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Ministry of Health of the Republic of Uzbekistan

Samarkand State Medical University

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Monograph

Tashkent -2024

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Authors:
Akhmedova Makhbuba Makhmudovna - Associate Professor, Department of Pediatrics and General Practice, Samarkand State Medical University, Candidate of Medical Sciences

Reviewers:

The monograph is about urinary syndrome, which is detected in young children with various pathological conditions and therefore requires careful decoding as a non-specific symptom.

Kidney damage in young children is a membranopathy in pneumonia, which is often accompanied by metabolic diseases (hyperoxal and uraturia). Tubular kidney dysfunction in patients with pneumonia is caused by the development of interstitial nephritis and stratification of secondary pyelonephritis.

The introduction of corrective therapy for pneumonia contributes to the stabilization of cytomembranes and the normalization of the urinary syndrome, while improving the homeostatic functions of the kidneys.

This monograph is relevant for nephrologists, pediatricians and neonatologists. The materials of the monograph can be used for teaching subjects such as "Nephrology", "Pediatrics", "Neonatology", for students of medical universities, as well as for postgraduate residents and clinical residents.

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LIST OF ABBREVIATIONS

AUS	-	abnormalities of the urinary system
HU	-	hyperuremia
DSMN	-	dysmetabolic nephropathy
DC	-	diene conjugates
DPh	-	Dimephosphon
LPhCh	-	lysophosphatidylcholine
MDA	-	malonol dialdehyde
MDG	-	mono-diglycerols
UA	-	uric acid
KSD	-	kidney stone disease
US	-	urinary syndrome
UFA	-	unrefined fatty acids
TL	-	total lipids
TCh	-	total cholesterol
LPO	-	lipid peroxidation
SFM	-	sphingomyelin
FCh	-	free cholesterol
BPhA	-	blood phospholipase activity
PhS		phosphatidylserine
PhCh		phosphatidylcholine
PhEA		phosphoethanolamine
ChSE		cholesterol esters

FOREWORD

Nephropathy with metabolic origin is a group that is difficult to distinguish and prone to hidden passage, requiring early differential correction depending on the type of dysmetabolism and the stage of development of kidney pathology.

The blurring of the clinical picture, the predominance of non-specific signs, and the frequency of hereditary and congenital forms of kidney pathology make the following complex studies necessary for kidney damage in pneumonia: genealogical analysis, biochemical screening of urine, ultrasound examination of the kidney, and partial kidney function. Membranotoxic damage to the kidney is clearly observed in pneumonia, but in patients with kidney damage, it should be carried out for at least two to three weeks and always with correction of dysmetabolism.

In young children with pneumonia with urinary syndrome, the duration of dispensary observation should be at least one year.

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INTRODUCTION

Kidney disease in children is a common pathology in the paediatric population, ranging from 16.6 to 54:1000. It is an urgent problem in modern paediatrics due to the frequency of hidden passages and the tendency to chronicity. The widespread application of clinical-genetic and biochemical research methods to clinical nephrology has made it possible to define changes in their nosological structure, resulting in an increase in the frequency of multifactorially defined clinical forms. Thus, according to the results of epidemiological studies, the main structure of nephropathies (34–40%) is nephropathies of metabolic genesis, including. 10–19% oxalate and 14–29% urate nephropathies. At the same time, kidney pathology is manifested and determined in connection with the organ pathologies of newborns in 34.2%, among premature children treated for various broncho-pulmonological diseases in 17–35%, and for staphylococcal infections in 78.6%. In addition, 24.2 to 31.5 percent of children are treated with residual urinary syndrome after the primary disease is eliminated. Taking into account the above and the existing ideas about urinary syndrome in children of this age, the most common broncho-pulmonological pathology in this period ("infectious kidney," "toxic kidney," "toxic-infectious kidney") poses a significant threat to the health of the living organism. There are several reasons for this: firstly, it is known that the development of toxic conditions in young children (including kidney damage) is not a random phenomenon, and toxicosis in pregnant women, the presence of hereditary metabolic diseases, and nephropathies play an important role in their development; secondly, it is an initial manifestation characteristic of hereditary, dysmetabolic, and congenital kidney diseases with the most serious prognosis. In practice, several episodes of urinary syndrome against the background of various intercurrent diseases are currently being evaluated as infectious or toxic kidney. At the same time, with modern methods of examination of children and relatives and the widespread introduction of genetic and biochemical methods into nephrological practice, kidney damage is difficult to differentiate from urinary syndrome. In such cases, the treatment of dysmetabolism without differential correction is mainly associated with the urinary syndrome, which can

lead to the stability and chronicity of the renal process, which is of independent medical and social importance. Most of the works are devoted to the study of kidney problems in respiratory diseases, and they mainly shed light on the homeostatic functions of the kidneys. A generally accepted view of the mechanisms of kidney damage in respiratory pathology. In fact, the role of heredity and metabolic and membranopathological aspects in this matter have not been studied from the point of view of modern clinical membranology, but the disturbance of antioxidant and membrane-stabilising systems has been clearly proven in broncho-pulmonological diseases.

CHAPTER 1. POLYORGAN MEMBRANOPATHY AND FUNCTIONAL CONDITION OF KIDNEYS IN PNEUMONIA OF CHILDREN. (LITERATURE REVIEW)

1.1. Polyorgan membranopathy and functional status of kidneys in pneumonia in children.

Despite significant progress in the diagnosis and treatment of kidney diseases in children, it remains an urgent problem in modern paediatrics due to the prevalence of 16.6 to 54 per 1000 children.

It is known that in 13.1–35.3% of cases of kidney pathology, the pathology of the respiratory system is preceded or accompanied by it. In addition, clinical observations in recent years against the background of the pathology of the respiratory tract are congenital and hereditary, including dysmetabolic nephropathies. Acute activation of cytomembrane LPO ("oxidative stress") in pneumonia caused by hypoxemia has been convincingly shown to be the main secondary pathogenetic basis for the appearance of almost all non-specific syndromes in this pathology: central nervous system, cardiovascular systems, causing critical conditions syndromes that cause polyorgan membranopathy in hypoxic conditions.

The importance of LPO stimulation of cytomembranes in various renal pathologies is also a proven fact.

According to modern concepts, kidney pathology is formed against the background of altered reactivity under the influence of unfavourable factors in the antenatal period, in which disorders develop in the entire functional maternal-placental-fetal system in conditions of oxygen deficiency and metabolic acidosis. (Acute and chronic diseases of the mother; nephropathy of pregnancy). In the intranatal period (asphyxia, birth injury, use of certain medicinal substances, and other factors) and postnatal development period (chronic diseases of nutrition, rickets, artificial feeding, frequent respiratory diseases),.

Among the reasons that lead to the appearance of urogenital system pathology, in addition to changes in the body's reactivity, it is necessary to take into account the values of genetic factors predisposing to kidney diseases, various enzymatic and non-enzymatic metabolic system disorders, and immunological defects. Thus, the problems of nephropathy in the neonatal period are the most relevant. Due to the absence of clinical manifestations, the predominance of nonspecific symptoms, the lack of clear diagnostic criteria, the frequency of hereditary and congenital forms, and late diagnosis, they form chronic kidney pathology in the elderly.

Most often, information on the prevalence of kidney diseases is presented in the literature according to the data on the visits of patients to the doctor, but this does not fully reflect the true prevalence of nephropathy since the forms of pathology that are not clinically visible in childhood prevail. In recent years, microbial-inflammatory diseases of the urinary system and dysmetabolic nephropathies have become common. At the same time, targeted examination of children from families with kidney pathology is very important for the early detection of nephropathy. There are several reports of families with a common, young onset of kidney disease.

In general, the frequency of hereditary and congenital kidney pathology is 21.7–46.3%, which manifests itself as an isolated urinary syndrome in the morning against the background of various intercurrent diseases. Hidden, difficult to diagnose, and difficult to differentiate nephropathies are usually unfavourable prognostically in young children, twice as many as manifest forms with active identification. Thus, in a retrospective analysis of large clinical material (2759 children, S.K. Abdurakhmanova, 1994), 63% (1738 children) of patients aged 4–14 years with kidney pathology diagnosed for the first time had episodes of urinary syndrome for more than 7 years (29%), showing that it was against the background of various intercurrent pathologies, and 25% for more than 5 years in the 4–7 age group. 8–14 years in 17.4%; that is, the onset of the disease usually corresponds to an early age, followed by a latent period. It should be noted that the existing

official statistics based on information on hospitalizations could not reflect the real state of affairs regarding the distribution and structure of kidney pathology in children. It is known that the type of screening to determine the hereditary and congenital pathology of the urinary system can aggravate heredity in terms of kidney pathology, many stigmata of dysembryogenesis, edema syndrome, urinary syndrome, and azotemia of any severity, which is often life threatening. In a child's first year, it is manifested against the background of various intercurrent diseases, especially respiratory diseases.

In children with pneumonia, the main link in the pathogenesis of functional changes in organs and systems is respiratory insufficiency, which is manifested in respiratory disorders of the lungs and tissues, hypoxia, and hypoxemia. Under the influence of hypoxia and hypercapnia, the functional state of the central nervous system and neuro-reflective regulation of vital functions are disturbed, and changes occur in metabolism and, first of all, in energy exchange. Thus, the development of tissue hypoxia in pneumonia can act as a universal trigger for the development of polyorgan membranopathy.

Hypoxia, bacterial toxins, metabolic disorders, changes in products, and hemodynamics can lead to toxicosis, the manifestation of hidden hereditary or congenital toxicosis, the manifestation of hidden hereditary or congenital pathology of the urogenital system, because they increase the activity of LPO and phospholipase, accompanied by activity and restructuring of the antioxidant defence system.

At the moment, it is known that the most important factors that cause kidney damage in the development of toxicosis are the age of the child, the presence of hereditary metabolic diseases, and maternal toxicosis during pregnancy. Thus, hypoxic, hemic, hemodynamic, and enzymatic disorders that appear in any pneumonia are clear factors that cause polyorgan membranopathy, which explains the involvement of various organs and systems in the pathological process.

A significant increase in kidney function is required to maintain homeostasis with hypoxia. Uncompensated sodium loss syndrome may occur in conditions of solipra consumption limited to diarrhoea, vomiting, increased sweating, or pneumonia. In such conditions, due to a decrease in sensitivity to mineralocorticoid effects, despite the acute lack of sodium, its massive excretion continues, which leads to hypovolemia, decreased glomerular filtration, oligoanuria, and azotemia. The kidney is the main executive organ in the management of osmohomeostasis; therefore, undoubtedly, in inflammatory diseases, their function of maintaining homeostasis is disturbed, and in the pathogenesis of nephropathy, various disturbances of the water-electrolyte balance are of great importance. Changes in the concentration of the osmotic fluid significantly affect the functional state of the vital organs and thus lose the properties of the cell membrane as water and sodium enter the cell. Cellular dehydration and cellular sodium intoxication are responsible for neurological symptoms. As a homeostatic organ, the kidney has a great compensatory capacity; therefore, the study of blood composition, which is widely used in practice, does not fully reflect the state of the partial functions of the kidneys. Adequate assessment of these functions can be made only by taking into account the influence of the kidney structures on changes in homeostasis caused by the introduction of various test tools. Only in such conditions do real reserve possibilities for intrarenal transport of substances appear.

Among the causes of death in children, pneumonia occupies a large place. However, according to VK Tatochenko (1979), in only 5% of children who died from 1 to 11 months, pneumonia was the main cause of death; in 50%, it was one of two or more competing causes. In addition, this did not play a leading role in the pathogenesis of pneumonia in 45% of cases. Hidden kidney pathology in children is caused by disorders in the formation of organs of the urinary system. Against the background of respiratory diseases, there is an opinion that without the aggressive secondary nature of such renal changes, they lead to serious consequences, as this is typical for most of the hereditary and dysmetabolic nephropathies with the most serious prognosis.

Only an active investigation can determine the true prevalence of diseases of the urinary system among the children's population, because neither the analysis of reports nor the study of statistical documents of medical institutions can reflect the special frequency of hidden kidney pathology in children, which manifests not only in kidney pathology but also in children (vaccinations against ARVI, bronchitis, pneumonia, AII). It recommends the use of a stepwise system, starting with screening tests for pathological changes in the urine and sequentially excluding people without pathology in the urinary system. According to the observations of Ostrapolus (1973), the frequency of involvement of the kidney in the pathological process in chronic pneumonia is: in the acute stage, proteinuria was found in 16.7%, hematuria in 12.9%, and leukocyturia in 26.9%. According to KakovUAiy-Addis, in the study conducted, hidden kidney damage was found in 17.8% of children who did not change during the general clinical examination. According to N.I.MrinUAaya (1974), in the examination of 282 sick children with various clinical forms of staphylococcal diseases, kidney damage was detected in 136 (48.1%), including 78.6% in children under 3 years of age. 25 out of 136 children with kidney damage were found to have changes in urine before hospitalization, 111 with pneumonia, ARVI, etc. were found in the process of clarifying the diagnosis. The stages of identifying diseases of the urinary system have proven themselves in practice. If at the first stage the problem of preliminary identification of kidney diseases is solved with the help of screening tests, then the purpose of the final examination of the disease process is to determine the functional status of the kidneys and the treatment and prognosis of the disease.

Deepening the examination of children and their relatives, with the wide introduction of genetic and biochemical research methods into nephrological practice, a number of metabolic diseases that are difficult to distinguish include "toxic-infectious" and "toxic" lesions of the kidney with nephritis-like urinary syndrome, which considers it to be a secondary, transitory, apparently harmless change of the kidneys observed in the middle of the pathological process. At the same time, if the urinary syndrome was once manifested

against the background of intercurrent diseases, it often recurs in the future. They take pictures of specific variants of kidney pathology and take a chronic course with all the consequences**.** Inadequate treatment without correcting metabolic diseases contributes to the development of the renal process, the stratification of pyelonephritis and interstitial nephritis, and, as a result, the development of chronic kidney failure. Depending on the type of dysmetabolism, there should be a diet for drug correction; of course, the approach should be individual. Therefore, treatment based on changes in urine without taking into account the type of dysmetabolism can lead to a paradoxical effect, that is, to the deterioration of the kidney process.

Thus, conclusions about the nature of urinary syndrome against the background of respiratory pathology are usually made based on the presence of the disease and the severity of toxicosis. The question requires justification: why, with the severity of bronchopulmonary disease, only a small percentage of children have kidney damage? This explains the persistent and sometimes progressive nature of changes in the kidneys after the elimination of symptoms of toxicosis and even complete clinical recovery from the underlying disease in other children, the kidneys are not affected. Undifferentiated damage to the kidney in respiratory diseases in young children is always a polyorgan membranopathy, which is confirmed by a systematic violation of the peroxide modification of phospholipids of cell membranes, and in almost all children, it is accompanied by symptoms of damage to many organs. Treatment of such patients should be taken into account during the medical examination. Thus, the frequency of kidney damage in pneumonia, their frequent stabilization and tendency to a chronic state point to the need for further study of this problem from the point of view of modern clinical memranology.

1.2. Study of stability of cytomembrane status in early-aged children with DSMN.

Cell membranes occupy one of the central places in the organization of living systems, they ensure the integrity and functional state of the cells in intracellular structures.

The biological role of membranes in the body is determined by the extreme importance and diversity of their functions. The main functions of membranes are: participation in the formation of cellular structures, active and passive transport of substances, maintenance of intracellular homeostasis, participation in phase-mechano-hemoreception processes, secretion absorption, gas exchange, tissue respiration, energy storage and change, implementation of intercellular interactions, construction and activation of histo-haematological barriers. Despite the fact that different biological membranes have specific characteristics related to the performance of specific functions by them, their structure is asymmetrically present in a continuous lipid bilayer with membrane proteins incorporated into it (E.M. Kreps 1981). Cholesterol and phospholipids make up the bulk of lipids. Membrane proteins determine their structure, perform the functions of enzymes, some of them are involved in membrane transport and the cell receptor system. They also determine the antigenic properties of cells. The physical state of membranes, microviscosity, liquid crystal structure, and membrane potential play an important role in the formation of pseudopodia and cell movement, phagocytosis and pinocytosis, membrane transport, cell aggregation, and the functions of membrane receptors and antigens. The lipid component of these membranes (mainly cholesterol and phospholipids) determines their fluidity or microviscosity, depending on the degree of ordering of the acyl chains of fatty acids. These properties also give membranes the degree of saturation of fatty acids since saturated analogues are less liquid. In particular, an increase in the ratio of cholesterol to phospholipids is accompanied by a decrease in membrane fluidity, an increase in the sensitivity of cells to the cytolytic effect of the component, and a change in the ion permeability of membranes. Advances in the field of membranology have made it possible to study the pathogenetic role of disturbances in the structure and function of cytomembranes in nephropathies in children. It has been shown that kidney diseases with hyperoxal, uraturia, and calcium-oxalate crystalluria are accompanied by a violation of the metabolism of cell membrane phospholipids and the

separation of nitrogenous components contained in phospholipids-seliatin, ethanolamine FEA, serine, and ethanolamine conjugates with fatty acids.

The complex complex of biochemical changes that determine the development of dysmetabolic nephropathies is primarily characterized by the disturbance of lipid metabolism, which is the main component of any biomembrane and determines the quality change of the functional properties of the cell.

Lipid peroxidation (LPO) reactions continue continuously with the formation of active products (free peroxide radicals, hydroperoxides, aldehydes, and ketones) in the tissues of a living organism, have a chain character, and are the natural background of ionizing radiation in the physiological restoration of membranes participates together with siri, ultraviolet radiation, is preserved to a certain extent, and is an important indicator of homeostasis.

The activation of LPO, excessive accumulation of free radicals in cell membranes, and the balance between the formation and decomposition of peroxides have a toxic effect and affect the structural and functional properties of cell membranes, primarily permeability and enzymatic activity. Changes lead to the breakdown of lecithin, slow cell division, and a decrease in energy supply to cells. This effect of oxygen radicals on tissues depends on the peroxidation of polyunsaturated fatty acids in membrane lipids and the formation of toxic aldehydes, as well as the inactivation of enzymes and their interaction with the DNK of the cell nucleus. Excessive activity of LPO processes leads to the formation of permeable pores through which ions and water pass; the fatty acid saturation of phospholipids decreases; and myelin-like structures are formed, which return the physical properties of membranes to cytomembranes reduce until it causes irreversible damage. Increased LPO activity in pneumonia, acute respiratory viral infections in children, bronchial asthma, septic pathology and hypoxic condition of newborns, obesity, peptic ulcer disease, anaemia in late toxicosis of pregnant women, rheumatism, rheumatoid arthritis, acute and chronic hepatitis and cirrhosis of the liver, and a number of other

diseases belonging to the group of free radicals are returned. Currently, the information available in the literature allows us to classify the majority of certain kidney diseases with their mechanisms of emergence, development, and chronicity as membrane pathologies. Thus, cell membrane damage is observed in a number of immune and non-immune nephropathies: pyelonephritis, interstitial nephritis, acute and chronic glomerulonephritis in children, oxalate nephropathies, urate nephropathies, urolithiasis, and the and the accumulation of LPO products in this membrane structure, manifested by an increase in their excretion with urine.

Most often, the detection of dysmetabolic nephropathy against the background of the above diseases, as well as the frequency in the genealogy of patients with dysmetabolic nephropathy is explained by the polyorganism of the membranopathological process. It is known that a predisposition to chronically existing somatic diseases can be acquired, whether congenital or hereditary; it is associated with the instability and permeability of cytomembranes, membrane transport disorders, and a decrease in the activity of membrane enzymes can be all kidney diseases, in one way or another, are associated with basement membrane or cellular pathology. In both cases, damage to the kidney membranes can be primary (genetically determined or caused by teratogenic effects on the foetus) or secondary (due to microbial-inflammatory, immune, toxic, or alimentary effects). Thus, kidney diseases can belong to the group of metabolic pathological conditions, the pathogenetic substrate or the source of the formation of low metabolites are previous changes in lipid metabolism, hereditary family characteristics, and specific characteristics of cytomembrane stability. Many researchers have confirmed the leading role of membrane pathology in the development of inflammatory processes. According to experimental studies, changes in the structural and functional properties of cell membranes under the influence of toxic, infectious, immunological, hypoxic, and other factors in the body are primarily associated with an increase in endogenous phospholipase activity and processes, and the level of free radical oxidation of lipids included in the membrane complex. It is known that this is especially evident in hypoxic processes. In recent years, severe forms of

pneumonia in children, due to severe hypoxia under the influence of bacterial toxins, metabolic disorders in the body, changes in the dynamic balance of blood coagulation and the anticoagulation system, which lead to the formation of microthrombosis, which leads to organ dysfunction, and especially a deep disturbance of the functional state of the kidneys.

In pneumonia, in hypoxic conditions, especially in children who are often sick with rickets, hypotrophy, anemia and deficiency (depletion) of antioxidant defence reserves (carotene, L-tocopherol, etc.) develop, then LPO restimulates cytomembranes, malonol dialdehyde, and diene conjugates in the cells until planning is observed. There is a certain familial tendency to encourage LPO in this way. Currently, the model of erythrocytes is often used to study the structural and functional properties of biomembranes in the body, as well as the functional metabolic processes in the body in general.

Structural and functional disorders of red blood cell membranes are one of the causes of microcirculation processes, including disorders at the level of bronchopulmonary and kidney tissues. It is known that the morphofunctional state of microcirculatory systems and their ability to maintain tissue homeostasis in respiratory diseases depend on the course and prognosis of the inflammatory process. In children with pneumonia, hemolysis of red blood cells in the vascular bed disrupts gas exchange and cell metabolism due to the intensification of LPO. Against the background of dyscirculatory disorders in the microcirculation system, the increase in erythrocyte aggregation can lead to a decrease in phagocytosis and irreversible tissue damage, and thus to kidney tissue damage. A multifaceted approach to the study of the pathogenesis of inflammatory diseases in early childhood is currently moving to the concept of the leading role of mechanisms and processes that stabilize cell membranes in their genesis. However, the implementation of this direction in relation to acute pneumonia in children requires a thorough analysis of various directions of dysmetabolic processes, the result of which is a state of membrane neutralization.

Thus, preliminary studies have shown that processes of membrane lipid peroxidation and endogenous phospholipases in young children and their importance in the formation of kidney pathology require special, in-depth study. It is also important critically analyze the genotypic environmental interaction of respiratory pathology and kidney damage through the prism of modern advances in membranology.

1.3. Antioxidant and membrane in pediatrics stabilization therapy.

The use of antioxidants in a number of membranopathological processes has been identified. L-tocopherol is a powerful physiological antioxidant active at the cellular level.

Currently, the use of L-tocopherol acetate in patients with impaired LPO-AOS in the body effectively inhibits the LPO reaction and thus provides a membrane stabilizing effect. From a clinical point of view, the ability of vitamin E to increase the body's resistance to hypoxia is of great interest. The mechanism of the protective effect of tocopherol, on the one hand, is related to the stabilization of cell membranes because it inhibits the decomposition of free radicals and the peroxidation reaction. The TYK of phospholipids, as well as the TYK residues of its side chain, have a stabilizing effect directly through the membrane. The direct interaction of vitamin E with TYK at the membrane level is reduced to the control of their addition to phospholipids and not to the physicochemical antioxidant effect. On the other hand, the antihypoxic effect of tocopherol is related to its direct participation in the oxidative phosphorylation system as an acceptor of hydrogen ions and as a stimulator of ubiquinone synthesis. These properties of tocopherol are the basis of its wide use in clinical practice. It was found that hypoxic conditions lead to a sharp increase in the body's need for vitamin E, which is confirmed by reports of the high effectiveness of including tocopherol in the complex therapy of pneumonia, bronchial asthma, and hypoxic brain damage in premature infants.

In recent years, vitamin E has been successfully used in neurological practice in children with glomerulonephritis, intestinal nephritis, and recurrent stone formation.

Targeted regulation of lipid peroxidation and endogenous phospholipase activity can be an important direction of therapy for the pathophysiological manifestations of hypoxia in children. From this point of view, the methods of controlling the processes of lipid peroxidation of cell membranes through the body's antioxidant systems are the most promising. In the literature in recent years, the prospects of research and clinical application of low-toxic phosphorus organic compounds, including derivatives of mono- and diphosphonic acids, have various physiological and therapeutic effects and have not yet been fully explored.

Dimephosphon (D): dimethyl ether 1, 1-ddimethyl 3-hydroxybutylphosphonic acid, synthesized at the IOPC named after A.E. Arbuzov, Kazan branch of the Russian Academy of Sciences, attracts more attention among the currently available diphosphonates. Experimental studies have proven its low toxicity. D is well absorbed from the gastrointestinal tract and does not accumulate in the body (the drug is removed within the first 5–6 hours after a single intake). D can be used for acute and chronic diseases. It does not have side effects; long-term use may cause a dyspeptic effect, which disappears when the drug is stopped.

Many experimental and clinical studies have shown a wide spectrum of its biological activity: immunomodulatory, antiacidotic, membrane stabilizing, antihypoxic, detoxifying effect, stimulation of regenerative processes, anti-inflammatory activity.

Not being an antioxidant in the literal sense of the word, that is, without acting as a trap for oxygen radicals, the drug having an affinity for membrane phospholipids is added to the membrane cells and destroys the phospholipids of the cell membrane ensures the preservation of qualitative and quantitative composition. By activating the glutathione oxidation-reduction system, D protects the sulfhydryl groups of blood proteins from oxidation, thereby reducing the accumulation of LPO products.

D enhances the reabsorption of bicarbonates, the processes of attido- and ammonogenesis in the kidneys, the ventilation function of the lungs, and normalising KAH in conditions of rickets modelling.

In response to the immunological effect, D reduces the formation of antibodies by separating cells, because of which the hypersensitivity of slow and fast tissues decreases, and finally, the ability to remove toxic metabolites included in the composition of this drug from the body determines the detoxification properties. The introduction of D in the complex treatment of pneumonia accelerates the normalization of KAH, patients begin to feel better, reduce shortness of breath, shorten the period of fever, reduce the duration of intoxication, and experience physical changes in the lungs. The antiacidic effect of the drug appears 2-3 hours after the first dose and increases the blood pH. In vitamin D-deficient rickets, administration of D resulted in normalization of blood Ca and P levels. A course of treatment for rickets-like diseases with acidosis improves the condition of patients within 1-2 months: fatigue and muscle weakness are reduced, and pH is normalized. D was used for pneumonia in newborns, acute respiratory viral infections, and bronchodestructive syndrome.

Thus, the analysis of the literature review confirms the feasibility of including drugs with antioxidant and membrane-stabilizing effects in the complex therapy of urinary syndrome in children with pneumonia.

A number of authors found that healthy young children have higher levels of uric acid in urine and blood compared to children of other age groups, which characterizes the tension of uric acid metabolism at an early age. Since pneumonia is accompanied by secondary hyperuricemia, that is, in the middle of the disease, an increase in the concentration of ammonia and uric acid in the blood and urinary excretion is determined, taking into account the activity of LPO and the suppression of the xanthine oxidase enzyme. In the complex treatment of hyperuricemia (HU), a number of authors use uricodepressors: orotic acid, purine antagonists (azathioprine, 6-mercaptopurine) in proteinuric and secretive types of

nephropathy, and enterosorbents are used to reduce intestinal absorption of UA. Although Y. Pitel (1985) recommended limiting the consumption of vitamin C, information was given about the uricosuric effect of ascorbic acid, since it is a source of oxalate formation. The most effective uric acid depressant is allopurinol. Allopurinol, commonly used as an anti-gout drug, is a strong inhibitor of xanthine oxidase, which eliminates the loss of purine bases, promotes the regeneration of hypoxanthine (which affects six times less energy than the new synthesis of ATF molecules), and is formed in the oxidation of hypoxanthine, which prevents the formation of superoxide ions. Allopurinol, a structural analogue of 4-oxypyrozolone, 3, 4-pyrimidine, and hypoxanthine, participates in UA synthesis, reduces the formation of UA in the blood, prevents the accumulation of UA salts in tissues, and inhibits the synthesis of pyrimidine ribonucleotides in the kidneys. By blocking YSH, allopurinol serves as "trap" free radicals and improves microcirculation. Allapurinol reduces the concentration of uric acid in blood, serum, and urine and reduces the amount of total lipids and free fatty acids. B-lipoproteins, cholesterol. During tissue hypoxia, intensive catabolism of energy-rich intracellular purines (ATP) occurs with the formation of their intermediate metabolism, hypoxanthine. It accumulates in high concentrations in ischemic tissues because, under these conditions, its reutilization is impaired (with the participation of hypoxanthine-gaunin-phosphoribosyltransferase), and its conversion to urates through xanthine is blocked or reduced. Elimination of hypoxia (inhalation of O2, improvement of microcirculation) is accompanied by oxidation of accumulated hypoxanthine in the presence of HCO, which leads to the irreversible loss of hypoxanthine from the nucleotide pool by the formation of uric acid and the abundant production of superoxide ion (O2) will lead to

$$\text{Hypoxanthine} + 2O_2 + H_2O \longrightarrow \text{xanthine} + 2O_2^{+} + 2H^{-}$$

YSH

$$\text{xanthine} \_ 2O_2 + H_2O \longrightarrow \text{uric acid} + 2O_2^{-} + 2H^{+}$$

Allopurinol is used as an antioxidant in neonatology.

Bode et al. (1984) used allopurinol at a dose of 20 mg/kg/day for three days in early respiratory distress syndrome (RDS). Children treated with allopurinol had a relatively short period of positive expiratory pressure. At the same time, there was a decrease in the level of UA in the blood serum and its excretion in the urine, an increase in the urine, and the excretion of creatine in the urine. New properties of allopurinol as a blocking product of LPO processes as a result of inhibition of the xanthioxidant enzyme, which leads to a decrease in the level of UA in the blood, can be used in hypoxic conditions that develop in pneumonia in young children. Thus, it follows from the review that the development of pathogenetic therapy methods in young children with pneumonia with metabolic nephropathy is an urgent problem in pediatrics. In this regard, drugs with antioxidant (vitamin E), membrane stabilizing (dimephosphon), and uricosuric (allopurinol) effects are promising. Despite many studies on the correction of metabolic nephropathies, data on the combined use of dimephosphonate, vitamin E, and allopurinol in the treatment of pneumonia in young children with dysmetabolic nephropathies show that the dynamics of phospholipid metabolism and lipid peroxidation indicators do not reflect partial renal function correction in pneumonia with urinary syndrome.

CHAPTER II. RESEARCH MATERIAL AND METHOD

2.1. Clinical characteristics of patients

Clinical observation was carried out in the pulmonology and nephrology departments of the regional multidisciplinary children's hospital in Samarkand during 1995–1998.

We analyzed the archival materials of 1003 children hospitalized for pneumonia aged 1 month to 3 years. We found the frequency of pneumonia complicated with cardiorespiratory syndrome in SS 177:1000, SS in obstructive syndrome in 64:1000,

neurotoxicosis in 88:1000, and intestinal syndrome in 56:1000 cases. It was observed in 67:1000 cases of pneumonia with cardiorespiratory syndrome and neurotoxicosis and in 24:100 cases of MS with intestinal and neurotoxicosis. Twenty of them developed acute renal failure, including six cases of death. The duration of urinary syndrome was 6–15 days; 208 children were discharged with SS. (Table 1). The obtained data made it possible to determine the frequency of pathological reactions to the components of urine (Table 2). In urinary syndrome, proteinuria was 51% (r = 0.51) and bacteriuria was 5% (r = 0.52). Saluria r = 1.12; erythrocyturia occurred with a frequency of r = 0.64. Among the combined positive urine reactions, erythrocytes + salts (r = 0.53) and leukocytes + erythrocytes + salts (r = 0.60) are more common. Studies have shown that 476 (47.5%) of 1003 patients hospitalized with pneumonia in early childhood had urinary syndrome.

Thus, urinary syndrome in pneumonia is noted with temporary changes in urine. The children were prescribed antibiotic therapy for pneumonia, their pneumonia was cured, but the kidney disease remained undiagnosed, so an appropriate correction of the urinary syndrome was not performed. In connection with the above, we examined 130 young children with dysmetabolic nephropathy pneumonia. The structure of nephropathy in children with pneumonia is presented in **Figure 1**.

FREQUENCY OF THE URINARY SYNDROME

IN CHILDREN WITH PNEUMONIA

Table 1

Indicators	Full time				Before the deadline				Total
	0-1 year		1-3 young		0-1 year		1-3 young		
	B	G	B	G	B	G	B	G	
	n=184	n=179	n=159	n=151	n=126	n=97	n=59	n=48	
Pneumonia (total), incl	184	179	159	151	126	97	59	48	1003
Cardiorespiratory s - m + urinary syndrome	42	41	29	33	12	9	7	4	177
Obstructive s - m + urinary syndrome	6	8	13	16	9	6	2	4	64
Neurotoxicosis s - m + urinary syndrome	11	13	14	18	16	12	2	2	88
s in the intestine - m + urinary syndrome	2	5	6	3	19	14	3	4	56
Cardiorespira + neurotoxicosis + urinary syndrome	11	8	11	4	14	11	6	2	67
Cardiorespir + Intensin + Neurotok + Urinary syndrome	1	3	3	1	7	6	2	1	24
including ACF	4	3	6	3	2	1	1	-	20
including death	1	1	-	-	2	1	1	-	6
Duration of urinary syndrome (days)	6-8	7-11	4-5	5-7	9-12	13-15	6-8	6-9	-
Issued with SS	34	36	33	32	22	18	16	17	208

Akhmedova M.M.

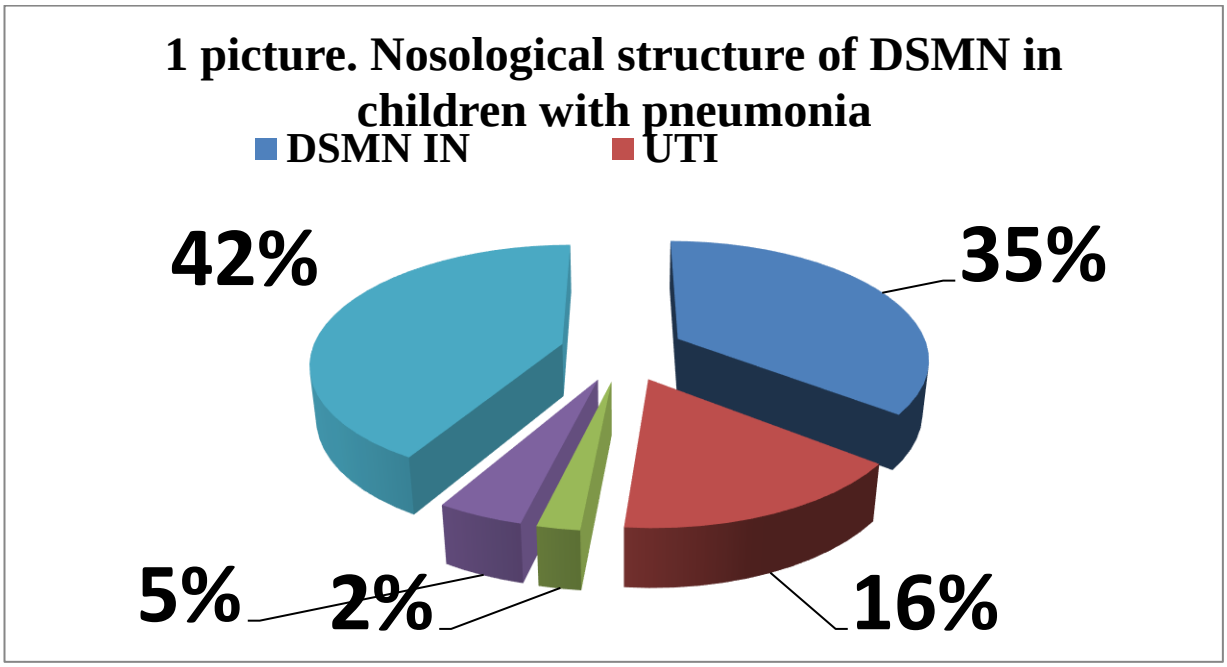
CHARACTERISTICS OF URINARY SYNDROME IN CHILDREN'S PNEUMONIA OF EARLY AGE (%)

Table 2

Indicators	Age and gender				
	up to 1 year = 248		up to 3 year = 228		Total
	B	G	B	G	
	n=130	n=118	n=121	n=107	n=476
Protein	12,0	13,0	12,0	14,3	51,3
Bacteriuria (Bk)	13,0	14,0	12,1	13,2	52,3
Leukocyturia (L)	13,1	14,2	12,1	12,1	51,5
Erythrocyturia (E)	14,0	19,0	13,0	18,0	64
Saluria (C)	25,0	29,0	30,0	28,2	112,2
L + E + S	14,0	15,0	15,1	14,8	59,9
L + S	13,1	13,0	12,0	12,4	50,5
E + S	14,0	13,0	14,0	12,2	53,2

Dysmetabolic KI in 54 children (41.5%), dysmetabolic IN in 46 children (35.4%), USA in 21 children (16%), tubulopathy in 3 children (2.3%). 6 children have OSS (4.6%). Since secondary BY with saluria was noted in children with USA and tubulopathy, this group of children was assigned to the group of patients with dysmetabolic BY in the course of development. There were 71 boys, 92 girls, boys often suffered from IN, USA, tubulopathy; girls DSMN, dysmetabolic and primary BY, urinary tract infection. Analysis of the structure of nephropathy in pneumonia in young children shows that dysmetabolic nephropathy takes the leading place in the structure of nephropathy in children. Thus, special studies conducted in children with pneumonia confirm the presence of dysmetabolic nephropathy in children and its tendency to the hidden path. The diagnostic criteria for dividing patients into groups were the results of clinical examination, analysis of the spectrum of kidney and extrarenal pathology in the pedigree, and biochemical studies. The level of uric acid in the blood is higher than 0.422 mmol / l, in the urine it is higher than 4.13 mmol / l, oxalates are more than 110 mmol / 24 hours, urate is more than 2.94 mmol / 24 hours.

A clinical group of 46 people with dysmetabolic interstitial nephritis on the background of pneumonia was determined based on the presence of bacterial damage to the kidney tissue, manifested by microhematuria from 3 to 5, with traces of average proteinuria up to 0.033 gr/l. Often complaints of pastosis of periodical lids r = 0.43 (43%), r = 0, b8 (68%) showed symptoms of intoxication. Traces of urinary syndrome proteinuria up to 0.66 g / l, pneumonia in 12 patients, leukocyturia from 8-10 to 20 in the field of vision, macrohematuria, p = 4 patients were observed. Urate crystalluria was found in r = 42 (42%), oxalate - r = 0.38 (36%), mixed urate-oxalate - r = 0.22 (22%). Nocturia was observed in 8 patients, the relative density of urine was 1009-1013.



The group of children suffering from dysmetabolic pyelonephritis against the background of pneumonia consisted of 63 patients. In the history of these patients, frequent respiratory tract infections, intestinal infections and helminthic invasion were noted. Manifestations of exudative-catarrhal diathesis were found in 12 children. Intoxication symptoms were found in 16 patients, 15 cases of gout pastosis, 12 cases of dysuric. Proteinuria from 0.033 to 1.2 g/l in the urinary syndrome, leukocyturia from 15-20 in the field of vision, and microhematuria in 12 patients were noted.

Oliguria was found in 16 patients, nocturia - in 12 patients. Crystalluria (uric acid, urate) was detected in 63% of patients, oxaluria in 37%. Kidney ultrasound revealed 15% pyelostasis. In 3 patients, doubling of the intestinal-pelvic system, in 2 patients congenital hydronephrosis, in 1 megaureter, i.e. in 10% of patients, secondary obstructive pyelonephritis developed due to congenital anomaly of the urinary system was detected. In 14 patients, as a result of ultrasound examination of the kidneys and bladder, salt deposits in the renal parenchyma were detected. Dysmetabolic diseases cause secondary pyelonephritis. In some cases, dysmetabolic disorders can have a secondary nature associated with membranolytic processes that occur in conditions of anatomical and tissue dysembryogenesis. Acute renal failure in 5 patients with pyelonephritis against the

background of abnormalities of the urinary system - oligoanuria (1-3 days) before anuria, azotemia with increased urea up to 12 mmol / l, creatinine 0.120 mmol / l. The diagnosis of pyelonephritis was confirmed by the bacteriuria of more than 100,000 microbial bodies in 1 ml of urine, and E. coli, Proteus, and Staphylococcus aureus prevailed in the urine culture. Duration of urinary syndrome was from 15 days to 1 month. Hemogram showed anemia in seven patients, leukocytosis in 10, ESR increase from 15 to 45 mm/s in 15 patients with pneumonia. 21 patients were diagnosed with urolithiasis on the background of pneumonia, two of them had spontaneous passage of stones, dyspepsia in early childhood, and two patients had enterobiosis. Three patients with congenital tubulopathy can be included in the same group - acidosis of renal tubules, bilaterally with numerous kidney stones. All of them had urinary syndrome in the form of obvious proteinuria, significant leukocyturia, micro-macrohematuria, crystalluria. Despite ongoing antibacterial treatment, the duration of urinary syndrome was significant.

Thus, dysmetabolic nephropathy against the background of pneumonia in young children is characterized by the latent course of the disease with the manifestation of the disease against the background of pneumonia, respiratory viral pathology, symptoms of intoxication, symptoms of dysuria, enuresis, oliguria, nocturia, urate-oxalate crystalluria. Patients suffering from pyelonephritis developed rapid homeostasis of the body with the development of acute kidney failure against the background of urinary system anomalies.

The analysis of the history of the disease in the observed early age patients showed that all patients DSMN: KSD IN, KI, AUS did not go to a special nephrology hospital, and pneumonia was first detected on the background of a targeted examination.Taking into account the high frequency of urinary syndrome in pneumonia in young children and their tendency to develop kidney pathology and the impact of pregnancy pathology on the formation of metabolic diseases, we were interested in studying the health of mothers during pregnancy and the history of obstetrics in the examined groups. (Table 3).

As shown in Table 4, complicated pregnancy (r = 0.55), maternal kidney disease (r = 0.47), hepatobiliary pathology (r = 0.48), hypertension (r = 0.53), hypotension (r = 0.4), ARVI during pregnancy (r = 0.5), cardiovascular diseases (r = 0.43), chronic tonsillitis is more common in children with IN on the background of pneumonia (r = 0 ,6), in patients with UTD on the background of pneumonia, a greater burden was found compared to patients with ENT diseases (4 times), hypertension (r = 0.25), gastrointestinal tract patients (3 times), kidneys (r = 0.4). In mothers of children with BY on the background of pneumonia, toxicosis occurs more often in the first and second half of pregnancy (r = 0.6) and is less common in UTD (r = 0.25). In these groups (r=0.5 t 0.4, respectively) and history (r=0.4 and 0.5), the rise of termination of pregnancy was also high. Judging from the above, the leading risk factors in the development of nephropathy were both genetic predisposition and the impact of various harmful factors on the fetus during pregnancy.

In addition, attention is paid to information about the relative role of genetic factors in the development of dysmetabolic nephropathy in children. The degree of susceptibility to nephropathy depends significantly on the degree of relationship.

We analyzed pedigrees with relatives with various nephropathies, 1 degree of consanguinity, and information on distant relatives was obtained mainly by cross-examination and examination of relevant medical records.

Table 3

HEALTH CONDITION BEFORE PREGNANCY AND OBSTETRICAL ANAMNESY OF MOTHERS CHILDREN'S NEPHROPATHY ON THE BACKGROUND OF DSMN (Character frequency)

Indications	Control	Pneumonia patient groups		
		IN	KD	UTD
	n=15	n=46	n=63	n=21
Practically healthy	0,9	0,75	0,7	0,6
Gastrointestinal pathology	0,1	0,15	0,28	0,3
Hypertension	-	0,2	0,58	0,25
Hypotension	-	0,2	0,4	0,5
UT diseases	0,05	0,06	0,43	0,2
Allergy	0,1	0,1	0,5	0,25
Chronic tonsillitis	0,2	0,6	0,3	0,4
Hepatobiliary pathology	0,1	0,08	0,48	0,2
Kidney disease	-	0,06	0,47	0,4
ARVI during pregnancy	-	0,1	0,5	0,35
Toxicosis of pregnancy:				
in the I-II half of pregnancy	-	0,05	0,6	0,25
Risk of miscarriage in pregnancy	-	0,06	0,5	0,4

History of abortion	-	0,08	0,4	0,5

Table 4

COMPOSITION OF THE PEDIGREE UNDER STUDIES AND FREQUENCY OF AUT DISEASE

Nosological group	DEGREE OF RELATIONSHIP					
	DEGREE I			D II	D III	total
	parents	siblings	in total			
Control n=20	56 (1) 1,8%	64 (3) 1,9%	120 (4) 3,3%	117 (4) 3,4%	369 (8) 2,2%	606 (16) 2,6%
KI n=54	21 (6) 28,5%	46 (9) 20,9%	64 (15) 23,4%	118 (8) 6,8%	119 (6) 5,0%	301 (29) 9,63%
IN n=46	35 (8) 22,8%	67 (11) 16,4%	102 (19) 18,6%	142 (11) 7,74%	161 (8) 4,96%	405 (38) 9,38%
UTD n=21	25 (5) 20%	56 (7) 12,5%	81 (12) 14,8%	102 (8) 7,84%	96 (3) 3,2%	279 (23) 5,73%
Note: The total number of cases of AUTD is shown in parentheses						

As can be seen from the table. 4, the percentage of diseases of the urinary system turned out to be significant, 63% in PI 9, respectively; UTD was 5.73% and IN was 9.38% compared to 2.6% in the control group.

In genealogy, the incidence of nephropathy was related to the degree of consanguinity to the proband. Thus, children with calcium oxalate lithiasis among relatives of the 1st degree, 14.8% (12 out of 81) of relatives with nephropathy, 23.4% (15 out of 64) with pyelonephritis, among relatives of the II degree of kinship 7.84% (102 8 out of 118) and 6.8% (8 out of 118) and III degree relatives 3.2% (3 out of 96) and 5.0% (6 out of 119), exceeded the indicators in the control group, where the following frequency depending on the degree of relationship observed: consanguinity degree 3.3% (4 of 120), II degree 3.4% (4 of 117) and III degree of consanguinity 2.2% (8 of 369). The increase in the frequency of CHI diseases in families of sick probands compared to controls (among relatives of the 1st degree of kinship - 8-10 times, among relatives of the 2nd-III degree of kinship - 3-5 times) indicates the important role of genetic factors in the development of nephropathies.

We analyzed the frequency of RKA in patients with dysmetabolic diseases, taking into account that small anomalies in the development of connective tissue (RKA) are a universal sign of instability of the connective tissue system and may indicate a high risk of congenital and hereditary kidney pathology. The most common external stigmata of dysembryogenesis were: wide bridge of the nose (h = 0.7), epicanth (h = 0.6), earlobe anomaly (h = 0.6), low umbilical ring (r = 0.5), hypertelorism (r = 0.6): eyes r = 0.3 and teats r = 0.3.

Thus, pneumonia is a trigger for the manifestation of hidden kidney pathology in young children, which is complicated by the development of interstitial nephritis and secondary pyelonephritis based on congenital and hereditary anomalies of the urinary system and dysmetabolic diseases. This situation requires a careful examination of each patient with pneumonia and urinary syndrome in order to determine the characteristics of the metabolic state, identify anomalies of the development of SUA, and then correct them.

2.2. RESEARCH SOLUTION AND METHODS.

Analysis of partial renal function and predisposing factors was performed in 130 children with various forms of pneumonia-related dysmetabolic nephropathy and 20 patients with pneumonia without kidney damage.

In patients diagnosed with urinary syndrome, characteristics of adaptation and development were analyzed in mothers during pregnancy and childbirth, in the newborn and infant period, in the past and in co-morbidities. Genealogical analysis of their pedigrees, quantitative study of urine sediment, Nechiporenko test and bacteriological study of urine were carried out.

Further clarification of the diagnosis was carried out in a specialized nephrology hospital. Here, in addition to biochemical studies usually accepted in nephrological hospitals (bacteriuria, urine sedimentation study, endogenous creatinine clearance, total protein and protein fraction), excretory urography and kidney ultrasound were performed. The function of the glomerular apparatus of the kidneys, that is, glomerular filtration, was evaluated by Van Slyke with the clearance of endogenous creatinine. The state of tubular functions of the kidney was evaluated by the cryoscopic method on the SUA 1 Ts-01 apparatus with urine osmolarity, the daily excretion rate of ammonia (according to Conway) [1551], titratable acids [127].

Tubular reabsorption of uric acid was determined by the formula of Nordin and Frozer (1954).

$$\%\mathrm{TRx} = 100\left(1 - \frac{\mathrm{Ux.\,Per}}{\mathrm{Px.\,Ucr}}\right)$$

TRx - the value of tubular reabsorption of the test substance in urine (%)

Ux is the level of the tested substance in urine (m- mol / s)

Ucr- creatinine content in daily urine (mmol/s)

Pcr - serum creatinine content (mmol / l)

Px- the concentration of the studied substance in the serum (mmol / l)

Quantitative determination of oxalates in urine was carried out according to N.V. Dmitriev [34]. The amount of uric acid in blood and daily urine was determined by the Müller-Seifert method based on the colorimetric determination of uric acid with phosphorus-tungsten Folin reagent, which consists of sodium tungstic acid and orthophosphoric acid [64]. To determine the daily excretion of urates in urine, the Hopkins method described by O. V. Travin was used [124]. This principle is based on the fact that uric acid is deposited in the urine in the form of an ammonia salt. The resulting precipitate is decomposed with strong H2SO4, titrated with 0.04 N KMnO4 solution of uric acid in 100 ml of urine until a pale pink color appears. The calculation is made according to the following formula: (KMnO4 x 3) + 3) x Diuresis = mg of urates per day. The index of endogenous production of uric acid [110] was calculated by the method of UA extraction from serum x UA extraction (mg/kg).

Total lipids were determined using the Bio-La-test kit manufactured by Lahem (Czechoslovakia). The principle of the method is based on the interaction of lipids with the phosphorus-vanillin reagent with the formation of a red color after hydrolysis with sulfuric acid.

After extraction of the lipid extract with Folch's mixture (chloroform-methanol 2: 1), the spectrum of serum lipids was determined by thin-layer chromatography on Silifol-254 plates, followed by densitometry on a Karl-Zeiss-Iene (GDR) densitometer. The composition of the following fractions was determined: phospholipids, non-esterified fatty acids, triglycerides, cholesterol esters.

The structural and functional state of cytomembranes was evaluated by the phospholipid spectrum of erythrocyte membranes determined by TLS according to V. I. Krylov [70] in the chloroform-methanol-water system (65: 25:4), followed by a 2% solution of phosphorus-molybdic acid was determined by spraying with and heating the

plates to 100°C for 10 minutes. Phospholipids were determined by color reactions and standards. The following phospholipid fractions were determined - lysophosphotidylcholine, phosphotidylethanolamine, sphingomyelin, phosphatidylcholine, phosphatidylserine.

The intensity of lipid peroxidation (LPO) processes was evaluated based on the content of unsaturated fatty acids and diene conjugates (DK) of malonic aldehyde (MDA) [118]. The total phospholipase activity of erythrocytes was determined by the method of H. Brokerhof and R. Jensen [150]. The essence of the method is mainly hydrolysis of A2 lecithin (phosphatidylcholine) by endogenous phospholipases. Accumulation of lysolecithin in this environment causes toxic hemolysis of erythrocytes. To characterize the activity status of kidney tissue phospholipases, we determined the activity of A2 phospholipase in urine according to S.A Tujilina and A.I Saluenya [125].

The complex of practical studies made it possible to reliably assess the metabolic state, state of cytomembranes and kidney function in young children with pneumonia. The obtained data were processed using the Student's test with the method of variance statistics and were considered reliable at $p < 0.05$.

CHAPTER III. DYSMETABOLIC

PARTIAL KIDNEY FUNCTIONS AND URINARY EXCRETION OF NEPHROTOXIC METABOLITES IN CHILDREN WITH PNEUMONIA WITH NEPHROPATHY.

Taking into account the polyetiology of factors affecting kidney tissue in pneumonia: infectious agents (viruses, bacteria, hypoxia), hypoxia, metabolic and hemodynamic disorders, iatrogenic factors (antibiotics, sulfonamides, pain relievers, diuretics, steroids non-inflammatory drugs) tried to emphasize the characteristics of kidney damage in patients with pneumonia against the background of dysmetabolic nephropathy.

We studied partial renal function in 20 patients with pneumonia with dysmetabolic nephropathy (DSMN) and 20 patients with pneumonia without dysmetabolic nephropathy (DSMN) (Table 5).

There was a trend toward a decrease in daily urine output in all patients with pneumonia, and 0.450 ± 0.038 ($p < 0.001$) in patients with pneumonia with DSM disorders compared to healthy children and 0.430 in patients with pneumonia without DSM. It decreased to 0.370 ± 0.013 compared to ± 0.021 ($p < 0.05$). In patients with pneumonia with DSMN, a decrease in glomerular filtration was found to 0.97 ± 0.17 ml / sec ($p < 0.05$), which is probably against the background of infectious-toxic damage to the lungs, hyperthermia, microcirculatory disorders associated with hemodynamic disturbances. In healthy children ($p<0.001$) and DSMN 42.0±1.2 ($p<0.05$) in children without pneumonia, DSMN 29.0±1.18 mmol/s is significant in ammonia excretion in patients with pneumonia decrease was observed.

Table 5

PARTIAL KIDNEY FUNCTION AND EXCRETION OF NEUROTOXIC METABOLITES IN PATIENTS WITH DSMN AND NON-DSMN PNEUMONIA

Indicators	**Healthy children n=15**	**Patients with pneumonia**	
		with DSMN	**There was no DSMN**
Diuresis (l)	0,450±0,036	0,430±0,021	0,370±0,013
		p<0,05	p<0,001
Glomerular filtration (ml/sec)	1,64±0,08	1,50±0,09	0,97±0,17
		p<0,05	p<0,001
Oxalates (mmol/s)	110,0±10,5	130,0±11,5	244,0±1,8
		p<0,05	p<0,001
Urates (mmol/s)	2,94±0,24	3,18±0,20	6,9±0,22

		p<0,05	p<0,001
Uric acid (mmol/s)	4,13±0,21	3,18±0,20	6,9±0,22
		p<0,05	p<0,001
Ammonia (mmol/s)	46,8±1,2	42,0±1,2	29,0±1,18
		p<0,05	p<0,001
Titratable acid (mmol/s)	51,0±2,8	48,8±2,2	23,37±4,25
		p<0,05	p<0,01
Osmolality (mmol/s)	627±61,7	648±19,0	920,00±26,5
		p<0,05	p<0,001

p - the reliability of the differences between the indicators of relatively healthy and sick children.

23.37 ± 23.37 ± 23.37 ± 2.8 ± 2.2 ($p < 0.05$) of DMN in children with pneumonia compared to 51.0 ± 2.8 mmol/v ($p < 0.001$) in healthy children and 48.8 ± 2.2 ($p < 0.05$) DMN Decrease to 4.25 mmol / s.

A decrease in acidoammonogenesis indicators in patients with pneumonia with DSMN indicates tubular kidney dysfunction that causes a violation of adaptive-compensatory functions of the body under hypoxia conditions, which increases metabolic acidosis in pneumonia. In children with pneumonia with DSMN up to 244.0 ± 1.8 μmol/o, compared to healthy children 110.0 ± 10.5 ($p < 0.001$) and with pneumonia without DSMN in the children's group 130, 0 ± 11.5 ($p < 0.05$) a significant increase in the excretion of urinary oxalate was detected, which may be of the nature of secondary oxaluria, which is an organism intoxication of the cytomembranes of the renal epithelium, hypoxia, inflammatory process in the lungs It is the result of instability against the background of electrolyte disturbances accompanying its development.

Considering that 2/3 of patients with pneumonia with uric acid syndrome have a genetic burden of oxalic acid and uric acid metabolism disorders, it was determined by

pedigree study and cross-examination of relatives, that the excretion of oxalate and urate is urate and along with oxaluria - uraturia, which is the result of hereditary instability of cytomembranes. Patients with pneumonia with DSMN had an almost two-fold increase in urate and uric acid excretion of 6.9 ± 0.36 mmol/s compared to healthy children ($p < 0.001$) and pneumonia without DSMN 3.18 ± 0.20 ($p < 0.05$) and 5.2 ± 0.3 ($p < 0.05$) and 5.2 ± 0.3 ($p < 0.05$) compared to children's indicators, respectively) formed. An increase in the excretion of urate and uric acid can be associated with an increase in catabolic processes against the background of hyperthermia, hypoxia and the breakdown of purine bases, which leads to an increase in the level of uric acid in the blood. Thus, functional disorders of the kidneys are characteristic of pneumonia in patients with pneumonia without DSMN, the concentration of nephrotoxic metabolites (urates, oxalates, SC) does not reach values with nephrotoxic effects, and as a targeted therapy of pneumonia, nutrition and water regime decreases, the concentration of urine, oxalate, uric acid, which corresponds to the disappearance of clinical manifestations such as eyelids, facial droop, normalization of diuresis. It should be noted that in patients with nephropathy of metabolic genesis, when the genetic load increases with a layer of pneumonia, the partial functions of the kidney - oliguria, a decrease in acid ammonia genesis and nephrotoxic metabolites, oxalates, urates, uric acid, thereby nephrotoxic effect In this case, it may be related to a violation of the process of adaptation at the cellular level as a result of the insufficient response of the individual to the influence of the individual stress factor, infection. Damage to the membrane structures of the renal tubules can lead to secondary changes in the processes of secretion and reabsorption in the tubules and cause the development of secondary tubulopathies.

The study of the partial functions of the kidneys, depending on the nosological forms of dysmetabolic nephropathies, was conducted in children with pneumonia with metabolic disorders (Table 6).

As can be seen from Table 6, the daily excretion of ammonia in dysmetabolic IN was 23.6 ± 3.9 mmol / s ($p < 0.05$), UTD was 29.6 ± 3.2 mmol / s ($p < 0.05$), and KI was 33.8 ± 2.6

(<0.05p) and was significantly reduced compared to healthy children. The most significant decrease in the excretion of titratable acids was IN 19.2 ± 7.9 mmol / s x 1.73 m2, ($p < 0.05$), and with PN and ICD it was 24.7 ± 3.85 mmol / s and 26.2 ± 4.7 mmol/s ($p<0.01$). The highest osmolarity of urine, UTD 976 ± 62.6 mmol/s ($p < 0.05$), IN 898 ± 62.5 mmol / s ($p < 0.05$), and KI 886 ± 58.74 mmol / s ($p < 0.05$), was observed in patients and was 627 ± 61.7 mmol/s when compared to healthy children.

Thus, in the group of patients with IN, in comparison with KI and UTD, a decrease in the function of acidoammonogenesis was found, which indicates that the compensatory-adaptive ability of the kidneys to maintain the body's acid-base status (CBS) is maintained in conditions of tissue hypoxia, respiratory acidosis, and metabolic acidosis in pneumonia. Compared with healthy children, excretion of nephrotoxic metabolites (oxalates) was 226.0 ± 23.0 μmol/s ($p < 0.05$), UTD 262.7 ± 49.8 μmol / s ($p < 0.01$) and IN 243, 2 ± 28.4 μmol / s ($p <0.05$) was noted to increase. Compared with healthy children, urate excretion in KI was 6.22 ± 0.52 mmol/s ($p < 0.01$), in ICD 8.3 ± 1.32 ($p < 0.001$), and in IN 6.36 ± 0.26 mmol / s ($p < 0.001$). The excretion of uric acid in patients with pyelonephritis was 7.1 ± 0.3 mmol / s ($p < 0.01$), ICD 6.26 ± 0.53 mmol / s ($p < 0.05$) and IN 6.55 ± 0.37 mmol / s ($p < 0.05$)). compared to healthy children was 4.13 ± 0.21 mmol/s.

Existing changes in the function of acidoammonogenesis are associated with a family history of metabolism of nephrotoxic metabolites, UA, and oxalate excretion. Against the background of pneumonia, intoxication, dehydration, respiratory failure, and hypoxia, the exacerbation of metabolic diseases lead to a violation of the homeostatic function of the kidneys.

Accordingly, the risk of nephrotoxic effects of urates and oxalates on the kidneys increases. In children with metabolic diseases, a sharp increase in the concentration of urates and oxalates against the background of pneumonia causes the risk of tubular obstruction with deterioration of urine output until the development of kidney failure. Clinically, such children showed eyelids, facial ptosis, a decrease in the amount of urine, - oliguria.

Table 6.

URINE OF A NUMBER OF NEPHROTOXIC METABOLITES IN NEPHROPATHY IN CHILDREN WITH PARTIAL KIDNEY FUNCTIONS AND PNEUMONIA

RELEASE

Indicators	**Healthy children n=20**	**Patients with pneumonia**		
		n=54	**n=21**	**n=46**
Diuresis (l)	0,45±0,036	0,370±0,03	0,360±0,02	0,380±0,04
		p<0,05	p<0,05	p<0,05
Glomerular filtration (ml/sec.)	1,64±0,08	0,98±0,07	0,93±0,14	1,0±0,07
		p<0,001	p<0,001	p<0,01
Oxalates (mmol/s)	110,0±10,5	226,0±23,0	262,7±49,8	243,2±28,4
		p<0,001	p<0,01	p<0,05
	2,94±0,24	6,22±0,52	8,30±1,32	6,36±0,26
		p<0,001	p<0,001	p<0,001
Urates (mmol/s)	4,13±0,21	7,1±0,3	6,26±0,53	6,55±0,37
		p<0,01	p<0,05	p<0,05
Uric acid (mmol/s)	46,8±1,2	33,8±2,6	29,6±3,2	23,6±3,9
		p<0,05	p<0,05	p<0,05
Ammonia (mmol/s)	51,0±2,8	24,7±3,85	26,2±4,7	19,2±7,9
		p<0,05	p<0,01	p<0,05
Titratable acid (mmol/s)	627±61,7	886±58,74	976±62,6	898±61,5
		p<0,05	p<0,05	p<0,05

Thus, in children suffering from dysmetabolic nephropathies, significant changes in the tubular functions of the kidneys, inadequacy of their adaptive reactions in conditions of hypoxia, and especially when exposed to infection are apparently factors affecting the process of pneumonia, contributing to the chronicity of pathological processes in the kidneys and increases the risk of interstitial nephritis, microcrystallization, and stone formation.

It is interesting to study the characteristics of uric acid metabolism in patients with pneumonia with urate nephropathy at an early age (Table 7). An almost 2-fold increase in the level of UA in the blood was found in patients with pneumonia with urate nephropathy to 0.232 ± 0.01 mmol/ll ($p < 0.001$) compared to healthy children, up to 0.432 ± 0.02 mmol/ll. Urinary excretion of uric acid in patients with pneumonia with urate nephropathy was 6.26 ± 0.98 mmol per day compared to healthy children ($p < 0.05$). Recalculated per kilogramme of body weight in patients with pneumonia with early-onset urate nephropathy, the concentration of UA in urine up to 0.77 ± 0.091 mmolg/kg / s compared to healthy children increased by 3 times (0.27 ± 0.02 mmolg/kg / s ($p < 0.001$) was determined.

In early age patients with pneumonia with urate nephropathy, the UA concentration in urine tended to increase to 2.07 ± 0.3 mmol/s ($p < 0.05$). The indicator of endogenous formation of UA also increased to 9.55±0.01, compared to the indicators of 8.69±0.04 ($p<0.05$) in healthy children, indicating an increase in catabolic processes. Hypoxia and pneumonia demonstrated the breakdown of cellular nucleosides against the background of acidosis.

At the same time, UA clearance in patients with pneumonia with HU had low values of 3.04 ± 0.30 ml/min compared to 5.44 ± 0.66 ($p < 0.001$) in healthy children. This may be related to the decrease in renal function filtrate, low diuresis, and the fact that during hyperthermia, part of the fluid was lost with sweat and through the **scin.**

Table 7

URIC ACID INDICATORS IN THE BLOOD AND URINE OF PATIENTS WITH URETHANEOUS NEPHROPATHY ON THE BACKGROUND OF PNEUMONIA

Indicators of the patient	**group SC in blood mol / l**	**In urine SC mol / sV**	**SC ml / min cleared**	**Endogenous. tas. symbol UA**	**in urine UA mol / kg / s**	**MK in 100 ml of urine mmol / s**	**TR MK %**
Healthy childrenn =20	0,242±0,01	4,13±0,24	5,49±0,66	8,69±0,40	0,27±0,02	1,34±0,7	51
Patients with pneumonia with urate nephropathy n=20	0,432±0,02	6,26±0,98	3,04±0,30	9,55±0,10	0,77±0,091	2,07±0,3	70
	p<0,001	p<0,05	p<0,001	p<0,05	p<0,001	p<0,05	

r - the reliability of the differences in the indicators of relatively healthy and sick children

Tubular reabsorption of UA (TR UA) in patients with pneumonia with GU was 70%, which is higher than that of healthy young children, which was 51%. TR UA in healthy older children is high - 91.3%. Increased SC reabsorption in the renal tubules apparently contributes to the maintenance of high levels of SC in the blood.

Thus, an increase in the level of UA in the blood and urine of patients with pneumonia, with impaired purine metabolism, was more clearly identified in patients with pyelonephritis. In the genesis of hyperuricemia, there may be an increase in the endogenous synthesis of UA due to the breakdown of cell nucleotides against the background of the catabolic direction of metabolism, viral-bacterial damage to lung and kidney tissues. An increase in the concentration of UA in the urine, along with an increase in the excretion of UA and urates, oxalates, damages the epithelium of the tubules of the kidneys, and an interstitial process develops in the kidneys. Tubular blockage with urate crystals and UA can lead to the development of ACF. Decreased UA clearance in patients with early-onset pneumonia indicates a lack of UA release secretory mechanisms due to impaired reabsorption-filtration processes.

To evaluate the pathogenesis of GU in patients with pneumonia with urinary syndrome, we analyzed the types of GU: at the same time, we evaluated the indicators of UA in blood, urine and UA clearance (Figure 3). GU metabolic type was determined in 12 patients (60%), the maximum values of UA in blood and urine were 0.559±0.03 mmol/l and 10.57 mmol/l, the clearance of UA was 5.5 ml/min, corresponding to the indicators in healthy children.

GU kidney type was observed in patients (40%), in which the minimum values of daily uricosuria were 4.39 ± 0.28 mmol / l, which corresponded to the values of healthy children, UA clearance 2, 76 ± 0.76 ml / min.

Thus, in patients with pneumonia with urinary syndrome, the metabolic type of hyperuricemia prevailed, which increases the biosynthesis of UA and enhances its excretion. The urinary syndrome of the metabolic type was characterized by significant crystalluria (oxalates, urates, UA), leukocyturia, microhematuria.

In the GU kidney type, due to impaired secretory and filtering mechanisms of the kidney, a decrease in UA excretion by the kidney was observed, which requires the use of uricosuric drugs. Identifying the types of GU in patients with pneumonia helps to reveal and determine the pathogenetic essence of GU and to carry out differential therapy of urinary syndrome in pneumonia.

As an illustration, we give an extract from the case history of patient Aliqulova Gulchera, 1 year 8 months, No. 12117. Girl, 3rd pregnancy, 2nd birth. In the second month of pregnancy, the mother suffered from cystitis. Pregnancy continued with nephropathy in the 2nd half. Birth weight 3000 g, length 52 cm. satisfactorily developed by 1 year 6 months.

At the age of 1 year and 6 months, the child fell ill: the temperature rose, acetone vomiting was observed, shortness of breath, difficulty breathing, cough, restlessness, alternating with lethargy, pale UAin, loss of appetite appeared. Objectively, the child's condition is moderately severe, consciousness is clear. Shortness of breath (54 times per minute), cyanosis of the nasolabial triangle. Hyperemia of the pharynx. Auscultative hard breathing in the lungs, moist wheezing with small bubbles. Cardiac parameters have not changed, tachycardia. Tones are off. Pulse 110/min. The abdomen is enlarged, the liver is 1.5 cm, the spleen is not enlarged. Stool liquid, 2 times a day. Free urination. Blood analysis 11/XI-95 Er-3.6 * 10/12 g / l; Hg - 110.0 color indicator - 0.9; leukocyte. - 10.2 * 10/9 g / l; s-ya - 40%; lymphocyte. - 48%; monisite- 7%; ESR - 45 mm / h. Urinalysis 11/X1-95. protein - 0.033 g / l; a large number of leukocytes; erit.-5-6-7 in the field of vision; urate salts, oxalates. Fecal analysis 11/X1-95 mucus, single leukocytes. Biochemical analysis blood

creatinine - 88.5 mmol / l, blood cholesterol - 3.2 mmol / l, blood urea - 6.9 mmol / l; residual nitrogen - 13.5 units.

Diagnosed: segmental pneumonia complicated by bowel syndrome. Shortness of breath 1 degree. In this regard, it is treated with antibiotics, detoxification therapy, hormone therapy, vitamin therapy, etc.

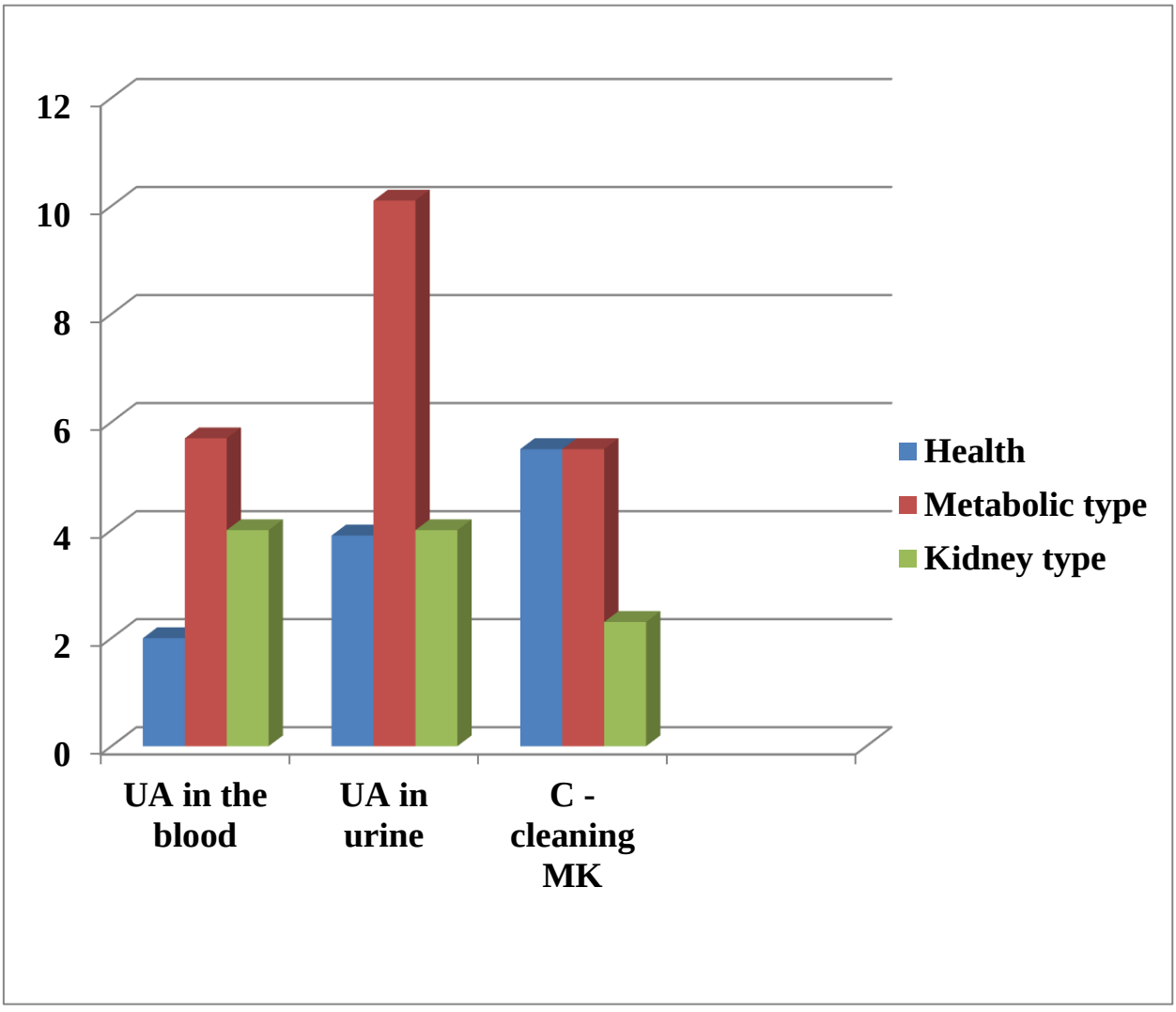


Fig. 2. UA indicators in blood, urine and clearance of patients with pneumonia with urate nephropathy.

ESR - 45 mm / h. Urine analysis 11 / X1-95 g protein - 0.033 g / l; a large number of leukocytes; erit.-5-6-7 in the field of vision; urate salts, oxalates. Stool analysis 10/X1-95 g mucus, one leukocyte. Blood biochemical analysis showed creatinine -

88.5 μmol / l, blood cholesterol - 3.2 μmol / l, blood urea - 6.9 mmol / l; residual nitrogen - 13.5 units.

The diagnosis was made: segmental pneumonia complicated by bowel syndrome. RF 1 degree. In this regard, it is treated with antibiotics, detoxification therapy, hormone therapy, vitamin therapy, etc. The girl's condition improved, her temperature returned to normal, but a repeated urine test revealed changes in the form of proteinuria (0.033 g / l, leukocyturia 7-15 p/e), hematuria (up to 10 new erythrocytes per p/e), soliurates and oxalates . Nechiporenko test leukocytes 6.0 * 10/9 g / l, erythrocytes 4 * 10/12 g / l. The child was transferred to the Nephrology Department of HMFCHD, where the same changes were detected. Acute pyelonephritis was diagnosed. He was treated with antibacterial drugs, vitamin therapy. The child was discharged home with "residual urine syndrome." 6 months after the treatment, the mother repeated the urinalysis in the polyclinic and found changes in proteinuria (0.033 g/l), leukocyturia (7-15 p/er), hematuria (up to 8-10 new erythrocytes. p/er). The child was again referred to KTBSH for inpatient treatment, due to the specific direction of the syndrome. When the child was admitted to the hospital, his health condition was satisfactory, he had no complaints. Correct physique, satisfactory obesity. Vesicular breathing in the lungs, the liver where protrudes 1 cm from the edge of the costal belt, it is painless. No signs of dysembryogenesis were found. Protein in urine 0.165 g / l, Nechiporenko test 9.0 * 10/9 l, erythr. - 7.6 * 10/12 l per day, urate salts in large quantities. Bacteriological examination of urine revealed 10,000 non-hemolytic staphylococcus aureus in 1 ml of urine. Residual nitrogen 13.96 mmol / L, cholesterol 5.72 mmol / L, blood uric acid level 0.307 mmol / L, urine 7.9 mmol / L. The amount of urine during the day is 312 ml; at night 164-ml. Specific gravity of urine fluctuates between 1020-1030. The average daily clearance of endogenous creatinine is 64 ml / min * 1.73 m2. Ammonia 36.5 mmol/sec, titrated acidity 41.92 mmol/day, urine osmolarity 800 ml/day

18 / V-96 in US the location of the kidneys and their size are normal, there is an expansion of the costo-pelvic system on the left side, and there is also a slight bend of the urethra without disturbing the flow of urine. In the kidney parenchyma - exo-positive inclusions.

From the anamnesis, it was found that the mother suffered from pyelonephritis, the father suffered from BTK, the maternal grandmother suffered from polyarthritis, obesity, and hypertension, and her 8-year-old cousin suffered from neuro-arthritic diathesis. It is clear from the pedigree that most of the examined relatives have metabolic diseases similar to the proband. Based on her family history and complex biochemical examination, the girl has dysmetabolic nephropathy complicated by secondary pyelonephritis. Hyperuricemia, uraturia were diagnosed.

Thus, in the example of this patient, it is possible to demonstrate that metabolic diseases are genetically determined and that a large burden is recorded in the maternal line. Manifestations of hereditary disorders are polymorphic (obesity, cholecystitis, salt deposition in the joints, hypertension, urolithiasis). Kidney damage is often manifested: in adults, as pyelonephritis, as in the mother and maternal grandmother, and in children, in the form of neuro-arthritic diathesis.

Revealing nephropathy against the background of the influence of a stress factor, in this case pneumonia, allows us to call this condition one of the manifestations of multi-organ membranopathy.

CHAPTER IV. MEMBRANOPATHOLOGICAL ASPECTS IN CHILDREN WITH DYSMETABOLIC NEPHROPATHY ON THE BACKGROUND OF PNEUMONIA

Specific characteristics of lipid metabolism and the stability of cell membranes in young children with pneumonia with kidney damage have not been specifically studied. In this regard, we studied the spectrum of serum lipids in patients with DSMN pneumonia 20 children and 20 children with DSMN pneumonia (Table 8). As can be seen from Table 8, in patients with pneumonia with DSMN, an average increase in the level of total lipids was found to 6.78 ± 0.19 g / l, while in healthy children it was 4.6 ± 0 .13 ($p < 0.05$) and 5.9 ± 0.21 ($p < 0.005$) in children with pneumonia without DSMN. In patients with pneumonia with PMD, FCh and UFA indicators were significantly increased compared with healthy children ($p < 0.05$) and in children with pneumonia without DSMN ($p < 0.05$), The FCh fraction decreased to 43.5 ± 0.54. At the level of 51.8 ± 0.6%, healthy children and children with pneumonia without DSMN 47.16 ± 0.37 ($p < 0.05$). The bioavailability of FCh is high due to their content of unsaturated acids; The formation of FCh is one of the methods of detoxification or removal of excess free fatty acids (UFA) from metabolic transformations. Changes in lipid metabolism in patients with DSMN pneumonia more clearly reflect C / TG, FCh /ChE indicators. Patients with pneumonia with DSMN have significantly increased V / TG ratios, the values in healthy children are 0.20, and in children with pneumonia without DNK, 0.43. In children with DSMN, FCh /ChE increased to 0.21, compared to 0.12 in healthy subjects and 0.17 in children with non-DNK pneumonia.

Table 8

SERUM LIPIDS IN PATIENTS WITH DSMN PNEUMONIA

Indicators Patient groups	**Healthy children n=20**	**Patients**	
		Pneumonia without DSMN n = 20 M ± m	**DSMN pneumonia n = 20 M ± m**
Total lipids (g / l)	4,6±0,13	5,9±0,21 x p<0,05	6,78±0,19 x p<0,05
FL (%)	3,2±0,12	3,0±0,16 p<0,05	2,8±0,12 p<0,05
MDG (%)	7,6±0,19	8,2±0,14 x p<0,05	9,43±0,20 x p<0,05
FCh (%)	6,4±0,29	8,2±0,13 x p<0,05	9,87±0,21 x p<0,05
NEYK (%)	4,06±0,17	6,1±0,11 x p<0,05	7,46±0,17 x p<0,05
TG (%)	14,6±0,17	14,2±0,17 p<0,05	15,21±0,21 p<0,05
ChE (%)	51,8±0,61	47,6±0,37 x p<0,05	43,56±0,54 x p<0,05
FCh/ChE	0,12	0,17	0,21
NEYK /TG	0,20	0,43	0,42

Note: X is the reliability of the differences between the indicators of healthy and sick children ($p < 0.05$)

P - reliability of the difference between indicators in patients with pneumonia and DSMN

An increase in the hypoxia coefficient may be associated with the predominance of lipolytic processes in early-aged children, as well as the mobilization of fat from the depot, which is accompanied by an increase in the tendency to increase the active transport form of lipids, MDG and TG.

An increase in the FCh/ChE index up to 0.21 compared to healthy children, compared to 0.12 healthy children, indicates a decrease in the processes of esterification of fatty acids, which are used faster in metabolism.

The decrease in the level of FL in blood serum can be explained by its consumption and participation in the structural renewal of biomembranes. The obtained data reflect the compensatory-adaptive reactions of the organism, on the other hand, they show deep metabolic changes in the lipid spectrum during hypoxia against the background of pneumonia.

To determine the phospholipid spectrum of erythrocyte membranes, characteristics of LPO processes, we examined 20 healthy young children, 20 with DSMN pneumonia, 20 patients with pneumonia on the background of DSMN. When determining the percentage of lipids in erythrocyte membranes, a decrease in phospholipids in choline was found, mainly in patients with pneumonia without DSMN, FX was 0.39 ± 0.01 mmol / L, and in patients with pneumonia with DSMN, it was 0.33 ± 0.01 ($p < 0.05$) and reached 0.44 ± 0.01 mmol / L compared to healthy children. The decrease in FX content was accompanied by an increase in the level of PC in erythrocyte membranes in patients with pneumonia without DSMN to 0.32 ± 0.01 mmol / L, compared to healthy children with DSMN 0.44 ± 0.01 (A significant increase in pneumonia ($p < 0.001$) was (0.3 ± 0.01 mmol / l (Table 9).In patients with pneumonia without DSMN, there was almost no change in the

composition of SFM, in patients with pneumonia with DSMN, it had a higher effect than in healthy children (0.45 + 0.01 mmol / L) (P<0.01). In children, in critical conditions, the binding of SFM membranes in the lipid bilayer structure is somewhat flexible, because in the conditions of increasing destabilization of cell membranes, the severe oxidation of these phospholipids and the metabolically inert class help to increase the relative stability of biomembranes (Sultonov A. T., 1989). Changes in the ratio of phospholipids in the structure of cell membranes, in particular, the accumulation of the cytotoxic fraction of LFX and the decrease in the endogenous bioantioxidant FX, which is important in pneumonia with DSMN, have a great impact on the destabilization of cell membranes. This was also shown by the LPhCh / PhX ratio, which increased to 0.14 in the peak period, but in patients with pneumonia with DSMN, this indicator was 0.1 and 0.08 in healthy children . During the period of recovery, the LPhCh / PhX ratio had a downward trend and was above the norm at 0.12. A decrease in PC synthesis in lung tissues is associated with hypoxia and inflammatory processes in bronchopulmonary tissues disrupting the synthesis of phospholipids, which is accompanied by a decrease in their amount in the blood.Pneumonia patients with DSMN showed a decrease in FS levels to 0.25 ± 0.01 (P < 0.01), whereas this phospholipid was unchanged in patients with pneumonia without DSMN (P < 0.05).

In patients with pneumonia with DSMN, the content of the endogenous bioantioxidant PhEA decreased to 0.29 ± 0.01 mmol / L (P < 0.05) and remained below the norm at discharge (P < 0, 05).

Table 9

INDICATORS OF THE PHOSPHOLIPID SPECTRUM OF ERYTHROCYTE MEMBRANES IN PATIENTS WITH PNEUMONIA

Indicators Patient groups	**Healthy children n=20**	**Patients**	
		Pneumonia without DSMN	**DSMN pneumonia**
LFX (mmol / l)	0,23±0,01	0,32±0,01 p<0,001	0,44±0,01 p<0,001
FX (mmol / l)	0,44±0,01	0,39±0,01 p<0,005	0,33±0,01 p<0,01
SMF (mmol / l)	0,35±0,01	0,36±0,01 p<0,13	0,45±0,01 p<0,01
FS (mmol / l)	0,32±0,01	0,33±0,01 p<0,15	0,40±0,01 p<0,01
FEA (mmol / l)	0,34±0,01	0,3±0,01 p<0,005	0,29±0,01 p<0,05
DC (nmol / 10/12 erit)	4,6±0,5	6,6±0,12 p<0,005	10,3±0,21 p<0,01
BPhA % hemoliz	15,01±1,4	17,02±0,2 p<0,1	21,25±0,3 p<0,05

Note: *R is the reliability of the differences between the indicators of sick and healthy children.*

Thus, pneumonia with metabolic nephropathy in young children is characterized by significant FL displacement of erythrocyte membranes, which indicates a disturbance in the metabolism of these compounds. Taking into account the variety of functional goals of PL, in particular, their participation in the structure of cell membranes, the blood coagulation process and the synthesis of pulmonary surfactants, it can be concluded that the identified changes may be important in the development and damage of pathogenetic kidney membranes. .

One of the mechanisms of structural and functional disorganization of cell membranes in children with pneumonia is the excessive accumulation of LPO products, which is confirmed by an increase in the content of the initial product of lipid-diene conjugates (DK) of their peroxidation in erythrocytes. In patients with pneumonia without DSMN, the level of DK was 6.6 ± 0.12 nmol / 10/12 er., ($P < 0.01$) to 4.6 ± 0.5 nmol / 10/12 er. i

ncrease was observed. Correlation analysis performed showed that LFX and DK ($r = + 0.50$ $P < 0.05$), FKh and DK ($r = - 0.780$, $P < 0.05$) in patients with pneumonia with DSMN, that is, lack of FX and excess of LFX presence creates the necessary conditions for the activation of the initial stages of lipid peroxidation.In patients with non-DSMN pneumonia, blood phospholipase activity increased, but with insignificant differences ($P < 0.05$), in patients with DSMN hemorrhagic pneumonia, FAA caused erythrocyte hemolysis by 21.25 ± 0.3% ($P < 0.001$) and achieved a hemolysis rate of 15.01± 1.4%.

Thus, the increase in the activity of endogenous BPhA helped the phospholipid layer of the renal epithelial membranes with the formation of phophorylated nitrogenous bases - FEA, FS and the development of secondary oxaluria, uraturia. Because calciuria was noted in patients with pneumonia with DSMN persistent crystalluria, which indicates the defeat of kidney membrane structures, the inclusion of kidneys in the pathological process can be said about the pathology of many organ membranes.

A comparative study of the activity of LPO processes in the state of cytomembranes in patients with pneumonia and dyspeptic diseases, depending on the nosological variants of kidney damage, is presented.

Table 10

PHOSPHOLIPID SPECTRUM OF ERYTHROCYTE MEMBRANES IN PATIENTS WITH PNEUMONIA ON THE BACKGROUND OF KIDNEY PATHOLOGY (M±m)

Indicators Groups	LFX (mmol / l)	SMF (mmol / l)	FS (mmol / l)	FX (mmol / l)	FEA (mmol / l)
Healthy childrenn = 20	0,23±0,01	0,35±0,01	0,32±0,01	0,44±0,01	0,34±0,01
IN n=46	0,32±0,02 p<0,05	0,35±0,01	0,35±0,01 p<0,05	0,30±0,01 p<0,01	0,26±0,01 p<0,01
KI n=54	0,36±0,01 p<0,05	0,45±0,01 p<0,01	0,411±0,03 p<0,05	0,28±0,01 p<0,01	0,24±0,01 p<0,01
UTD n=21	0,29±0,01 p<0,01	0,37±0,01 p<0,05	0,35±0,01 p<0,01	0,33±0,01 p<0,01	0,28±0,01 p<0,01

Note: *r is the reliability of the differences between the indicators of sick and healthy children.*

As can be seen from Table 10, an increase in the cytotoxic fraction of LPC (0.29 ± 0.01 mmol / L, P < 0.01) was observed in patients with BTK pneumonia, while the FX fraction was 0.33 ± 0.01.A decrease of 0.44±0.01 (P<0.01) was observed compared to the increase in healthy children. In patients with IN 0.30 ± 0.01 mmol / L (p < 0.01), the level of phosphotidylcholine decreased, especially in pyelonephritis 0.28 ± 0.01 (P < 0.01), and the LFX fraction of patients KI 0, It exceeded 36 ± 0.01 mmol / L (p < 0.05), and in patients with IN, it was 0.52 ± 0.02 mmol / L (P < 0.05).

FEA fraction decreased in all types of kidney pathology, especially in pyelonephritis ($P < 0.01$), and SFM in pyelonephritis reached 0.405 + 0.01 mmol / l ($P < 0.01$). Thus, considerable disorganization of cytomembranes was revealed with the reorganization of phospholipids with IN and KI.

A comparative assessment of the severity of LPO processes in patients with pneumonia at different stages of the development of kidney damage showed that in children with KSD there is a tendency to increase in the level of DK ($P < 0.01$), IN and KI, significant DK values are upward was noted (8.06 ± 0.14 nmol / 10/12 erythrocytes. In addition, 10.24 + 0.01, respectively, $P < 0.01$ and $P < 0.001$) compared to the indicator in healthy children 4, 6 ± 0.5 nmol er. (Table 11).

Blood BPhA also increased in patients with IN, KI, 22.0 ± 0.01% hemolysis in patients with KI ($P < 0.05$), 19.8 + 0.8% hemolysis in patients with interactive nephritis ($p < 0.01$). The level of the final product of LPO - malondialdehyde increased in patients with IN, KI, compared to healthy children, with the highest values in patients with KI 8.3 ± 0.21 nmol/mg lipids ($P < 0.001$). In patients with IN, KI 5.5 + 0.25 nmol/s and 6.8 ± 0.3, the increase in the level of DK in urine compared to healthy children was 3.55 ± 0.35 ($P < 0, 01$ and $P < 0.01$), increased activity of phospholipase A2 in urine is more pronounced in pyelonephritis, which indicates the involvement of renal epithelial membranes in the pathological process.

Correlation analysis in patients with pneumonia with DSMN showed that UA and LFX ($r = 0.37$, $P < 0.05$) UA and DK ($r = 0.38$; $P < 0.05$), (Fig. 4) GU confirm the effect on the development of membranopathological processes.

In the correlational analysis of patients with pneumonia with DSMN, a close correlation was found between the level of UA and LFS ($r = 0.37$, $P < 0.05$) and the level of MC and DC ($r = 0.38$; $P < 0.05$), (Fig. 4), which confirms its effect on the development of GU membranopathological processes. The inverse correlation between UA and FX level ($r = - 0.50$, $P < 0.05$), UA and FEA ($r = - 0.60$, $P < 0.05$)

indicates a decrease in the level of endogenous bioantioxidants, structural and ionic state of cytomembranes.

With the deepening of the pathological process in the kidneys, the layering of the infectious-inflammatory process, LPO processes are activated, the structure of cytomembranes is disturbed not only in the lung tissues, but also based on the inflammatory processes in the kidneys. Studies have shown that in patients with pneumonia with dysmetabolic nephropathy, there are significant changes in LPO processes, structural and ionic disorganization of the cytomembrane, a combination of the micro-inflammatory processes in the kidneys, and blood flow depending on the level of SC in the blood increase was found.

Table 11

INDICATIONS OF LPO PROCESSES IN PATIENTS WITH PNEUMONIA WITH KIDNEY PATHOLOGY

Indicators **Patient groups**	**Healthy n = 20**	**BTK n=21**	**BY n=63**	**IN n=46**
Blood:				
QFF % erythrocyte hemolysis	15,01±1,4	17,7±0,5 p<0,05	22,0±0,1 p<0,05	19,8±0,8 p<0,01
DK nmol / 10/12 erythrocytes	4,6±0,5	5,79±0,21 p<0,01	10,25±0,01 p<0,05	8,06±0,14 p<0,01
MDA nmol/mg	5,8±0,26	6,59±0,28 p<0,05	8,3±0,21 p<0,001	7,59±0,28 p<0,05
lipids				
Urine:	3,55±0,35	4,3±0,3 p<0,05	6,8±0,3 p<0,01	5,5±0,25 p<0,01

DK mmol / l	5,2±0,45	5,8±0,3 p<0,05	9,3±0,32 p<0,01	7,8±0,3 p<0,01

Note: r is the reliability of the differences between the indicators of sick and healthy children.

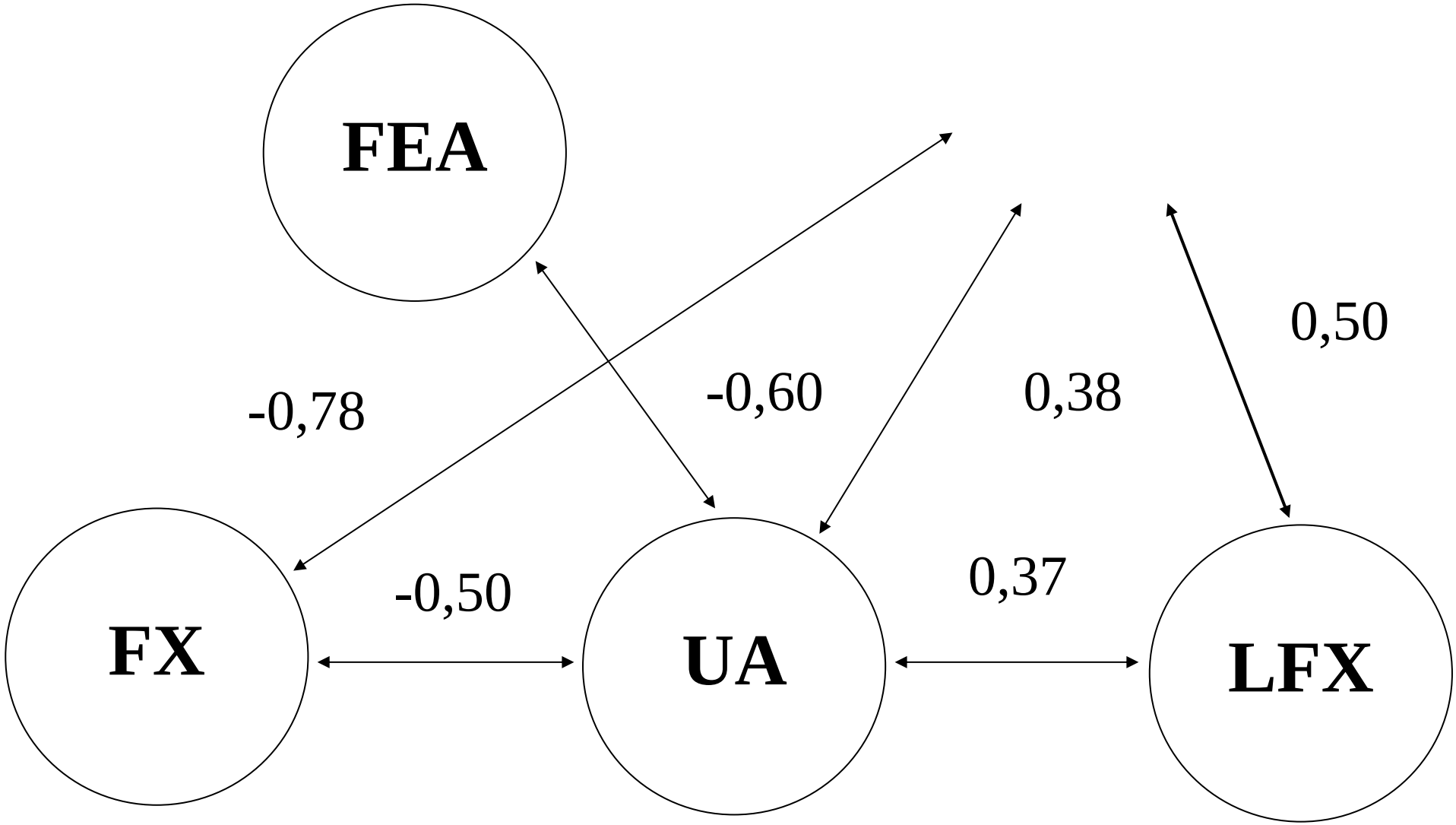


Figure 3. Correlation between UA parameters and the state of cytomembranes

CHAPTER V. EFFECT OF PNEUMONIA ON LIPID METABOLISM AND PARTIAL KIDNEY FUNCTION

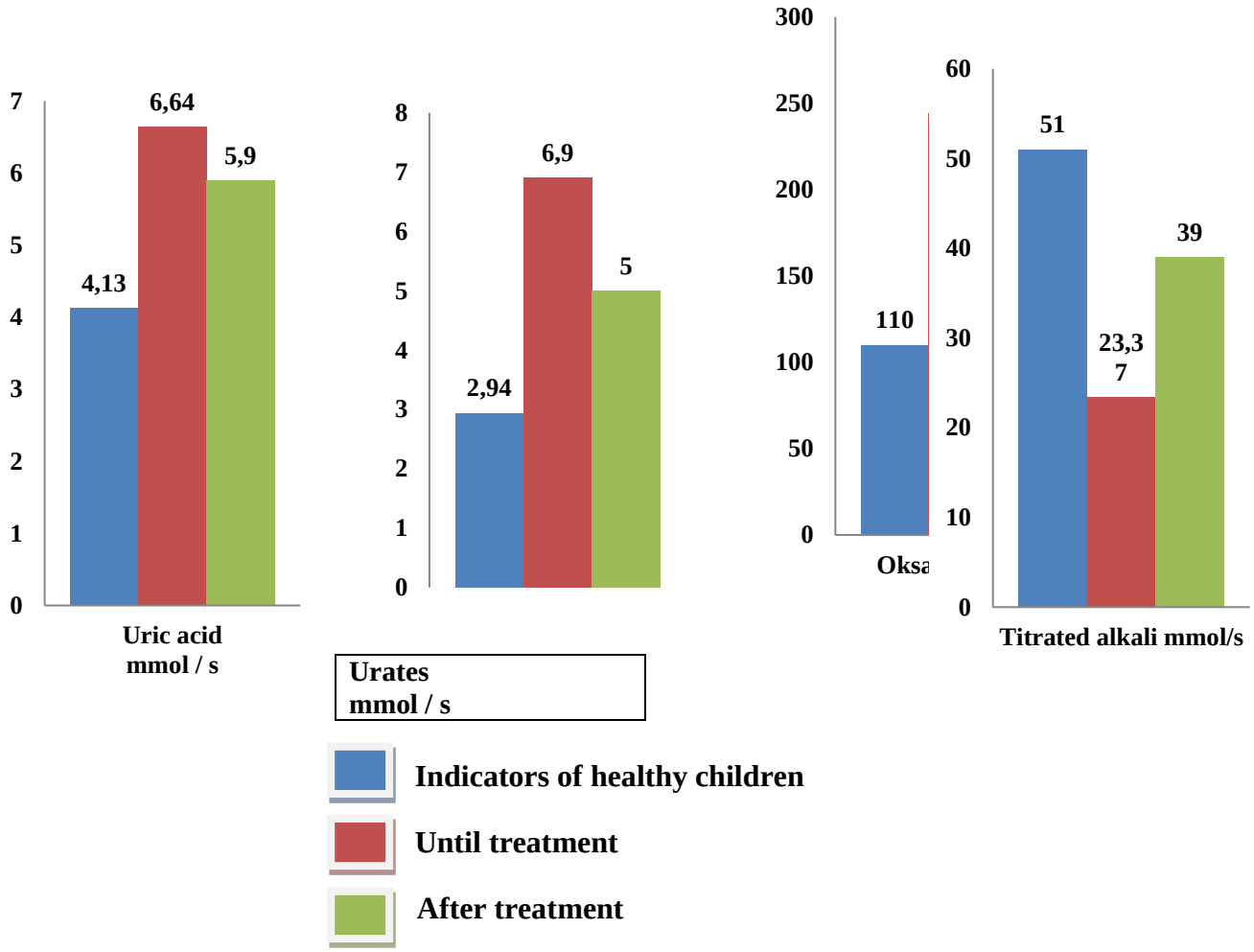
The acute stimulation of lipid peroxidation with the accumulation of cytotoxic metabolites in pneumonia in young children found during the research is consistent with the data available in the literature (Bobomuradov TA, 1994), clinical course and ongoing therapy. In the group of patients with pneumonia with kidney damage, these changes are more pronounced and stable, and they remain even during the stage of clinical recovery from pneumonia. The above aims to address the need for established dietary drug therapy alongside conventional therapy for pneumonia. Even in young children, the presence of hyperuricemia, uricosuria, hyperoxaluria suggests taking into account the specific features of the metabolic state when creating a child's menu. It is known that diet therapy for DSMN is not only a means of correcting metabolic diseases, enhancing general treatment efforts, but also serves as a method of patient rehabilitation. When prescribing a diet for oxaluria, we took into account oxalic acid when introducing foods rich in easily digestible calcium (cottage cheese, cheese), dysmetabolism, excluding foods rich in purine bases, pH, and salt content of urine in magnesium (rolled oats, buckwheat) fibre (vegetables, fruit), which helps to reduce the absorption of calcium in the intestine. When determining the diet, the pH and salt content of urine, limiting products rich in chelic acid, purine-based products, limiting products rich in easily digestible calcium (cottage cheese, cheese) in oxaluria, products containing magnesium (hercules, oats, buckwheat), and products rich in fibre (the introduction of vegetables, fruits) help to reduce the absorption of calcium in the intestine.

For older children, grape and pomegranate juice, lemon, dried apricots, compote, and potato-cabbage diets were used to alkylate urine and prevent hypokalemia. In uraturia, meat broth is excluded, meat is recommended in boiled form, and phytotherapy is used. In the morning, taking into account the daily rhythm of the

nephron, proteins of animal origin are recommended. In metabolic diseases of oxalic acid, sodium benzoate, solubility of oxalates in 10-14 day cycles, burnt magnesia at the level of 0.1-0.2 g per day, phytin in a dose of 10 mg/kg body weight are prescribed.

The first group of patients consisted of 15 children without extrarenal clinical manifestations of pneumonia with transient UA on the background of hyperoxal - and (or) uraturia, who were treated with conventional pneumonia treatment and diet. The second group of patients consisted of 20 children with pneumonia on the background of dysmetabolism - IN, KI who were injected with dimephosphon in the complex of traditional therapy (D). D was used in the form of a ready dosage - 15% aqueous solution for oral administration in 100.0 vials produced by Tatfarmhim drugs. The dosage was 10-15 mg per day at 50 mg/kg body weight. In the third group, 20 patients with pneumonia with DSMN received the same dose of dimephosphon, and at the same time, they received vitamin E at a dose of 3 mg/kg body weight for 10-12 days orally or intramusculally.

In patients with pneumonia with hyperoxal-uraturia without renal manifestations, conventional therapy and diet therapy reduced the excretion of oxalate and urate from the initial level (170.0 ± 13.0 mmol / s and 5.0 ± 0.3 mmol / s) in urine by 20 % and contributed to the normalization of urinary pH. (Figure 4). There was a tendency to restore the function of acidoammonogenesis, a decrease in uric acid excretion to 5.9 ± 0.3 mmol / s compared to 51.0 ± 2.8 mmol / s in healthy people ($p < 0.05$), ammonia excretion to 38.0 ± 2.0 mmol/s ($p < 0.01$) and tetaturic acids increased to 39.0 ± 1.8 mmol/s.



Picture. 4. Dynamics of partial kidney functions in patients with pneumonia with DSMN during diet therapy

The imbalance of blood lipid fractions, phospholipids, and lipid peroxidation processes did not change in patients who received diet therapy along with traditional therapy. Therefore, conventional therapy and diet are not enough to normalize LPO processes, lipid fractions and phospholipids, which determines the need to prescribe drugs that, normalize the stability of cytomembranes in membrane-destabilizing stress conditions.

In the second group, the use of dimephosphon in complex treatment contributed to a more favorable course and outcome of pneumonia. In children, such phenomena as infectious toxicosis, febrile period, percussion and auscultation of the lungs,

hypoxia and shortness of breath stopped in a short time. Acceleration of the processes of stabilization of the pathophysiological manifestation of the disease helped to reduce the average stay of patients in the hospital by 1/3.

MS in young children with pneumonia stopped faster during treatment with Dimephosphon: diuresis was restored ($p < 0.05$), saluria disappeared, proteinuria, leukocyturia, hematuria decreased compared to conventional treatment and diet therapy. During D treatment, the excretion of urate, uric acid, and oxalates approached the norm compared to the control group ($p < 0.001$) (Figure 4).

In healthy children, the excretion of ammonia, titratable acids was restored ($p < 0.01$), these indicators were reduced against the background of traditional therapy and diet therapy, which indicates the use of D in restoring the acidogenetic capacity of the kidneys, its antioxidative effect on kidney mechanisms of acid-base control. A decrease in the excretion of indirect oxalates in the background of D indicates the stabilization of the cytomembranes of the renal epithelium.

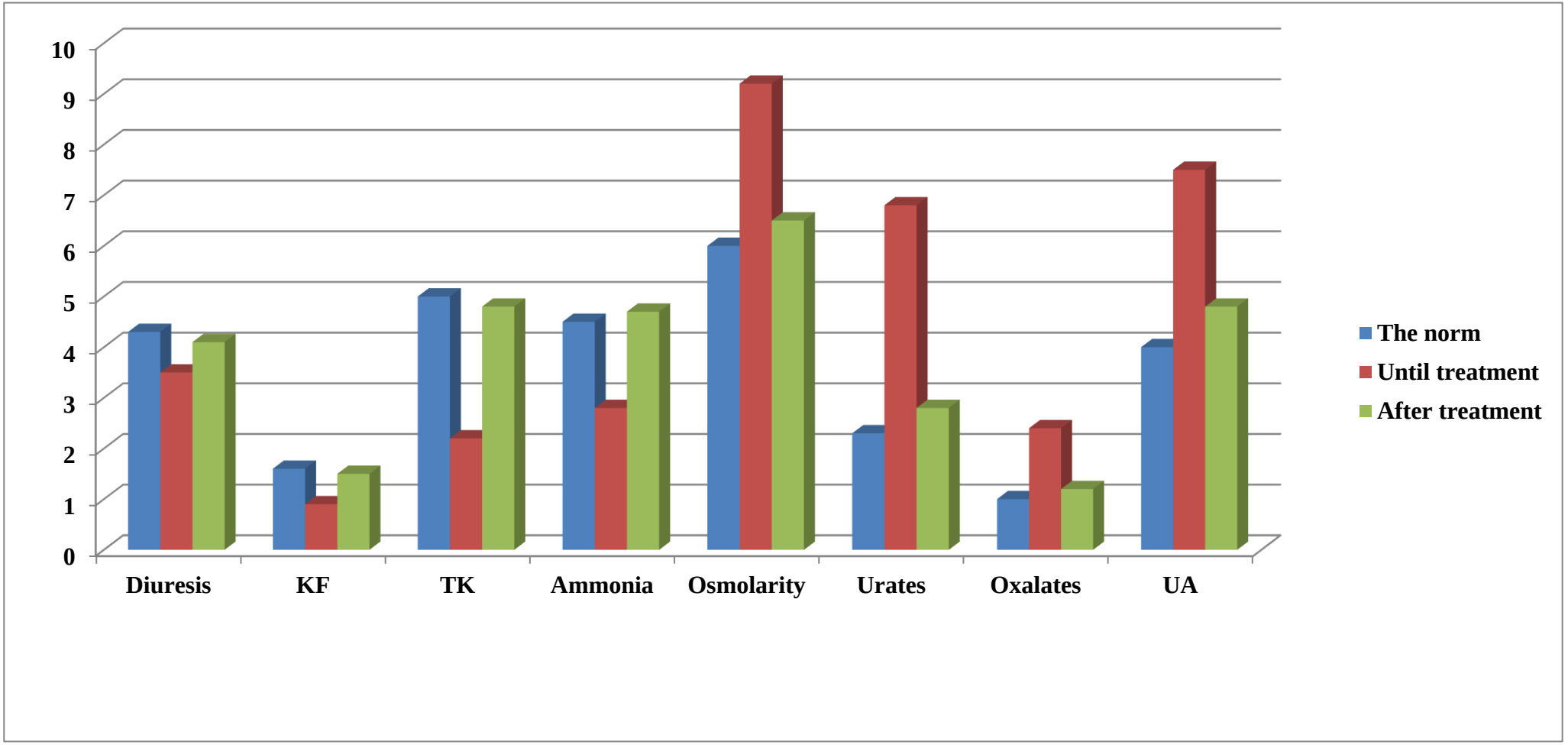


Fig. 5. Effect of D on partial kidney functions in patients with pneumonia with DSMN

A decrease in MC levels in the blood to 0.301 ± 0.02 mmol / l ($p < 0.01$) was detected along with a decrease in the excretion of uric acid and urate in the urine.

In parallel with the improvement of the partial functions of the kidneys, the urinary syndrome was eliminated.

Against the background of the use of D, the level of total lipids in patients with pneumonia decreased by 4.8 ± 0.12 g / l, and in patients undergoing conventional and diet therapy, the indicator decreased by 5.8 ± 0.13 ($p < 0.001$) (Table 12). The level of FL increased compared to sick children in group I, while the fraction of non-esterified fatty acids decreased significantly, approaching the norm ($p < 0.05$), which characterizes the positive effect of D on lipid metabolism. In the background of D, the level of XSE increased from 42.0 ± 0.3% ($p < 0.001$) to 52.4 ± 0.6% in patients of group I, which is very important, because esterified fatty acids, which are part of cholesterol esters, are the fastest in metabolism is used.

Normalization of phospholipid fractions of erythrocyte membranes against the background of D treatment was also noted. In the background of D, the level of LFX decreased to 0.24 ± 0.01 mmol / L, in patients with traditional treatment and diet therapy, it remained at the level of 0.43 ± 0.01 ($p < 0.01$) ol / l. The level of endogenous bioantioxidant FX increased to 0.42 ± 0.01 mmol/L ($p < 0.05$), its indicator in group I patients was 0.36 ± 0.01 ($p < 0.05$).

EXS fraction decreased from 0.42 ± 0.01 to 0.34 ± 0.01 mmol/l in group I patients ($p < 0.05$).

Table 12

THE EFFECT OF DIMEOFFON ON LPO, LIPID AND PHOPLIPID SPECTRUM OF ERYTHROCYTE MEMBRANES IN PATIENTS WITH PNEUMONIA ON THE BACKGROUND OF URINARY SYNDROME (M±m)

Indicators Patient Groups	Healthy n = 20	n = 20 patients with DSMN pneumonia		
		before treatment	after treatment	
			The main diet therapy	Dimephosphon
UL (g / l)	4,6±0,13	6,78±0,19	5,8±0,13	4,8±0,12xx
FL (%)	3,2±0,12	2,8±0,12	2,85±0,12	3,2±0,2
EXS (%)	6,49±0,29	11,6±0,23	9,87±0,24	7,3±0,3
EYK (%)	4,06±0,17	9,19±0,21	7,8±0,2	4,3±0,3xx
XSE (%)	51,8±0,41	38,5±0,29	42,0±0,3	52,4±0,6xx
LFX mmol / l	0,23±0,01	0,44±0,01	0,43±0,01	0,24±0,01x
FX mmol / l	0,44±0,02	0,33±0,01	0,36±0,01	0,42±0,01x
SFM mmol / l	0,35±0,01	0,45±0,01	0,42±0,01	0,34±0,01x
DK nmol / 10 erit.	4,6±0,5	10,3±0,21	9,8±0,23	5,8±0,3xx
MDA nmol / g lipid	5,8±0,26	8,3±0,21	8,0±0,18	6,4±0,2xx
BPhA % hemoliz	15,01±1,4	21,25±1,5	20,0±1,8	17,0±20,x

Note: x - reliability of group differences (p < 0.05), xx - (p < 0.001)

main therapy + dimephosphone.

Thus, D has a positive effect on the composition of phospholipids in cell membranes, thereby affecting the structural and functional structure of all membranes.

Dimephosphone significantly contributed to the normalization of the LFX / FX ratio from 0.54 to 0.52, while in the control group it was equal to 1.3, which simultaneously stabilizes the structure of cell membranes and, due to phosphatidylcholine, cells determines optimal antioxidant activity.

The use of D against the background of the main therapy helped to normalize LPO products, the level of DK decreased to 5.8 ± 0.3 mmol / 10 12 nmol / 10 12 erythrocytes, which compared to the indicators of group I patients according to conventional therapy was 9.8 ± 0.23 (p < 0.001). The level of MDA reached 6.4 ± 0.2 nmol / g lipids, and the index in the group was 8.0 ± 0.18 (p < 0.001), the initial level in patients before treatment was 8.3 ± 0.21 nmol / g lipids did.During treatment, QFF decreased to 21.25 ± 1.5% compared to patients before treatment, erythrocyte hemolysis decreased to 17.0 ± 2.0% compared to hemolytic patients, this indicator was 20.0 ± 1.8% (p < 0.05) in patients of group I.

The obtained data show the membrane-stabilizing effect of D in early childhood pneumonia, which is complicated by the development of kidney damage, which creates the necessary conditions for a more favorable course of pneumonia and relief of SS. LPO processes can be explained by the addition of D to the membranes due to its affinity to the phospholipids of the cell membranes, the effect on the body's protective antioxidant system.

By suppressing the intensity of lipid peroxidation, dimephosphon reduces the level of inflammatory alterations, while exerting an anti-inflammatory effect, which is confirmed by faster clinical remission.

But during treatment with D, the level of LPO-DK and MDA products, as well as the blood level in patients with DSMN pneumonia, are not completely normalized, so we used vitamin E together with dimephosphon as an antioxidant.

In the group of patients who received vitamin D and E, SS normalized faster in 5-6 days, crystalluria, leukocytes and erythrocyturia disappeared. Concentration function of kidneys improved and daily diuresis increased (Figure 5). Excretion of oxalate and urate in healthy children reached 112.6 ± 18.4 mmol/s ($p < 0.001$) and 2.3 ± 3.0 mmol/s ($p < 0.001$).

The functions of acidoammoniogenesis were restored: the level of ammonia increased to 46.2 ± 4.0 mmol/s, the initial level was 36.5 ± 1.18 mmol/s ($p < 0.01$), the level of titrated acids was 50.4 ± increased to 6.4 mmol / s ($p < 0.01$).

Therefore, the combined use of dimephosphon and antioxidant vitamin E made it possible to normalize SS, reduce the excretion of nephrotoxic metabolites - oxalates, urates and improve the function of distal tubules.

With the combination of vitamin D and E, we found positive shifts in the blood plasma lipid spectrum: phospholipid fractions of erythrocyte membranes, reduction of lipid peroxidation products. An increase in the fraction of XSE ($p < 0.01$) (Fig. 7) and a significant decrease in the cytotoxic part of LFX ($p < 0.01$), an increase in the level of endogenous antioxidant FX ($p < 0.05$) and the level of total phospholipids ($p < 0.05$).

The level of MDA also decreased (Fig. 8) to 5.9 ± 0.23 nmol / g of lipids, in healthy people its level was 5.8 ± 0.26 ($p> 0.05$), DK to 4.8 ± 0.4 nmol / 10 12 erythrocytes

(p> 0.05) . Blood QFF erythrocyte hemolysis during D ± E treatment decreased to 15.8 ± 2.0% (p < 0.05).

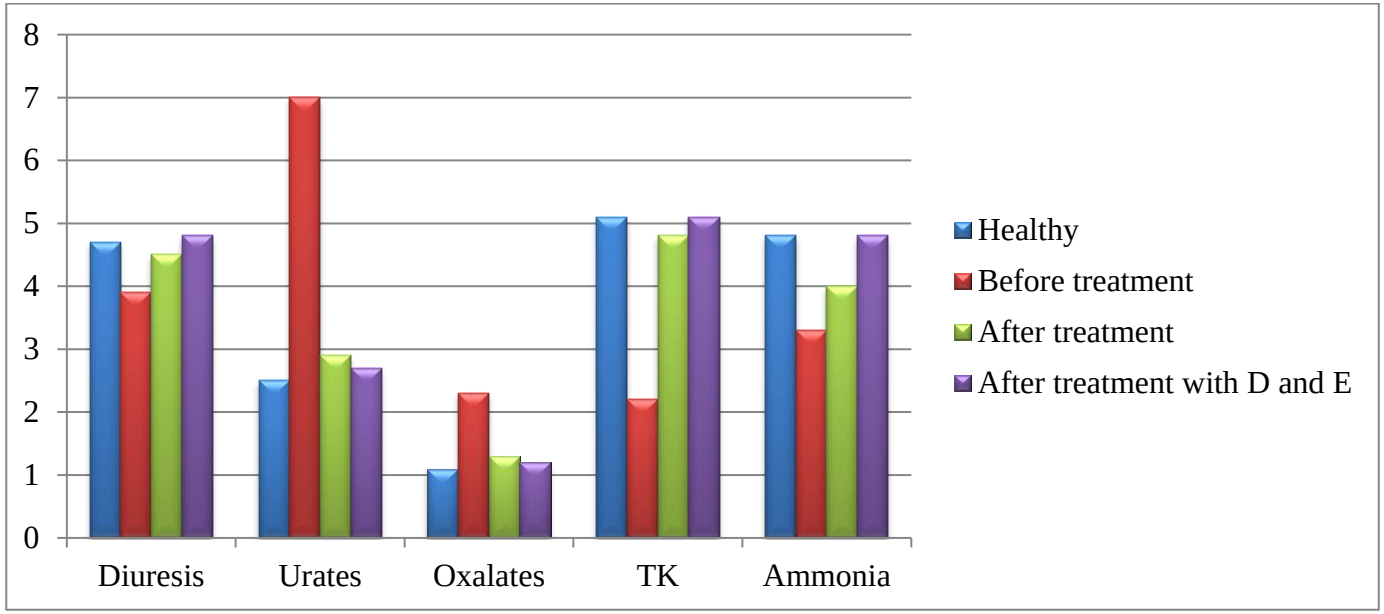


Figure. 6. In patients with pneumonia with DSMN, partial improvement with maintenance therapy with dimephosphonate and vitamin E
dynamics of kidney functions.

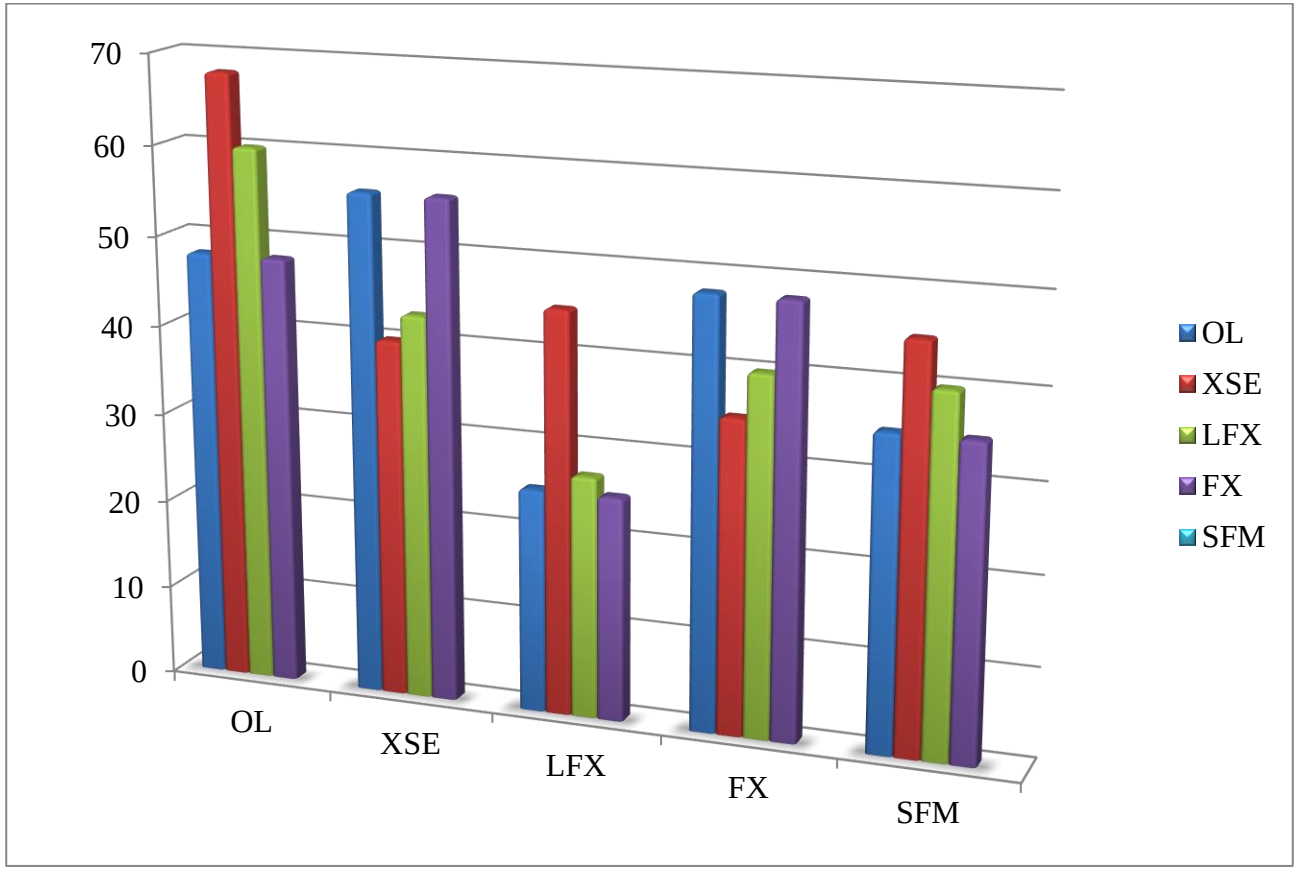
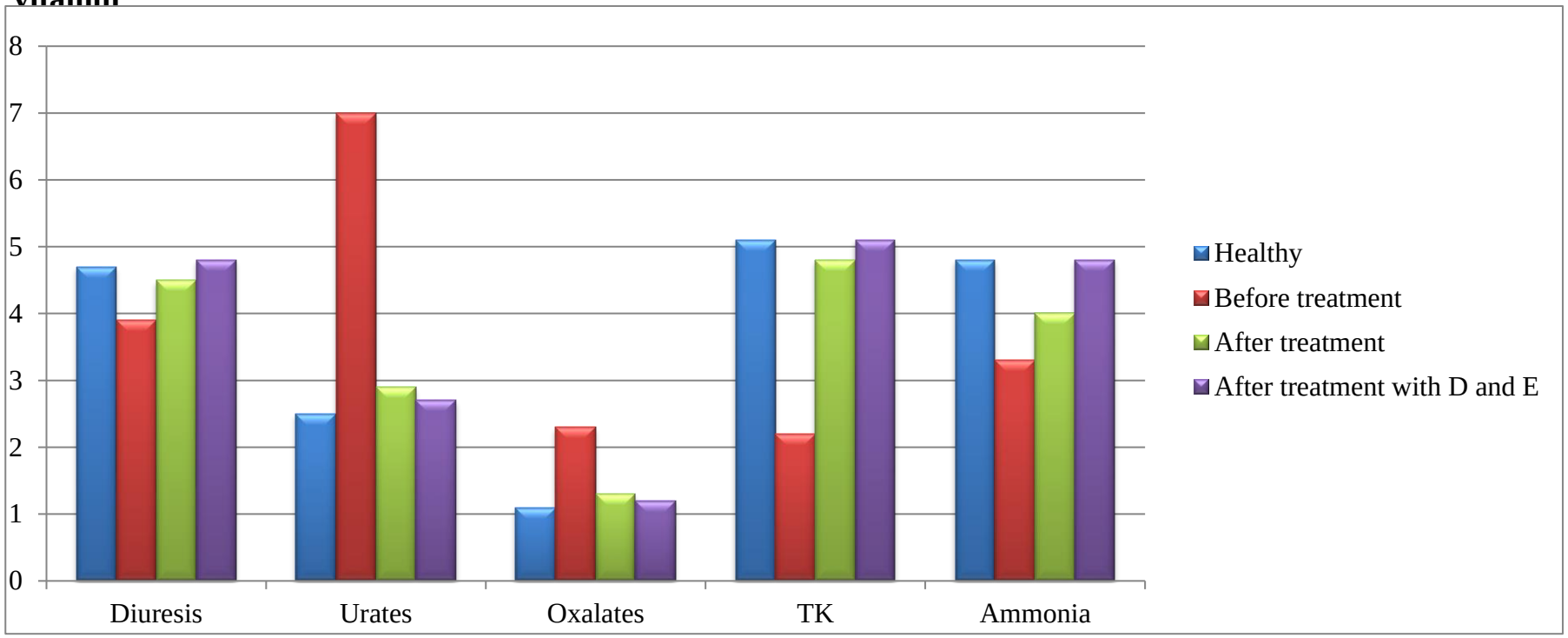


Figure 7. Dynamics of some parameters of lipids, phospholipid fractions

during treatment with dimephosphon and

vitamin E.


8
7
6
5
4
3
2
1
0
Diuresis
Urates
Oxalates
TK
Ammonia
Healthy
Before treatment
After treatment
After treatment with D and E

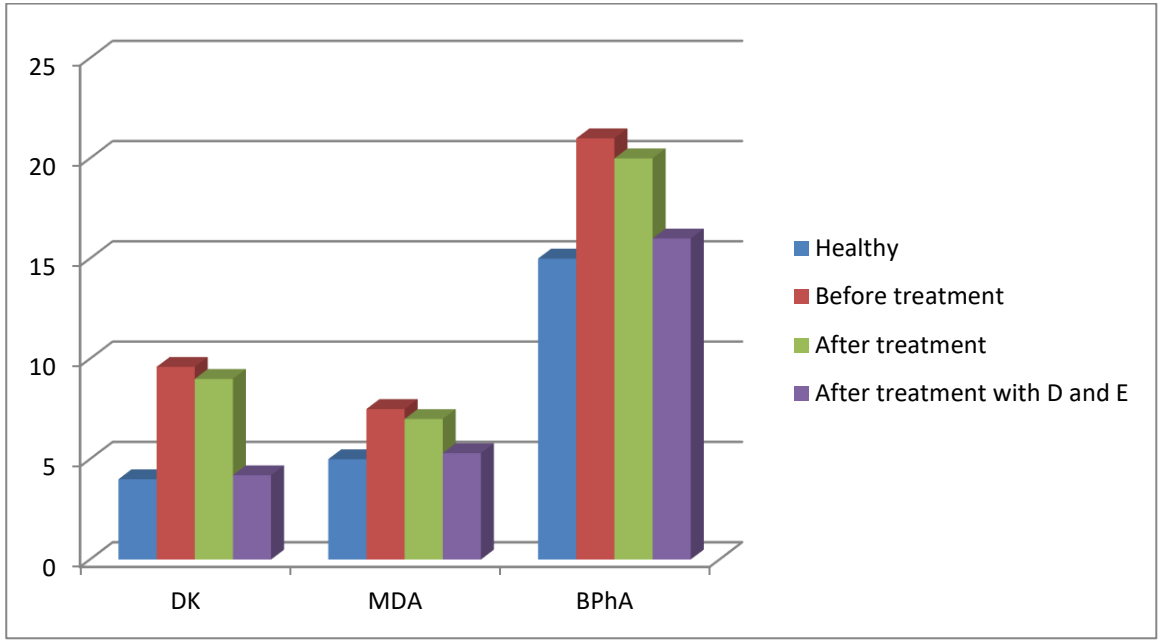


Figure. 8. Effects of D and Vitamin E on LPO and BPhA indicators in blood in children with DSMN pneumonia

Thus, the effectiveness of combined therapy with vitamin D and E is manifested not only in their membrane-stabilizing effect, but also in the significant suppression of excess lipid peroxidation, blood phospholipase activity, since the points of application of these drugs to the body are different. The stabilizing and antioxidative effect of membrane D together with the effect of antiphospholipase and the increase in antioxidant activity of biomembranes because of the antioxidant effect of vitamin E contribute to a more favorable course of pneumonia and relief of SC. The combination of vitamin D and vitamin E is especially effective in pneumonia with IN, BY layers, which may be due to their increased immunomodulatory effect. Protein in the urine was constantly normalized, leukocytes, erythrocytes in the urine sediment and saluria disappeared. The clearance of uric acid increased to 4.08 ± 0.22 ml/min, which characterizes the restoration of filtering and secretory functions of the kidneys. There was a tendency for the index of endogenous formation of SC to decrease to 9.0 ± 0.02, which may indicate a decrease in indirect membranolytic processes, a decrease in protein catabolism. However, the level of uric acid in blood and urine, UA clearance is not fully restored at the time of discharge. Based on this, we tried to further modify the treatment to bring SC levels in blood and urine to a strict standard.

Allopurinol drug was used. Allapurinol affects the process of formation of uric acid and related concretes, inhibits reabsorption of uric acid in kidney tubules.

The main indications for the combined use of dimephosphonate, vitamin E and allopurinol were the following clinical symptoms: severe respiratory failure of the II-III degree, acetone vomiting, stable hyperthermia, manifestations of severe neurotoxicosis, structural and functional cytomembranes against the background of disorders, dysfunction of the cardiovascular system, activation of LPO, as well as an increase in UA values in blood to 0.430 mmol / L and in urine to 6.28 mmol / L in patients with pneumonia with SS. In children who received this multicomponent

therapy, allopurinol was prescribed to children at a dose of 10 mg / kg / h for 3-5 days, the symptoms of shortness of breath quickly stopped and urine output increased. Compared with the previous group of 0.301 ± 0.01, a decrease in serum UA level to 0.240 ± 0.02 mmol / l and 4.08 ± 0.50 mmol / s excretion of UA with urine was observed.

At the same time, UA clearance reached even higher numbers, approaching the norm of 5.60 ± 0.28 ml / min ($p < 0.01$), which indicates the recovery of the filtering secretory function of the kidneys (Table 13).

Indices of endogenous SC formation were restored at the level of 8.5 ± 0.03 in healthy children, which indicates a decrease in catabolic processes. An almost 2.5-fold decrease in the concentration of UA in urine was noted, 0.30 ± 0.02 compared with the initial 0.77 ± 0.09 mmol / kg / s ($p < 0.01$). Allapurinol is an inhibitor of the enzyme xanthine oxidase, which initiates lipid peroxidation, and helps to reduce the processes of lipid peroxidation and decrease the level of UA in blood and urine. Thus, the complex use of dimephosphonate membrane stabilizer and vitamin E antioxidant, allopurinol reduces not only the level of UA in blood and urine, but also the processes of suppression of lipid peroxidation, effectively affects the pathogenetic mechanisms of the development of pneumonia caused by SS.

We present the following clinical observation to demonstrate the effectiveness of dimephosphonate, allapurinol vitamin E in complex therapy in patients with pneumonia with (urinary syndrome).

Table 13

UA INDICATORS IN BLOOD AND URINE IN PATIENTS WITH PNEUMONIA WITH URINARY SYNDROME TREATED WITH DIMEPHOSPHONE, VITAMIN E AND ALLOPURINOL

Indicators Patient groups	Healthyn = 20	Patients with DSMN pneumonia	
		D+vit. E	D+vit.E+allopurinol
UA in blood serum (mmol / l)	0,242±0,01	0,432+0,02 0,301+0,01	0,432+0,02 0,240+0,02x
UA in urine	4,13±0,24	6,26+0,98 5,01+0,30	6,26+0,98 4,08+0,50xx
(mol/s)	0,450±0,036	0,313+0,029 0,420+0,018	0,313+0,029 0,460+0,03x
Diuresis (l)	5,49±0,66	3,04+0,30 4,08+0,22	3,04+0,30 5,60+0,28xx
UA clearance	8,69±0,04	9,55+0,01 9,0+0,02	9,55+0,01 8,5+0,03xx
(ml/min)	0,27±0,02	0,77+0,09 0,50+0,04	0,77+0,09 0,30+0,02xx
Indicator of endogenous formation of MC	1,34±0,7	2,07+0,3 1,8+0,2	2,07+0,3 1,30+0,6

Note: the numerator contains readings before treatment with antioxidants and membrane stabilizers, and after treatment with these drugs, the numerator contains readings. x - reliability of differences in the main therapy group ($p < 0.05$), xx - ($p < 0.001$) + D + vit. E + allopurinol.

Patient Safarov Ural 9 months, medical history No. 9930.

RHCHD 4 / XII-96 came on the 7th day of the illness with complaints of high temperature 39.6, shortness of breath, cough and weakness.

From the analysis of the child from the second pregnancy and the second birth. The mother's pregnancy continued with toxicosis in the second half. The child was born on time, in grade II asphyxia, 6 points on the Hangar scale, weight 3100 g, height 51 cm. Grows up in satisfactory material and household conditions. He experienced frequent colds and intestinal dysfunction. His mother suffered from pyelonephritis, his father suffered from kidney stones, and his grandmother suffered from hypertension. Objectively: the reception is difficult, the child is sluggish, capricious. Shortness of breath (56 min), involvement of auxiliary muscles in respiratory movement, cyanosis of the nasolabial triangle, pallor, acetonomic vomiting, throat hyperemia was observed. Pulmonary auscultation shows harsh breathing, moist small bubble wheezing against the background of weakened breathing. Liver +3 cm, soft, spleen is not palpable. Faeces formed. Free urination without pain.

Blood analysis hemoglobin 110 g / l, er. - 4.5 x 10/12 L, leukocytosis 9.5 x 10/9 L, ESR - 36 mm / h. Urinalysis: proteinuria 0.065 g / l, leukocytes 10-15 v p / er, erythrocytes 5-6 v p / er, bactericides everywhere, urates 5.6 mmol / l / s (2.94 ± 0.24 mm / s in the norm), the concentration of UA in the blood increased to 0.430 in the norm 0.422 + 0.08 mmol / l. UA in urine is 6.28 mmol/s. Diuresis is reduced (306 ml), specific gravity is 1020. On ultrasound, the size of the kidney is normal, its location is normal. Expansion of the calcium-pelvic system. There is sand in the kidney parenchyma. Lipid metabolism and phospholipid spectrum indicators UL 6.8%, FL 2.8%, MDG 9.4%, EXS 9.8%, EYK 7.5%, TG 15.2%, XSE 43.6%, LFX 0.44 mmol / L, FX 0.33 mmol / L, SMF 0.45 mmol / L, FS 0.25 mmol / L, FEA 0.29 mmol / L, DK 10.3 nmol 10/12 solution. , BPhA 21.3% hemolysis.

In the chest x-ray: the roots are strengthened, heaviness in the right lower lung area, there is clear infiltration.

Diagnosis: Complex segmental pneumonia with cardiorespiratory syndrome, acute course, RF II degree.

Kidney: dysmetabolic nephropathy complicated by secondary pyelonephritis, hyperuricemia.

Treatment: carbinicillin 100 units/kg per day, gentamicin 5 mg/kg body weight, intravenously, glucose solution, 2.4% euphylline solution, cocorbaxilose 50 mg. Therapy was started, including dimephosphone 50 mg/kg body weight per day and vitamin E 10% - 3 mg/kg body weight per day IM, allopurinol 10 mg/kg per day for 3 days. On the 4th day from the beginning of therapy, this condition improved significantly, the incidence of shortness of breath and intoxication decreased, the temperature decreased, and the number of wheezing decreased. On the 6th day, the urinary syndrome returned to normal: proteinuria (g/l), oxaluria reached 115 mmol/s, the concentration of SC in the blood decreased to the norm of 0.222 mmol/l in the urine to 4.13 mmol/s. Diuresis increased to 450 ml, beating weight.

Lipid metabolism and phospholipid spectrum indicators UL - 4.6 g / l, FL - 3.2%, MDX - 7.6%, EXS - 6.4%, EYK - 4.06%, TG - 14.6 %, EXO - 51.8%. LFX - 0.23 mmol / L, FX - 0.44 mmol / L, SMF - 0.35 mmol / L, FS - 0.32 mmol / L, FEA - 0.34 mmol / L, DK - 4.6 nmol 10/12 solution, BPhA -15% hemolysis.

Because of complex therapy, the patient was discharged home on the 18th day in a satisfactory condition.

Summarizing the observational data on the nature of hemostasis, the state of lipid metabolism and the clinical picture, we came to the conclusion that it is optimal to use a combination of drugs: membrane stabilizer (dimephosphone), antioxidant (vitamin E) and allapurinol, depending on the severity of hemostasis disorders ,

prevents an individual dose of stopping the formation of salts and preventing the inflammatory process in the kidneys.

CHAPTER VI

CATAMNESTIC FOLLOW-UP OF YOUNG CHILDREN WITH URINARY SYNDROME IN PNEUMONIA

A comparative evaluation of the effectiveness of the complex treatment of nephropathies in pneumonia in young children with the simultaneous use of drugs with antioxidant and membrane stabilizing properties showed the high effectiveness of this approach.

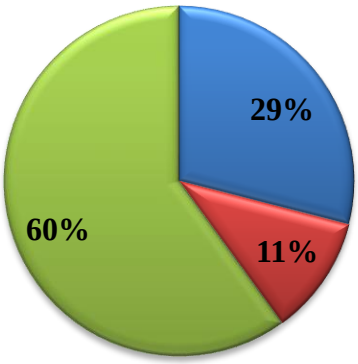
For this purpose, we considered it necessary to study the observations of patients with urinary syndrome (US) with pneumonia in young children who were discharged from the hospital after a traditional course of treatment and a modified therapy method. Until now, this issue has not attracted the attention of nephrologists, although the prevalence of repeated kidney disease cases among this contingent of children is not only decreasing, but has a growing tendency. Comparison of the changes in biochemical and functional parameters obtained using different treatment methods was carried out taking into account the dynamics of clinical symptoms of diseases.

After discharge from the hospital, we continued to observe children with acute pneumonia with urinary syndrome for 1 year. In the first group of children discharged from the hospital, children underwent regular monitoring and observation, while in the second group, preventive correction was applied with the implementation of antioxidant, membrane stabilization and uremic treatment.

In-group I, 28 patients with SS who received conventional treatment on the background of pneumonia (Fig. 9), 11% (3 children) showed US pyelonephritis (KI), 29% of cases (8 children) showed interstitial nephritis (IN) and 60% (17 patients)

underwent rehabilitation. At the same time, in the II group of observed patients (36 children), increased IN was observed in 6% (2% of children), 3No. (1 child) KI and complete rehabilitation was observed in 91% (33 children), that is, in these years did not have bronchopulmonary pathology, if it was, it was not severe, but there were no signs of U

Picture. 9. Clinical-catamnestic analysis of examination of early-aged children with nephritis



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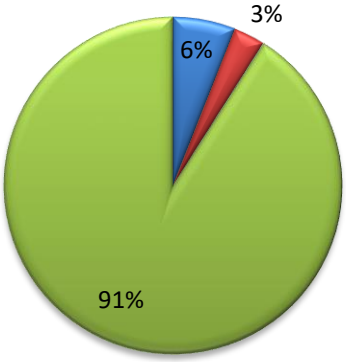

II gr.

It is noteworthy that such a significant difference was not accidental, but was strictly based on obtaining reliable final results of clinical outpatient and follow-up examinations.

The results of the study of partial liver functions, in comparison with children who received only the traditional method of therapy, confirm the modified therapy method in the hospital.

Group I, consisting of 28 children with pneumonia with US group, were discharged from the hospital with no indication of the cause that led to the development of US. Group II - 36 children with pneumonia with a nephrological diagnosis who received antioxidant membrane stabilizing drugs and dietary therapy. At the time of discharge, these patients were in our dispensary observation, underwent clinical and laboratory examination once a quarter to determine the nature of partial kidney functions and metabolic diseases (Table 14).

In group I, 11 children had a cold, while in the modified group, the disease was observed in 3 children. The use of modified treatment in group 11 contributed to a more favorable course and outcome of pneumonia. Cases of infectious toxicosis, febrile period, hypoxia and shortness of breath in children were stopped in a short time. On the background of modified treatment in group II, US and diuresis in young children were restored faster, saluria disappeared, protenuria, leukocyturia and hematuria decreased compared to children of group I who were not treated with modified. In group II, on the background of modified treatment, excretion of urate, oxalate, uric acid approached the norm compared to group I without modified treatment.

Table 14

RESULTS OF CLINICAL AND LABORATORY STUDIES OF CHILDREN IN CONVENTIONAL TREATMENT AND MODIFIED TREATMENT

Indicators	**Healthy n = 15**	**Group I n = 28**	**Group I n = 28**
Clinical signs			
1. The frequency of wind patients	1	11	3
2. Urinary syndrome	-	10	2
Laboratory studies			
1. Diuresis (l)	0,450±0,36	0,352±0,013	0,420±0,018 P<0,05
2. Glomerular filtration ml / s	1,64±0,08	1,26±0,17	1,62±0,13 P<0,05
3. Oxalates mmol / s	110,0±10,5	240,0±12,0	115,0±10,5 P<0,01
4. Urates mmol / s	2,94±0,24	4,8±0,22	2,8±0,20 P<0,01
5. Uric acid mmol/s	4,13±0,21	6,28±0,36	4,3±0,28 P<0,05
6. Ammonia mmol / s	46,8±1,2	36,5±1,18	46,0±3,0 P<0,05
7. Titratable acid	51,0±2,8	38,1±1,4	50,0±3,0 P<0,05
8. Osmolarity mmol / m	627±61,7	810±26,5	630±11,0 P<0,05

Excretion of ammonia, titrated acids in-group II was restored in healthy children, while in children of group I these indicators were reduced. Thus, the complex treatment, modified with metabolic, antioxidant and membrane stabilizing drugs, restores the octogenetic ability of the kidneys, regulates the acid-base status of the body and stabilizes the cytomembrane of the renal epithelium.

Scheme of complex metabolic correction in the post-hospital rehabilitation phase Fig. 10.

Prophylactic treatment against metabolic relapse includes: children with acute pneumonia with urinary syndrome were given 50 mg/kg of demiphosphon per day, vitamin E 3 mg/kg of body weight for 10-12 days after discharge from the hospital. In addition, special diet therapy is prescribed for metabolic diseases. Patients were given these treatment complex 3-6-12 months after discharge from the hospital.

The appointment of metabolic therapy allows full recovery of clinical and laboratory indicators in these patients and reduces the patient's stay in the hospital to 4-8 days.

Thus, as a result of our work, we can conclude that the commonality of the pathological mechanisms of dysmetabolic nephropathies in young children, due to common biological laws, contributes to the uniformity of their clinical symptoms. One of the main links in the development of these processes this is hypoxia, intoxication, disruption of functional adaptive processes and genetic load. In such conditions, the kidney becomes a "shock" organ and reacts by disrupting the processes of cellular bioenergetics and metabolic changes.

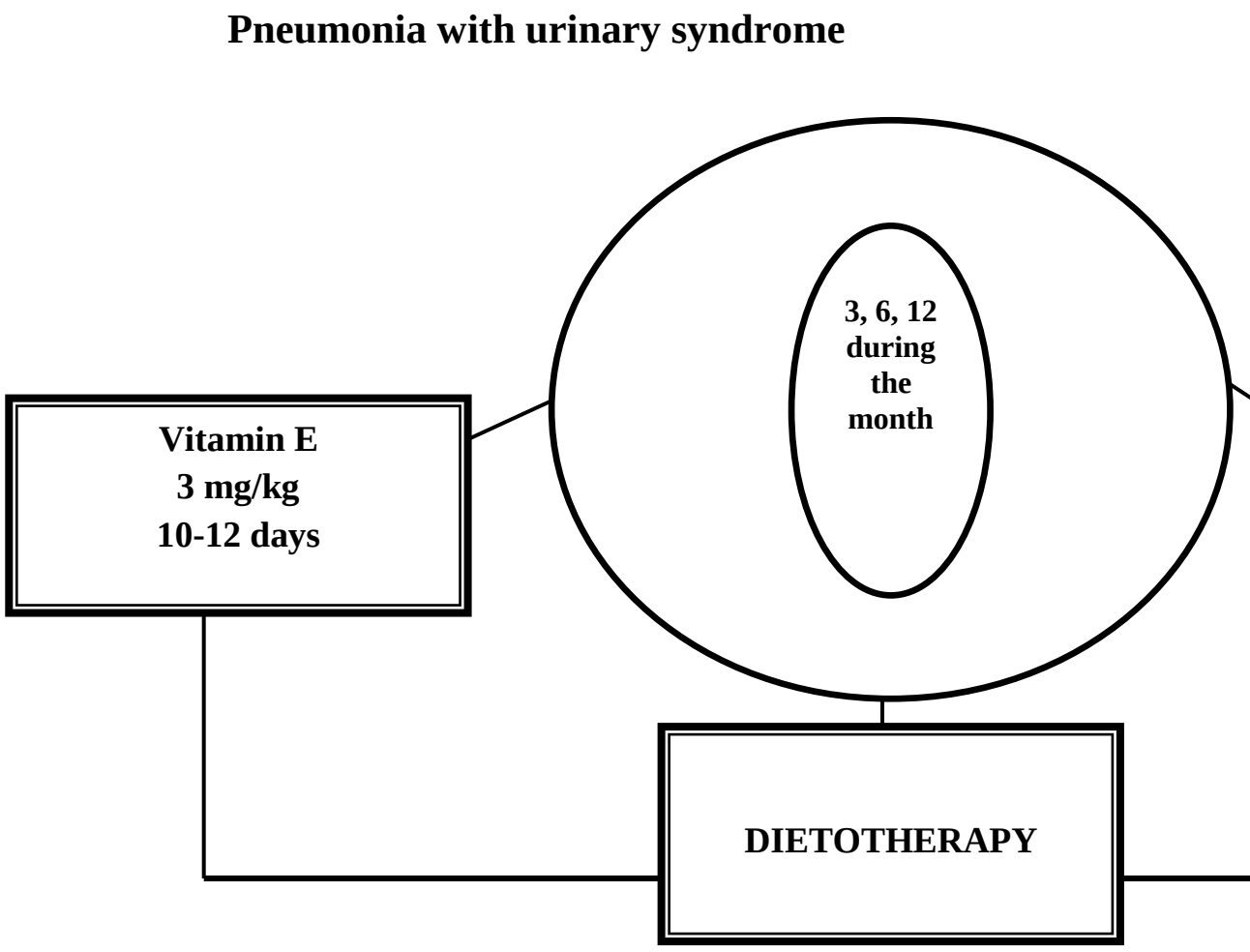

Figure 10. Scheme of complex metabolic correction in

the post-hospital stage

Correction of metabolic processes is a fundamental pathogenetic factor. Reducing hypoxia, intoxication, and metabolic diseases under the influence of drugs with antioxidant and membrane stabilizing effects helps to restore the homeostatic functions of the kidneys at the cellular level.

CONCLUSION

Children with pneumonia of early age bacterial toxins under the influence of hypoxia in the body, appear severe metabolic diseases which lead to kidney damage.

Toxic nephropathy is a common type of pathology, accounting for up to 20% of the total number of nephropathies of various etiologies. Functional changes in the kidneys in pneumonia, even without the development of urinary syndrome, have been noted by many authors. It is known that pneumonia is a pathology of many organ membranes. Taking into account the above, the effect of pneumonia on the metabolism and function of the kidneys in children with hereditary metabolic diseases of oxalic acid and uric acid is of primary importance, since dysmetabolic nephropathy often manifests itself against the background of respiratory pathology. will then take a torpid flow.

In this regard, the purpose of this work was to develop a principle of early differential diagnosis, treatment and prevention of a comprehensive study of lipid peroxidation of erythrocyte membranes and partial functions of the kidneys in children suffering from pneumonia with kidney damage.

Based on the above, evaluation of the effectiveness of combined treatment with dimephosphonate and vitamin E in patients with pneumonia with kidney pathology.

To perform the task, the followings are necessary:

- to determine the frequency and nosological structure of nephropathy in young children with pneumonia.

- to determine the relationship between cell membranes, partial functions of kidneys and changes in the biochemical composition of urine in pneumonia in children with kidney damage.

- determination of LPO characteristics, partial functions of kidneys depending on the nosological structure of nephropathy in pneumonia in young children suffering from metabolic diseases (hyperoxal-uraturia).

- to evaluate the effectiveness of early corrective therapy, taking into account the type of diabolism that causes kidney damage.

Due to the frequent occurrence of nephropathies in young children with pneumonia with urinary syndrome, an analysis of archival materials of 1003 children aged 1 month to 3 years treated in the pulmonology department of the Samarkand Regional Children's Clinical Hospital was conducted.

The analysis showed that 476 (47.5%) of 1003 patients hospitalized with early-onset pneumonia had pathological urinary syndrome.

In order to determine the nosological structure of the pathology of the urinary system and analyze the risk factors, 130 patients with pneumonia with DSMN were examined in a specialized nephrology hospital: dysmetabolic BY 54 children (41.5%), dysmetabolic IN 46 children (35.4 %), UTD in 21 children (16%), tubulopathy in 3 children (2.3%) and OSS in 6 children (4.6%). Tubulopathies and SSOA manifested themselves as secondary dysmetabolic pyelonephritis. There were 71 boys and 92 girls. Boys often suffer from IN, UTD, tubulopathy; girls suffered more from DSMN, dysmetabolic and primary KI, urinary tract infections.

Thus, the analysis of the structure of neuropathy in pneumonia in young children shows that dysmetabolic nephropathy takes the leading place in the structure of nephropathy in children. Special studies conducted in children with pneumonia confirm the early age of dysmetabolic nephropathy in these children, which naturally requires appropriate therapeutic tactics. Obstetric anamnesis, data on the course of the perinatal period in the observed patients revealed the following features: pregnancy and delivery complications, somatic pathology on the part of the mother,

as well as hereditary predisposition to metabolic diseases and family history of severe kidney disease.

Pneumonia, along with the development of IN, KI, UTD, is a triggering factor in the manifestation of hidden kidney pathology in young children based on congenital and hereditary anomalies of the urinary system and dysmetabolic diseases.

The study of partial kidney function tests in patients with pneumonia with DSMN showed a decrease in acidoammonogenesis, increased metabolic acidosis in conditions of hypoxia, compared to patients with oliguria, DMN and pneumonia without a healthy child ($p<0.001$).

In pneumonia with DSMN, compared to healthy children and patients with pneumonia without DSMN, a significant increase in the excretion of urinary oxalate was found ($p < 0.001$), which indicates secondary oxaluria, which is due to the different thickness of the cytomembranes of the renal epithelium. Precipitating factors: infections, hypoxia, hyperthermia, electrolyte disturbances, and possibly the result of genetically determined instability of the membranes.

In patients with pneumonia with DSMN, there was an almost 2-fold increase in the excretion of urates and uric acid ($p < 0.001$), which can be considered as a result of catabolic processes of the breakdown of purine bases in the tissues, in addition, 58 of the patients It was found that there is a genetic predisposition to disorders of uric acid metabolism in %.In patients with pneumonia with dysmetabolic IN, KI and KID, partial kidney function is impaired, a decrease in the function of acidoammonogenesis, which manifests itself as an oversaturation of urine with oxides, crystals of uric acid and uric acid, and an increase in urine osmolality. It's done. In patients with pyelonephritis, due to a decrease in the level of excretion of ammonia and titratable acids due to a decrease in the level of excretion of ammonia and titratable acids, a significant impairment of kidney function was found, which reduces the adaptive capacity of the kidneys to maintain the acid-base state of the

body in conditions of tissue hypoxia, and can increase respiratory and metabolic acidosis in pneumonia in young children.

In patients with pneumonia with urate nephropathy, there was an almost 2-fold increase in the level of uric acid in the blood to 0.432 ± 0.02 mmol / L, compared to healthy children by 0.242 ± 0.01 ($p < 0.001$), in urine MC release increased by 6.26 ± 0.98 mmol/s ($p < 0.05$).

In patients with early-onset pneumonia, when recalculated per kg / body weight, MS concentration in urine was 0.77 ± 0.09 mmol / kg / s compared to healthy children, a 3-fold increase was observed (0.27 ± 0.02 mmol/kg/s ($p<0.001$).

The concentration of uric acid tended to increase in patients with pneumonia ($p> 0.001$). The indicator of endogenous formation of MS also tended to increase to 9.55 ± 0.01 compared to the indicator of 8.69 ± 0.04 mmol / L ($p> 0.05$) in healthy children, which is catabolic processes, the breakdown of cell nucleotides in the background of hypoxia, showed acidosis in pneumonia.

The clearance of uric acid in patients with pneumonia was 5.44 ± 0.66 ($p> 0.001$) lower than that of healthy children, 3.04 ± 0.30 ml / ml, which is a decrease in the filtration function of the kidneys, may be associated with low diuresis.Tubular reabsorption of uric acid TR UA in patients with pneumonia with urate nephropathy was 70%, which is higher than in healthy young children, where this figure was 51%.

Thus, in the genesis of hyperuricemia in young children with pneumonia against the background of urate nephropathy, the following are pathogenetic: increased endogenous synthesis of SS as a result of the catabolic direction of metabolism, breakdown of cellular nucleotides against the background of viral-bacterial damage to lung and kidney tissues , increased renal tubule SC reabsorption along with a decrease in uric acid clearance. An increase in the concentration of uric acid in the urine, as well as its increase in the urine, urate and oxalate crystals damage the

tubular epithelium of the kidneys, causing the development of interstitial processes in the kidneys. Tubular blockage with urate crystals and uric acid can lead to the development of kidney failure.

A decrease in UA clearance in patients with pneumonia indicates a lack of secretory mechanisms for UA release due to impaired reabsorption-filtration processes.

In patients with pneumonia, the metabolic type of HU was mainly present (60%), which indicates an increase in UA biosynthesis and its excretion. In the renal type (40%), there was a decrease in the renal excretion of UA, a decrease in the clearance of SC due to a violation of the secretory and filtering mechanisms of the kidney, which requires the appointment of uricosuric drugs. As a result of the study of the spectrum of lipids in the blood serum, an increase in the level of total lipids, EXS, EYK, and a decrease in the fraction of FL and XSE, which characterize the catabolic scheme of lipid metabolism, were revealed. An increase in the EYK / TG ratio by 0.42, and in healthy people it can be equal to 0.20, this is probably the predominance of lipolytic processes in young children, the mobilization of fats from the depot, this is an increase in EYK, the active transport of lipids comes with a form. An increase in the EXS / XSE index of 0.21 indicates a decrease in the esterification of fatty acids that are used faster in metabolism. The decrease in the level of FL can be explained by the increase in consumption and participation in the structural renewal of biomembranes. The obtained data reflect the participation of lipids in the body's stress reactions and show the effect of hypoxia on lipid metabolism.

In the study of the structural and functional state of erythrocyte membranes in patients with pneumonia without DSMN, in patients with pneumonia with DSMN, phospholipid fractions containing choline, mainly in patients with pneumonia without DSMN 0 revealed a decrease in FL up to .39 ± 0.01 mmol / L, 0.33 ± 0.01 mmol / L in patients with pneumonia with DSMN ($p < 0.01$). At the same time, the

cytotoxic fraction of phospholipids LPX increased ($p < 0.001$), and the endogenous antioxidant FEA fraction decreased. Thus, pneumonia with DSMN is characterized by significant shifts in the phospholipid spectrum of erythrocyte membranes, which may be of pathogenetic importance in renal membrane damage. One of the mechanisms of structural and functional disorganization of cell membranes in children with pneumonia is the excessive accumulation of LPO products, which is evidenced by the increase in the content of the initial LPO product in erythrocytes - diene conjugates, with DSMN 10.3 ± 0.21 nmol / 10, Up to 2 erythrocytes is more significant in patients with pneumonia ($p < 0.01$). LPX and DK in pneumonia patients with DSMN ($r = 0.5$) $p < 0.05$; FX and DK ($r = -0.7$) $p < 0.05$, ie. FX shortages and surpluses create the necessary conditions to activate the initial stages of LPO. In patients with pneumonia with DSMN, the QFF of blood reached 21.25 + 0.3% of erythrocyte hemolysis ($p < 0.001$).

Thus, an increase in the activity of endogenous BPhA in the blood contributed to the destruction of the phospholipid layer of membranes with the appearance of phosphorylated nitrogenous bases - FEA, FS and secondary oxaluria, uraturia. Since patients with pneumonia with DSMN have persistent crystalluria, calciuria, which indicates damage to the kidney membrane structures, it is possible to talk about the pathology of polyorgan membranes with the involvement of the kidneys in the pathological process.Patients with pneumonia had a lower cytotoxic fraction of LFX ($p < 0.01$) and a decrease in FX fraction by 0.33 ± 0.01 compared to healthy children ($p < 0.01$). The level of endogenous bioantioxidant - FX in KID, IN, patients also decreased, especially with pyelonephritis up to 0.28 ± 0.01 mmol / l ($p < 0.01$) and significantly increased LPC fraction in patients with pyelonephritis ($p < 0 .05$).

FEA fraction decreased in all types of kidney pathology, especially in pyelonephritis ($p < 0.01$), KID reached 405 ± 0.01 mmol / l ($p < 0.01$) in pyelonephritis. Thus, in IN, pyelonephritis, significant disorganization of cytomembranes with rearrangement of phospholipid components was revealed.

A comparative assessment of the severity of LPO processes at different stages of kidney damage in patients with pneumonia with dyspeptic diseases showed that the constant indices were significantly increased in patients with IN, KI ($p < 0.01$; $p < 0.001$). Blood BPhA also increased in patients with IN, KI, and increased LPO product - MDA, with the highest values in patients with BY - 8.3 ± 0.21 nmol/mg lipids ($p < 0.001$). In patients with IN, PN ($p < 0.01$ and $p < 0.001$), an increase in the level of DC in the urine, an increase in the activity of phospholipase A2 in the urine, which is more pronounced in pyelonephritis, indicates the involvement of the renal epithelial membranes in the pathological process.

A positive correlation analysis was established between UA and LFX ($r = 0.37$; $P < 0.05$), UA and DK levels. ($r = 0.38$, $P < 0.05$) confirms the effect of GU on the development of membranopathological processes. The inverse correlation between the levels of UA and FX ($r = -0.50$; $P < 0.05$), UA and FEA ($r = -0.60$; $P < 0.05$) indicates a decrease in the level of endogenous bioantioxidants, a change in the structural and functional state of cytomembranes show.

With the deepening of the pathological process in the kidneys, the stratification - infectious - inflammatory process is activated, the LPO processes are activated, the disorganization of cytomembranes increases, which is the basis of inflammatory processes not only in the lung tissue, but also in the kidneys.

Thus, the presented data show the important pathogenetic importance of cell membrane destabilization, the activity of LPO processes in the course of pneumonia in young children on the development of pathological processes in the kidneys.To correct these disorders, we used dimephosphon - 50 mg/kg of body weight per day for 10-15 days, along with the commonly accepted complex therapy and pneumonia diet therapy. The use of D in the complex treatment of pneumonia contributed to a more favorable course and outcome of pneumonia. Urinary syndrome was eliminated faster when treated with D treatment: diuresis was restored, saluria

disappeared 5 days earlier, and proteinuria disappeared 6.3 ± 0.2 days earlier compared to conventional treatment. Normalization of the elements formed in urine - 5 ± 0.72 days ago. Excretion of urates, uric acid, oxalates approached the norm, the function of acidoammoniogenesis was restored, which indicates the improvement of the asystenetic function of the kidneys, the antikidotic effect of D, and the effect on the renal mechanisms of the control of the acid-base state of the body. A decrease in the excretion of oxalate, urate, and uric acid indirectly indicates the stabilization of the cytomembranes of the kidney epithelium. Positive shifts were observed in the lipid spectrum of blood serum - an increase in the level of FL, EKS, and the normalization of the balance of phospholipid fractions of erythrocyte membranes was noted. Dimephosphon simultaneously stabilizes the structure of cell membranes and contributes to the normalization of the LF / FX ratio to 0.54 in patients with 1.3 pneumonia, which determines the optimal antioxidant activity of cells due to phosphatidylcholine. The inhibitory effect of D on the intensity of LPO processes can be explained by the addition of D to membranes due to its affinity for phospholipids of cell membranes, which stimulates the effect on the body's protective antioxidant system. At the same time, LPO indicators - DK, MDA, as well as blood BPhA were not returned to their initial values when the membrane protector was prescribed, therefore, as an antioxidant, we used vitamin E at 3 mg/dose per body weight orally or intramuscularly for 10-12 days. The effectiveness of the combined use of vitamin D and E was shown not only in their membrane-stabilizing effect, but also in the significant suppression of excess lipid peroxidation, blood phospholipase activity, because the points of application of these drugs are different.

Stabilizing and antioxidative action of membrane D, combined with antiphospholipase effect, and increased antioxidant activity of biomembranes as a result of antioxidant effect of vitamin E, contribute to a more comfortable course of pneumonia, alleviation of urinary syndrome, and normalization of dysmetabolic changes.

But in patients with pneumonia with urate nephropathy, the level of uric acid in the blood and urine, the clearance of UA during discharge was not completely restored, and therefore, to correct these parameters, they used allopurinol in a dose of 3 mg to 10 mg / kg / h . Allopurinol is an inhibitor of the enzyme xanthine oxidase, which initiates lipid peroxidation, and not only reduces the level of uric acid in the blood and urine, but also normalizes the index of endogenous production of UA, decreases the concentration of UA in urine by almost 2.5 times, increases clearance of uric acid, and contributes to a decrease in membranolytic processes. Thus, the obtained clinical and biochemical data are convincing evidence for the need to use membrane stabilization, antioxidant drugs for young children with pneumonia with US, as well as hyperuricemic drug - allapurinol can be used in the treatment of pneumonia caused by uricopathy. In addition, one of the phytopreparations of the German company Bionorica, registered in the Republic of Uzbekistan, which normalizes the normal passage of urine, regulates the rhythm of urination, prevents the high concentration of salts in urine, and the formation of large crystals, is the "Kanefron "N" drug. The drug improves blood supply to the kidneys, helps to reduce proteinuria and the pathological crystallization of urine. Flavonoid components included in Kanefron N drug have angioprotective, anti-inflammatory and antispasmodic effects. Due to its antioxidant properties, Kanefron N has a clear protective effect against free radical damage. Kanefron N can be indicated for all types of metabolic nephropathy.

Akhmedova M.M.

Kanefron N: instructions for use

• Complex treatment of chronic bladder (cystitis) and kidney (pyelonephritis) infections.

• With non-infectious chronic inflammation of the kidneys (glomerulonephritis, interstitial nephritis)

• As a remedy against calculus (even after removing stones from the urine)

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