Central Asian Journal of Medicine

FEATURES OF ULTRASOUND EXAMINATION OF THE THYROID GLAND IN PATIENTS WITH HYPOTHYROIDISM

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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.

Key words: Peroxide-antioxidant system, chronic migraine, hypothyroidism.

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 in natural conditions is the enzyme superoxide dismutase (SOD), the oxidative capacity of which allows inactivation of free radicals at the site of formation, preventing their diffusion. Indeed, a huge amount of data shows that SOD is an important component of

human antioxidant protection. SOD contains metals necessary for their catalytic function - copper and zinc.

The aim of the study. To identify the role of biochemical biomarkers oxidative stress in patients with hypothyroidism and chronic migraine

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

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

The peroxide-antioxidant system of blood serum was assessed based on the level of malonic dialdehyde (MDA), diene conjugates (DC), superoxide dismutase (SOD) activity, glutathione peroxidase, glutathione reductase, catalase, cytochrome -C and nitric oxide (NO), determined by spectrophotometric method (L.P. Andreeva et al., 1988; Dubinin B.B. et al., 1983).

Results and their discussion. The results of the study of lipid peroxidation (LPO) processes in the blood plasma of the examined patients showed that the MDA values in patients of group 1 were 3.12±0.4 nmol /ml, in patients of group 2 4.18±0.8 nmol /ml, in group 3 - 2.87±0.6 nmol /ml, i.e. the MDA values exceeded the values of the control group in group 1 by 2.6 times, in group 2 by 3.5 times and in group 3 by 2.4 times. Diene conjugates (DC) in group 1 amounted to 1.89±0.21 μmol /l , in group 2 2.15±0.01 μmol /l , in group 3 1.55±0.1 μmol /l. DC exceeded in group 1 by 1.45 times, in group 2 by 1.65 times and in group 3 by 1.2 times in relation to the control group (Table 1).

Table 1

Comparative indices of LPO in blood plasma in examined patients

Groups	SEX indicators	
	MDA nmol/ml	DK μmol /l
Control group n=20	1.2±0.1	1.3±0.01
1 group (n = 38) HM up to 5 years + hypothyroidism	3.12±0.4 ***	1.89±0.21 ***
Group 2 (n=41) HM more than 5 years + hypothyroidism	4.18±0.8 ***	2.15±0.01 ***
Group 3 (n=39) HM without hypothyroidism	2.87±0.6 ***	1.55±0.1 ***

Note: *P<0.01; **P<0.05; ***P<0.001; – reliability of differences with control values.

As can be seen from Table 1, malonic Dialdehyde (MDA) is a product of lipid peroxidation and is used as a biomarker of oxidative stress. This suggests that patients in Group 2 have higher levels of oxidative stress than patients in Groups 1 and 3.

The presence of mitochondrial abnormalities in migraine patients has been known for a long time, and a disturbance in the energy phosphate metabolism has been described in the initial phase of migraine [1]. This leads to anaerobic metabolism, which makes the cell prone to oxidative stress [2]. In addition, other mechanisms play a role in the development of oxidative stress in headache patients, namely: the release of proinflammatory cytokines during headache [3] the formation of nitric 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 that can cause oxidative stress and, finally, the associated psychological stress, which imperceptibly causes oxidative damage to the body [5].

In the metabolism of every cell, an oxidation reaction occurs. The presence of oxygen in the internal environment is, on the one hand, important for the functioning of the cell; 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.

The study of antioxidant protection indices revealed their decrease. At the same time, the content of AOS parameters in the blood plasma of patients with CM and hypothyroidism showed a decrease in indices as the duration of headache increased to more than 5 years ($P < 0.01$).

Oxidative stress is a disturbance of the prooxidant -antioxidant balance in favor of the former, which leads to the accumulation of free radicals and reactive oxygen species. 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 superoxide radical to 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 reactive oxygen species. If reactive oxygen species are formed in excess or the protective antioxidant mechanisms are ineffective, oxidative stress develops [3]. The most important action of free radicals and reactive oxygen species is lipid peroxidation, which causes the destruction and

breakdown of cell membranes. Imbalance between the formation and removal of reactive oxygen species plays a role in the pathogenesis of many diseases.

Our results suggest that decreased SOD activity and levels in patients with CM and hypothyroidism make them more susceptible to oxidative damage caused by reactive oxygen species (ROS) (Table 2).

Table 2.

Comparative indices of AOS in patients with HM and hypothyroidism

Groups	Analyzed indicators of AOS				
	Catalase mk cat / mg protein	Superoxide dismutase units/mg protein	Glutathione reductase mM/min g protein	Glutathione peroxidase mM/min g protein	Glutathione transferase mmol/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
1 group (n=38) HM 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***
Group 2 (n=41) HM 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***
Group 3 (n=39) HM 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 values.

In patients of group 1, 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 were characterized by a 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 group 3, the AOS indices 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.

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

Conclusion: Our results suggest that decreased SOD activity and levels in patients with CM and hypothyroidism make them more susceptible to oxidative damage caused by reactive oxygen species.

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