## PURINE METABOLISM IN BLOOD OF PATIENTS WITH CHRONIC PYELONEPHRITIS ASSOCIATED WITH ARTERIAL HYPERTENSION

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Changes in concentration purine metabolites in blood plasma of patients are investigated at arterial hypertensia (AH), chronic pyelonephritis (ChP) and a pyelonephritis associated with arterial hypertensia (ChP+AH). The concentration of uric acid in blood plasma of patients with ChP and isolated AH is higher than a control parameter of 25 % and 42 % accordingly.

The expressed increase in concentration of adenine is established both at arterial hypertensia and at chronic pyelonephritis. There is fixed authentic growth of xantine and hypoxantine in blood plasma of patients with AH and ChP+AH. It can be the precondition of damage of endothelium and of development of AH.

**Keywords:** chronic pyelonephritis, arterial hypertensia, purine metabolism an exchange

Now one of the most significant directions of medical and biologic researches is studying of the role of extracellular products of purine metabolism in maintenance of normal ability to live of an organism and in development of pathological conditions. For example, the increase in concentration of uric acid in blood is considered as one of steps of atherosclerosis and diabetes mellitus pathogenesis [1, 2, 3, 4]. There are data on that superfluous accumulation of adenine nucleotides in plasma induces change of platelets functions in blood of patients with a diabetes Extracellular purine act as inflammatory or anti-inflammatory mediators. Increase in level of adenosine in blood plasma of spontaneously hypertensive rats has allowed to assume its participation in mechanisms of development of an arterial hypertensia [6]. The opinion about adenosine vasa-construction influence on renal microcirculation and about its probable synergism with renin-dependent mechanisms of regulation of arterial pressure is stated [7, 8].

In this connection is especially interesting to study the condition of contents of purine and their metabolites in blood of pa-

tients with a chronic pyelonephritis and secondary arterial hypertensia.

## Patients and methods

Patients have been included in 3 groups. The first group included 23 patients with chronic pyelonephritis (ChP). The second group was made by 15 patients with the arterial hypertensia (AH) which has been not connected to a pathology of kidneys. 10 patients with an arterial hypertensia and chronic pyelonephritis (AH+ChP) were included in third group. As the control blood of clinically healthy 15 donors was investigated.

All patients were inspected physically and by tools that were based on routine (interview, objective research, including palpation, percussion, auscultation of internal organs) and additional (measurement of blood arterial pressure (BP), electrocardiogram (ECG), special probes of urine by Nechiporenko and Zimnitskiy) methods. Arterial pressure was defined on Korotkov's standard method. The arterial hypertensia was diagnosed as on the basis of results of measurement of BP during surveys, and also on the basis of studying the anamnesis and official medical documents (epicrisis, reports of inspection, including daily monitoring the BP).

Authenticity of a self-estimation and self-checking of patients with constant AH of 2-3 degrees was studied also.

The arterial hypertensia was classified on degrees according to criteria of experts of World Health Organization (WHO). The surveyed patients had 2-d (160-180 mm Hg of systolic BP and 90-105 mm Hg of diastolic BP) or 3-d (over 180 mm Hg of systolic BP and is higher than 105 mm Hg of diastolic BP) degrees AH.

Stratification of risks of vascular events was carried out according to recommendations of the NY Heart association (NYHA), maintained the WHO. Groups of risk to which the surveyed patients concerned, were identified as 3-d (86% of patients) and 4-d (14% of patients). Obligatory researches were the general analyses of blood and urine, biochemical tests for activity of transaminases, levels of blood plasma creatinine and urea. Estimation of speed glomerular filtrations and tubular reabsorbtion were carried out by method Reberg-Tareyev. Also the approaches that had been suggested of *Cocroft-Gault* were used

Diagnostics of a chronic pyelonephritis was carried out on the basis of the criteria offered by O.L. Tiktinskiy (1990), T.Z. Sejsembekov (1984,1995), I.A. Borisov (1997).

The maintenance of purine metabolites in blood plasma was defined on I.V.Madjanov method [9]. A level of a uric acid was defined with the help of sets of firm Lahema.

Results processed a method of variational statistics with use of Student's criterion.

## Results and their discussion

It is established, that in blood plasma of patients with arterial hypertensia at absence of kidney pathology the guanine level exceeded control on 42% (p <0,05) whereas the adenine concentration was higher than the control over 91% (p < 0,001).

The levels of hypoxantine and xantine in blood plasma of patients with AH was

higher than at healthy persons 42% and 38% accordingly (p <0,05).

At patients with a chronic pyelonephritis the same parameters of guanine and adenine were higher than the control accordingly 21% and 42% (p <0,05). At the same time levels hypoxantine and xantine not so considerably exceeded control parameters - on the average on 20% - if to compare their ratio in the first and control groups.

Guanine and adenine plasma concentration at pyelonephritis associated with arterial hypertensia was higher than the control over 30% (p <0,05) and 36% (p <0,05). Levels of hypoxantine and xantine were higher than control parameters 33,3% and 28% accordingly (p <0,05).

The concentration of uric acid in plasma of blood of patients with ChP was higher, than in control group on 25%. At patients with arterial hypertensia this parameter exceeded norm on 42% (p <0.05). At ChP in combination to arterial hypertensia concentration of uric acid insignificantly exceeded control grout level.

The phenomenon of more expressed increase in concentration of adenine in patients' blood both is established by arterial hypertensia, and chronic pyelonephritis. At ChP, associated with an arterial hypertensia, the degree of increase of adenine and guanine concentration is comparable with each other. In blood plasma of patients with AH and ChP+AH authentic growth of hypoxantine and xantine is fixed, that has negative value owing to vessel consrictive effect of these metabolites and abilities to damage endothelial barrier [10]. In this context the big interest causes the found out trend of increase xantine and hypoxantine concentration in blood plasma of ChP- patients. Obviously, it is necessary to consider this fact as the precondition of damage of epithelial tissue and development of AH. As xantine is a substratum for xantinaxidase so increase in its concentration is possible to serve as the indicator of growing generation of superoxidanions which are capable to induce microvascular dysfunction and development of oxidizing stress.

The increase in uric acid in blood plasma is considered as an independent risk factor of development of kidney dysfunctions at patients with arterial hypertensia [11, 12]. It is shown, that at reduction in initially raised level of uric acid in blood plasma of patients with kidney dysfunction, the control of arterial pressure improves and rates of progressing of kidneys illness are slowed down [13]. On the other hand, the uric acid, being one of low-molecular non-enzyme antioxidants of blood plasma, can render protective effect, limiting development of the oxidizing stress caused by superfluous generation of superoxidanions [14].

Such ambiguity of effects of uric acid demands carrying out of the further researches role of purine metabolites in development of defeats of kidneys and arterial dysfunction.

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