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FEATURES OF PURINE METABOLISM AND MICROALBUMINURIA OF ISCHEMIC HEART DISEASE IN PATIENTS WITH METABOLIC SYNDROME

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Abstract:
To study the state of purine metabolism and microalbuminuria in patientswith metabolic syndrome.

Keywords: Metabolism And Microalbuminuria, Metabolic syndrome

INTRODUCTION: Metabolic syndrome (MS) accelerates the development and progression of vascular pathology and increases the risk of developing cardiovascular complications (CVD). The term "metabolic syndrome" (MS) is widely used in the modern literature. Its prevalence among the general population reaches 15% [1], and among the population aged 40-60 years, signs of MS are already observed in 20-25% of cases [2]. The basis of MS is hyperinsulinemia, which in turn leads to the development of dyslipidemia, the formation of arterial hypertension and obesity [3]. Metabolic syndrome is a combination of risk factors for the development of type 2 diabetes mellitus and cardiovascular diseases (CVD), which include abdominal-visceral obesity, dyslipoproteinemia, hypertension, insulin resistance with compensatory hyperinsulinemia, as well as hyperuricemia, changes in the pre-thrombotic homeostasis system [4, 5]. Vascular pathology manifests itself micro- and macrovascular disorders. The pathophysiological basis of macrovascular complications is atherosclerosis [1]. This is one of the most widespread diseases in the world, responsible for ischemic CVD, with high morbidity and mortality, which has been developing asymptomatically for many years [2]. MS is a complex of interrelateddisorders of carbohydrateand purine metabolism, as well as mechanisms of regulationof arterialpressure and endothelial reticule. The development of these disorders is based on a decrease in the sensitivity of tissues to insulin – insulin resistance (IR) [9]. Glucose and insulin are important factors in uricacid homeostasis,contributing to its secretion and reabsorption. An imbalance of theseindicators leads to either hypouricemia or hyperuricemia. Thus,-hyperemiacontributes to uricosuria, so the level of uric acid in the blood of patients with decompensated diabetes mellitus of any type maydecrease.The effects of insulin onuric acid extraction are oppositeфектам глюто those of glucose. At the same time, normal insulin levels have almostno effect on renal hemodynamics, glomerularfiltration, and thepermeability of the renal filter to albumin [4,8]. Therefore, hyperuricemia (HUK) and microalbuminuria (MAU) are closely interrelated processes that characterize the clinical manifestation of MS. However, studies of purine metabolism and microalbuminuriain MS patients are insufficient and this problem needs further comprehensive research.

OBJECTIVE: To study the state of purine metabolism and microalbuminuria in patientswith metabolic syndrome.

MATERIALS AND METHODS. Weexamined 62 patientsaged 29-59 years with MS, taking into account risk factorsand target organ damage. В условиях стационара обследованы 24 male (34.7%) and 38 female (65.3%) patientsaged from 30 to 55 years were examined in a hospital setting, who were randomly assigned to the following 3 groups: I (control) – healthy individuals aged 25-40 years – 15 people; II - patients with arterial hypertension-18 people in 30-59; III the age group Group III – patients with MS-32 aged 30-59 years. To determine metabolic disorders in patients, the level of total cholesterol (CH), triglycerides, very low-density lipoproteins (VLDL), LDL, high-density lipoproteins (HDL), and the coefficient of atherogenicity were studied (the lipid spectrum was determined biochemically by the "Reflotron-Roche"express analyzer). The state of purine metabolism was determined enzymatically bythe colorimetric method from the level of uric acid in blood serum onan automatic analyzer Stat Fax Awareness technology INC (Italy), using Hospitex diagnostics s.r.l reagents. The results of clinical studies were processed using statistical processing applications of the Excel program, as well as by the method of variational statistics using tables Student's t-criteria. Differences between the arithmetic mean values were considered statistically significant at $p<0.05$.

RESULTS AND DISCUSSION. What additional risk factors for the development of cardiovascular morbidity and mortality are associated with kidney pathology?

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They can be grouped into several groups. Factors associated with increased permeability of the renal membranes (MAU and PU). Activation of the renal renin-angiotensin system (RAS) (hypersecretion of angiotensin II). Renal hypertension. Renal anemia associated with reduced erythropoietin synthesis. Accumulation of toxic metabolites and uremic toxins due to their reduced renal clearance.

In most MS patients, the disease was associated with a hereditary factor (31.5%), obesity (30.0%), alimentary factor (28.4%), and low physical activity (inactivity – 10.1%). In the alimentary factor group, patients indicateexcessive consumption of carbohydrates and fats. Overweight and obesity are considered the main components of MS. However, the relationship between MS components is of particular interest. The больных Quetelet index (IC), a body mass index, and the degree of abdominal obesity (AO)were determined in the examined patients. Measurement of the waist circumference in Igroup I showed 78.8±1.14 cm, in IIgroup II-80.3±0.46, and in C MC-102.5+±1.5 cm (Table -11). In patients with AH, AO was 1.9% higher than in the control group, i.e. the parameters were almost identical. When examining the IR in the control group, this indicator showed 24.3±0.77 м^{m2}, and in IIthe second group, the IR was equal to 26.7±1.3,3м^{m2}. In the GB group, IR was higher by 4.9%, the indicators almost did not differ. In MS, the IC averaged 32.6±0.88 м^{m2}, was 35% higher than in the control groupII, and 28.6% higher than in the second group. The results suggest that blood pressure and glycaemia levels are related to body weight. Purine metabolism was evaluated based on the determination of uric acid concentration in fasting venous blood plasma samples. Hyperuricemia MC levels above 0.45 mmol / L weredetected in 52.6% of patients with MS and 37.1% of patients with AH. This indicator is the earliest marker of kidney damage in DM and represents highly selective urinary albumin excretion in the range from 30 to 300 mg / day, which is not detected by routine urine testing methods. Currently, MAU is a generally recognized marker of not only kidney tissue damage, but also a risk factor for the development of cardiovascular diseases. For a more in-depth analysis of the relationship between the level of uricemia and otherMS parameters, we divided all the examined individuals according to the resultsof the study into 3 clinical groups.

Asthe clinical picture of the syndrome grew, the prevalence of hyperuricemia also increased: in the AH group in 22.2% of cases and in the MS group in 50.7% of cases. The table shows the average values of purine metabolism indicators, as wellas other parameters under study that reflect the severity of disorderscharacteristic of MS. A significant increase in the degree of uricemia occurred in the MS groups, i.e. at the stages when there was a statistically significant increase in the concentration of TG and parameters of obesity. Although the deterioration of diastolic function correlated with an increase in the degree of hyperuricemia, the change in this parameter in patients with MS acquired a significant (comparedto patients with the absence of the mentioned syndrome)character much earlier than in them the concentration of significantly increased. We did not find a reliable relationship between the uricemia value and the blood pressure level, but in patients with the metabolic syndrome clinic without hypertension, the level was statistically significantly lower than in patients with MS, andthere was a tendency for thisparameter to be lower compared to all groups of patients with MS with hypertension. individual distribution of MC concentration values among individuals of all clinical groups, we concluded that the level of uricemia characteristicof MS is the index of 0.45 mmol/l and higher. Patients withшие MS had this level of significantly moreoften than those with hypertension ($p<0.05$).

Table -11
Indicators of purine metabolism, blood pressure, fat and carbohydrate metabolism in patients with varying degrees of MS severity (M ± m)

Indicator	MS indicator		
	I group	II group	III group
Age, years	44,6±1,22	46,5±1,6	50,2±2,2*
MK, mmol / l	0,337±1,77	0,59±2,0*	0,71±2,0*
waist circumference	78,8±1,14	80,3±0,46, **	102,5+±1,5 ***
Quetelet Index, kg / sq. m	24,3±0,7	26,77± 1,3**	32,66± 0,8***

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SAD, mmHg.	125,66±1,8,8*	150,0±2,9	152,4±5,0
DBP, mmHg.	85,2,2±2,22	101,2±2,1	100,7±1,7

Note: * p<0.05, * * p<0.01, * * * p<0.001

In our study, hyperuricemia was detected in 52.6%of patients with MS, which is slightly higher than the data of other authors. However, the frequency of purine metabolism disorders depended on the presence of concomitantcomponents of MS: in its absence, it was only 22.2%, increasedas the clinical picture of the syndrome progressed, and reachedгала максиthe maximum of 68.6% in patients with MS. In addition, we noted that the concentration of in the blood significantly correlated withthe severity of obesity, hyperinsulinemia, triglyceridemia, and glycemia -parameters that reflect the state of IR. Таким обThus, the data obtained indicate that hyperuricemiais ametabolic disorder and one of the components that contribute to themetabolic syndrome.

We examined MS patients for the presence of MAU. Patientswere divided into groups. The criteria for the formation of groups were the stagesof diabetic nephropathy: group 1 – patients with normoalbuminuria:urinary albumin excretion below 30 mg/day; group 2 - patients with MAU: urinaryalbumin excretion from 30-300 mg / day; group 3 - patients with proteinuria (PU), detected in the presence of the study of daily protein excretion in the urine and withpreserved renal nitrogen-releasing function (serum creatinine levelbelow 110 mmol / l). The results of the study showed that MAU was expressed in 22.44% of cases in AH patients, and in 75.22% of cases in MS patients. In thepresence of MS, non-selective proteinuria is observed in 80.1,1%of cases. The degree of MAU and PU directly correlated with the degree of DN: in the initialstage of DN, MAU is determined at the level of microalbuminuria (<30 mg / day), in IIstage II DN, MAU is determined from 30-300 mg/day, and in degree III – IV DN, PU is determined. The degree of PU severity is directly proportional to the degree of DN. In IIIstage III DN, PU was 1.47±0.7 g / day, IV and 2.7±1.9 g/day in stage IV DN.

Thus, the presence of normo albuminuriain MS patients indicates an adaptive-compensatory vascular response aimed atovercoming the developing kidney pathology. The presence of MAU means that the stage of MAU can be reversible with timely initiation of treatment and will slow down the progression of DN and its transition to the stage of PU and CRF.

Most cases of MS occur against the background of long-term coexistence of risk factors, which include an increase in TG, LDL cholesterol, and a decrease in HDL levels in blood plasma [2,10]. There are also studies that emphasize that these parameters cannot fully explain the variability of the clinical course of MS.

As can be seen from Table 2, the maximum level of total cholesterol, triglycerides, LDL is observed in III group III, compared with the control and II groups. In comparison with the control group, the total cholesterol level in patients with hypertension increased by 30.4%, and in those with MS - by 47.8%. The triglyceride content in III group III exceeded the control value by 71%, in II group II by 44.4%. The LDL level in II group II exceeded the control group by 53.8%, the LDL content in III group III increased by 99.7% compared to the healthy group. HDL in groups II and III was reduced compared to the control group. When comparing the first and second groups, the difference in blood glucose level was 8.8%, and in groups I and III-46.6%. When comparing the first and second groups, the difference in blood glucose level was 7.1%, and in groups I and III – 47.6%.

Table 2.
Content of lipids and glucose in blood serum in practically healthy patients with arterial hypertension and metabolic syndromeдромом

Groups	Total CHOLESTEROL, mmol/l	Triglycerides, mmol / l	LDL, mmol/L	HDL, mmol / l	VLDL, mmol/l	Atherogen index, units	Glucose Plasma glucose, mmol / l
I Group I	4,6±0,1	1,5±0,1	2,6±0,2	1,4±0,1	0,4±0,1	2,8±0,3	4.5±0.2
II Group II	6,0±0,2	1,8±0,2	4,0±0,2	1,2±0,3	0,5±0,2	4,0±0,2	4,9±0,2
III Group III	6,8±0,3	2,6±0,1	5,2±0,3	0,9±0,4	0,7±0,3	5,2±0,2	6,6±0,3
P 1-2	P<0.001	P<0.05	P<0.001	P<0.05	P<0.05	P<0.01	P<0.05
P 1-3	P<0.001	P<0.001	P<0.001	P<0.05	P<0.05	P<0.001	P < 0.001
P 2-3	P<0.05	P<0.001	P<0.01	P<0.05	P<0.05	P<0.001	P<0.01

According to some authors, it is difficult to separate MS from hyperuricemia, as well as to determineлить при-

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cause-and-effect relationships, because, accordingto modern concepts of the pathogenesis of MS, these conditions mutually induce the occurrence and consolidationof each other. Hyperuricemia is detected in 25%of MS patients. The importance of the relationship between hyperuricemia and the development of MS, atherosclerosisand CHD is shown by the relationship of hyperuricemia as a factor.

CONCLUSION. Today, we are faced with the fact that the number of patients with cardiovascular diseases is steadily increasing, and complications associated with them still occupy the first place among the main causes of death and disability.

Таким обThus, the data obtained indicate that hyperuricemiais a metabolic disorder and one of the components that contribute to themetabolic syndrome. The severity of HY is directly proportional to the increasein the clinical picture of MS. In MS patients, the presence of normoalbuminuria indicates an adaptive-compensatory vascular response aimed atovercoming the developing kidney pathology. The presence of MAU means that the stage of MAU can be reversible with timely initiation of treatment and will slow down the progression of DN and its transition to the stage of PU and CRF. The presence of MAU indicates glomerular hypertension and a decrease in glomerular filtration. Today, MS is considered as one of the fundamental causes of CVD development. Recent decades have seen a significant increase in MS cases worldwide. Risk factors and pathogenesis of CHD, optimal methods for diagnosing MS, and the need to individualize approaches to its correction, which meets the main goals of CHD prevention, are widely discussed.

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