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CORRECTION OF PROTEIN OXIDATIVE MODIFICATION WITH QUERTINE IN PATIENTS WITH URONEPHROLITHIASIS COMORBID WITH METABOLIC SYNDROM

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Urate nephrolithiasis is one of the most common types of urolithiasis (KSD), developing most often in young and middle-aged people [1]. Depending on the place of residence in Europe, this disease affects from 5 to 10% of population, i.e. about 2000 people per 1 million inhabitants annually. Moreover, the incidence of urate nephrolithiasis is increasing regardless of age, sex and race [10].

There is no single theory of the urate nephrolithiasis pathogenesis. As a multi-etiological disease, UN stems from the consequences of disorders of the urinary system, gastrointestinal tract, as well as metabolic, hormonal and genetic disturbances. With urate nephrolithiasis, various physicochemical processes occur in the body in general, and in the kidneys and urinary tract in particular. "Unmodified factors" – ethnic, sexual, geographical, genetic – play an important role among the causes of urate nephrolithiasis. "Modified factors" such as metabolic syndrome (MS), hypertension, atherosclerosis, obesity, and diabetes are also of great importance [12].

For many years, MS has been called a "non-infectious epidemic." It is in highly developed countries that the population leads a sedentary lifestyle, overeats, and is often exposed to stress. MS is a combination of metabolic and functional disorders and consists of several pathological processes based on obesity, impaired insulin resistance, hypertension, impaired lipid metabolism manifested as elevated triglycerides and high levels of high-density cholesterol. In the pathogenesis, it is very important to take into account the metabolic disorders that are characteristic of MS and urate nephrolithiasis. The reasons for the development of these pathologies include lithogenic properties of urine, the patient's diet, the environment for the formation of urate stones, inflammation in the kidneys, various molecular changes that are the basis for the emergence and progression of MS. Changes in substances in the urine and the activation of oxidative stress should also be considered [9].

It is known that urate nephrolithiasis comorbid with MS occurs against the background of the formation of reactive oxygen species and the peroxidation activity of biosubstrates [6]. We proved that in patients with urate nephrolithiasis comorbid with MS there was an increase in TBA-reactive substances, diene and triene conjugates in the serum, as well as a decrease in the activity of the antioxidant system (AOC) – the level of endogenous α-tokeferol as a non-enzymatic part, and the activity of glutathione reductase, as the enzymatic part in the serum. Moreover, the progression of lipid peroxidation was more significant in patients with urate nephrolithiasis comorbid with MS. This feature of the pathogenesis is due to the fact that the kidneys are exposed to xenobiotics. In this case, unsaturated fatty acids are a substrate for the processes of free radical oxidation of lipids. Microorganisms are activated, causing an inflammatory process and stimulating the phagocytes, which leads to the release of a large number of reactive oxygen species [11].

Reactive forms of oxygen damage molecules and proteins of the plasma membrane which leads to dysfunction of the antioxidant system and the progression of free radical oxidation of lipids and oxidative modification of proteins (OMB) in cells. Proteins of the amaged plasma membrane activate the release of reactive oxygen species and deplete antioxidant protection. OMB metabolites contribute to DNA damage. It is believed that free radical oxidation of proteins is an earlier indicator of OS than the processes of lipid peroxidation. Protein peroxidation (PPO) triggers the development of urate nephrolithiasis, with oxidative stress activity progressing more rapidly when urate nephrolithiasis is associated with MS. Thus, obtaining the results of OMB makes it possible to assess the level of progression of peroxidation of the affected cells and the reserve-adaptation state of the patient in general [4].

Given that the mechanisms of PPO are insufficiently studied in urate nephrolithiasis comorbid with MS, it is very important to maintain homeostasis by

prescribing drugs with antioxidant effects [6].

In the treatment of urate nephrolithiasis comorbid with MS, it is important to use drugs that have a complex action, namely anti-inflammatory, membrane stabilizing, antioxidant, hypoglycemic, hypolipidemic, nephroprotective, cardioprotective, metabolitotropic, diuretic, immunomodulatory and antispasmodic. For the treatment of these pathologies it is promising to use the flavonoid quertin, which consists of aglycone plant flavonoid glycosides, including rutin [5].

In this regard, the study of the diagnosis and treatment of urate nephrolithiasis comorbid with MS is an urgent problem of clinical medicine for optimal and radical metaphylaxis of KSD.

The aim of this research is to study the effect of quertin on the processes of POM in patients with urate nephrolithiasis, comorbid with MS.

Material and methods

The research was conducted on the basis of the urology department of the Community Non-Commercial Establishment 'Zaporizhzhia Central District Hospital'. Patients were divided into control, comparison and main groups depending on the presence of comorbid pathology in combination with urate nephrolithiasis, and the nature of drug treatment. A total of 118 people were included in the study. Indicators obtained from healthy individuals (donors) were taken as normal.

Control group 1 (n = 38) included 19 men and 19 women aged 22 to 73 years (average age was 45.27 ± 1.93 years) with urate nephrolithiasis. In males,abdominal circumference before treatment was 89.17 ± 0.88 (cm), and in females it was 78.42 ± 0.41 cm; it totally made 84.80 ± 0.88 cm. The weight of patients with urate nephrolithiasis reached 72.36 ± 0.96 kg. The patients' height was 172 ± 10 cm, body mass index (BMI) was 24.34 ± 0.14 kg / m^2. Patients with urate nephrolithiasis received traditional therapy: anticholinergic drug riabal 1 tablet (30 mg) 3 times a day or antispasmodic drotaverine 1 tablet (40 mg) 3 times a day, nonsteroidal anti-inflammatory drug dexalgin 50 mg in the dose of 2 ml for intramuscular pain,granulated Uralit-Uin the dose of 1 teaspoonful (2.5 g) 2-3 times a day depending on the pH of fresh urine (6.2-6.8), water blast.

In Group 2 (experimental group) (n = 42), there were patients with urate nephrolithiasis comorbid with MS (16 males and 26 females), whose age varied from 30 to 80 years (on average,59.14 ± 1.67 years old). Before treatment, abdominal circumference in men was 109.48 ± 1.59 cm, in women – 110.73 ± 1.34 cm, totally– 110.30 ± 1.01 cm. The weight of patients with urate nephrolithiasis comorbid with MS was 96.62 ± 1.13 kg. The height averaged to 169 ± 10 cm. BMI was 33.90 ± 0.38 kg / m^2. Patients with urate nephrolithiasis comorbid with MS received traditional therapy and conventional drugs that correct cardiometabolic disorders: atorvastatin 1 tablet (20 mg) per day in the evening, metformin 1 tablet (1000 mg) 1-2 times a day, allopurinol 1 tablet (100 mg) per day, liprazidum ½ - 1 tablet (20 mg) per day in the morning, vitamin B6 1 tablet (50 mg) 2 times a day, magnesium oxide 1 tablet (0.5 g) 2 times a day.

Main Group 3 (n = 38) was made up of patients with urate nephrolithiasis comorbid with MS, who received traditional therapy and conventional drugs that correct metabolic disorders, on the background of bioflavonoids. The group included 10 males and 28 females whose age ranged from 40 to 78 years (averaging to 59.89 ± 1.34 years). The weight of patients in this group was 99.18 ± 1.15 kg, the height – 168 ± 10 cm, and BMI was 35.02 ± 0.45 kg / m^2. In men, the circumference of the abdomen before treatment was 110.50 ± 1.82 cm, in women – 112.32 ± 1.37 cm, totally – 110.50 ± 1.82 cm. Patients of the main group received traditional therapy and conventional drugs, which correct metabolic disorders, on the background of quertinein the dose of 1 tablet (40 mg) 3 times a day.

In the group of healthy individuals (n = 30), there were 13 males and 17 females aged between 21 and 68 years (average age was 34.83 ± 2.04 years).

The study of the condition of patients with urate nephrolithiasis and urate nephrolithiasis comorbid with MS was performed by means of anamnestic, objective, clinical-laboratory, radiological, ultrasound, radioisotope, biochemical methods, according to the protocol approved by the Order of the Ministry of Health № 1-1/152 (item 2) "Urolithiasis, kidney stones" dated March 6, 2003.

The diagnosis of MS was made according to the recommendations of the 2005International Diabetes Federationand was based on the central obesity (waist circumference in men over 94 cm and over 80 cm in women, BMI ≥25) and two additional MS criteria, found in patients with urate nephrolithiasis

All patients were examined after obtaining their informed consent in accordance with the requirements of GSR INS.

Enrollment criteria included the following: confirmed urate nephrolithiasis or urate nephrolithiasis comorbid with MS; age between 18-80 years; a patient's informed consent to the study and pharmacotherapy.

Exclusion criteria were the following: the presence of concomitant oncological, psychoneurological, pulmonary and other somatic diseases (e.g. gout); refusal of the proposed treatment and re-examination; intakeof drugs absent from the standards of urate nephrolithiasis and MS treatment; alcoholism and drug addiction; for women – pregnancy and lactation.

According to the protocol, the participants underwent a number of laboratory and instrumental studies: general blood and urine test, urine pH test; measurement of abdominal circumference, body weight, and body mass index;kidneys ultrasonography, Doppler imaging, X-ray examination of the kidneys (plain and excretory urography), radioisotope renography, electrocardiography, blood pressure control.

To study the state of PPO, the indicators of POM were analysed according to the method designed by E.E. Dubininaet al. The optical density of diphenylhydrazones was recorded on a spectrophotometer at a wavelength of 356 nm and 370 nm (for neutral aldehyde and ketone derivatives) and 430 nm and 530 nm (for alkaline aldehyde and ketone derivatives) [4].

The study of POM in serum was followed up before treatment and 7 days, 14 days, 1.5-2 months, 3-6 months after it. The results were statistically processed using software packageStatistica 13.0.

Results and Discussion

As a result of the study of POM indicators, it was found that in the course of treatment in the control, the main, and the experimental group, ambiguous changes in the level of diphenylhydrazones were observed. Thus, before treatment the content of neutral ketoderivatives POM-356 and POM-370 increased in the group of patients with urate nephrolithiasis by 33.4% and 21.5%, respectively, compared with the group of healthy individuals (Figure1). In the groups of patients (main and experimental) with urate nephrolithiasis comorbid with MS, the level of these indicators increased even more significantly – by 56.2%, 42.3% and 69.6%, 59.7%, respectively. The level of alkaline aldehyde derivatives POM-430 increased even more significantly, especially in the groups of patients with urate nephrolithiasis comorbid with MS (by 53.3% and 73.3%, respectively).

Thus, in patients with urate nephrolithiasis in pretreatment period there was activation of PPO and pronounced changes in the state of biological membranes, which exhausts protective mechanisms. The presence of MS in patients with urate nephrolithiasis enhanced the processes of proteins oxidative modification. The data in Figure 1 indicate a significant progression of the content of diphenylhydrazones, which belong to neutral aldehyde and ketone derivatives. Thus, after 3-6 months of follow-up in the control group of patients with urate nephrolithiasis, the level of POM-356 and POM-370 increased significantly by 37.04% and 46.14%, respectively. The content of alkaline aldehyde derivatives OMB-430 also increased by 28.51% after 3-6 months.

Data of the POM study in groups of patients receiving traditional therapy and conventional drugs that correct metabolic disorders are shown in Figure 1. This allows to observe that the pronouncement and direction of changes in indicators was ambiguous.

The level of POM-356 in patients with urate nephrolithiasis comorbid with MS (experimental group) slightly decreased after 1.5-2 months (by 14.78%) and after 3-6 months (by 20.47%). At the same time, in quertine-receiving patients of the main group, the content of POM-356 decreased moderately after 7 days (by 27.19%) and after 14 days (by 37.12%), and significantly after 1.5-2 months (by 40.65%) and after 3-6 months (by 42.63%). A moderate decrease in the level of POM-370 was observed in patients of the experimental group after 14 days, 1.5-2 months and 3-6 months of treatment with traditional and conventional drugs that improve metabolic processes (by 19.86%; 16.65% and 18.01% respectively). Simultaneously, the level of POM-370 decreased in patients receiving quertine: moderately after 7 days (by 28.50%) and after 14 days, and significantly after 1.5-2 months (by 41.33% and 50.0% respectively) and after 3-6 months (by 35.86%).

Likewise, the level of POM-430 decreased in experimental group patients (by 17.95% and 21.44%, respectively) after 1.5-2 months and 3-6 months of treatment. At the same time, the level of OMB-430 decreased moderately in patients receiving quertine after 7 days of treatment (23.0%) and significantly after 14 days, 1.5-2 months and 3-6 months of follow-up (by 30.69%, 42.93% and 39.27%, respectively).

Assessing the results of the study integrally, it is possible to draw the following conclusions. The level of phenylhydrazones, which belong to natural aldehyde and ketone derivatives POM-356 and

Table1 (Figure 1)

Dynamics of changes in protein peroxidation in patients with urate nephrolithiasis (UN) (control group), urate nephrolithiasis comorbid with metabolic syndrome (UN + MS) (experimental group) and the main group of patients with urate nephrolithiasis comorbid with metabolic syndrome against the background of quertine intake (UN+MS+quertine)

Indicator	Group	Before treatment	After 7 days	After 14 days	After 1.5-2 months	After 3-6 months
POM-356, c.u..	Healthy individuals	0.224±0.040				
	Control	0.300±0.020*	0.290±0.020* p>0.05	0.360±0.040* p>0.05	0.370±0.020* p<0.05	0.420±0.010* p<0.05
	UN+MS	0.350±0.020*	0.320±0.020* p>0.05	0.310±0.016* p>0.0	0.300±0.010*/§ p<0.05	0.280±0.020*/§ p<0.05
	UN+MS+quertine	0.380±0.040*	0.280±0.010* p<0.05	0.240±0.01§/+ p<0.05	0.230±0.010§/+ p<0.05	0.220±0.020§/+ p<0.05
POM-370, c.u.	Healthy individuals	0.288±0.006				
	Control	0.350±0.030*	0.360±0.020* p>0.05	0.380±0.010* p>0.05	0.390±0.010* p>0.05	0.510±0.020* p<0.05
	UN+MS	0,410±0,030*/§	0,370±0,030* p>0,05	0,330±0,010*/§ p<0,05	0,330±0,010*/§ p<0,05	0,340±0,020* p<0,05
	UN+MS+quertine	0.460±0.040*/§	0.330±0.010* p<0.05	0.270±0.01§/+ p<0.05	0.230±0.01§/+ p<0.05	0.300±0.01§ p<0.05
POM-430, c.u..	Healthy individuals	0.150±0.003				
	Control	0.200±0.005*	0.180±0.007 p>0.05	0.220±0.016* p>0.05	0.220±0.005* p>0.05	0.250±0.018 p<0.05
	UN+MS	0.230±0.015*	0.220±0.009*/§ p>0.05	0.210±0.009* p>0.05	0.188±0.005 p<0.05	0.180±0.014§ p<0.05
	UN+MS+quertine	0.260±0.022*/§	0.200±0.007* p<0.05	0.180±0.006§ p<0.05	0.150±0.008§/+ p<0.05	0.160±0.06§ p<0.05
POM-530, c.u.	Healthy individuals	0.060±0.001				
	Control	0.050±0.004	0.048±0.002 p>0.05	0.051±0.003 p>0.05	0.051±0.001 p>0.05	0.052±0.001 p>0.05
	UN+MS	0.062±0.004	0.056±0.002 p>0.05	0.059±0.003 p>0.05	0.055±0.003 p>0.05	0.055±0.007 p>0.05
	UN+MS+quertine	0.065±0.006	0.064±0.006§ p>0.05	0.063±0.003§ p>0.05	0.060±0.003§/+ p>0.05	0.061±0.007§/+ p>0.05

*Note: * – reliability in relation to a group of healthy people; § – reliability in relation to the group of patients with urate nephrolithiasis; + – reliability in relation to the group of patients with urate nephrolithiasis comorbid with metabolic syndrome on the background of quertine intake; p<0.05 - reliability in relation to treatment and in the process of observation during the treatment of patients.*

POM-370, increased moderately in patients with urate nephrolithiasis before treatment. A significant increase in these indicators occurred in groups of patients before treatment for urate nephrolithiasis comorbid with MS. The level of alkaline aldehyde derivatives POM-430 also increased moderately in patients of the control group and significantly in patients of the main and experimental groups, which indicates the activation of PPO in patients with comorbidity to MS. Thus, the violation of metabolic processes in patients with urate nephrolithiasis contributed to POM activation.

Activation of oxidative stress in patients with urate nephrolithiasis, along with DNA degradation and progression of PPO processes, causes fibroblast damage, stimulates thromboxane formation, reduces local defense activity, increases epithelial and endothelial permeability, increases mucus secretion. Such mechanisms are the basis of oxidative stress processes and are one of the links in the pathogenesis of urate nephrolithiasis, which is caused by oxidative damage, inhibition of membrane enzyme activity, deepening changes in physicochemical properties of proteins of the plasma membrane.

In the process of monitoring a group of patients with urate nephrolithiasis, a moderate increase in the level of POM-356 was observed after 1.5-2 months. A pronounced increase in POM-356, POM-370, POM-430 was noticed after 3-6 months. Treatment of patients with traditional therapy and drugs that improve metabolic processes (experimental group) was accompanied by a moderate decrease in PPOindicators after 1.5-2 months and after 3-6 months of treatment. In patients receiving quertinecomplementary to the main therapy, there was a significant decrease in the level of POM-356 to 0.22 ± 0.02 c.u., the level of POM-370 to 0.30 ± 0.01 c.u., the content of POM- 430 to 0.16 ± 0.006 units, which was close to that of healthy individuals.

Positive results of treatment in patients with urate nephrolithiasis comorbid with MS (experimental group) were achieved due to complex treatment with diet, water regimen and pharmacotherapeutic agents Uralit-U, allopurinol, atorvastatin, metformin, liprazidum, vitamin B_6, MgO.

In patients with urate nephrolithiasis comorbid with MS after treatment with quertine, an improvement in general condition and increased vitality were observed. In most patients, lumbar pain, headache, tinnitus, fainting decreased, and blood pressure returned to normal.

The experiments are consistent with other studies that demonstrate how changes in renal function are associated with the processes of LPO and AOS in patients with urate nephrolithiasis [7]. Disorders of purine, carbohydrate and lipid metabolism exacerbate disorders of the functional state of the kidneys and the processes of free radical oxidation of lipids [7].

Primarily,quercetin is a scavenger of free radicals and has the ability to activate the body's own antioxidant enzymes. It has an anti-inflammatory effect due to the blockade of the lipoxygenase pathway of arachidonic acid metabolism, decreased synthesis of leukotrienes, serotonin and other mediators of inflammation. Quercetin increases the activity of phagocytes, T- and B-lymphocytes and antibody production, thus reducing the manifestations of secondary immunodeficiency [5].

Having a powerful antioxidant effect based on the neutralization of free radicals, the drug "Quercetin" leads to the stabilization of cell membranes, which has a pronounced activating effect on the enzyme system of the body's own antioxidant defense system [5]. In addition, the drug prevented the increase of LPO in the mitochondria of the heart, as evidenced by a decrease in TBA-reactive substances and anincrease in the rate of reduced glutathione [6].

Conclusions

1. In patients with urate nephrolithiasis comorbid with MS, the development of OS was observed, which was manifested by the accumulation of the content of POM products.

2. The use of quertine in combination with traditional therapy and drugs that affect metabolic disorders, as well as differentiated uricostatic and uricolytic drugs, contributed to the normalization of the content of POM products.

3. Further studies of the complex treatment of patients with urate nephrolithiasis comorbid with MS with quertine, traditional drugs, conventional drugs that correct metabolic disorders, differentiated use of uricolytic and uricostatic therapy, based on the results obtained, will allow in the future to correct POM states, AOS and LPO processes, as well as indicators of the functional state of the kidneys, carbohydrate, lipid, purine and electrolyte metabolism, and to improve patients'health and quality of life.

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CORRECTIONOFPROTEIN OXIDATIVE MODIFICATION WITH QUERTINE IN PATIENTS WITH URONEPHROLITHIASIS COMORBID WITH METABOLIC SYNDROME

Key words: quertine, urate nephrolithiasis, metabolic syndrome, protein oxidative modification.

The aim of the research was to study the effect of quertine on the processes of protein oxidative modification in patients with urate nephrolithiasis comorbid with metabolic syndrome.

118 patients, divided into three groups, were examined and treated. Group 1 (control) included patients with urate nephrolithiasis, who were prescribed traditional therapy. Group 2 (experimental) embraced patients with urate nephrolithiasis comorbid with metabolic syndrome, who were prescribed traditional therapy and generally recognized drugs that correct metabolic disorders. Group 3 (main) involved patients with urate nephrolithiasis comorbid with metabolic syndrome, who received traditional therapy and drugs that correct metabolic disorders. To study the state of protein peroxidation, the indicators of protein oxidative modifications were analysedwith the method designed by E.E. Dubininaet al. The study of indicators in the blood serum was performed before treatment and after 7 days, after 14 days, after 1.5-2 months, after 3-6 months.

In patients with urate nephrolithiasis, prooxidant activation was observed in the course of traditional therapy: the level of neutral aldehyde and ketone derivatives as well as of alkaline aldehyde derivatives increased. Patients with urate nephrolithiasis and urate nephrolithiasis comorbid with metabolic syndrome demonstrated the development of oxidative stress, manifested by the accumulation of the content of products of protein oxidative modification. After the treatment with quertine, which contains the aglycone of various plant flavonoid glycosides, a more significant decrease in the indicators of protein oxidative modification was noted comparing to experimental group patients; this indicates the antioxidant properties of quertine.

Thus, the use of quertine together with traditional therapy and drugs that affect metabolic disorders, differentiated between uricostatic and uricolytic agents, contributed to the normalization of the content of protein peroxidation products.

Conflict of interests. The authors declare no conflict of interests.

С.І. Білай

КОРЕКЦІЯ КВЕРТИНОМ ОКИСНОЇ МОДИФІКАЦІЇ БІЛКІВ У ХВОРИХ НА УРАТНИЙ НЕФРОЛІТІАЗ КОМОРБІДНИЙ З МЕТАБОЛІЧНИМ СИНДРОМОМ

Ключові слова: квертин, уратний нефролітіаз, метаболічний синдром, окисна модифікація білків.

Метою дослідження було вивчення впливу квертину на процеси окисної модифікації білків у хворих на уратний нефролітіаз коморбідний з метаболічним синдромом.

Обстежено та проведено лікування 118 пацієнтів, які були розділені на три групи. 1-а група (контрольна) – хворі на уратний нефролітіаз, яким застосовували традиційну терапію. 2-а група (порівняння) – хворі на уратний нефролітіаз коморбідний з метаболічним синдромом, яким застосовували традиційну терапію та загальноприйняті лікарські засоби, які коригують метаболічні порушення. 3-я група (основна) – хворі на уратний нефролітіаз коморбідний з метаболічним синдромом, яким застосовували квертин, традиційну терапію та лікарські засоби, які корегують метаболічні порушення. Для вивчення стану перекисного окиснення білків досліджували показники окисної модифікації білків по методиці Є.Є. Дубініної та співавторів. Дослідження показників дифенілгідрозоніву сироватці крові супроводжувалося: до лікування, через 7 днів, через 14 днів, через 1,5-2 місяців, через 3-6 місяців. У хворих на уратний нефролітіаз протягом лікування традиційною терапією спостерігалася прооксидантна активація – підвищувався рівень альдегідних, кетонових похідних нейтрального характеру, альдегідопохідних лужного характеру.

У хворих на уратний нефролітіаз коморбідний з метаболіч-

ним синдромом до лікування спостерігався істотний розвиток оксидативного стресу, що проявлялося накопиченням вмісту продуктів окисної модифікації білків. Після проведеного лікування квертином, який містить аглікон багатьох рослинних флавоноїдних глікозидів, було відзначено більш суттєве зниження показників окисної модифікації білків, ніж у хворих групи порівняння, що вказує на антиоксидантні властивості квертину.

Таким чином, застосування квертину спільно з традиційною терапією та лікарськими засобами, які впливають на метаболічні порушення, диференційовано урикостатичних та уриколітичних засобів, сприяло нормалізації вмісту продуктів перекисного окиснення білків.

С.И. Белай

КОРРЕКЦИЯ КВЕРТИНОМ ОКИСЛИТЕЛЬНОЙ МОДИФИКАЦИИ БЕЛКОВ У БОЛЬНЫХ УРАТНЫМ НЕФРОЛИТИАЗОМ КОМОРБИДНЫМ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ

Ключевые слова: квертин, уратный нефролитиаз, метаболический синдром, окислительная модификация белков.

Целью исследования было изучение влияния квертина на процессы окислительной модификации белков у больных с уратным нефролитиазом коморбидным с метаболическим синдромом.

Обследовано и проведено лечение 118 пациентов, которые были разделены на три группы. 1-я группа (контрольная) – больные с уратным нефролитиазом, которым назначали традиционную терапию. 2-я группа (сравнения) – больные с уратным нефролитиазом коморбидным с метаболическим синдромом, которым назначали традиционную терапию и общепризнанные лекарственные средства, которые корректируют метаболические нарушения. 3-я группа (основная) – больные с уратным нефролитиазом коморбидным с метаболическим синдромом, которым применяли, традиционную терапию и лекарственные средства, которые корректируют метаболические нарушения. Для изучения состояния перекисного окисления белков исследовали показатели окислительной модификации белков по методике Е.Е. Дубининой и соавторов. Исследование показателей дифенилгидрозонов в сыворотке крови сопровождалось: до лечения, через 7 дней, через 14 дней, через 1,5-2 месяцев, через 3-6 месяцев. У больных с уратным нефролитиазом на протяжении лечения традиционной терапией наблюдалась прооксидантная активация – повышался уровень альдегидных, кетоновых производных нейтрального характера, альдегидопроизводных основного характера.

У больных с уратным нефролитиазом и с уратным нефролитиазом коморбидным с метаболическим синдромом наблюдалось развитие оксидативного стресса, что проявлялось накоплением содержания продуктов окислительной модификации белков. После проведенного лечения квертином, который содержит агликон многих растительных флавоноидных гликозидов, было отмечено более существенное снижение показателей окислительной модификации белков, чем у больных группы сравнения, что указывает на антиоксидантные свойства квертина.

Таким образом, применение квертина совместно с традиционной терапией и лекарственными средствами, которые влияют на метаболические нарушения, дифференцировано урикостатических и уриколитических средств, способствовало нормализации содержания продуктов перекисного окисления белков.

Автор заявляє про відсутність конфлікту інтересів.

Внесок автора:

Білай С.І. – ідея, дизайн, набір клінічного матеріалу, статистичне опрацювання, аналіз літератури і клінічного матеріалу, висновки, анотація, корекція статті.

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ГЕПАТОПРОТЕКТОРНА ЕФЕКТИВНІСТЬ СПОЖИВАННЯ МАКУХИ З НАСІННЯ ВИСОКООЛЕЇНОВОГО СОНЯШНИКА ЩУРАМИ З ЕКСПЕРИМЕНТАЛЬНИМ ДИСБІОЗОМ

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Вступ

Печінка відіграє центральну роль у взаємодії макроорганізму з ендогенною мікробіотою [1]. Антимікробна функція печінки забезпечує захист усіх систем тканин і органів організму від дії бактерій та їх токсинів [2]. Водночас наявність кишкового дисбактеріозу, який обумовлює високу токсичну дію на печінку, в основному, кишко-