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## **Some indicators of lipid and phosphorus-calcium metabolism in children with rickets receiving conventional treatment**

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**Abstract**---In children with rickets and when rickets was aggravated by pneumonia and malnutrition, after the traditional method of treatment, there was no normalization of phosphorus-calcium metabolism and some indicators of lipid metabolism. Along with the clinical recovery, total blood and fecal lipids, SFA, and alkaline phosphatase activity remained elevated, while the content of calcium, phosphorus, and USEFA were below normal, which indicates an “incomplete recovery” and apparently requires further correction of biochemical parameters.

**Keywords**---lipid, phosphorus-calcium metabolism, rickets, children.

### **Introduction**

### **Relevance**

Rickets still occupies an important place in the structure of morbidity in young children and remains an urgent problem in pediatrics [1,3,5,11,22]. This problem requires special attention to the problem of rickets, which has a negative impact on the reactivity of the organism, the course and outcome of somatic diseases, especially in children of the first year of life [2,7,8,10,14]. This is a disease of childhood, caused by a lack of vitamin D in the body, characterized by a violation

of phosphorus-calcium metabolism, bone formation and a disorder in the function of the nervous system and internal organs.

It is clear that the basis of any complex method of treating rickets is the use of vitamin D preparations. However, the data accumulated in the literature [4,12,21] indicate that in some children, course treatment with vitamin D preparations is not effective enough. These data in the wider world reflect the experience of domestic and world practice, indicating that a number of manifestations of this disease are persistent and insufficiently correctable when treated with vitamin D preparations [13,17,19]. The schemes proposed by a number of authors [16,18] using various dosage forms and dosages of vitamin D, as a rule, do not lead to complete recovery by the end of the course of treatment and, at the same time, in some cases it is accompanied by the occurrence of complications - manifestations of hypervitaminosis D [6,9,20].

The traditional treatment complex for rickets, along with drugs with a specific effect (group D vitamins), includes pharmacological agents for pathogenetic and symptomatic therapy (group B vitamins, ascorbic acid, calcium gluconate, and others) against the background of the mandatory widespread use of a complex of nonspecific measures that provide optimal conditions for harmonious development child's body. This is a properly organized regime, strict observance of sanitary and hygienic standards for child care, rational nutrition.

Specific therapy for rickets, against the background of the described complex of non-specific measures, was carried out with a 0.5% alcohol solution of vitamin D. With rickets of the I degree of severity, taking into account the nature of the course of the disease, patients receive 400-600 thousand IU of vitamin D per course. With rickets of the II degree of severity, the course dose of vitamin D increased to 600-800 thousand IU, which the children received in the acute course of 15-20 days, and in the subacute course of 45-60 days. In none of the observed cases, we noted increased sensitivity to vitamin D preparations, side effects and phenomena of D-vitamin intoxication.

### **Purpose of the study**

To study in dynamics the clinical and biochemical parallels in the observed children in the course of a comparative analysis of the clinical features of the course of the disease and the dynamics of some indicators of lipid metabolism during the traditional therapy of children with rickets.

### **Material and research methods**

We examined 47 patients who received traditional therapy. Of these, 11 children with rickets, 15 children with rickets aggravated pneumonia and 21 children with rickets aggravated pneumonia and malnutrition. Analysis of fatty acids in blood serum was carried out by gas-liquid chromatography. Among the methods of chromatographic analysis, gas chromatography is promising due to its high separating power, sensitivity, and expressivity, becoming one of the most used methods in analytical chemistry [15]. We determined the qualitative and quantitative composition of fatty acids on a Tsvet-100 chromatograph, model 165

with a flame ionization detector, in the laboratory of the Department of General Chemistry of Samarkand State University. The determination of total lipids in blood and feces, calcium, phosphorus and alkaline phosphatase was carried out using kits from Biolatest.

### Research results

We analyzed the initial indicators of biochemical variants, which reflect the state of some indicators of lipid metabolism, as well as some features of their dynamics in children against the background of the traditional method of treatment. The results obtained are presented in Table 1 and in Fig.1. Initially, let us consider the dynamics of the studied values in the group of examined children (Table 1). At the time of admission to the hospital, their total lipids were higher than the control values (4.61 g/l) and amounted to 6.95 g/l of total lipids ( $P < 0.001$ ).

Table 1  
Some indicators of lipid metabolism in children with rickets, receiving traditional treatment

Indicators	Healthy M $\pm$ m	On admission M $\pm$ mP	In the dynamics (for 5-7 days) M $\pm$ m P	At discharge M $\pm$ mP
Totallipids, g / l	4.61 $\pm$ 0.28	6.95 $\pm$ 0.30 < 0.001	6.61 $\pm$ 0.30 < 0.001	5.64 $\pm$ 0.20 < 0.02
Total feces lipids, g / l	0.42 $\pm$ 0.05	0.85 $\pm$ 0.03 < 0.001	0.79 $\pm$ 0.03 < 0.001	0.71 $\pm$ 0.02 < 0.001
C(16 : 0)	28.17 $\pm$ 1.37	30.87 $\pm$ 1.53 > 0.2	29.50 $\pm$ 1.43 > 0.5	28.96 $\pm$ 0.43 < 0.01
C(16 : 1)	2.70 $\pm$ 0.22	1.32 $\pm$ 0.62 < 0.05	1.45 $\pm$ 0.53 < 0.05	1.62 $\pm$ 0.30 < 0.05
C(18 : 0)	26.13 $\pm$ 1.32	28.13 $\pm$ 1.04 > 0.2	27.88 $\pm$ 0.92 > 0.2	27.67 $\pm$ 0.82 > 0.2
C(18 : 1)	0.90 $\pm$ 0.13	0.60 $\pm$ 0.14 > 0.2	0.66 $\pm$ 0.11 > 0.2	1.76 $\pm$ 0.10 > 0.2
C(18 : 2)	33.32 $\pm$ 2.51	29.73 $\pm$ 2.34 > 0.5	30.10 $\pm$ 2.12 > 0.5	30.74 $\pm$ 2.10 > 0.5
C(18 : 3)	2.41 $\pm$ 0.45	2.56 $\pm$ 0.50 > 0.2	2.44 $\pm$ 0.48 > 0.2	2.11 $\pm$ 0.45 < 0.05
C(20 : 4)	3.56 $\pm$ 0.60	2.68 $\pm$ 0.60 > 0.2	2.32 $\pm$ 0.56 > 0.2	2.10 $\pm$ 0.51 < 0.05
UFAs	54.30 $\pm$ 2.69	59.00 $\pm$ 2.57 < 0.05	57.38 $\pm$ 2.35 > 0.2	56.63 $\pm$ 2.10 > 0.2
EFAs	42.89 $\pm$ 3.91	36.93 $\pm$ 4.20 < 0.05	36.98 $\pm$ 3.80 < 0.05	37.33 $\pm$ 3.62 < 0.05
K= UFAs /EFAs	0.80	0.63	0.64	0.65

P – significance of differences between indicators in the group of patients and healthy

When analyzing the fatty acid spectrum of the blood serum of the examined children upon admission to the hospital, it was revealed that almost all of its indicators are C (16:0), C (18: 0), C (18: 1), C (18: 2), C (18:3), C (20:4) had no significant differences compared with healthy children ( $P > 0.2$ ), ( $P > 0.5$ ) and only C (16:1) tended to decrease ( $P < 0.05$ ).

The content of total lipids in the feces of healthy children was  $0.42 \pm 0.05$  g/kg, and at the time of admission to the hospital in children this figure was  $0.85 \pm 0.03$  g/kg, which was higher than in healthy children ( $P < 0.001$ ) When the children were re-examined, out of 5-7 days of their stay in the hospital, it was found that the level of total lipids in the blood serum became equal to an average of  $6.95 \pm 0.3$  g/l ( $P < 0.001$ ), the content of total fecal lipids (TLC ) decreased and was equal to  $0.79 \pm 0.03\%$  ( $P < 0.001$ ). The content of C(16:0) tended to decrease and amounted to  $27.50 \pm 1.43\%$  ( $P > 0.5$ ), while C(16:1) increased, it turned out to be  $1.45 \pm 0.53\%$  ( $P < 0.05$ ) .

The level of C (18:0) in the blood serum decreased and averaged  $27.88 \pm 0.92\%$  ( $P > 0.2$ ), while C (18:1) tended to increase and amounted to  $0.66 \pm 0.11\%$  ( $P > 0.2$ ). The content of C (18:2) also tended to increase  $30.10 \pm 2.12\%$  ( $P > 0.5$ ), while C (18:3) decreased, averaging  $2.44 \pm 0.48\%$  ( $P > 0.2$ ) . The C(20:4) level averaged  $2.32 \pm 0.56\%$  ( $P > 0.2$ ) i.e. tended to decrease. Further studies were carried out by the time the children were discharged. As a result, by the time of discharge in children of this group, the studied parameters were: the content of OL was  $5.64 \pm 0.3\%$  ( $P < 0.02$ ), which was higher than in the control group. OLK decreased, the indicator corresponded to  $0.71 \pm 0.02\%$  ( $P < 0.001$ ).

The concentration of C(16:0) at the time of discharge was  $28.96 \pm 1.28\%$  ( $P < 0.001$ ). C(16:1) was  $1.62 \pm 0.43\%$  ( $P < 0.01$ ) and remained below the standard values. C(18:1) content  $0.76 \pm 0.10\%$  ( $P > 0.2$ ), i.e. lower than in healthy children. The C(18:2) level at discharge was  $30.74 \pm 2.10\%$  ( $P > 0.5$ ), which also tended to decrease, and C(18:3) was  $2.11 \pm 0.45\%$  ( $P < 0.05$ ) i.e. lower than in healthy children. The content of C(20:4) was  $2.10 \pm 0.51$  ( $P < 0.05$ ), also below the norm.

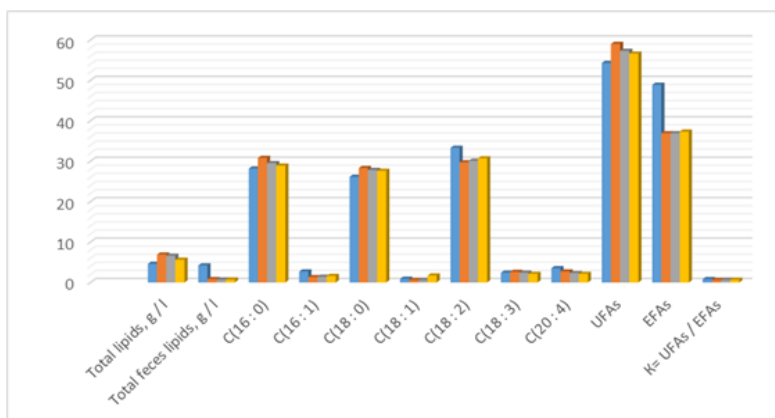


Figure 1. The dynamics of some indicators of lipid metabolism (spectrum of high fatty acids) in children with rickets, against the background of traditional therapy.

As can be seen from the table. 1 data, in children who received conventional treatment, along with a decrease in the clinical manifestations of the disease, there was a tendency to reduce the violations of some indicators of lipid metabolism, which is retained in sick children, despite the treatment. Figure 1 clearly demonstrates that the analyzed parameters changed quite differently, both qualitatively and quantitatively, even within the same link of lipid metabolism. The studies were carried out three times: upon admission to the hospital, on days 5-7 and at discharge from the hospital.

The stability of lipid dysmetabolism under the influence of the generally accepted complex of treatment is probably due to the fact that the effect of specific therapy in the body is primarily aimed at correcting phosphorus-calcium metabolism. It can be assumed that the lack of noticeable positive dynamics of the fatty acid spectrum under the influence of generally accepted complex therapy makes it difficult to implement the action of vitamin D in the body, since it has been proven that under the influence of lipid dysmetabolism, there is a decrease in the level of 1,25-dihydroxycholecalciferol in plasma, which is one of the most active vitamin D metabolites [1,10]. This is probably due to a violation of its renal metabolism in terms of lipid dysmetabolism. The redistribution of 24,25-dihydroxycholecalciferol, which is important in the processes of osteogenesis, also changes in the tissues of the body.

## **Discussion**

Our clinical observations and biochemical studies have shown that the use of conventional therapy for rickets did not adequately normalize the studied parameters of lipid metabolism, which is probably due to the stability of lipid dysmetabolism in the examined children. In all observed children, after a course of conventional complex therapy, the parameters of the fatty acid spectrum of blood serum, the content of inorganic phosphorus, calcium, alkaline phosphatase activity, total lipids of blood and feces were studied. Case histories of observed children with rickets are given as clinical examples of the therapeutic and biochemical effectiveness of the traditional method of treatment.

We believed that such a presentation of the materials of our own observations should most effectively and adequately reflect the reliability of general conclusions, conclusions and practical recommendations. An illustration of the influence of the conventional therapy of rickets on the dynamics of the symptoms of the disease, the studied indicators of lipid, phosphorus-calcium metabolism in the blood can be the following our observations:

The child Farhod D., aged 6 months, was under observation in the children's polyclinic N 1 in Samarkand. The boy was born full-term from the 4th pregnancy and childbirth. The mother's pregnancy proceeded with toxicosis of the first half, which was manifested by nausea, vomiting, loss of appetite. The mother received no treatment for this. The child's body weight at birth was 3400 g, height 50 cm. He was attached to the breast on the second day, sucked actively. The umbilical cord fell off on the 4th day and the child was discharged home in a satisfactory condition. From the age of 3 months he was artificially fed. Specific prophylaxis of rickets was not carried out. complaints when examining a child for periodic anxiety

for no apparent reason, sleep disturbance, excessive sweating, irritability, capriciousness, decreased appetite.

### **Objective examination**

The general condition of the child on examination is satisfactory. However, the child is pale, there is muscle hypotonia, lethargy, the child is of satisfactory nutrition. As a result of pronounced hyperplasia of the osteoid tissue, the head has an irregular shape. No signs of osteomalacia were found in the bones of the skull. A large fontanel measuring 2.0 by 2.0 cm, its edges are supple. "Harrison's furrow", "rachitic rosary" are clearly defined on the chest. No pathology was found in the lungs. Heart sounds are muffled, there are no murmurs. The abdomen is enlarged, hypotension of its muscles is determined. The spleen is not enlarged. According to the mother, the child has constipation. Urination is free.

Due to pronounced muscular hypotension and weakness of the ligamentous apparatus, the boy lags behind in statokinetic development, does not sit on his own, does not roll over onto his stomach. In a clinical blood test: 110 g / l, erythrocytes - 3.8, leukocytes - 10.2, neutrophils - 74, monocytes - 10, ESR - 8 mm / h. General analysis of urine without features. X-rays of the bones show signs of osteoporosis. The content of calcium in the blood serum is 1.78 mol / l, inorganic phosphorus - 1.19 mmol / l, alkaline phosphatase activity - 6.68  $\mu$ kat / l, total blood lipids - 7.25 g / l, total stool lipids - 0,84 g/kg. Indicators of the fatty acid spectrum of blood (in %):

C(16:0) - 30.17 C(18:2) - 29.75  
 C(16:1) - 1.35 C(18:3) - 2.58  
 C (18:0) - 28.03 C (20:4) - 2.67  
 C (18:1) - 0.60

Clinical diagnosis: rickets II degree, subacute course, peak period. The child was prescribed a complex treatment, including an organized regimen with the correct alternation of sleep, active wakefulness, with sufficient exposure to fresh air, in conditions of natural insolation, a balanced rational diet by prescribing "Baby", pureed vegetable dishes, grated apple, juices. The child received vitamins: pyridoxine 5% 0.5 ml in the morning on an empty stomach 30 minutes before meals, ascorbic acid 0.15% x 3 times a day inside for 20 days. As a specific anti-rachitic therapy, the child received a 0.5% alcohol solution of vitamin D, 20 thousand I.U. per day for 40 days (the course dose was 800 thousand IU).

By the end of treatment, serum calcium was 2.19 mmol/l, inorganic phosphorus was 1.33 mmol/l, alkaline phosphatase activity was 6.56 mkat/l, total blood lipids were 5.64 g/kg, total stool lipids were 0.72 g / kg, and the indicators of the fatty acid spectrum of the blood were (in%):

C(16:0) - 28.86 C(18:2) - 30.54  
 C(16:1) - 1.50 C(18:3) - 2.50  
 C(18:0) - 27.32 C(20:4) - 2.12  
 C(18:1) - 0.81

Studies have shown that in children with rickets there are changes in some indicators of lipid metabolism, which are expressed by dyslipidemic changes in the blood serum. From this it follows that the traditional therapy of this disease does not provide complete relief of the rickets process, although clinically it is expressed by an improvement in the general condition of such children. After 2 weeks from the start of specific therapy, general massage and physiotherapy exercises were connected.

Under the influence of the ongoing treatment from 10-14 days from the beginning, the child became calmer, sleep improved, and excessive sweating decreased. Somewhat later, 20 days after the start of treatment, there was a tendency to seal the edges of the large fontanel. And only 3 weeks after the end of the course of treatment, muscle tone and motor function improved. The child began to sit with support and rise.

Consequently, in children with rickets who received complex therapy, including drugs D, first of all, there is a normalization of functional changes in the nervous system, such as anxiety, excessive sweating, and sleep disturbance. A longer time is required to restore muscle tone, statokinetic functions, and positive dynamics of bone changes.

An analysis of the dynamics of some indicators of lipid and phosphorus-calcium metabolism in the examined children who were on the traditional method of treatment is presented in Table 2 and Figures 2.1 and 2.2. From the data in Table 3.2, it follows that in children with rickets of 1 and 2 severity, the calcium content significantly decreased ( $P<0.01$ ), ( $P<0.02$ ), as well as the phosphorus content ( $P<0.02$ ), ( $P<0.05$ ) compared with the corresponding indicators in healthy children. Alkaline phosphatase activity was significantly ( $P<0.05$ ) increased compared to healthy children. In rickets aggravated by pneumonia and malnutrition, similar changes were observed, which, apparently, also depend on vitamin D deficiency and the layering of concomitant diseases is not the cause of the aggravating violation of phosphorus-calcium metabolism.

Table 2  
Dynamics of some indicators of lipid and phosphorus-calcium metabolism in examined patients who were on traditional treatment

Indicators	Healthy	Rickets I degree		Rickets II degree	
		I	II	I	II
Ca	2,51±0,04	1,49±0,06	2.16±0.06	1.49±0.07	2.17±0.13
P	1,66±0,05	1,23±0,11	1.23±0.12	1.12±0.13	1.26±0.18
AP	6,10±0,20	6.78±0.07	6.60±0.06	6.72±0.34	6.60±0.12
OL of blood	4,61±0,28	6.96±0.74	5.39±0.27	6.98±0.27	5.57±0.33
OL feces	0,42±0,05	0.74±0.07	0.71±0.06	0.85±0.07	0.60±0.08
PUFA	48,89±3,97	36.57±2.16	41.67±2.07	37.45±2.35	36.52±1.81
NLC	54,30±2,09	58.10±1.54	56.34±1.36	59.08±1.22	56.76±1.54
PUFA/UFA	0,80	0,63	0,74	0,63	0,64

Note: I - data at admission; II - data at discharge

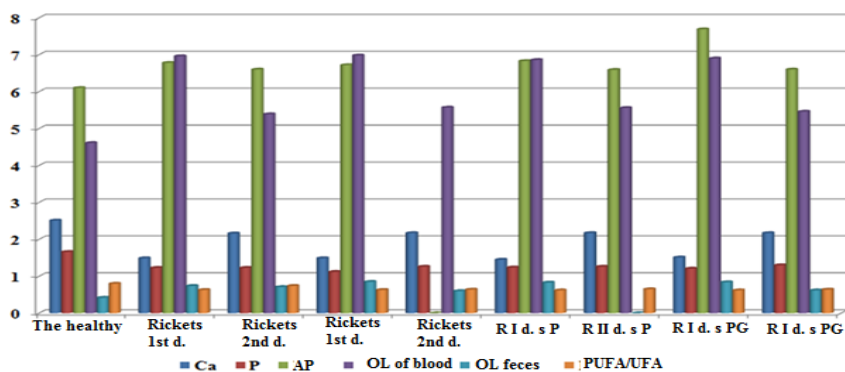


Figure 2.1 Dynamics of indicators of phosphorus-calcium metabolism, total blood and fecal lipids in children with rickets who were on the background of traditional therapy

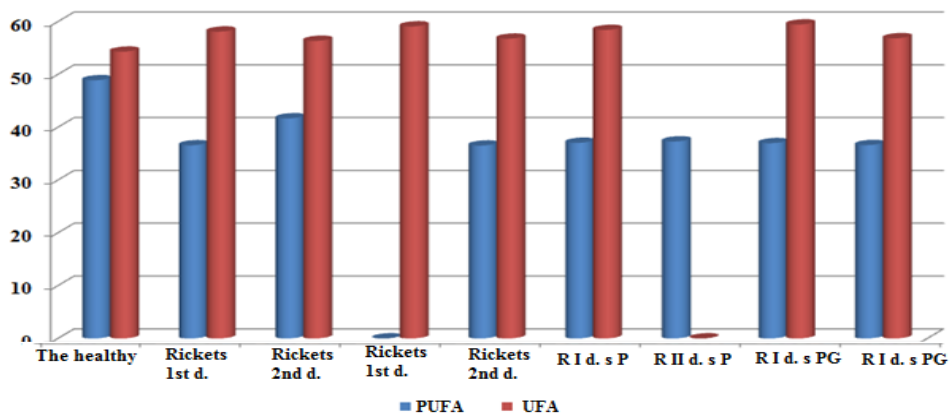


Figure 2.2 Dynamics of some indicators of lipid metabolism (HFA spectrum) in children with rickets who were on the background of traditional therapy

The level of total blood lipids under the influence of the generally accepted complex therapy for rickets remains unreliably elevated ( $P>0.1$ ) in rickets of the I degree of severity, in the II degree of the disease it is significantly increased ( $P<0.05$ ). A slight decrease in the elevated content of total blood lipids can be noted, although there was no complete normalization of their content in the blood. The content of total lipids in feces at the I degree of rickets remained significantly increased ( $P<0.01$ ), at the II degree, similar changes were unreliably ( $P>0.1$ ) compared with the indicators of healthy children.

The amount of EFA in rickets of the I degree of severity decreased unreliably ( $P>0.1$ ), in the II degree of the disease it decreased significantly ( $P>0.5$ ), and the amount of EFA remained unreliably increased ( $P>0.1$ ), ( $P>0.5$ ) compared with the norm. The coefficient of unsaturation of fatty acids in rickets of the I degree of severity was 0.74, and in the II degree of the disease it decreased to 0.64, not reaching the rate of healthy children.

When burdening rickets with other diseases, a decrease in the amount of unsaturated fatty acids is also noted, especially with pneumonia and malnutrition. The amount of EFA was increased with rickets of the II degree of severity and with aggravation of rickets by pneumonia and malnutrition. Therefore, comorbidity exacerbates the deficiency of unsaturated fatty acids in rickets. The coefficient of unsaturation of fatty acids is slightly reduced with rickets of the I degree of severity, with rickets of the II degree of the disease and the layering of concomitant pathology, it is significantly reduced, especially when rickets is aggravated by pneumonia and malnutrition to 0.62.

### Conclusions

In children with rickets and when rickets was aggravated by pneumonia and malnutrition, after the traditional method of treatment, there was no normalization of phosphorus-calcium metabolism and some indicators of lipid metabolism. Along with the clinical recovery, total blood and fecal lipids, SFA, and alkaline phosphatase activity remained elevated, while the content of calcium, phosphorus, and USEFA were below normal, which indicates an "incomplete recovery" and apparently requires further correction of biochemical parameters.

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