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Biochemical Biomarkers of Oxidative Stress in Patients with Hypothyroidism and Chronic Migrenia

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Abstract In the membranes of erythrocytes in patients with hypothyroidism against the background of chronic migraine, a significant violation of pro- and antioxidant homeostasis is revealed. When studying the indicators of antioxidant protection, their decrease was revealed. At the same time, the content of AOS parameters in the blood plasma in patients with CM and hypothyroidism was noted to decrease in indicators as the duration of headache increased for more than 5 years.

Keywords Peroxide-antioxidant system, Chronic migraine, Hypothyroidism

1. Introduction

The relevance of the problem of patients with hypothyroidism against the background of chronic migraine is determined by the significant frequency of occurrence. It has been proven that hypothyroidism has an adverse effect on the body's systems and is a powerful factor in the activation of oxidative processes, the main manifestation of which is endothelial dysfunction.

The main mechanism of antioxidant protection under natural conditions is the superoxide dismutase (SOD) enzyme, the oxidation capacity of which allows for the inactivation of free radicals at the site of formation, preventing their diffusion. Indeed, the vast number of data shows that SODs are important components of human antioxidant defense. SODs contain metals necessary for their catalytic function - copper and zinc.

Purpose of the research. Identification of the role of biochemical biomarkers of oxidative stress in patients with hypothyroidism and chronic migraine.

2. Materials and Methods of Research

The study included 118 patients with chronic migraine (CM). The diagnosis of migraine was made according to the International Classification of Headaches (ICHD-III).

The patients were divided into 3 groups. The 1st group consisted of 38 patients with chronic headache lasting up to 5 years and hypothyroidism. The 2nd group included 41 patients with CM with a headache duration of more than 5 years and hypothyroidism. The 3rd group consisted of 39 patients with CM without hypothyroidism. A control group was also created, which included 20 practically healthy individuals.

Assessment of the peroxide-antioxidant system in blood serum was carried out by the level of malondialdehyde (MDA), diene conjugates (DK), the activity of superoxide dismutase (SOD), glutathione peroxidase, glutathione reductase, catalase, cytochrome C, and nitrogen oxide (NO), determined by the spectrophotometric method (L.P. Andreeva et al., 1988; Dubinin B.B. et al., 1983).

3. Results and Their Discussion

Table 1. Comparative indicators of LPO in blood plasma in the examined patients

Groups	Gender Indicators	
	MDA nmol/ml	DK µmol/l
Control group n=20	1,2±0,1	1,3±0,01
1st group (n=38) CM up to 5 years old + hypothyroidism	3,12±0,4***	1,89±0,21***
2nd group (n=41) CM more than 5 years + hypothyroidism	4,18±0,8***	2,15±0,01***
3rd group (n=39) CM without hypothyroidism	2,87±0,6***	1,55±0,1***

Note: *P<0,01; **P<0,05; ***P<0,001; – significance of differences with control indicators

The results of the study of lipid peroxidation processes (LPO) in blood plasma in the examined patients showed that MDA indicators in patients of the 1st group were 3.12±0.4 nmol/ml, in patients of the 2nd group 4.18±0.8 nmol/ml, in the 3rd group - 2.87±0.6 nmol/ml, i.e., MDA indicators exceeded the values of the control group in the 1st group by 2.6 times, in the 2nd group by 3.5 times, and in the 3rd group

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by 2.4 times. Diene conjugates (DCs) were 1.89±0.21 μmol/l in the 1st group, 2.15±0.01 μmol/l in the 2nd group, and 1.55±0.1 μmol/l in the 3rd group. DK was 1.45 times higher in the 1st group, 1.65 times higher in the 2nd group, and 1.2 times higher in the 3rd group compared to the control group (Table 1).

As can be seen from the table. 1 Malonic dialdehyde (MDA) is a lipid peroxidation product and is used as a biomarker of oxidative stress. This indicates that the level of oxidative stress in patients of the 2nd group is higher than in patients of the 1st and 3rd groups.

The presence of mitochondrial anomalies in migraine patients has long been known, and the disruption of energy phosphate metabolism has been described in the initial phase of migraine [1]. This leads to anaerobic metabolism, making the cell susceptible to oxidative stress [2].

In addition, other mechanisms play a role in the development of oxidative stress in patients with headaches, namely: the release of pro-inflammatory cytokines during headache [3] formation of nitrogen oxide (NO) in the endothelium and perivascular spaces during a migraine attack [4], which, being an unstable molecule, quickly turns into peroxynitrite NO-mediated release of arachidonic acid metabolites, which can cause oxidative stress and, finally, associated psychological stress, which subtly causes oxidative damage to the body [5].

In the metabolism of each cell, an oxidation reaction occurs. The presence of oxygen in the internal environment is, on the one hand, important for cell functioning; on the other hand, it is a threat that causes oxidative damage due to the formation of free radicals. Superoxide dismutase is the only antioxidant enzyme that removes the superoxide anion, converting this free radical into oxygen and hydrogen peroxide, thereby preventing the formation of peroxynitrite and further damage.

When studying the antioxidant protection indicators, their decrease was revealed. At the same time, the content of blood plasma AOS parameters in patients with CM and hypothyroidism decreased as the duration of headaches increased by more than 5 years (P<0.01).

Oxidative stress is the disruption of prooxidant-antioxidant balance in favor of the former, leading to the accumulation of free radicals and active oxygen forms. The body's antioxidant defense systems include various enzymatic and non-enzymatic mechanisms. The most important antioxidant enzymes are superoxide dismutase, catalase, and glutathione peroxidase.

Superoxide dismutase catalyzes the conversion of the superoxide radical into hydrogen peroxide. Catalase and glutathione peroxidase remove hydrogen peroxide, and glutathione peroxidase can also reduce lipid peroxides. Under normal conditions, there is a balance between the formation and removal of active oxygen. If active forms of oxygen are formed in excess or protective antioxidant mechanisms are ineffective, oxidative stress develops [3]. The most important action of free radicals and active oxygen forms is lipid peroxidation, which causes the destruction and destruction of cell membranes. Disruption of the balance between the formation and removal of active oxygen forms plays a role in the pathogenesis of many diseases.

Our results indicate that a decrease in the activity and level of SOD in patients with CM and hypothyroidism makes them more susceptible to oxidative damage caused by active forms of oxygen (AFC). (Table 2).

In patients of the 1st group, the level of catalase was 39.24±1.09, superoxide dismutase 10.84±0.5, glutathione reductase 1.61±0.4, glutathione peroxidase 1.57±0.5, glutathione transferase 2.28±0.12.

In the 2nd group, these indicators differed in their decrease, i.e., catalase 36.88±1.02, superoxide dismutase 8.28±0.6, glutathione reductase 1.22±0.8, glutathione peroxidase 1.19±0.7, glutathione transferase 1.76±0.26.

In the 3rd group, the AOS indicators were as follows: catalase 41.2±1.81, superoxide dismutase 12.36±0.71, glutathione reductase 1.87±0.03, glutathione peroxidase 1.99±0.06, glutathione transferase 2.88±0.09.

Table 2. Comparative indicators of AOS in patients with CM and hypothyroidism

Groups	Analyzed AOS indicators				
	catalase mc Kat/mg protein	**Superoxide dismutase units/mg protein**	**Glutathione reductase mM/min g protein**	**Glutathione peroxidase mM/min g protein**	**Glutathione transferase mM/min g protein**
Control group n=20	45,7±1,4	14,2±0,6	2,1±0,02	2,2±0,03	3,92±0,16
1st group (n=38) CM up to 5 years +hypothyroidism	39,24±1,09***	10,84±0,5***	1,61±0,4***	1,57±0,5***	2,28±0,12***
2nd group (n=41) CM more than 5 years + hypothyroidism	36,88±1,02***	8,28±0,6***	1,22±0,8***	1,19±0,7***	1,76±0,26***
3rd group (n=39) CM without hypothyroidism	41,2±1,81*	12,36±0,71	1,87±0,03***	1,99±0,06**	2,88±0,09***

Note: *P<0.05; **P<0.01; ***P<0.001- significance of differences with control indicators

In conclusion, it should be noted that AOS indicators were significantly reduced in patients of the 2nd group, which indicates a decrease in the body's antioxidant system and damage to the cell membrane.

4. Conclusions

Our results indicate that a decrease in the activity and level of SOD in patients with CM and hypothyroidism makes them more susceptible to oxidative damage caused by active forms of oxygen.

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