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## **STUDY OF GLUTAMATE AND ASPARTATE IN BLOOD PLASMA OF PSORIATSC PATIENTS**

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Introduction. Recently it has been prevailing view that psoriasis is a systemic disorder of multifactorial etiology with genetic and exogenous factors involvement, that results in escalation of functional disorders, metabolic violation and launching of the mechanisms of pathological process formation [1-3]. Despite fundamental studies of psoriasis, its many aspects remain poorly understood, especially regarding its combination with somatic diseases. These aspects include issues of cooperative interaction and the role of integrative systems of the body – neurological, endocrine and immunological [4-6]. Amino acids glutamate and aspartate are widespread excitatory CNS neurotransmitters and play important role in homeostasis providing [7, 8].

The goal of this study was to study the level of glycine, glutamate, and aspartate in blood plasma of the patients suffering from isolated psoriasis and psoriasis combined with hypertension depending on severity of disease.

The object and methods of the study. The study was conducted on two groups of patients, age range from 40 to 65 having confirmed diagnosis of psoriasis, that were examined and treated in an outpatient dermatologic city clinic № 5 in Kharkiv (Ukraine). The first group consisted of 74 patients with isolated psoriasis, 40 of which had mild course of disease, 24 – moderate, and 10 – severe course. The second group consisted of 48 patients with psoriasis combined with hypertension, 22 of which had mild course of disease, 16 - moderate and 10 - severe course. Precise anamnesis and laboratory examination were conducted, that consists of general clinical and biochemical analysis of the peripheral blood. Control group comprised 30 practically healthy patients.

Blood plasma level of glutamate and aspartate were defined by liquid chromatographic analysis with amino acidic analyzer AAA-339 (Czech Republic). For calibration tests and quantitative evaluation of chromatographs there were used standard technical solutions of amino acids (the firm "Lachema"), that accompanied the reagent kit of amino acid analyzer. For processing and analysis of statistical information a computer kit Statistica 6.0 was used for mathematical analysis of the obtained numeral material.

Results of the study and its discussion. During the conducted study there were determined changes of the level of excitatory amino acids in blood plasma (Table 1). There was statistically reliable increase of the level of glutamate and aspartate in patients with isolated psoriasis of moderate and severe course in comparison to the control group.

Table 1 The level of glutamate and aspartate in blood plasma of patients with psoriasis depending on its severity (M m).

| Indicator | Control group | Severity of disease |                |                |
|-----------|---------------|---------------------|----------------|----------------|
|           |               | mild                | moderate       | severe         |
|           |               | isolated            | psoriasis      |                |
| glutamate | 21,2 ± 1,9    | 24,3 ± 2,5          | 30,2 ± 3,0*    | 37,3 ± 3,6*    |
| aspartate | 5,23 ± 0,50   | 5,92 ± 0,53         | 6,36 ± 0,60*   | 8,23 ± 0,77*   |
|           |               | psoriasis           | combined with  | hypertension   |
| glutamate | 21,2 ± 1,9    | x32,4 ± 3,0*        | x40,2 ± 3,7*   | x48,7 ± 4,6*   |
| aspartate | 5,23 ± 0,50   | 6,86 ± 0,63*        | x10,95 ± 0,97* | x15,21 ± 1,34* |

Note: unit of measure is mmol/l; \* - reliability in comparison to the control group ( $p < 0,05$ ); x – reliability in comparison to isolated disease ( $p < 0,05$ ).

The raise of the level of glutamate was 42% and 76 % respectively, of aspartate – 22% and 57%. In case of isolated psoriasis of mild severity these indicators practically did not differ from the control group. In case of psoriasis combined with hypertension there was significant raise of the level of glutamate and aspartate, 53% and 31% in the group of mild severity, 90% and 109% in the group of moderate severity, and 130% and 191% in the group of severe course.

It should be noted that the level of excitatory amino acids was statistically higher in patients with moderate and severe course of psoriasis combined with hypertension, in comparison to patients with isolated psoriasis. Such results indicate that psoriasis, especially combined with hypertension, is accompanied by the release of excitatory mediators. As a consequence hyper stimulation of NMDA-receptors of N-metil-D-aspartate develops, that provokes dilatation of Ca-channels, massive entrance of Ca into the cell with consequent activation of proteases and phospholipase. Hyper enzymatic activity leads to interruption of integrity of the cell membrane and its organelles, first of all the internal membrane of mitochondria that significantly deepens energetic disturbances. Glutamate receptor activation leads to synthesis of free radicals by activation of Ca-dependent arachidonic acid cascade, nitric oxide synthesis [7]. As previous studies have showed, psoriasis is accompanied by activation of glutamate receptors, that is one of launching mechanisms of free radical generation [9].

Conclusions. Severe course of psoriasis, especially combined with hypertension is characterized by excitatory and inhibitory mechanisms imbalance with signs of increased release of excitatory amino acids, that determines the severity of psoriasis in this contingent of patients. Psoriasis accompanied by Glutamate receptor activation, than is one of starters of free radical generation. Changes of the level of excitatory amino acids in blood plasma of psoriatic patients indicate their obvious participation in formation of cerebral circulation disturbance and autonomic regulation of peripheral vessels disorder. Biochemical monitoring of the level of neuro active amino acids in blood plasma of psoriatic patients allows to monitor the effectiveness and validity of treatment.

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## THE SERUM CYTOKINES LEVEL DYNAMICS IN PATIENTS WITH HEART FAILURE

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Introduction. The heart failure (HF) syndrome is characterized by impaired systolic and/or diastolic function and various clinical signs such as fatigue, dyspnea, fluid retention, and cachexia. An inflammatory activation in CHF patients has long been recognized. Indeed, immune mechanisms modulate interstitial fibrosis, cardiomyocyte apoptosis, and hypertrophy, all of which are central processes leading to maladaptive remodeling in response to a variety of stimuli [7].

Several reports have demonstrated enhanced expression and release of inflammatory cytokines, as well as several chemokines in HF patients [8]. Ivabradine is a new therapeutic agent designed to reduce heart rate at rest and during exercise by selective inhibition of a novel receptor (If channel) located on the pacemaker-cell membrane within the sinoatrial node. As such, ivabradine joins a list of rate-limiting medications already available to prescribers for the control of heart rate in coronary artery disease (CAD) and HF with systolic dysfunction [5]. The  $\omega$ -3 polyunsaturated fatty acids (PUFA), such as docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), are known as anti-inflammatory factors, and are used for HF treatment [5]. The data for ivabradine influence for cytokine' cascade are poor; but for PUFA – controversial.