appropriate dosages for severe muscular hypotension.

The child's age determines the diet. For the first three to four months of life, natural feeding or extracted breast milk is the healthiest option. This is especially important for preterm newborns who have rickets. When utilizing tailored mixes for mixed and artificial feeding, the doctor should adhere to the WHO/UNICEF guidelines for baby artificial feeding. Vitamin D and other vitamins should not be administered to a baby who is fed mixed or mixed milk and gets tailored combinations. As a result, 400 ME of vitamin D2 per liter is part of the Detolakt mixture's makeup. Acute respiratory infections are treated holistically. In order to correct bone mineralization, vitamin D3 is recommended.

Furthermore, calcium preparations are recommended for phosphate diabetes, phosphate crystalluria, and magnesium preparations. Acidosis, electrolyte imbalances, hypovitaminosis A, B1, B2, B5, and B6 correction, and etiopathogenetic management of gastrointestinal and nephrological disorders are all practiced. Children with a phosphopenic form of rickets are administered 0.5 ml of adenosine triphosphoric acid (ATK) intramuscularly once day or every other day for a course of 15 to 20 injections to enhance energy metabolism.

Massage and physiotherapy exercises are part of the complex of treatment approaches for all ill children two weeks following the initiation of medication therapy. Following the conclusion of therapy, parents are taught the massage technique and a series of therapeutic gymnastics at a children's polyclinic. These procedures are then carried out at home for a period of one and a half to two months. A regimen of vitamin D3 may be followed with a prescription for mercury-quartz lamp irradiation. After evaluating each person's susceptibility to ultraviolet radiation (biodoses), UV lamp radiation is administered daily or every other day for a total of 1/2–1/4–4 biodoses to distinct locations with a focal length of 50–100 cm. The treatment course lasts for 20–25 days.

It is advised that children older than six months get balneotherapy in the form of herbal, saline, or coniferous therapeutic baths. Children who are easily agitated should take coniferous baths. Ten liters of water at 36 °C are mixed with one teaspoon of natural liquid coniferous extract or a standard briquette strip. After five minutes, the steam bath is prolonged to six to ten minutes; a total of twelve to fifteen baths are advised for the course. Every day or every other day, they are performed.

Children who are slow, lethargic, and have a noticeable low subcutaneous fat base are the greatest candidates for salt baths.

Two teaspoons of table or sea salt are dissolved in ten liters of water that is between 35 and 36 degrees Celsius. Every day, eight to ten salt baths are performed. The youngster is given clean water after the salt bath. Children with exudative diathesis are advised to take herbal remedy baths. Equivalent amounts of plantain leaves, horsetail, chamomile, calamus root, and oak bark are combined, and the mixture is cooked at a rate of one tablespoon per liter of water.

The primary dietary sources of calcium are milk and dairy products, which, depending on the kind of product, can contain anywhere from 120 to 1000 mg of calcium per 100 g (see table). Therefore, 20–30% of the daily calcium need can be met by a single glass of milk (200 ml). The remaining food categories have comparatively low calcium contents (10–50 mg/100 g), with the exception of legumes, which have 100–150 mg/100 g.

In contrast to chocolate, which has a high calcium content (up to 215 mg/100 g) combined with a high oxalic acid content that hinders adequate calcium absorption, dairy products, particularly cheese and coIDAge cheese, are not only rich in calcium but also contain it in an easily digestible form and in an ideal ratio with phosphorus.

Product	Calcium content, mg /	The amount of the
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	100 g	product containing the physiological norm of calcium need (800-1200 mg)
Milk, kefir 3,2%	120	650-100 ml
Yogurt, 3,2%	119-122	650-100 ml
Cream, 10%	90	1000-1300 g
CoIDAge cheese, 9%	164	500-730 g
Hard cheese	1000	100-120 g
Legumes (peas, beans)	115-150	500-1200 g
Milk chocolate	150-215	500 g
Vegetables, fruits, berries	20-50	1,5-6 kg
Meat	10-20	4-12 kg
Fish	20-50	1,5-6 kg
Soybeans	300	250-350 g
Bread	20-40	2-6 kg

Therefore, a sensible diet that gives kids vital and irreplaceable nutrients in addition to calcium and vitamin D is a good way to avoid osteopenia and osteoporosis.

Phosphorus-calcium metabolism disruption is recognized as one of the primary signs of rickets. This results from variations in the rate at which minerals are absorbed, used, and excreted. Many researchers utilize blood calcium and phosphorus levels as a diagnostic tool for rickets and to track how well treatment is working for the condition.

Although this approach is obviously far less expensive, a more precise indicator—the measurement of active metabolites of vitamin D in the blood—must be used to diagnose rickets. It should be mentioned that in industrialized nations, measuring the amount of the liver metabolite 25(OH)D₃ is the benchmark for figuring out how much vitamin D is needed to prevent and cure rickets. The

following marginal amounts of this percentage in serum have been determined by both domestic and international authors: 10-30 ng/ml (32); 36.2 ng/l (Ageikin); 20-40 ng/ml (30.31).

Depending on race, age, season, and food, normative values of 25 (OH) D₃ and less than 1.25 (OH) 2D₃ and 24.25 (OH) 2D₃ vary. They also rely on the techniques employed. In Russia, rickets prevention and treatment are conducted in compliance with the 1990 USSR Ministry of Health methodological guidelines. At three to four weeks of age, full-term infants are recommended to take 400–500 IU of vitamin D daily as a preventative measure. First-degree premature children are provided 400–1000 IU of vitamin D per day for two years, except the summer, starting at 10–14 days of age.

For second- to third-degree premature neonates, vitamin D is administered daily at a dose of 1000–2000 IU for the first year and 500–1000 IU for the next year, except the summer. In Russia, 1000 ME per day is the recommended daily dosage to prevent rickets, whereas 400,000 ME is the recommended course dose. The recommended daily dosage for treating rickets is between 20,000 and 40,000 ME. The course dose for first-degree rickets is 600,000 ME, for second-degree rickets it is 800,000 ME, and for third-degree rickets it is 1,000,000 ME.

There are inconsistencies in the preventive dosages here since, although the daily therapeutic dose for rickets therapy is 2000–5000 ME, it is replaced with a prophylactic dose (400–500 ME) for two years when a therapeutic effect is achieved for 30–45 days. It is sufficient to increase the prophylactic dose of vitamin D by 1.5–2 times during the first degree of rickets and up to 1600–2000 ME (course dose of 200,000 ME) during the manifestation period. Under the supervision of the Sulkovich reaction, the daily dose of vitamin D can be increased to 4000 ME during the second and third degree manifestation periods (course dose of 400,000–600,000 ME).

The "half-effect technique" is employed in several nations, which involves introducing 100,000 MED of vitamin every three months (or two weeks for preterm kids) beginning at one month. However, prophylactic vitamin D is not

necessary for children who live in mid-latitudes, are nursing (one liter of breast milk contains 20–60 MED of vitamin), and spend 30–120 minutes a week in the sun (on their arms and head).

Rickets has been effectively avoided for many years by using vitamin D preparations in the form of oil solutions or fish oil. However, these medications' release and dose forms, together with their distinct taste and smell, have made providing them to young children challenging. Sadly, this has frequently resulted in non-compliance with medical advice. It should be mentioned that there are now differing views in pediatrics on the necessity of rickets prevention and the best ways to carry it out. Many clinicians mistakenly believe that prophylaxis is optional, despite recent research on vitamin D and the decline in the prevalence of rickets in youngsters.

Large follicular size and modest volume of posthypoxic encephalopathy are not contraindications for the prescription of preventive dosages of vitamin D, according to several research. However, children with hemorrhagic encephalopathy younger than one and a half months should not be prescribed vitamin D dosages. It is necessary to administer vitamin D to infants throughout their first year of life in order to avoid rickets. State-level health officials are responsible for implementing this preventative action. Naturally, the occurrence of severe and moderate types of rickets will be eliminated as a result of the methodical execution of this approach.

The disease's incidence is still somewhat high, though. Despite receiving preventative doses of vitamin D, some kids have mild types of rickets. It is evident that the preventative aim has not been met. It should be mentioned that treating phosphorus-calcium metabolism issues in children growing up in the present world requires adequate prevention and treatment of rickets. As a result, only when there is adequate protein in the diet could rickets be prevented and treated.

For three years, children with moderate to severe rickets should be monitored by a doctor. Every three months, they are inspected. For the first two

years of life, specific prevention is done in the fall, winter, and spring; for the third year, it is only done in the winter. Rickets is a minor condition when diagnosed early and treated appropriately. If moderate and severe rickets is not treated, children with this condition frequently develop flattening and deformation of the scapula, chest deformities, flat feet, the development of myopia, and cavities of numerous teeth in addition to cosmetic flaws (deformity of the head, curvature of the legs).

Infants have a higher risk of respiratory conditions, pneumonia, chronic inflammation, and serious gastrointestinal disorders. Due to lumbar lordosis, women who have had childhood rickets may have smaller lung entrance and exit volumes, which frequently results in a cesarean section at the conclusion of the birthing process. However, it should be noted that a change in the echo signal during an exoencephalogram of the brain is one of the earliest indicators of rickets development in babies.

The ventricular index varies between 2.0 and 2.6. In children with perinatal disease, rickets symptoms are identified at 3–4 months and are characterized by an increase in the frequency of echoes and pulsations from the medial walls of the ventricular system. A two-peak M-exo echo revealed a 7-9 mm rise in the third ventricle.

When rickets symptoms were seen in children without prenatal illness between the ages of two and five months, they were accompanied with a somewhat noticeable hydrocephalic condition. Increased pulsing of echo signals and uneven development of the skull's interior diameter were noted, while the amplitude values were lower.

As a result, the authors were able to draw the conclusion that exoencephalographic examinations of rickets patients aid in the early detection of hypertension-hydrocephalic syndrome, differential diagnosis, and pathogenetic therapy. It should be mentioned that the liver produces microsomal enzymes that render vitamin D and its metabolites inactive when anticonvulsants (phenobarbital, diphenin) are taken for an extended period of time to treat newborns with perinatal

encephalopathy. Furthermore, they disrupt calcium homeostasis without influencing the metabolism of vitamin D. This is particularly crucial since, because of the serious effects and quick recurrence, the issue of hypoxia encephalopathies has not lost its substance or significance.

Four to twenty percent of full-term babies have this condition, while preterm babies have it twice as frequently. Standards for the efficacy of rickets prevention and treatment:

1. Young children show no symptoms of the illness.
2. The incidence of rickets in young children has decreased, as has the specific gravity of moderate and severe forms of the condition.
3. Improvements in the health index, a decrease in overall morbidity in children, and positive dynamics of physical development markers in early children.

Particularly in the pathways leading to bone mineralization and bone mass formation, vitamin D is essential for maintaining calcium and phosphorus homeostasis.

Recent research has revealed that vitamin D has significant extracellular effects in addition to these traditional skeletal effects, which can make the progression of rickets more difficult. Since there aren't many natural sources of vitamin D, dietary supplements are the primary way to prevent and cure rickets. Thus, the question of rickets' significance in young children's pathology may now be approached from a different angle thanks to major advancements in early childhood ricket prevention, the eradication of its societal roots, and our growing understanding of the disease's progression.

Without a doubt, the findings of the study and the observations of pediatricians working in clinics for infants in their first year of life regarding rickets prevention should aid in the creation and enhancement of strategies for preventing and treating this illness. It should also be determined by the amount of 25(OH)D₃ present in the blood serum. The metabolically active forms of vitamin D are taken into consideration when correcting for rickets in both domestic and international nations. Regretfully, our Republic does not have any such studies.

In this observation, we aim to identify latent vitamin D deficiency and thus prevent the development of rickets in children, as well as to prevent hypocalcemic seizures.

CHAPTER II. RESEARCH MATERIALS AND METHODS

2.1. Characteristics of the children under study

Clinical examinations, questionnaires, and outpatient records from the Central Hospital of Akdarya District and polyclinics No. 3, 6, 8, and 12 in Samarkand were used to evaluate the children. The parents were informed of the study's goal, and their formal agreement was acquired. The research eliminated those children who had taken vitamin D supplements for at least one month prior to the visit, had illnesses, or were not exposed to enough vitamin D. A questionnaire in Uzbek was given to all moms. Pregnancy, development, sunlight exposure, medical history, and ethnic origin were among the subjects covered in this questionnaire.

466 infants between the ages of one and twelve months were observed. Every kid, who was thought to be in generally good health, had a physical examination to check for the clinical symptoms of rickets, which include pale skin, excessive perspiration, delayed teething, rachitic rosary, wide wrist, muscular weakness, dystrophy, delayed closure of the huge fontanelle, and psychomotor development. Consequently, the kids were split up into three groups: Children in Group 1 (healthy) (263 children, 56.4%), Group 2 (rickets-symptomatic 130 infants, 27.9%), and Group 3 (children with the effects of prenatal nervous system injury, 73 children, 15.7%) (Fig. 2.1.1).

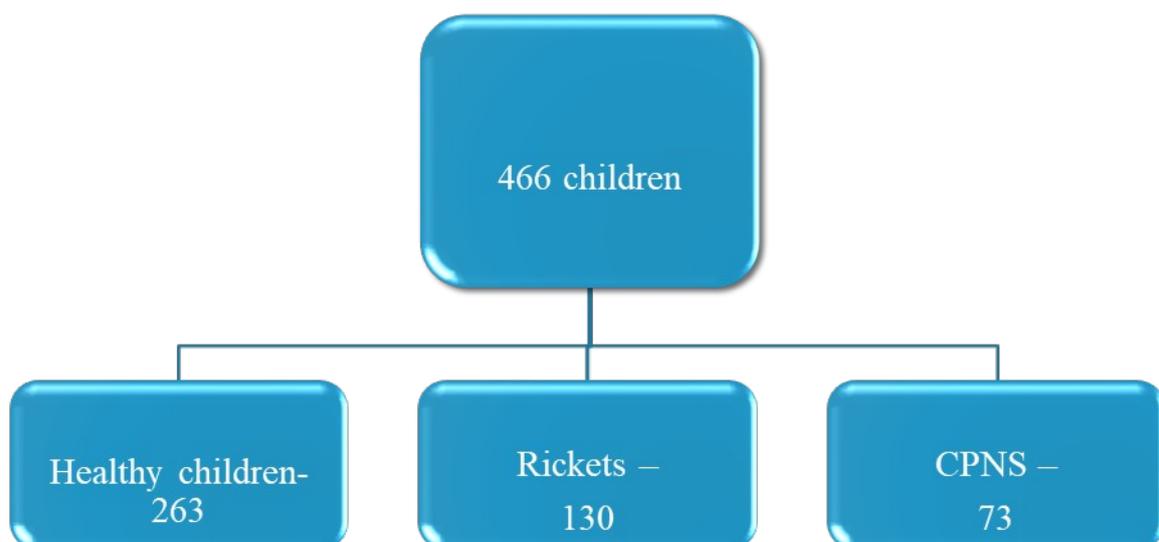


Figure 2.1.1. Distribution of examined children by groups.

The distribution of children by age is shown in Figure 2.1.2. As can be seen from Figure 2.1.2, there were 44 (33.9%) children with rickets at the age of 6 months, and 56 (43.1%) at the age of 12 months. The number of children under 3 months was 30 (23%).

Figure 2.1.2 Distribution of examined children by age.

The distribution by gender is shown in Figure 2.1.3. As can be seen from the presented material, we did not identify significant gender differences. The number of boys with rickets was 70 (53.9%), and the number of girls was 60 (46.1%).

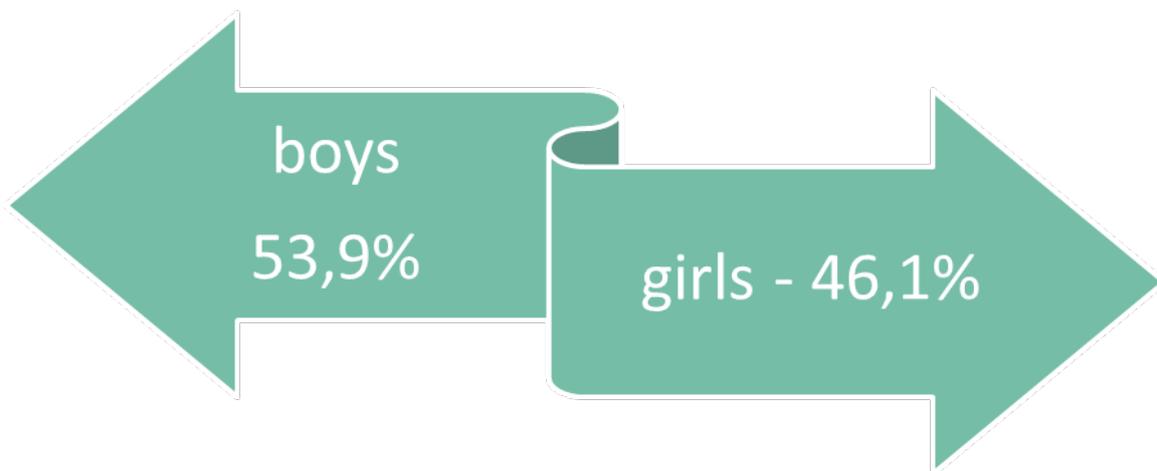


Figure 2.1.3. Distribution of examined children by gender.

An important factor in the development of rickets is the weight of children at birth. The study of this indicator showed that 12 (9.2%) children had a birth weight of 2000 - 2500 g, 104 (80%) - 2600-3500 g, 11 (8.5%) children - 3600-4000 g, 3 (2.3%) children - more than 4000 g.

Table 2.1.1

Weight of children diagnosed with rickets at birth

Birth weight, grams	2000-2500	2600-3500	3600-4000	high 4000
number of children	12	104	11	3
% children	9,2	80	8,5	2,3

2.2. Research methods

Biochemical (2 oh) d3, calcium and phosphorus, shown in Figure 2.4 in Figure 2.4 of the diagnosis). A consultation was given by the doctors of the narrow specialty.

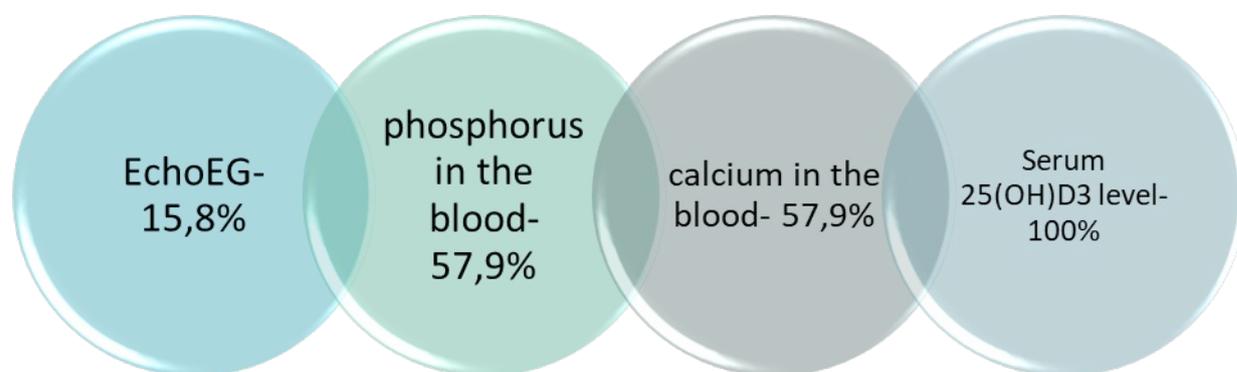


Figure 2.1.4. The volume of studied research.

Biochemical methods.

calculating the blood serum's 25 (Oh) D3 content. Each youngster was given two milliliters of Vienna blood. For ten minutes, the serum was separated by centrifugation at 3000 AIL/min. Blood has been kept at 200 degrees Celsius. Radioemuns (Netherlands) in Rotterdam measured the blood serum's 25 (Ohs) D3 content. A result less than 30 nol/l was considered to be indicative of vitamin D3 deficiency.

Determination of phosphorus in the blood.

The process of creating a malachite green complex with phosphomolybdic acid was used to measure phosphorus. The usual amount of inorganic phosphorus in blood serum or plasma is 1-2 mmol/l, or 3-6 mg per 100 ml. measurement of blood serum calcium levels. The method of color reaction with murexide in the presence of glycerol was used to detect calcium. The calibration table was followed when doing the computation. 2.3-2.75 mmol/l (9–10 mg per 100 ml) is the typical amount.

Instrumental methods.

The capacity of ultrasonic vibrations to reflect off the borders of different brain areas is the basis for the ultrasound echolocation recording technique known as exoencephalography. The "Exo-12" portable exoencephalograph equipment (norm 7.0-9.0) was used to conduct the procedure.

Methods of statistical processing of research results and data analysis.

Using the variational statistics approach, the findings were analyzed and processed on a Pentium 4 SyncMaster 793 DF personal computer using the Microsoft Excel correlation database. The so-called analysis package, a collection of data analysis capabilities included with Microsoft Excel, is intended to address challenging statistical and engineering issues. Using widely recognized techniques, the arithmetic mean (M), its error (m), and the standard deviation were computed in order to statistically analyze the findings (41). The Studen test (t) was used to assess the dependability of quantitative data differences (42).

The least squares approach was used to analyze correlation. We employed the Chi-square test of conjugation tables with Yetsa adjustment for continuity (degree of freedom in each case $v = 1$) to determine risk variables. Using SPSS software version 10.1, data on the measurement of 25(OH)D3 in blood serum were examined. For data with a normal distribution, the difference between the mean and the standard deviation ratio was computed. P-values less than 0.05 were regarded as statistically significant. Children were deemed to have a biochemical deficit if their 25(OH)D3 level was less than 30 mmol/l.

CHAPTER III. RISK OF RICTIS DEVELOPMENT IN CHILDREN AND COMPARATIVE CHARACTERISTICS OF SERUM 25(OH)D3

3.1. Assessment of risk factors for vitamin D deficiency in children

We surveyed 466 healthy children who had not received treatment in the preceding month in order to determine the risk factors for the onset of vitamin D insufficiency in one-year-olds. Serum 25 (OH) D3 levels were reported to be low in 360 children (77.2%) and normal in 106 children (22.8%). A thorough clinical evaluation revealed that 15.7% of infants had the effects of prenatal injury to the nervous system (CPNS), and 27.9% of children had rickets. In order to detect different premorbid conditions of phosphorus-calcium metabolism, we employed CPNS as a second control group. We divided children into 2 groups according to the level of the main metabolite of vitamin D (Table 3.1.2).

Table 3.1.2

Frequency of normal and low serum 25 (OH) D3 levels in the examined children.

Children	low level of	normal	Total
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	25(OH)D3	25 (OH) D3 levels	
Healthy children	198 (75,2%)	65 (24,8%)	263 (56,4%)
Children with signs of rickets	112 (86,1%)	18 (13,9%)	130 (27,9%)
Children with CPNS	50 (68,4%)	23 (31,6%)	73 (15,7%)
Total	360 (77,2%)	106 (22,8%)	466 (100%)

Table 3.1.2 shows that children in the CPNS group and healthy children had the same low level of 25(OH)D3. In 86.1% of children with rickets, a tendency toward a more severe shortage of the vitamin D metabolite was seen. Early childhood diseases that increase the risk of developing rickets are quite significant. According to the investigations that were done, respiratory infections were the most prevalent illness in all groups, although they were more prevalent in CPNS and rickets. For IDA, there was no discernible difference between healthy children and CPNS, but for rickets, this signal was three times greater.

We can say that when IDA is added to latent vitamin D deficiency, it manifests itself with rickets (Table 3.1.3.).

Table 3.1.3

Frequency of diseases in children in the anamnesis

Clinical diagnosis	Healthy %	Rickets %	CPNS %
URVI	45,2	52,3	58,9
Diarrhea	2,8	3,0	1,3
IDA	16,3	50,0	13,6

A different sample of 141 children up to 6 months old was chosen in order to ascertain the relationship between vitamin D insufficiency and food type. It was discovered that children with rickets and healthy infants were mostly breastfed, and that mixed feeding was more prevalent for children with rickets. Just 10.5% of healthy children and 12.5% of children with clinical indications of rickets were on

artificial feeding. Of the 141 children, only 74 (52.5%) were breastfed, and 46 (62.1%) of them were healthy, according to the analysis of absolute markers. Just 5.4% (4) of rickets-affected youngsters were on natural feeding, and only 32.4% of them were. Thus, it may be concluded that one of the most effective ways to avoid rickets is by nursing.

(Table 3.1.4).

Table 3.1.4

Distribution of children under 6 months of age by type of nutrition

Feeding	Healthy	Rickets	CPNS	Total
Breast milk	62,1%	32,4%	5,4%	74 (52,5%)
Artificial	10,5%	12,5%	-	14 (10,0%)
Mixed	54,7%	18,8%	22,6%	53 (37,5%)

To identify risk factors for the development of pathologies in young children, we analyzed risk factors from mothers based on a questionnaire (Table 3.1.5).

Table 3.1.5

Prenatal risk factors for the development of rickets

Risk factors	Number of mothers	
	abs.	%
Lack of vitamin D intake during pregnancy		
Iron deficiency anemia	397	85,1
Unbalanced diet	289	62
Maternal age (up to 20 years)	275	59
Low level of education	252	54
Risk factors	226	48,4
Complicated births	223	47,8
Toxemia of pregnancy	64	13,7

Total	466	100
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The primary risk factors for the development of rickets in young children, according to a questionnaire survey given to mothers during pregnancy, were iron deficiency anemia (62%), unbalanced nutrition (59%), low vitamin D intake (85%), and the mother's young age at the time of the first pregnancy (54%). Only 13.7% of pregnant women had toxicosis, and less than 50% of cases had both difficult delivery and low maternal education. We selected 88 women with IDA based on the questionnaire data in order to shed light on the relationship between iron deficiency anemia during pregnancy and using iron-containing medications and the development of rickets in offspring.

Of them, 63.6% did not get any therapy, while 32 (36.4%) received ICD. When we examined 25(OH)D levels after the babies were born, we discovered that 81.2% of the infants whose mothers had taken ICD during pregnancy had low levels, whereas 87.5% of the children whose mothers had not taken ICD had somewhat higher levels. (Table 3.1.6).

Table 3.1.6

Serum 25(OH)D3 levels in children according to maternal ICD intake during pregnancy.

	Low level 25(ON)D	Normal level 25(ON)D
IDA + ICD =32	81,2%	18,8%
IDA – ICD =56	87,5%	12,5%

It was shown that infants whose mothers got long-term ICD throughout pregnancy had lower levels of calcium and phosphorus (Ca and P, respectively), measuring 1.93 ± 0.04 and 0.81 ± 0.02 mmol/l. Ca and P levels were 2.09 ± 0.05 and 0.97 ± 0.01 mmol/l in infants whose mothers did not get ICD, respectively.

It is reasonable to expect that offspring of pregnant women who had long-term ICD may later have low serum Ca and F levels. We discovered that low serum 25 (OH) D3 levels and poor rickets prevention during the first year of life are the two main risk factors for children developing rickets. (Table 3.1.7).

Table 3.1.7.

Postnatal risk factors for the development of rickets

Risk factors	Number of children	
	Abs.	%
Low serum 25(OH)D3 levels in children	360	77,2
Inadequate prevention of rickets with vitamin D in the 1st year of life	321	68,8
Severe acute respiratory infections	230	49,3
Insufficient exposure to fresh air (less than 20 min per day)	180	38,6
Time of birth (winter-autumn season)	151	32,4
Perinatal factors	150	32,1
Iron deficiency anemia	119	25,5
Low blood calcium levels	112	24
Low blood phosphorus levels	80	17,1
Birth weight more than 3500 g	76	16,3
Increased intraventricular complex on echocardiography	67	14,3
Mixed feeding (children under 6 months)	50	10,7
Premature birth	43	9,2
Total	466	100

Rickets development is significantly influenced by a number of factors, including prior acute respiratory infections (49.3%), less than 20 minutes of fresh air per day

(38.6%), the time of birth (autumn-winter period) (32.4%), perinatal factors (32.1%), and IDA (25.5%). Body weight greater than 3500g and mixed feeding are less significant. A closer look revealed that the prevalence of preterm birth in the general population was 8.7%, despite the fact that 9.2% of premature newborns were detected. Therefore, the mother's young age and inadequate vitamin D consumption during pregnancy were the most frequent prenatal causes for the development of rickets.

Future rickets development was significantly influenced by low maternal skills, education, and difficult childbirth; pregnant women's toxicosis had no effect. B. Mamatkulov states that rickets occurs if the parents have an incomplete secondary education (62.6%) and are students (81.8%). A.I. Ryvkin states that rickets develops as a result of maternal obstetric pathology and extragenital (24.5%) factors.

According to an analysis of postnatal variables, the primary cause of rickets development in our region is the conventional preventive measures used, which result in a vitamin D deficit in the blood of 77.2% of children.

IDA, autumn-winter births, and recurrent colds also contributed. There is evidence that breastfeeding can effectively prevent vitamin D insufficiency.

According to our statistics, 79.3% of children with rickets had recurrent bronchopulmonary illnesses, and 7.9% had iron deficiency anemia (Mukhamedov Kh.T. and T. Ryvkin A.I. A.I. Ryvkin claims that just 18.8% of youngsters employed natural protective factors, 68.7% used targeted prevention, and 72.2% used at least three hours of fresh air. This implies the necessity of creating rickets prevention strategies and a unique vitamin D therapy strategy.

In conclusion, I would like to state that the most frequent variables that predispose children and their mothers to acquire rickets were vitamin D deficiency, malnutrition, and the lack of preventative interventions such therapeutic gymnastics, massage, and outdoor walks. This implies the necessity of creating rickets prevention strategies and a unique vitamin D therapy strategy.

3.2. Features of the symptoms of rickets in children with perinatal damage to the nervous system

As previously mentioned, of the 466 children that were analyzed, 360 (77.2%) had vitamin D insufficiency, 130 (21.9%) had clinical rickets, and 73 (15.7%) had ASD. Birth asphyxia, persistent intrauterine hypoxia, intracranial birth trauma, cerebral circulation problems, and preterm delivery were among the diagnoses made for patients with ASD who frequently had respiratory conditions in their anamnesis.

It is evident that not every youngster with a vitamin D deficit develops rickets. We performed a comparative study of the variables that contribute to children developing phosphorus-calcium metabolism problems.

Rickets were categorized using Lukyanova Ye.M., Antipkin Yu.G., and Omelchenko L.I. (1991). Figure 3.2.5 shows that 84 (64.7%) of the children had mild rickets, with 53 (63%) having an acute course and 31 (37%) having a subacute course. Of the 46 children, 130 (35.3%) had moderate rickets, of which 25 (54.3%) had an acute episode and 21 (45.7%) had a subacute course.

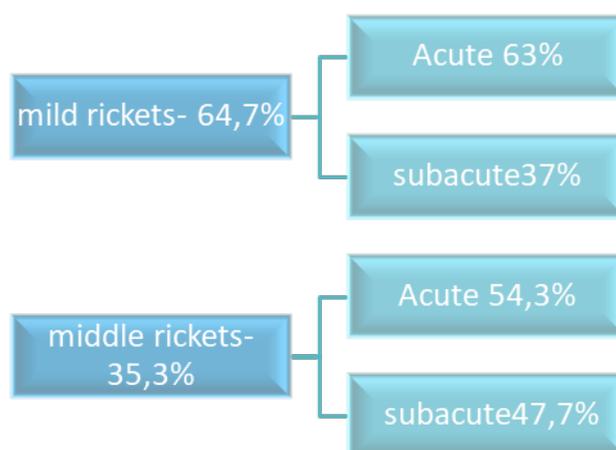


Figure 3.2.5. Distribution of children by severity and course of rickets.

When children with rickets symptoms were examined, 112 out of 130 (86.1%) had a low level of 25(OH)D₃ in their blood serum, including 77 out of 84 (91.6%) children at the early stages of the illness. Vitamin D insufficiency was found in 35 out of 46 youngsters (76.0%) during the height of rickets development.

Of the 18 (13.8%) children whose blood 25(OH)D3 levels were normal, 7 out of 84 children (8.3%) had the first stage of rickets, and 11 out of 46 children (23.9%) had the peak period.

According to our research, vitamin D insufficiency is a significant factor in the early phases of a child's growth, and at the peak time, it is also associated with a lack of phosphorus-calcium complexes. It should be mentioned that the first and second pregnancies accounted for 61.5% (80) of the children born, the third pregnancy for 25.3% (33) and the fourth and later pregnancies for 13% (17). Children with rickets have a history of early birth, colds, lack of sunshine, poor nutrition, and inactivity.

To determine the risk factors for the emergence of pathological illnesses and disorders, a multifactorial analysis was performed on all children. The analyzed children's current pregnancy and delivery, as well as their mothers' somatic and obstetric-gynecological anamnesis, were also investigated.

The chi-squared test was computed using Yetsa's adjustment for consistency (in each instance, the degree of freedom $v = 1$) for conflict tables in order to evaluate risk factors for rickets. (Table 3.2.8). Chi-squared values were dependable for evaluating prenatal risk factors in children with clinical signs of rickets. These included iron deficiency anemia (4.096; $P < 0.043$), malnutrition during pregnancy (10.064; $P < 0.002$), and lack of vitamin D intake during pregnancy (40.059; $P < 0.0001$), as well as maternal age at one pregnancy (up to 20 years) (0.418; $P > 0.518$). There were no notable variations discovered. In mothers of children with ASD, the chi-squared value was as follows: iron deficiency anemia was a significant risk factor for rickets (20.132; $P < 0.0001$), as was a lack of vitamin D intake during pregnancy (8.609; $P < 0.003$), malnutrition during pregnancy (3.37; $P < 0.072$), and the first gestational age (up to 20 years) was 0.761 ($P < 0.383$). No significant differences were found.

Table 3.2.8

X²-squared criterion for prenatal rickets risk factors

maternal risk factors	Rickets	CPNS
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Iron deficiency anemia	χ^2 -square = 4.096 P < 0.043	χ^2 -square = 20.132 P < 0.0001
Insufficient vitamin D intake during pregnancy	χ^2 -square = 40.059 P < 0.0001	χ^2 -square = 8.609 P < 0.003
1 pregnancy (up to 20 years)	χ^2 -square = 0.418 P > 0.518	χ^2 -square = 0.761 R < 0.383
Poor nutrition during pregnancy	χ^2 -square = 10.064 P < 0.002	χ^2 -square = 3.237 P < 0.072

Furthermore, it was interesting to see how the chi-square test was calculated to elucidate postnatal risk variables (Table 3.2.9). Additionally, we found that the following risk factors for rickets were highly reliable: iron deficiency anemia 7.083 (P < 0.008), time of birth (autumn-winter period) 5.014 (P < 0.025), perinatal factors 8.516 (P < 0.004), lacking vitamin D prophylaxis in the first year of life 4.334 (P < 4.337), and inadequate exposure to the outside world (less than 20 minutes) 9.395 (P < 0.002).

Table 3.2.9

Chi-squared test for postnatal risk factors for rickets

Risk factors in children	Rickets	CPNS
Insufficient prophylaxis with vitamin D in the 1st year of life	χ^2 - square = 4.334 P < 0.037	χ^2 - square = 1,55 P < 0.25
Insufficient exposure to fresh air (less than 20 minutes)	χ^2 - square = 9.395 P < 0.002	χ^2 - square = 17.171 P < 0.0001
Time of birth (autumn-winter period)	χ^2 - square = 5.14 P < 0.025	χ^2 - square R > 0.5
Iron deficiency anemia in children	χ^2 - square = 7.083	χ^2 - square R > 0.5

	P < 0.008	
Perinatal factors	x ² - square = 8.516 P < 0.004	x ² - square = 4.228 P < 0.040
Premature birth	x ² - square R >0.05	x ² - square = 30.559 P < 0.0001

The x²-squared value shown good reliability for the following risk factors in children with CPNS: limited environmental exposure, perinatal variables, and premature delivery. However, as risk factors, IDA and the time of birth were unreliable.

Therefore, the examination of risk factors for rickets development during pregnancy will reveal that prognostic indications include an imbalanced diet, the mother's IDA, and inadequate vitamin D consumption throughout pregnancy. Low environmental exposure, prenatal variables, the child's birth time, and vitamin D prophylaxis are among postnatal factors that can also be used as predictive markers for the development of rickets.

Premature delivery and other perinatal variables were more significant in the development of CPNS at the same period. Furthermore, rickets in children is one of the elements that contribute to the development of CPNS, suggesting that these disorders are related in some way.

3.3. Comparative comparison of 25(OH)D3 levels and biochemical parameters in the examined children.

As was mentioned in the previous chapter, rickets is largely caused by inadequate vitamin D consumption. We examined biochemical markers and attempted to ascertain the connection between vitamin D insufficiency and blood levels of calcium and phosphorus in order to assess the significance of calcium and phosphorus consumption. It should be mentioned that the vitamin's hydroxylated derivative, which guarantees regular intestinal absorption of calcium, is its active form. According to the investigations, just 18 (13.9%) of the patients in the group

of children with rickets had a normal level of 25(OH)D3 in their blood serum, while 112 (86.1%) of the patients under examination had a low level.

The amount of this metabolite in children with CPNS was low in 50 (68.4%) of the patients who were evaluated, and normal in 23 (31.6%) of the children. (Fig. 3.3.6).

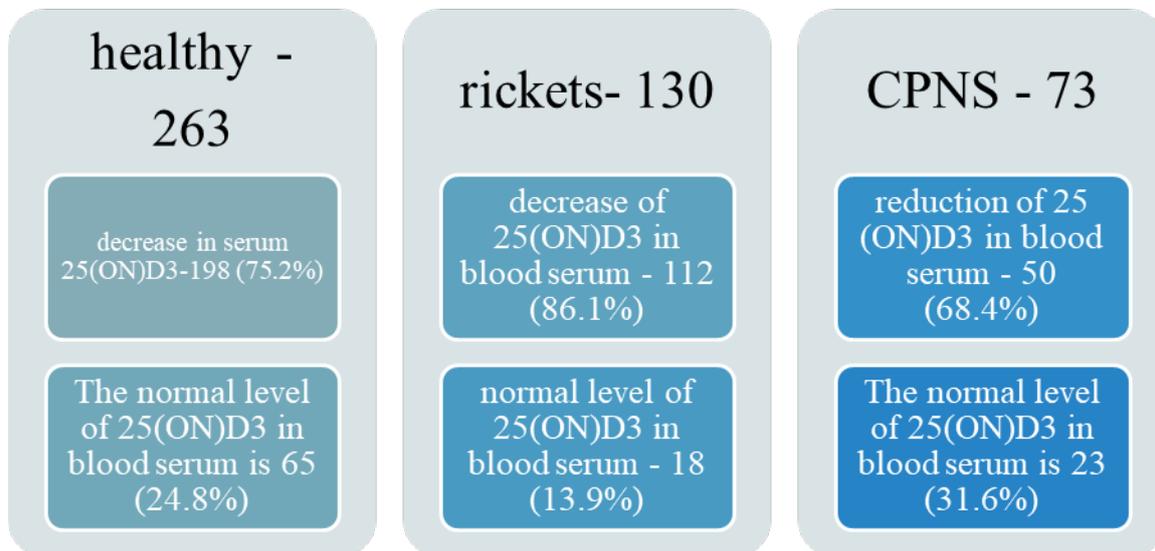


Figure 3.3.6. Frequency of occurrence of normal and low levels of 25 (OH) D3 in the blood serum of the examined children.

According to Figure 3.3.6, of the youngsters in the healthy group, 75.2% had low blood serum levels of 25 (OH) D3, whereas 24.8% had normal levels. The normal level was 77.34 ± 11.36 nmol/l, whereas the low level in the group of kids with CPNS was 16.39 ± 0.78 nmol/l. According to an analysis of the metabolite's composition in the group of children with rickets, the low level of 25 (OH) D3 was 13.77 ± 0.35 nmol/l, while the normal level was 62.72 ± 9.17 nmol/l.

Determining the biochemical parameters in rickets based on the blood serum level of the primary metabolite of vitamin D was intriguing. (Figure 3.3.7).

Blood Ca and P indicators depend on the amount of 25(OH) D3 in the blood serum in rickets

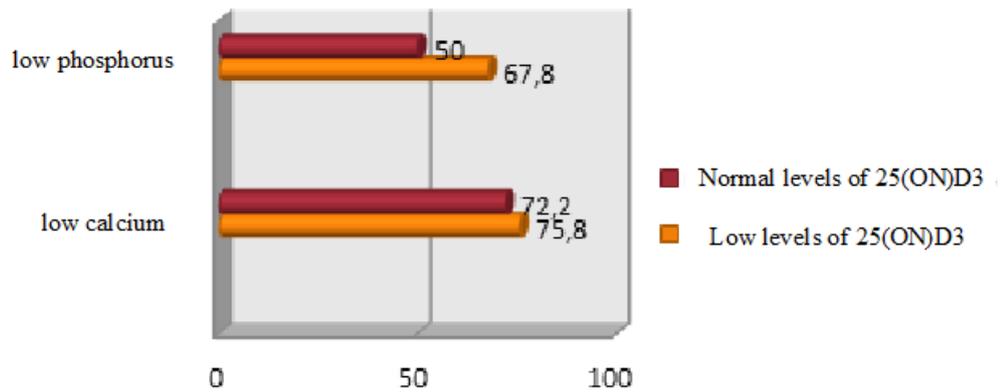


Figure 3.3.7. Blood Ca and P indicators depend on the amount of 25(OH) D3 in the blood serum in rickets.

As can be seen from Figure 3.3.7, calcium and phosphorus levels were normal in 44.4% of cases, while low levels were noted in 16.6% of cases, with a normal level of 25(OH) D3 in the blood serum. Therefore, we may state that a kid has vitamin D-resistant rickets if their blood serum level of 25(OH) D3 is normal but their calcium and phosphorus levels are low. We did not, however, consider such youngsters. 15.1% had normal calcium and phosphorus levels but a low 25(OH) D3 level.

Low blood 25(OH)D levels of 19.8 ± 1.98 nmol/l and low serum calcium and phosphorus levels (1.99 ± 0.019 and 0.92 ± 0.011 mmol/l, respectively) were seen in children with rickets (Fig. 3.3.8). Meanwhile, 25(OH)D levels were 5.04 nmol/l in 36.7 and 0.0524 and 0.044 mmol/l in 2.22 and 0.044 mmol/l in 1.21 in children with CPNS.

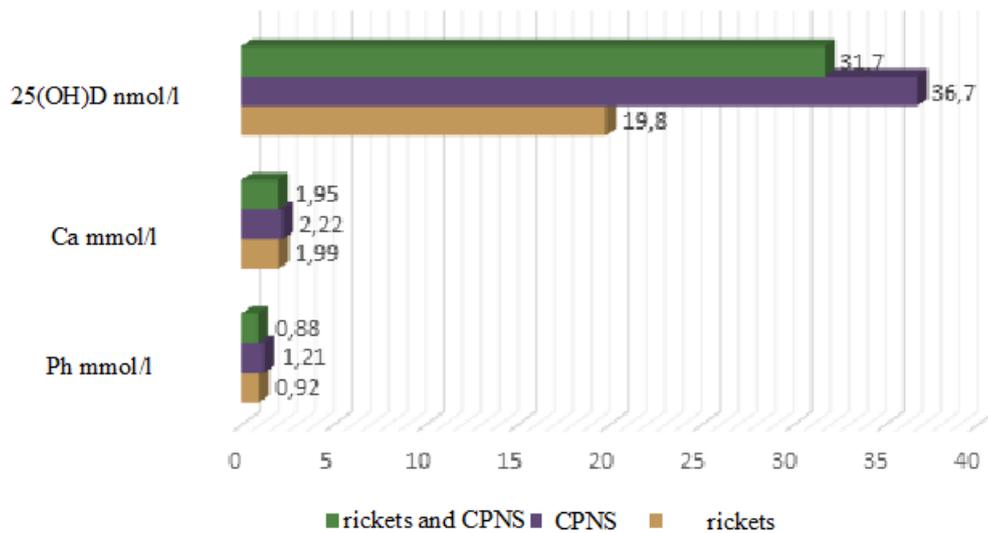


Figure 3.3.8. Analysis of serum Ca and P levels in children with rickets, CPNS, and their combination.

Serum calcium and phosphorus levels in the children with CPNS and rickets were 1.95 ± 0.04 and 0.88 ± 0.028 mmol/l, respectively. These levels were marginally lower than those in the babies with rickets, and the level of 25 (OH) D was 31.7 ± 14.8 nmol/l.

As is evident from the information provided, low blood serum levels of calcium, phosphorus, and the primary metabolite of vitamin D promote the development of rickets, but these markers have a lower value when CPNS is present. It was also interesting to see how the youngsters under examination correlated with the aforementioned indications.

To ascertain the link between serum 25(OH)D3 concentration and serum Ca concentration, we employed the Pearson (r) correlation coefficient.

Figure 3.3.9. Correlation between serum 25(OH)D3, calcium, and phosphorus in rickets.

Research has indicated that the positive association between 25(OH)D3 and blood calcium levels ($r = -0.82$) and 25(OH)D3 and serum phosphorus levels ($r = -0.87$) vanishes in a sample of children with rickets. Meanwhile, there is still a positive association ($r = 0.71$) between serum phosphorus and calcium levels.

(Figure 3.3.9).



Figure 3.3.10. Correlation between serum 25 (OH) D3, calcium and phosphorus in children with ASD.

In the group of children with ASD (Figure 3.3.10), the positive correlation between 25 (OH) D3 and serum calcium ($r = - 0.146$), and between 25 (OH) D3 and serum phosphorus ($r = - 0.204$) also disappears. At the same time, there is a positive correlation between serum phosphorus and calcium ($r = 0.736$).

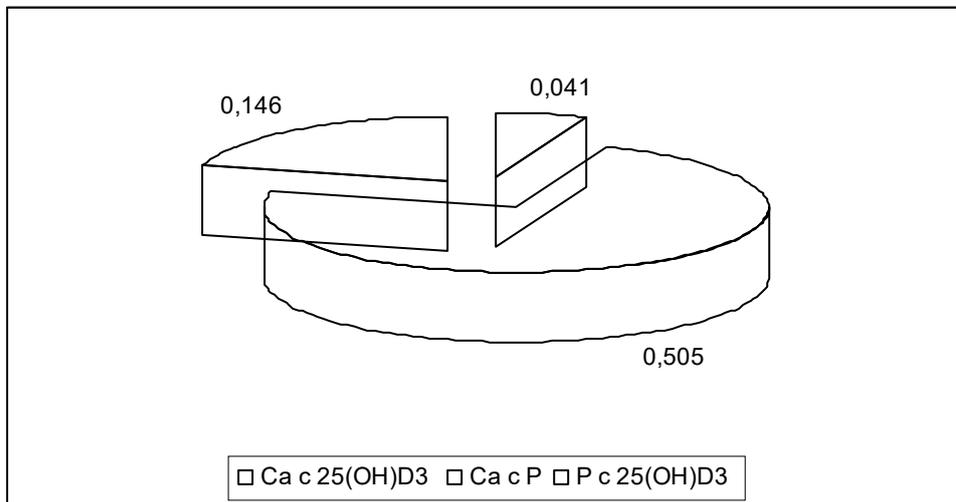


Figure 3.3.11. Correlation between serum 25(OH)D3, calcium and phosphorus in children with rickets and CPNS.

There was a $r=0.041$ association between 25(OH)D3 and calcium, a $r=0.146$ correlation between 25(OH)D3 and phosphorus, and a $r=0.505$ correlation between phosphorus and calcium in blood in the group of children with rickets and CPNS (Figure 3.3.11). It is evident that low blood 25(OH)D3 slows down the intestinal absorption of calcium because it does not aid in the intestinal epithelium's production of metallothioneins. This means that in order to stop rickets from developing, preventative actions must be used in outpatient settings. However, the findings also suggest that young children should be fed a healthy diet that includes more items high in calcium and phosphorus.

3.4. The importance of risk factors depending on the level of 25(OH)D3 in the blood serum.

Determining the amount of 25(OH)D3 in the blood serum is essential for establishing the actual causes of rickets in infants within the first year of life in order to ensure the validity of our findings. We can only identify youngsters at risk for rickets, determine the cause, and—most importantly—produce differentiated therapy with such a comprehensive assessment. This kid distribution enables us to evaluate the mother's and child's risk factors for rickets development as well as the effectiveness of the preventative measures implemented.

Depending on the amount of 25(OH)D3 in the blood serum, all of the children who were tested were split into two groups: group 1 consisted of children whose blood serum levels were normal, and group 2 consisted of children whose blood serum levels were lower. There were 22.7% ($n=106$) youngsters in the first group and 77.2% ($n=360$) in the second. Given that the prevalence of childhood diseases is based on the level of 25(OH)D in blood serum, as the data above shows, the proportion of children with low levels of 25(OH)D3 in blood serum is much higher (77.2%), suggesting a latent vitamin D deficiency in the bodies of the children under examination.

Its proportion of CPNS, iron deficiency anemia, and rickets incidence (Fig. 3. 4. 12) suggests a connection between the frequency of co-occurrence of these

diseases and the function of diet, as well as therapeutic and health-promoting interventions, in halting their development.

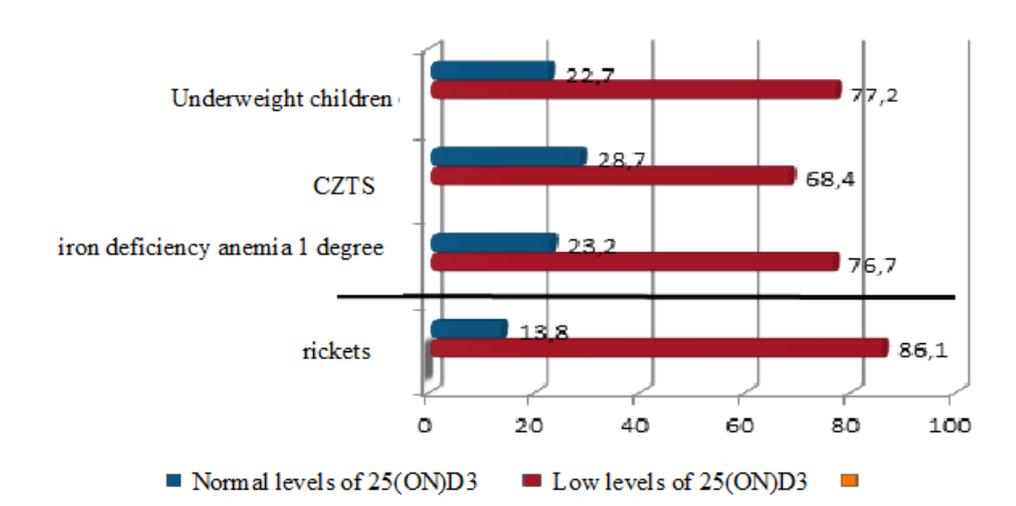


Figure 3.4.12. Distribution of diseases by serum 25(OH)D3 levels.

Clarifying the connection between risk factors for the development of rickets in children and levels of the primary metabolite of vitamin D3 was also of interest (Table 3.4.10). Over 77.5% of children have low blood 25(OH)D3 levels, which is mostly caused by inadequate preventive usage of vitamin D. Children with normal serum 25(OH)D3 levels and those with low levels have the same frequency of other variables.

(3.4.10) The table indicates that inadequate vitamin D intake during the first year of life (73.6%; a normal level would be 52.8%), URVI (45.5%; a normal level would be 62.2%), and exposure to the environment (up to 20 minutes) (36.3%; a normal level would be 46.2%) are the causes of rickets in children with low blood serum levels of 25(OH)D3.

Table 3.4.10

Frequency of risk factors in children depending on the level of 25(OH)D3 in the blood serum

Risk factors	Normal levels of	Low levels of
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	25(OH)D3 n=106		25(OH)D3 n=360	
	abs.	%	abs.	%
Iron deficiency anemia	34	32	85	23,6
Low phosphorus levels in the blood	9	8,4	76	21,1
URVI in a child	66	62,2	164	45,5
Lack of outdoor exposure (less than 20 minutes a day)	49	46,2	131	36,3
Childbirth period (autumn-winter season)	37	34,9	114	31,6
Insufficient prevention of rickets with vitamin D	56	52,8	265	73,6
Perinatal factors	17	16	61	16,9
Birth weight more than 3500 g	27	25,4	49	13,6
Mixed feeding				
Low calcium levels	13	12,2	85	23,6
Premature birth	14	13,2	29	8

It was also interesting to examine the correlation between the primary risk factors in mothers and the blood serum level of 25(OH)D3 (Table 3.4.11). Iron deficiency anemia during pregnancy (56.1%; normal: 82%), poor vitamin D intake during pregnancy (84.1%; normal: 88.6%), and irregular diet during pregnancy (53.3%; normal: 78.3%) were the mother's main risk factors.

Table 3.4.11

The frequency of occurrence of risk factors in mothers depending on the level of 25(OH)D3 in the blood serum

Risk factors	Normal levels of 25(OH)D3 n=106		Low levels of 25(OH)D3 n=360	
	abs.	%	abs.	%
Vitamin D deficiency during pregnancy	94	88,6	303	84,1
Iron deficiency anemia	87	82	202	56,1
Irregular diet during pregnancy	83	78,3	192	53,3
Mother's age (up to 20 years) in 1 pregnancy	64	60,3	187	51,9
Complicated labor	19	17,9	53	14,7
Low level of education	74	69,8	152	42,2
Toxemia of pregnancy	19	17,9	45	12,5

Accordingly, the study found that 112 (86.1%) of the children with lower blood 25(OH)D3 levels and 18 (13.8%) with normal serum 25(OH)D3 levels had rickets. This demonstrates once more how prompt preventive vitamin D medication might stop rickets from developing and lessen its severe forms. We compared the tables with normal and lowered serum 25(OH)D3 levels using the proportionality score criteria. The Student's t-test critical values table was used to assess reliability.

Iron deficiency anemia during pregnancy $Z=3.12$ $P<0.002$, difficult labor $Z=1.46$ $P<0.2$, housewife $Z=1.2$ $P<0.5$, and maternal toxicosis during pregnancy $Z=1.8$ $P<0.1$ were the four significant risk variables that were found among the mother's listed risk factors. Therefore, iron deficiency anemia in the mother during pregnancy influences a drop in the level of 25(OH)D3 in the child's blood serum.

Six major risk factors were found for the child: low blood phosphorus $Z = 15.9$ $P < 0.0001$; URVI $Z = 1.5$ $P < 0.02$; time of birth (autumn-winter period) $Z = 1.6$ $p < 0.2$; walking in the fresh air (less than 20 minutes per day); and lack of vitamin D

in the first year of life $Z = 6.9$ $P < 0.00001$. The child's iron deficit is $Z = 16$ $P < 0.01$; $Z = 1.5$ $p < 0.02$. Using statistical technology, two more substantial risk variables for the kid were found: Iron deficiency anemia in the kid $P < 0.01$; lack of vitamin D prophylaxis in the first year of life $P < 0.00001$.

Serum 25(OH) D3 levels in children should currently serve as the basis for recommendations about the preventative and therapeutic use of vitamin D3 preparations. Furthermore, safety evaluation ought to be carried out indirectly, by directly identifying the blood's metabolites of vitamin D rather than by measuring the levels of calcium and phosphorus. The measurement of blood levels of 25-oxycholecalciferol (25-OH-D) is presently the most accurate way to evaluate the delivery of exogenous vitamin D.

Chapter IV. SUBSTITUTION OF TRADITIONAL AND MODIFIED RICKETS PROPHYLAXIS IN ASSESSMENT OF THE ESSENCE OF THE MEASURES CARRIED OUT

4.1. Features of changes in the content of 25 (OH) D3 in the blood serum of children during traditional prevention of rickets

The prevention of vitamin D deficiency rickets is a key component of the complex of actions done to improve the health of young children. Simultaneously, the prompt prophylactic administration of vitamin D 3 preparations, taking into consideration the degree of negative influence of risk factors on the child's body, is crucial to the efficiency of disease preventive measures. Given the extent to which risk factors negatively affect a child's body, this condition is rather common in Uzbekistan.

Even if we don't see grade III rickets, we still diagnose mild and moderate cases in children as young as one year old. By 1999, 30% of 1-year-old toddlers at Samarkand City polyclinics had been found to have primary and peak rickets. In pediatrics, a specialized approach to the diagnosis, treatment, and prevention of rickets and childhood impairments is necessary due to the high frequency of rickets in young children, particularly latent vitamin D insufficiency.

Prior research revealed that 77.2% of children, including 27.8% of those in their first year of life with clinical signs of rickets, suffer from vitamin D insufficiency. This suggests that conventional rickets preventative measures are insufficiently successful. Only 79 children were able to have their 25(OH)D levels re-determined after we modified the prevention of instances of vitamin D insufficiency in all of the children who were tested. Children were split into two groups: 466 (83%) of the children in the first group were protected against rickets using the normal method, while 79 (16.9%) of the children in the second group were protected against rickets using the modified plan. 130 youngsters, or 27.8% of the total, showed symptoms of rickets. Rickets affected 27 children (19.8%) in urban settings and 103 children (37.7%) in rural

ones.

We conducted interviews with the parents of these youngsters because we had no doubts about the diligence of medical professionals. It turns out that the majority of them never purchased this medication, and even those who did did not give it to their kids. Only 128 (27.4%) of the total number of youngsters got vitamin D during the routine rickets prophylaxis, according to the survey. It should be mentioned that the mother neglected to give the child vitamin D on a regular basis, despite the local doctor writing a prescription and prescribing it.

336 (72.1%) of the 466 children who got the usual rickets prophylaxis did not exhibit clinical indications of rickets, whereas 130 (27.8%) had mild to severe severity. Low levels of 25(OH)D₃ were found in the blood serum of 112 (86.1%) children who had rickets symptoms, whereas the remaining children's levels varied within the normal range. Remarkably, we discovered that 248 (73.8%) children who showed no symptoms of rickets had low levels of the primary metabolite of vitamin D (Fig. 4.1.13). It is evident that this was caused by the unique lifestyle and dietary habits of mothers and their children as well as the absence of preventative measures.

Of the 106 (22.7%) children whose blood serum level of 25(OH)D₃ was within the normal range, 88 (26.1%) did not exhibit any rickets symptoms, whereas 18 (13.8%) did.

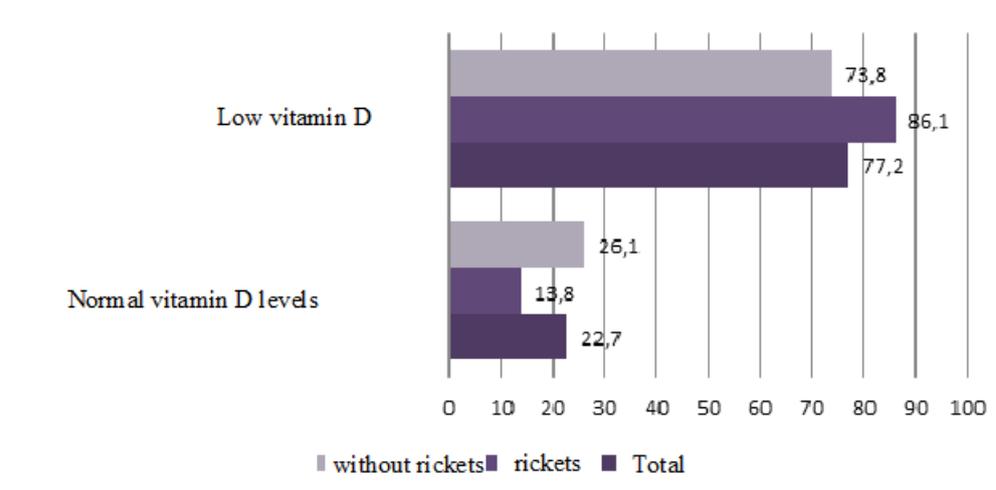


Figure 4.1.13 The content of 25(OH)D₃ in the blood serum of children during traditional prophylaxis.

Therefore, the high frequency of rickets and the low level of 25(OH)D3 in children's blood serum demonstrate that traditional rickets prophylaxis is still ineffective. There is little significance in mothers' continuous work or their inadequate use of preventative measures.

4.2. The effect of modified rickets prevention on the content of 25(OH)D3 in the blood serum of children.

The use of conventional methods to prevent rickets did not provide fruitful outcomes, as was shown in the preceding subsection. We changed the vitamin D intake and made the decision to let nursing consumers be in charge of taking their medications. This was because taking vitamin D on a regular basis was inconvenient, and occasionally parents just forgot to provide the medication. Accordingly, it was determined to alter both the plan and the way this medication is administered in order to improve the dependability of prevention. The doctor is certain that the youngster will take these drugs, and this approach does not add to the workload of secondary medical staff.

Furthermore, our findings indicated that around 5% of parents oppose giving any kind of medicine to children who are healthy. Approximately the same proportion of parents are keen to take "some action" to enhance their child's development. Although most parents are optimistic, they frequently disregard the pediatrician's advice and fail to see the significance of preventative techniques.

According to our approach, a modified rickets prevention strategy was applied for the 79 children in the second group. This strategy involved developing and implementing a direct vitamin D3 delivery technique for each individual kid. Parents, nurses, and physicians all actively participated in this. Preventive medication consumption can only be ensured in this interconnected chain. Our hypothesis was validated by examination of the children's health. According to the following plan, the district nurse administered vitamin D directly to all children: 4000 ME of vitamin D (vigantol, akvadetrim, and devarone) once a week

from the child's birth until the end of the first year, under the supervision of the child's guardian (course dose 160,000-180,000 ME). We chose to administer a course of vitamin D therapy and reassess the amount of the active metabolite of vitamin D in the blood serum of 79 children after 10–12 weeks in order to allay any concerns. Notably, 71 (89.8%) children were able to normalize their blood level of 25(OH)D3 after receiving modified prophylaxis in 79 children (Fig. 4.2.14). Fourteen (17.7%) children did not exhibit clinical indications of rickets, while 65 (82.2%) children had both the beginning and manifestation periods of rickets.

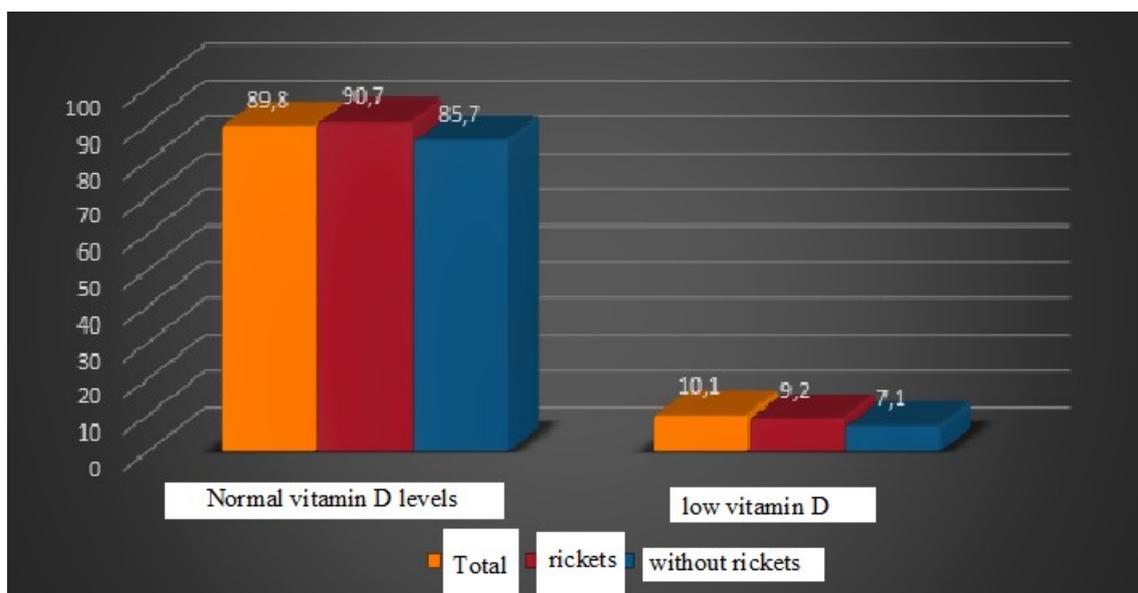


Figure 4.2.14. Serum 25(OH) D3 levels in children on modified rickets prophylaxis.

Therefore, only 6 (9.2%) of the children with rickets symptoms who were tested had a low level of 25 (OH) D3, and 59 (90.7%) normalized following modified rickets prophylaxis. In contrast, just one kid (7.1%) had low serum 25 (OH) D3 levels among children who did not exhibit any symptoms of rickets, while 12 (89.8%) had normalized levels. The child's body weight improved, their appetite and sleep returned to normal, they stopped perspiring excessively during meals, and their general well-being improved. Following preventative measures, the

children's overall health at examination was satisfactory. Side effects and dosage development were not disclosed by the medication we utilized.

4.3. Treatment of rickets in children, depending on its severity and serum 25(OH)D3 levels

As mentioned before, rickets was identified in 130 youngsters. There were 84 children with mild rickets ($64.6\pm 4.19\%$), 53 with an acute course ($40.7\pm 4.30\%$), and 31 with a subacute course ($23.8\pm 3.73\%$). Children were frequently irritable, timid, anxious, sleep deprived, and less hungry. Children also experienced perspiration, particularly on the scalp, and red dermographism. Mild symptoms of acute osteomalacia in children included soreness in the scapular margins and skull bones.

Of the children analyzed, 20 had a subacute course of rickets, 25 had an acute course ($19.2\pm 3.45\%$), and 46 ($35.3\pm 4.19\%$) had moderate rickets. Children exhibited brachycephaly, asymmetry of the head, flattening of the back of the skull, and craniofacial anomalies in addition to the symptoms listed above. A rickets "hump," chicken breast, and muscular hypotension were some of the symptoms of osteoid hyperplasia that some children with a subacute course exhibited. 26 (20%) rickets-affected children displayed symptoms of spasmophilia brought on by acidosis and hypocalcemia. Serum 25(OH)D3 levels were low in 19 (73%) of these kids.

Serum 25(OH)D3 levels in children with rickets were found to be low in 112 (86.1%) of the patients who were evaluated and normal in 18 (13.8%). In children who had rickets, the mean serum 25(OH)D3 levels were 15.23 ± 1.32 nmol/l and 18.75 ± 2.76 nmol/l, respectively, mmol. Children with CPNS with rickets had blood levels of 25(OH)D3, calcium, and phosphorus of 17.4 ± 4.24 nmol/l, 1.96 ± 0.05 , and 0.88 ± 0.03 mmol/l, respectively. Blood levels of calcium and phosphorus were found to be normal in children who were given only vitamin D and no extra calcium supplements (2.48 ± 0.07 and 1.11 ± 0.40 mmol/l, respectively).

The information gathered suggests that the methods of treating rickets should be reexamined, with laboratory parameters being taken into account in order to determine a personalized therapeutic dosage of vitamin D.

Children with mild rickets who had normal calcium and phosphorus levels (2.48 ± 0.07 and 1.11 ± 0.40 mmol/l, respectively) and a serum 25(OH)D₃ level (15.23 ± 1.32 nmol/l) were prescribed 2000 IU of vitamin D per day. A prophylactic dose of vitamin D was administered for 1 to 1.5 months at a rate of 4000 IU per week until 25(OH)D normalized.

Calcium and phosphorus levels dropped (1.73 ± 0.02 and 0.77 ± 0.2 nmol) in patients with moderate to severe rickets, and the dosage was 4000 IU/day. The serum 25(OH)D₃ level was 18.75 ± 2.76 nmol/l. Rickets was treated with prophylactic dosages of vitamin D -4000 ME/week and normalization of 25 (OH) D for 30 to 45 days. Throughout the whole duration of therapy, children were also given calcium preparations in calcipenic forms.

Since rickets is associated with a blood serum level of 25 (OH) D (17.4 ± 4.24 nmol/l), calcium, and phosphorus (1.96 ± 0.05 and 0.88 ± 0.03 mmol/l, respectively), vitamin D dosage was raised to 8000 ME/day for 1–1.5 months until the biochemical parameters returned to normal and the clinical symptoms (sweating, anxiety, sleep, and appetite disorders) subsided.

Therapeutic gymnastics and massage are utilized in the intricate treatment of rickets.

All of the children who were followed showed positive therapeutic outcomes from the complex therapy of rickets.

By days ten or twelve, the condition had improved, hunger had grown, the huge fontanel's edges were less pliable, and the autonomic nervous system was functioning normally again as a result of the treatment. It took a while for muscular tone and motor functions to return, and only after two weeks had positive dynamics been shown. Children with rickets saw increases in calcium, phosphorus, and 25(OH)D₃ concentration in their blood serum of 6.46, 1.5, and 1.7 times throughout the course of therapy.

In mild rickets, the 25(OH)D level was 105.49 ± 8.98 nmol/l ($R < 0.001$), in moderate to severe rickets it was 124.39 ± 14.36 nmol/l, and the calcium and phosphorus levels were 2.97 ± 0.02 mmol/l ($R < 0.05$) and 1.52 ± 0.17 mmol/l ($R < 0.01$), respectively (Fig. 4.3.15 and Fig. 4.3.16).

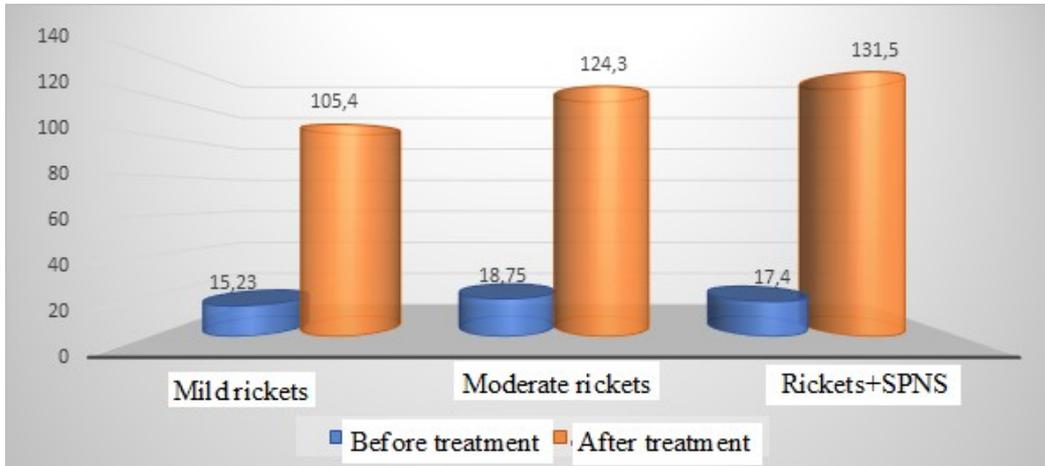


Figure 4.3.15 Serum 25(OH)D3 levels before and after treatment.

Both the 25(OH)D3 level and the blood levels of calcium and phosphorus rose dramatically in children treated with vitamin D in conjunction with CPNSs, reaching 131.54 ± 21.14 nmol/l, 2.01 ± 0.21 , and 1.83 ± 0.13 mmol/l, respectively. The outcomes show that the suggested course of therapy for rickets is quite successful.

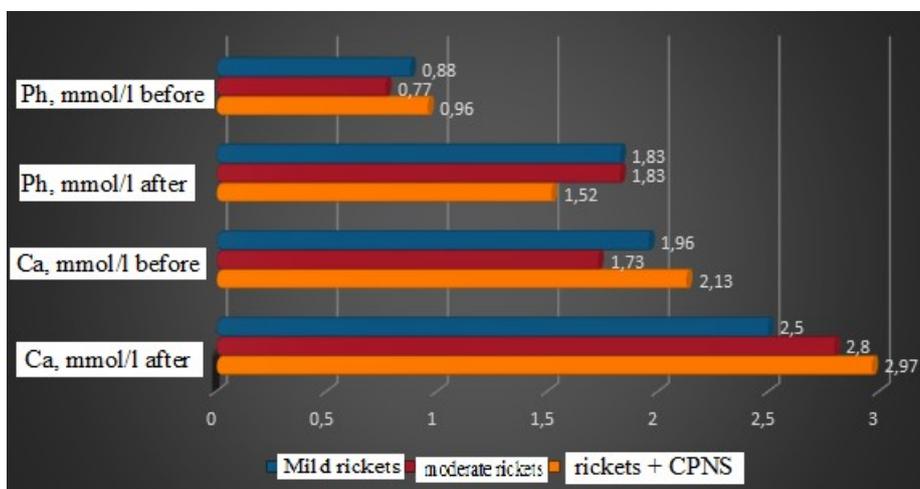


Figure 4.3.16. Serum Ca and P levels before and after treatment.

Therefore, the plan of giving vitamin D supplements to children who have rickets on an individual basis together with calcium supplements and general strengthening therapy enabled a considerable improvement in treatment efficacy. While the clinical indications of rickets are lessened, its aftereffects, such as spasmophilia and a rise in the blood serum's 25(OH)D₃ level, are noted. This makes it possible for modified rickets treatment to be widely implemented throughout the nation.

4.4. Assessment of the effectiveness of traditional prevention of rickets in children

The family's medical activity is one indication of how well treatment and preventative strategies are working. The term "family medical activity" refers to any parental behavior that is directly connected to or directed at the health of the child and the family. Given the high incidence of rickets in infants within the first year of life, it is necessary to examine family medical practices and keep an eye on current guidelines for implementing preventative interventions against rickets development with parents' free participation. Figure 4.4.17. The incidence of certain disorders in families with various medical groups participating in traditional rickets preventative measures is shown.

Ninety percent of mothers reject the need that doctors give vitamin D to infants during the first year of life, according to the findings of preventative measures for 466 children under supervision from one to twelve months. While this was going on, the prevalence of CPNS, anemia, and rickets was twice as low in households that practiced rickets prevention as in those that did not. This suggests that patronage nurses must engage with families and conduct educational activities that explain the advantages of taking preventative action.

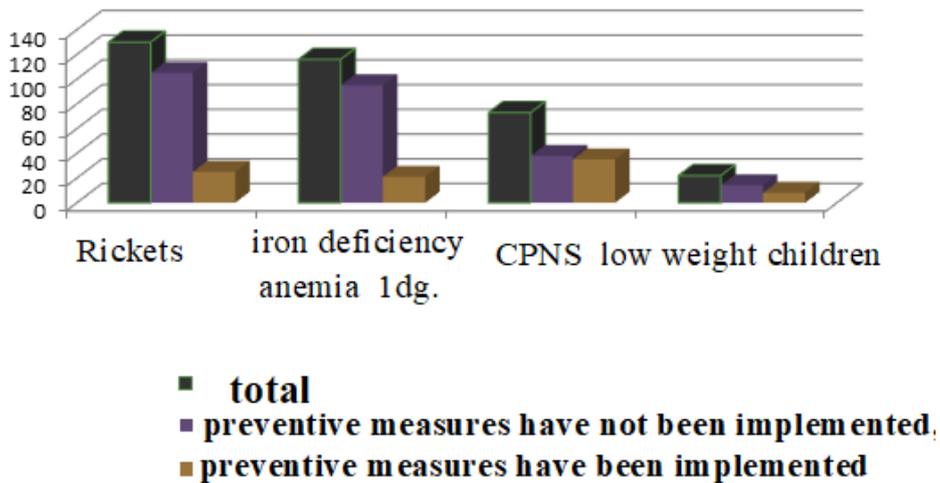


Figure 4.4.17. The influence of family medical activities on the development of certain diseases during traditional prophylaxis of rickets in children in the first year of life, %.

Usually, at follow-up, which occurs 6–12 months later, the efficacy of a given therapy or preventative strategy is evaluated. Only 20 (4.2±0.92%) of the 466 children who had previously been evaluated had blood calcium levels < 2.0 mmol/l, according to a repeat measurement of blood calcium levels conducted during the children's follow-up examination 12 months later. The information gathered demonstrates the necessity of repeating measurements of serum 25 (OH)D3 or, at the at least, the calcium and phosphorus levels in these kids so that the vitamin D3 dosage may be promptly adjusted.

As a result, the dosage and duration of vitamin D3 must be chosen individually. All of the aforementioned evidence supports the necessity of administering these medications to the kids directly in addition to prescribing preventive through advice and prescriptions. Follow-up monitoring of these youngsters was conducted in collaboration with the polyclinic's physicians. It should be mentioned that these kids did well in school and that the parents became more confident about taking precautions. Furthermore, they said in their own words that they had promptly advised vitamin D and told their friends about its efficacy.

According to our approach, we think that the chain of efficacy of preventive measures is caused by both rickets and the rise in the number of women who trust the efficiency of preventive measures in general.

It may be inferred that a woman seeks advice from friends and acquaintances who are older than herself more frequently on the development and growth of her child. In this sense, comparing our findings with laboratory data (specifically, the amount of 25(OH)D₃ in the blood serum for rickets in a particular group of mothers) as well as clinical data can assist to boost confidence in treating more parents.

Therefore, it was shown that administering preventative vitamin D dosages by itself did not stop the emergence of severe rickets. A modified approach to rickets prevention contributes to a notable decrease in the incidence of respiratory and diarrheal illnesses in children. It became out that the widely recommended method of preventing rickets in youngsters did not work, which might lead to rickets development. According to the plan we created, the course of vitamin D₃ treatment resulted in a notable rise in the amount of 25(OH)D₃ in children's blood serum and a dramatic reduction in the condition of vitamin D insufficiency.

This is the first research on vitamin D insufficiency prevalence in Samarkand that we are aware of, and it demonstrates that most babies have serum 25(OH)D₃ levels below 30 nmol/l. This is made possible by the absence of a medical strategy to address vitamin D insufficiency by supplementation. The available supplements are too costly for middle-class households and are imported. Iron and vitamin D pills are not given to many kids. Because of their poor socioeconomic standing and limited financial resources, residents in rural regions cannot afford to purchase vitamin D supplements.

Nearly all of these kids were nursed throughout the experiment, and the ones who were exhibited signs of vitamin D inadequacy. Breast milk cannot be the sole supply of vitamin D since its content is only 15–50 IU/l, but the required amount is 400 IU/l.

CONCLUSION

A sickness that affects young children is called rickets (Greek: rhachis, which means spine). It happens when the body's phosphorus-calcium metabolism is disrupted because of insufficient vitamin D. Artificial feeding, starvation, and early delivery are common causes of rickets. A child's body does not absorb enough vitamin D if they are not properly cared for, do not enjoy the sunshine and fresh air, and are fed incorrectly. Additionally, if they are not exposed to UV rays, the skin's ability to produce vitamin D is impaired, which exacerbates rickets.

Rickets is also brought on by the child's recurrent illnesses and the mother's nutrition during pregnancy. Rickets causes metabolic problems and interferes with the way different organs and systems work. The metabolism of mineral salts like calcium and phosphorus is very badly disturbed in this illness. Bone fragility, tissue softening, and disturbance of the neurological system and internal organs are caused by alterations in the intestinal absorption of calcium and its subsequent deposition in the bones.

Changes in the patient's neurological system occur in the early stages of the disease: the kid starts to become frightened, irritable, capricious, or irritable; he also sweats a lot, and when he feeds, his face and back are covered in perspiration. The youngster rubs his head on the pillow, forcing his back to fall out, because he is itching.

The muscles weaken and twitch as the disease worsens; the child walks more slowly than a healthy child, his eyes swell, his stomach often hardens or sucks, and later the bone system changes: the large cranial fossa does not ossify in time, the ribs near the chest thicken, the shoulder blades flatten, the head gets bigger, the forehead and crown of the head protrude, the forehead becomes convex, and the bones in the skull and occipital region soften. The child's legs bend in an X or O and the chest changes shape, either protrude forward or sinks inward.

Rickets is as ancient as the globe, and research has been done on its pathophysiology, treatment, and management. It is assumed that there is nothing to consider and that everything is obvious. However, there are still a lot of unknowns around this issue. This issue is still significant today even though rickets has been known for a long time. Rickets was eradicated in several nations in the first half of the 20th century once the function of vitamin D and its preventative measures were discovered. In the first year of life, 100% of children are diagnosed with mild rickets, and 25–55% with moderate rickets.

Rickets mostly affects low-income areas of Africa and Asia, although it has lately been seen to be on the rise in wealthier nations as well. Because vitamin D prophylaxis is lacking in Africa and other "sunny" nations, rickets is quite

prevalent there. Despite receiving enough sun exposure, 39% of youngsters in subtropical Saudi Arabia are diagnosed with rickets during the first six months of their lives. Thirty percent of infants in their first year of life had rickets, according to an examination of Samarkand practitioners' surveys and outpatient cards.

Premature newborns, twins, and children with morphofunctional indications are more likely to have rickets. Calcium and phosphorus absorption is decreased by certain anticonvulsant medications that induce the liver to produce microsomal enzymes that deactivate vitamin D and its metabolites. Lack of calcium in the diet may be more important in the development of rickets in children older than one year than a lack of vitamin D. The low calcium content of foods also contributes to this insufficiency, particularly in Asian and African-American nations where dietary customs and mindsets heavily impact the nutrition of children under two.

One of pediatrics' most pressing issues at the moment is the multifactorial study of risk variables for rickets development and the creation of risk groups for preventative treatments based on them. It is important to approach multifactorial analysis with consideration for geographical, climatic, and other factors. One of our research's objectives was to determine the importance of prognostic criteria for evaluating children's health and to evaluate risk factors for phosphorus-calcium metabolism abnormalities in order to design updated rickets preventive plans.

The importance of rickets in the pathology of young children can thus be approached from a new angle thanks to major advancements in early childhood ricket prevention, the eradication of its social causes, and the growth of our understanding of the disease's progression. The findings of the study, along with the observations of pediatricians at children's polyclinics regarding the prevention of rickets in infants under one year of age, should surely aid in the advancement and enhancement of strategies for preventing and treating this illness. At the same time, the blood serum's 25(OH)D₃ content should be determined.

The metabolically active forms of vitamin D are taken into consideration when correcting for rickets in both domestic and international nations. Regrettably, our Republic does not have any such studies. The purpose of this observation is to

detect latent vitamin D insufficiency in order to avoid hypocalcemic seizures and rickets in youngsters. Based on a review of clinical examinations, questionnaires, and outpatient records, children were examined at Samarkand's polyclinics No. 3, 6, 8, and 12 as well as the Central Hospital of Akdarya District between 2004 and 2007. 466 infants between the ages of one and twelve months were observed.

Physical examinations were performed to check for clinical indications of rickets, which included delayed closure of the big fontanel, rachitic rosary, wide wrist, muscular weakness, dystrophy, pale complexion, excessive perspiration, delayed teething, and psychomotor development. All of the infants were deemed to be in nearly perfect health. Consequently, the kids were split up into three groups: Group 1 consisted of 263 infants (56.4%) who were in good health; Group 2 consisted of 130 newborns (27.2%) who showed symptoms of rickets; and Group 3 consisted of 73 children (15.6%) who had prenatal nervous system impairment. It should be mentioned that the levels of calcium and phosphorus, as well as the amount of the metabolite 25(OH)D₃ in children's blood serum, are the primary markers of phosphorus-calcium metabolism.

By agreement, serum samples from the children who were examined were sent to the Santa Clara Hospital laboratory in Rotterdam, the Netherlands, to investigate the level of 25(OH)D₃. The biochemical laboratory of Children's City Hospital No. 1 in Samarkand was used to determine the calcium and phosphorus content.

We surveyed 466 healthy children who had not received treatment in the preceding month in order to determine the risk factors for the onset of vitamin D insufficiency in one-year-olds.

Serum 25 (OH) D₃ levels were reported to be low in 360 children (77.2%) and normal in 106 children (22.7%). A thorough clinical evaluation revealed that 15.6% of infants had the effects of prenatal injury to the CPNS nerve system, and 27.8% of children had rickets. CPNS served as a second control group to detect different premorbid problems of phosphorus-calcium metabolism. Children were split into two groups based on their levels of the primary vitamin D metabolite.

75.2% of children in the CPNS group and 68.4% of children in the healthy group had low 25 (OH) D levels. It was shown that 86.1% of children with rickets had a propensity toward a deeper deficit of the vitamin D metabolite. In healthy children, the typical blood serum level of 25(OH)D is -24.7%; in children with ASD, it is -31.5%; and in children with rickets, it is -13.8%. Lack of sunshine, dietary variables, prenatal factors, and other illnesses are the primary causes and risk factors for the development of rickets, according to the literature. For multivariate analysis, their significance should thus be considered and computed.

Childhood illnesses are a major factor in the development of rickets. According to the research, respiratory tract infections were the most prevalent illness across all categories, but they were more prevalent in rickets and ASD. For IDA, there was no discernible difference between healthy children and ASD, but for rickets, this signal was three times greater. With the classification of clinical IDA, we may state that latent vitamin D insufficiency presents as rickets.

When vitamin D shortage is shown in children younger than six months, mixed feeding is more prevalent in rickets-affected children, whereas breastfeeding is helpful in healthy children. This helps determine if vitamin D deficiency is dependent on the kind of feeding. Just 10.5% of healthy youngsters and 12.5% of children with clinical indications of rickets are on artificial feeding. Of the 141 children, only 74 (52.4%) were breastfed, and 46 (62.1%) of them were healthy, according to the analysis of absolute markers. Just 5.4% (4) of rickets-affected youngsters were on natural feeding, and only 32.4% of them were. Thus, it may be concluded that one of the most effective ways to avoid rickets is to breastfeed.

The primary risk factors for the development of rickets in young children, according to a questionnaire survey given to mothers during pregnancy, were iron deficiency anemia (62%), unbalanced nutrition (59%), low vitamin D intake (85%), and the mother's young age at the time of the first pregnancy (54%). Only 13.7% of pregnant women had toxemia, and less than 50% of cases had both difficult delivery and low maternal education.

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Children whose mothers had taken long-term ICD throughout pregnancy had lower levels of calcium and phosphorus (1.93 ± 0.04 and 0.81 ± 0.02 mmol/l, respectively), according to an analysis of these children's levels. Ca and P levels were 2.09 ± 0.05 and 0.97 ± 0.01 mmol/l in infants whose mothers did not take ICD, respectively.

Children whose moms used long-term ICD throughout pregnancy are likely to have lower blood calcium and phosphorus levels in the future.

Low blood 25 (OH) D₃ levels and inadequate rickets prophylaxis during the first year of life are the most significant risk factors for the development of rickets in children, according to our assessment. Rickets development is significantly influenced by a number of factors, including prior acute respiratory infections (49.3%), less than 20 minutes of fresh air per day (38.6%), time of birth (autumn-winter period) (32.4%), perinatal factors (32.1%), and IDA (25.5%). Body weight greater than 3500g and mixed feeding are less significant. 9.2% of preterm newborns were found, which may seem like a small percentage at first, but a further investigation revealed that the prevalence of premature birth in the general population was really 8.7%.

According to an analysis of postnatal variables, the primary cause of rickets development in our region was the conventional preventive measures used, which resulted in a vitamin D deficit in the blood of 77.2% of children. IDA, frequent colds, and autumn-winter deliveries were additional factors. Vitamin D insufficiency was effectively prevented by breastfeeding. As previously mentioned, of the 466 children who were tested, 360 (77.2%) had a vitamin D deficit, 130 (27.2%) had clinical rickets, and 73 (15.6%) had CPNS.

Diagnoses of asphyxia, persistent intrauterine hypoxia, intracranial birth trauma, impaired cerebral circulation, and preterm delivery were made for patients with CPNS in their anamnesis, who frequently had respiratory conditions.

Rickets were categorized using Lukyanova Ye.M., Antipkin Yu.G., and Omelchenko L.I. (1991). Of the 84 (64.6%) children with mild rickets, 53 (40.7%) had an acute course and 31 (23.8%) had a subacute course. Of the 46 children, 130 (35.3%) had moderate rickets, of which 25 (19.2%) had an acute episode and 21 (16.1%) had a subacute condition. Examining children exhibiting rickets symptoms revealed that 112 out of 130 (86.1%) had low serum 25(OH)D₃, including 77 out of 84 (91.6%) during the early stages of the illness. At the height of rickets development, 35 out of 46 children (76.0%) had vitamin D insufficiency.

Of the 18 (13.8%) children whose blood 25(OH)D₃ levels were normal, 7 out of 84 children (8.3%) had the first stage of rickets, and 11 out of 46 children (23.9%) had the peak period. Our findings support the notion that vitamin D insufficiency is a significant factor during the early stages of a child's growth, and that phosphorus-calcium compound deficiencies also contribute during the peak time.

The accuracy of prenatal risk factor assessment for iron deficiency anemia was 4.059 ($R < 0.043$), lack of vitamin D intake during pregnancy was 40.059 ($R < 0.0001$), and disordered eating during pregnancy was 10.064 ($R < 0.002$), according to statistical correlation analysis of risk factors for the development of rickets. Therefore, maternal age at 1 pregnancy was not a significant risk factor (up to 20 years old) at 0.418 ($R < 0.518$).

Iron deficiency anemia was found to significantly increase the risk of rickets -7.083 ($R < 0.008$), the child's birth time (autumn-winter period) -5.14 ($R < 0.025$), prenatal factors -8.516 ($R < 0.004$), low exposure to fresh air (no more than 20 minutes) -9.395 ($R < 0.002$), and the child's lack of vitamin D-based rickets prevention during the first year of life -4.334 ($R < 0.037$). Therefore, prognostic indications include the existence of IDA in the mother, the

identification of risk factors for the development of rickets during the prenatal period, vitamin D deficiency during pregnancy, and an imbalanced diet.

Low environmental exposure, prenatal variables, the child's birth timing, and vitamin D prophylaxis are among postnatal factors that can also be used as predictive markers for the development of rickets. Premature delivery, another perinatal risk, was considerably more significant in the development of CPNS at the same period. Furthermore, rickets in children is one of the elements that contribute to the development of CPNS, suggesting a connection between both disorders.

The results of the tests revealed that, among the children with rickets, only 18 (13.8%) had normal levels of 25(OH) D₃ in their blood serum, while 112 (86.1%) had low levels. In contrast, 23 (31.5%) and 50 (68.4%) of the patients in the group of children with ASD had normal and low levels of this metabolite, respectively. Serum 25(OH)D₃ levels were found to be low in 75.2% of the sample of healthy youngsters and normal in 24.7% of them.

Of the youngsters, 44.4% had normal calcium and phosphorus levels within the normal range of blood 25(OH)D₃, whereas 16.6% had low levels. Therefore, we may state that a kid has vitamin D-resistant rickets if their blood 25(OH)D₃ level is normal but their calcium and phosphorus levels are low. We did not, however, consider such youngsters. 15.1% had normal calcium and phosphorus levels in the low range of 25(OH)D₃.

Children with rickets had low serum levels of calcium and phosphorus (1.99 ± 0.019 and 0.92 ± 0.011 mmol/l, respectively), as well as a low serum 25(OH)D level of 19.8 ± 1.98 nmol/l. Meanwhile, 25(OH)D levels in children with CPNS were 36.7 ± 5.04 nmol/l, and their serum levels were 2.22 ± 0.0524 and 1.21 ± 0.044 mmol/l. Serum calcium and phosphorus levels in the children with CPNS and rickets were 1.95 ± 0.04 and 0.88 ± 0.028 mmol/l, respectively. These levels were marginally lower than those in the babies with rickets, and the level of 25(OH)D was 31.7 ± 14.8 nmol/l.

It was also interesting to see how the content of the aforementioned indicators correlated with the youngsters under examination. We employed the Pearson (r) correlation coefficient to ascertain the association between the blood concentrations of Ca and R and 25(OH)D3. According to the research that were done, the positive association between the levels of 25(OH)D3 and calcium in the blood serum ($r = -0.82$) and phosphorus in the blood serum ($r = -0.87$) vanishes in the group of children who have rickets.

Meanwhile, there is still a favorable association ($r = 0.71$) between the blood serum levels of calcium and phosphorus. There was a connection of $r = 0.041$ between the level of 25(OH)D3 and calcium in the group of children with rickets and CPNS (Fig. 3.3.11), $r = 0.146$ between the level of 25(OH)D3 and phosphorus, and $r = 0.505$ between the level of phosphorus and calcium in the blood serum.

Determining the amount of 25(OH)D3 in the blood serum is essential for establishing the actual causes of rickets in infants within the first year of life in order to ensure the validity of our findings. Every kid who was tested was split into two groups based on the amount of 25(OH)D3 in their blood serum: group 1 consisted of children whose blood serum levels were normal, and group 2 consisted of children whose blood serum levels were lower.

There were 22.7% ($n=106$) youngsters in the first group and 77.2% ($n=360$) in the second. The aforementioned data shows that, in relation to the prevalence of childhood diseases as determined by the level of 25(OH)D in the blood serum, a significantly higher percentage of children (77.2%) had low levels of 25(OH)D3 in the blood serum, indicating a latent vitamin D deficiency in the bodies of the children under examination. Its percentage suggests a connection between CPNS, iron deficiency anemia, and rickets, as well as the prevalence of co-occurrence of these conditions, the importance of nutrition, and the use of preventative and therapeutic measures.

Clarifying the connection between risk factors for a child's development of rickets and the concentration of the primary metabolite of vitamin D3 is of interest.

The causes of rickets in children with low blood serum levels of 25(OH)D3 include inadequate vitamin D intake during the first year of life (73.6%; normal level: 52.8%), URVI (45.5%; normal level: 62.2%), and exposure to the environment (up to 20 minutes) (36.3%; normal level: 46.2%).

Iron deficiency anemia during pregnancy (56.1%; normal level: 82%), inadequate maternal vitamin D intake during pregnancy (84.1%; normal level: 88.6%), and irregular diet during pregnancy (53.3%; normal level: 78.3%) are the main risk factors. We compared the normal and decreased serum 25(OH)D3 levels using the proportionality test. The Student's t-test critical value table was used to assess reliability.

Four important risk factors were found out of the ones the mother had mentioned: Maternal toxicosis during pregnancy $Z=1.8$ $P<0.1$; housewife $Z=1.2$ $P<0.5$; difficult labor $Z=1.46$ $P<0.2$; iron deficiency anemia during pregnancy $Z=3.12$ $P<0.002$. The youngster discovered six important risk variables, including: Low blood phosphorus $Z=15.9$ $P<0.0001$; URVI $Z=1.5$ $P<0.02$; time of birth (autumn-winter season) $Z=1.6$ $p<0.2$; walking in fresh air (less than 20 minutes per day) $Z=1.5$ $p<0.02$; iron deficit in a kid $Z=16$ $P<0.01$; vitamin D insufficiency in the first year of life $Z=6.9$ $P<0.00001$.

Using statistical technologies, two additional significant variables were found from these risk factors in children: iron deficiency anemia in a kid $P < 0.01$ and lack of vitamin D prophylaxis in the first year of life $P < 0.00001$. The factors causing the high incidence of rickets in children are still relevant today, even with the notable improvements in the social conditions of children's lives and upbringing, the widespread promotion of a healthy lifestyle among the populace, and the rise in the cultural level of parents.

As a result, it is essential to increase parents' accountability for their children's health and create radically new strategies for encouraging a healthy lifestyle. Vitamin D insufficiency is a significant factor in preventing rickets in the complex of steps done to improve the health of young children. We discovered that 77.2% of children, including 27.8% of 1-year-old children with clinically

noticeable rickets, suffer from vitamin D insufficiency. This suggests that conventional rickets prevention measures are insufficiently successful. All of the children who were tested had their vitamin D deficiency prophylaxis changed, but we were able to recalculate the 25(OH) D levels in 79 of them.

The children were split into two groups: 466 (83%) of the children in the first group were protected against rickets using the normal method, while 79 (16.9%) of the children were protected against rickets using the modified strategy. 130 youngsters, or 27.8% of the total, showed symptoms of rickets. Rickets affected 27 children (19.8%) in urban settings and 103 children (37.7%) in rural ones. 336 (72.1%) of the 466 children exhibited no clinical indications of rickets, whereas 130 (27.8%) had mild to severe severity despite the usual prevention of rickets.

Low blood serum levels of 25(OH)D₃ were found in 112 (86.1%) children exhibiting rickets symptoms, whereas the other children's levels varied within the normal range. Remarkably, we also discovered that 248 (73.8%) children who showed no symptoms of rickets had low levels of the primary metabolite of vitamin D. It is clear that this was caused by a combination of factors, including the unique lifestyle and dietary habits of mothers and their children, as well as the absence of preventative measures.

Of the 106 (22.7%) children with blood 25(OH)D₃ levels within the normal range, 88 (26.1%) did not exhibit any rickets symptoms, whereas 18 (13.8%) did. Therefore, the high frequency of rickets and low blood 25(OH)D₃ levels in children demonstrate that conventional rickets prophylaxis is still ineffective. Mothers' continual job, bad lifestyle, and lack of preventative measures are likely contributing reasons to the low medical culture.

The second group of children (79 children) underwent modified rickets prevention using our technique, which entails developing and implementing a method of direct vitamin D₃ delivery to each individual kid. Parents, nurses, and physicians all actively participated in this. Our supposition was confirmed by an analysis of the children's condition. In this regard, the district nurse administered

vitamin D directly to all children in accordance with the following plan: once a week, under the supervision of the child's guardian, 4000 ME of vitamin D (vigantol, Akvadetrim, and devarone) from the age of one month until the end of the first year (course dose 160,000-180,000 ME).

We chose to administer a course of vitamin D therapy and reassess the amount of the active metabolite of vitamin D in the blood serum of 79 children after 10–12 weeks in order to allay any concerns. It should be mentioned that 71 (89.8%) of the 79 children who received modified prophylaxis were able to return their blood serum levels of 25(OH)D₃ to normal. Fourteen (17.7%) children had no clinical indications of rickets, whereas 65 (82.2%) children had both the beginning and manifestation periods of rickets. As a result, following modified rickets prophylaxis, 59 (90.7%) of the children with rickets symptoms had normalized levels of 25(OH)D₃, whereas only 6 (9.2%) had low levels.

In contrast, 12 (89.8%) of the children without rickets symptoms had normal blood 25(OH)D₃ levels, while just 1 (7.1%) had low levels. The child's health, appetite and sleep patterns returning to normal, excessive perspiration during meals stopping, and weight improvement were all noted. The children's overall health was satisfactory during the examination that followed the use of preventative measures. There were no reports of drug overdoses or adverse consequences.

When 25(OH)D₃ levels in the blood serum of children with rickets were measured, 112 (86.1%) of the patients had low levels, while 18 (13.8%) had normal levels. The average levels of 25(OH)D₃ in the blood serum of children with rickets were 18.75 ± 2.76 nmol/l and 02/l, respectively, with average values of 15.23 ± 1.32 nmol/l. Children with rickets and CPNS had blood levels of 25(OH)D₃, calcium, and phosphorus that were 17.4 ± 4.24 nmol/l, 1.96 ± 0.05 , and 0.88 ± 0.03 mmol/l, respectively. Children who received only vitamin D and no extra calcium supplements had normal blood levels of calcium and phosphorus (2.48 ± 0.07 and 1.11 ± 0.40 mmol/l, respectively). We treated rickets on an individual basis based on these findings.

Vitamin D was prescribed at a dose of 2000 IU/day for children with mild rickets who had a serum 25(OH)D₃ level of 15.23±1.32 nmol/l, normal calcium and phosphorus levels of 2.48±0.07 and 1.11±0.40 mmol/l, respectively. A prophylactic dose of vitamin D was administered for 1–1.5 months at a rate of 4000 IU/week until 25(OH)D normalized. The recommended dosage for moderate to severe instances of rickets was 4000 IU per day. The course of treatment included normalizing 25 (OH) D levels and administering a preventive dose of vitamin D at a rate of 4000 IU per week for 30 to 45 days. Throughout the duration of therapy, children were also given 0.5 g of calcium preparations daily in calcipenic forms. The vitamin D dosage was raised to 8000 IU per day for one to one and a half months in rickets patients until the biochemical parameters returned to normal and the clinical symptoms (sweating, anxiety, sleep, and appetite disturbances) subsided. Therapeutic gymnastics and massage are utilized in the intricate treatment of rickets. All of the children who were followed showed positive therapeutic outcomes from the complex therapy of rickets. By the tenth or twelfth day, the condition had improved, hunger had grown, the huge fontanel's edges were less pliable, and the autonomic nervous system was functioning normally again. Positive dynamics were only noticed after two weeks, and the recovery of motor functions and muscular tone was gradual. Children with rickets saw increases in blood serum levels of 25(OH)D₃, calcium, and phosphorus of 6.46, 1.5, and 1.7 times during therapy. Calcium and phosphorus levels are 2.97±0.02 mmol/l (R<0.05) and 1.52±0.17 mmol/l (R<0.01), while the level of 25 (OH) D is 105.49±8.98 nmol/l (R<0.001) in mild rickets and 124.39±14.36 nmol/l in moderate to severe rickets.

The blood serum levels of 25 (OH) D₃, calcium, and phosphorus rose considerably to 131.54±21.14 mmol/l, 2.01±0.21, and 1.83±0.13 mmol/l when children received vitamin D in addition to CPNS. Therefore, the plan of giving vitamin D preparations to children with rickets on an individual basis together with calcium medications and general strengthening therapy enabled a considerable improvement in treatment efficacy. Meanwhile, there are evidence of

spasmophilia, rickets's aftereffects, clinical manifestations, and elevated blood 25(OH)D3.

This makes it possible for modified rickets treatment to be widely implemented throughout the nation.

LIST OF ABBREVIATIONS

25(OH)D3 – 25-hydroxycholecalciferol (calcitriol)

ATP – adenosine triphosphoric acid

IUI – intrauterine infection

IGC – intragastric complex

ICBI – intracranial birth injury

CP – Cerebral palsy

IDA – iron deficiency anemia

ICD – iron-containing drugs

CCD – cerebral circulatory disorders

ARVI – acute respiratory viral infection

CPNS – consequences of perinatal damage to the nervous system

P – phosphorus

Ca – calcium

UE – Ultrasound examination

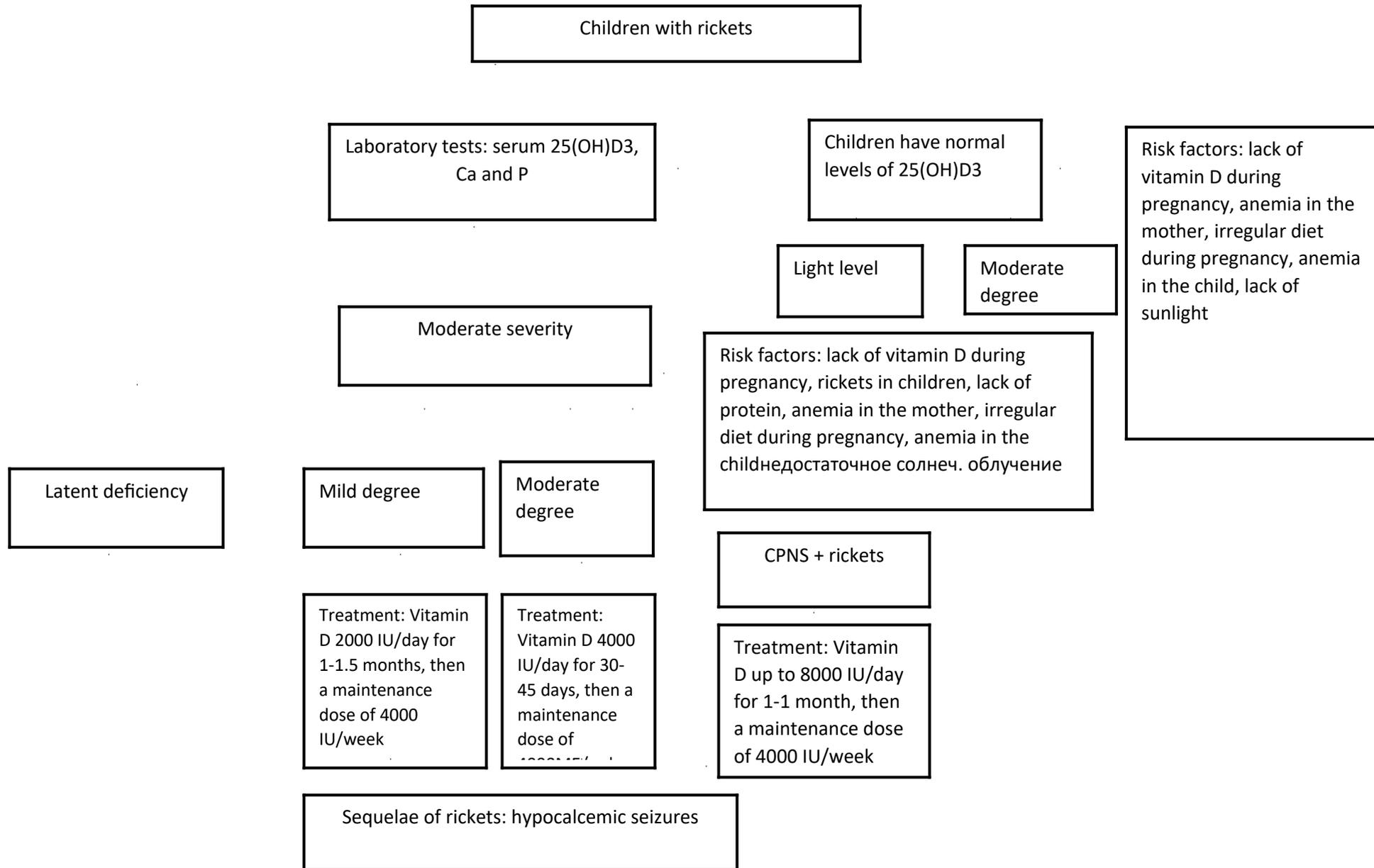
UV lamp – ultraviolet lamp

CHIFH – chronic intrauterine fetal hypoxia

CNS – Central nervous system

ExoEG – exoencephalography

Algorithm for examination and treatment of rickets in young children and prevention of the consequences of rickets



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