Volume 2, Issue 02, February 2024 **ISSN (E):** 2938-379X

METABOLISM DISORDERS IN CHRONIC NUTRITIONAL DISORDER WITH ACUTE INTESTINAL INFECTION IN PREMIUM CHILDREN

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Abstract:

Chronic nutritional disorder (dystrophy) takes a significant place among the pathologies of early-aged children, and it causes a decrease in the body's resistance to infectious diseases, immune reactivity, and endangers the child's health and life. As a result of severe dystrophy (hypotrophy 1st, 2nd, 3rd degree, paratrophy) in early children, it causes the child to lag behind in neuro-spiritual and intellectual development [1,4,9,11,14,21,23].

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Introduction

As a result of chronic malnutrition, tissue and organ malabsorption of nutrients occurs. The most common causes of chronic malnutrition are violations of the rules of breastfeeding and infection with infectious diseases. In addition, it can be caused by a violation of the mother's nutrition in the antenatal and postnatal period, a number of acute and chronic diseases, toxic substances and side effects of some drugs. Everyone knows that during pregnancy, the demand for food and its nutrients increases, which in turn determines the health of both the mother and the unborn child [2,3,8,12,15,19,24].

Unbalanced nutrition during and after pregnancy, the main nutrients in food; lack of proteins, fats, carbohydrates, minerals and vitamins has an adverse effect on the development of the fetus and causes the development of various defects in the fetus. Disruption of the metabolism process in the fetus and newborn child leads to a violation of physical and mental development [6,7,13,17,22,25,26]. It should be noted that even mild forms of chronic malnutrition can lead to serious and long-lasting diseases of breathing, digestion, urination and other organs, and as a result, a deep disturbance of the metabolic process is observed, and a characteristic "defective circulation" occurs. In recent years, in addition to the usual forms of chronic nutritional disorders, i.e., violations of the rules of nutrition (hypotrophy, paratrophy) and forms that develop due to infectious diseases, there are also forms that are somewhat complicated and difficult to treat, as well as those that cause severe damage to the child's organism in the antenatal and intranatal period. meets. One of these forms is dystrophy, which occurs as a result of metabolic disorders. Such forms of chronic malnutrition are caused by the deficiency of certain enzymes involved in the metabolism [3,4,9,13,17,24,25]. In dystrophy associated with exchange disorders, the usual

treatment does not work. We cannot say that there has been enough targeted research in this area. Currently, in the literature published in the world and in our country, there is a lot of information about investigations related to the violation of protein, fat, carbohydrate, trace elements, water-salt exchange in the body in chronic malnutrition, but the information about the violation of amino acid exchange is not sufficiently covered [12,16,20,21,22,24]. The purpose of the work is to determine the levels of etiological factors and proteins and their fractions, amino acid metabolism disorders that lead to antenatal and postnatal forms of dystrophy in premature children with chronic nutritional disorders complicated by acute intestinal infections [15,17,21,23,26].

Materials and Methods:

154 newborns and their mothers were examined for factors that lead to chronic nutritional disorders in the antenatal period, based on specially prepared cards. 46 healthy children aged 1 month to 2 years were observed to determine normal body weight and height in early childhood. 200 people; The amount of free amino acids in blood serum and daily urine was checked in 12 healthy children aged 3-6 months, 10 children aged 6-12 months, 24 healthy children aged 1 to 2 years, and 154 children with chronic malnutrition complicated by acute intestinal infection. The amount of total protein and its fractions in blood serum was checked by the refractometric method using the Human reagent on the Mindray apparatus. The amount of amino acids in blood serum and the amount of amino acids in daily urine were determined by the chromatographic (colorometric) method. The mother's anamnestic data, diseases during pregnancy, somatic diseases, and the course of pregnancy were studied. It is important to study the disorder of protein-amino acid metabolism in the case of acute intestinal infection in children with chronic malnutrition, because the main part of the protein in the body is used for the child's growth.

Table №1 **Indicators of serum protein and its fractions in hypotrophy complicated by acute intestinal infection**

Forms of hypotrophy complicated by acute intestinal infection	Indicators %			
	Total protein	Albumin	Alpha globulins	Beta globulins
Hypotrophy I degree	6,0±0,2	55,8±0,4	10,4±0,4	8,1±0,2
Hypotrophy II degree	5,4±0,2	53,2±0,6	7,8±0,2	7,0±0,2
Hypotrophy III degree	5,2±0,4	50,4±0,7	6,4±0,2	6,5±0,2
Healthy	5,2-6,8	55,1-64,3	7,2-13,5	7,2-10,2

The investigation showed that in healthy children, even when the disease was moderate and severe, there was no sharp decrease in the amount of total protein, which turned out to be due to the compensatory capabilities of the organism. In hypotrophic children, it was observed that protein and its fractions were decreased in blood serum, based on this, it can be said that sick children are very sensitive to protein deficiency.

Table №2 **Daily excretion of free amino acids (mg) in healthy and chronically malnourished children.**

Distro clinical form of fiya ri	Age	Monoaminocarbon		Dibasic		Dicarbon		Glycine	
		Isolated form	Isolated form complicated with acute intestinal infection	Isolated form	Complicated with acute intestinal infection	Isolated form	Complicated with acute intestinal infection	Isolated form	Complicated with acute intestinal infection
1	2	3	4	5	6	7	8	9	10
Hypotrophy 1st degree	3-6 months	94±6	126±12	30±3	38±3	32±2	37±4	5,7±0,9	6,2±0,7
		92±8	104±12	32±3	35±4	28±2	33±4	6,2±1,0	7,1±0,8
	6-12 months	92±7	112±1,1	28±2	36±4	24±2	34±3	6,3±1,0	8,3±0,8
		81±7	94±12	31±3	32±3	20±1	33±3	6,5±0,8	7,2±1,0
	1-2 Age	92±8	114±10	28±2	32±3	24±2	30±3	7,0±0,8	9,1±1
		84±7	94±10	26±2	25±2	20±2	27±2	7,2±0,9	8,0±1,3
Hypotrophy 2nd degree	3-6 months	110±10	128±10	44±3	47±3	30±2	34±3	8,1±1,2	7,0±1,3
		95±8	106±12	38±2	41±3	30±2	25±2	7,1±0,9	7,2±1,2
	6-12 months	106±9	120±10	34±3	42±3	28±2	32±3	8,7±1,2	7,3±0,9
		92±7	96±10	35±2	35±3	26±1	28±2	8,6±0,8	6,0±1,0
	1-2 Age	104±9	122±10	33±3	42±4	24±2	35±3	10,4±1,0	5,2±1,4
		92±7	108±10	33±5	25±1	26±2	27±3	5,2±1,4	7,1±1,4
Hypotrophy 3rd degree	3-6 months	125±10	140±12	76±4	84±6	44±3	46±4	5,0±0,9	5,6±1,0
		104±14	126±12	44±2	58±5	32±2	33±3	5,7±1,0	6,3±0,9
	6-12 months	122±10	144±18	64±4	78±7	36±3	42±3	5,±1,0	6,1±1,2
		98±7	124±14	44±3	60±6	28±2	28±3	6,0±0,9	5,6±1,1
	1-2 Age	124±10	132±14	56±4	71±5	33±2	36±3	6,3±1,2	6,2±1,4
		91±7	104±12	42±2	51±5	28±2	32±3	7,2±1	7,1±1,2
Paratrophy with normal body weight and height	3-6 months	144±10	156±14	80±3	87±6	34±2	34±3	8,4±1,1	8,8±1,6
		114±9	114±12	60±3	65±6	30±3	30±2	7,0±1,0	7,4±1,1
	6-12 months	140±11	158±13	66±4	76±4	28±2	32±2	9,2±1,3	11,1±1,2
		108±7	111±12	52±3	54±4	23±1	26±2	8,4±0,9	7,5±1,3
	1-2 Age	138±10	165±16	62±4	74±5	24±2	32±2	11,3±1,6	10,4±1,5
		106±9	102±12	52±2	59±5	22±2	24±3	8,7±1,3	8,1±1,1
Paratrophy with excess weight Hypotrophy 1st degree	3-6 months	136±10	164±18	83±6	90±10	33±2	34±4	8,1±1,5	10,2±2,1
		112±8	148±16	52±2	73±8	30±2	32±3	7,2±1,3	9,3±1,8
	6-12 months	140±10	164±18	72±6	83±8	36±2	50±4	9,5±1,3	12,7±1,5
		108±8	134±15	52±4	64±6	28±2	28±3	8,2±1,1	11,4±2
	1-2 Age	155±12	178±19	71±5	75±6	28±2	30±3	13,3±1,3	12,1±1,2
		108±7	125±14	48±3	54±4	24±2	27±2	11,4±1,1	9,2±1,6
Healthy children	3-6 months	84±7	-	36±3	-	26±2	-	8,4±1,2	-
	6-12 months	82±6	-	30±2	-	16±2	-	7,9±1,1	-
	1-2 Age	78±6	-	26±2	-	16±1	-	7,8±1,8	-

According to investigations, mild disaminoacidemia, hyper and disaminoaciduria were observed in hypotrophy of the 1st degree depending on age. That is, the younger the child's age, the more dysaminoacidemia and dysaminoaciduria were observed. Some deep disturbance of amino acid metabolism was observed in the case of hypotrophy of the 1st degree in addition to acute intestinal infections. Hyperaminoaciduria in the urine was observed due to increased excretion of histidine, phenylalanine, tryptophan, aspargine and glutamic acids, cystidine. Disaminoacidemia and disaminoaciduria were slightly higher in 2nd degree hypotrophy. Monoaminocarbon amino acid levels were observed in children aged 3-6 months and 1 to 2 years of age, and dibasic and dicarbon amino acid disaminoacidemia was observed in children of all ages examined. In hypotrophic children of all ages, it was found that glycine and arginine content in daily urine was uniformly lower than normal. In grade 2 hypotrophy complicated by acute intestinal infection, dysaminoacidemia and dysaminoaciduria were observed. It was found that biochemical normalization of amino acid metabolism was not always observed during clinical recovery. Taking into account the above, after inpatient treatment, it is indicated that rational nutrition norms should be developed. In hypotrophy of the 3rd degree, deep disorders of amino acid metabolism in the form of disaminoacidemia and disaminoaciduria are observed. In addition, disaminoacidemia and aminoaciduria of monoaminocarbon, dibasic and dicarbon amino acids were observed even after clinical recovery. A similar situation was observed in hypotrophy of the 3rd degree complicated by acute intestinal infection. When the amount of amino acids was checked in children with paratrophic patients with normal weight and height, high concentration of amino acids in daily urine was revealed, along with hypoaminoacidemia in the blood serum. It was found that hyperaminoaciduria has an account of histidine, tryptophan, lesine, arginine, cystidine. Some profound hypoaminoacidemia and hyperaminoaciduria were observed in paratrophic children with excess amino acid metabolism. In cases complicated by acute intestinal infections, total hypoaminoacidemia has been observed, and clinical improvement has been observed for a long time. Based on this, it can be said that the disorder of amino acid metabolism in overweight paratrophic children is considered to be long-lasting, and it indicates that corrective treatment should be continued even after the inpatient treatment. According to the obtained results, the total amount of monoaminocarbon, dibasic and dicarbon amino acids did not change in the group of healthy children. It was determined that the amount of urine content depends on the age, that is, the younger the child, the more they excrete with urine.

Conclusion

Factors causing dystrophy in the prenatal period were found to be cardiovascular diseases in mothers, toxicosis late in pregnancy, nephropathies, irrational diet (lack of protein, vitamins and carbohydrates in food), diabetes, anemia.

The results of clinical-biochemical investigations have shown that the pathogenesis of any form of chronic eating disorder lies in a metabolic disorder. In some cases, metabolic disorders lead to chronic nutritional disorders, and in other cases, they appear as a

complication. Therefore, it is necessary to develop and implement rational nutrition standards even after clinical recovery in children with chronic nutritional disorders complicated by acute intestinal infection.

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