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Changes in Key Indicators of The Antioxidant and Endothelial Systems in Chronic Kidney Disease Developed on The Basis of Nephropathies of Various Genesis

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Abstract: The article examines changes in lipid peroxidation and endothelial function markers in patients with chronic kidney disease (CKD) of various etiologies. The study analyzed the levels of malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and free nitric oxide metabolites (NOEM) in patients with CKD developed on the background of non-diabetic and diabetic nephropathies.

According to the obtained results, an increase in lipid peroxidation products and a decrease in the activity of antioxidant enzymes were observed. It was also shown that the use of calcium dobesilate has a positive effect on improving metabolic parameters.

Keywords: Chronic kidney disease, antioxidant system, lipid peroxidation, endothelial function.

Introduction: The antioxidant system refers to systems that prevent the formation of highly active free radicals. The production of free radicals that lead to oxidation of cell membrane lipids is one of the most powerful stimuli causing damage to cells and tissues. Reactive oxygen species are chemically highly aggressive: they damage proteins and DNA, and most importantly, they trigger lipid peroxidation (LPO), which leads to serious damage to membranes. A free radical is characterized as a special type of molecule capable of existing independently and containing one or more unpaired electrons. Free radicals and various reactive oxygen species can be formed during basic metabolic processes or under the influence of external sources. If free radicals are not inactivated, they can damage all types of cellular macromolecules, including proteins, carbohydrates, lipids, and nucleic acids, leading to cell damage. On the other hand, the local formation of such molecules by immune cells can be an important factor in the body's fight against bacteria and in protecting it from infection. For many diseases, oxidative stress is a consequence of the underlying pathology; the uncontrolled spread of toxic radicals causes more cellular damage than the primary disease itself. This stress participates in the emergence of generalized changes in capillary permeability and tissue diffusion characteristic of multiple organ failure. Under normal physiological conditions, a small amount of

oxygen consumed by mitochondria is continuously converted into superoxide anions, hydrogen peroxide, and hydroxyl radicals. Excessive production of these radicals is a damaging factor, and in this regard, the cell possesses a natural antioxidant system. It is represented by enzymes, among which superoxide dismutases (SOD), glutathione peroxidase (GPO), glutathione reductase (GR), and catalase (CAT) are of particular importance. SOD converts superoxide anions into hydrogen peroxide, which is then converted into water by other enzymes — GPO and CAT. Under pathological conditions, specifically, the content of LPO products increases and the reactivity of antioxidant systems often changes — these are referred to as "free radical" processes. In particular, in our study we examined how these enzymes change in chronic renal failure.

Aim of the Study

To evaluate the disturbances in antioxidant system and endothelial function markers in patients with chronic kidney disease (CKD) of various genesis, and to determine their clinical and diagnostic significance.

METHODS

The diversity of etiological factors leading to chronic kidney disease (CKD) and the prognosis associated with it necessitate a thorough and comprehensive analysis of this issue. In particular, currently more than 50% of renal failure cases consist of CKD formed on the basis of diabetic nephropathies. Likewise, non-diabetic primary nephritides such as autoimmune, infectious, and congenital kidney diseases leading to renal failure have not yet lost their significance. Therefore, in this chapter of our research, we studied the markers of kidney damage (urinary syndrome) and the picture of renal functional status in the following patient groups: Group 1 (n=75), consisting of patients with Stage II and III-A CKD formed on the basis of non-diabetic nephropathies, divided into subgroups 1A (n=35) and 1B (n=40); and Group 2 (n=45), consisting of patients with Stage II and III-A CKD formed on the basis of diabetic nephropathies, divided into subgroups 2A (n=20) and 2B (n=25). Patients in subgroup A received only conventional therapy in accordance with CKD treatment guidelines. Patients in subgroup B, in addition to this therapy, received calcium dobesilate — a drug with ADP receptor inhibitory properties — for a period of six months. Based on prospective studies conducted over six months, the following results were obtained. Accordingly, we studied the dynamics of changes in the antioxidant system and endothelial damage in chronic kidney disease formed on the basis of nephropathies of various genesis. In all participants, the following markers of the antioxidant system and endothelial function were assessed: 1. Malondialdehyde (MDA). 2. Superoxide dismutase (SOD). 3. Catalase (CAT). 4. Free metabolites of nitric oxide (NO).

RESULT

The Result of our research, malondialdehyde — a product of lipid peroxidation that accumulates and has a damaging effect on the glomerular basement membrane — was found to be significantly elevated in both groups compared to the control group at the start of the study. Superoxide dismutase and catalase, enzymes that neutralize lipid peroxidation products and are essential for the body, were found to be significantly decreased in both groups compared to the control group at the start of the study. Free metabolites of nitric oxide showed analogous results as reflected in the table. (Table 1.1)

Table 1.1

Changes in LPO/AOS and Endothelial Function Markers in Patients with Chronic Kidney Disease of Non-Diabetic Genesis

Parameters	MDA, nmol/ml	SOD, U/g Hb	CAT, act. U	NO free metabolites, µmol/L
Control group (n=20)	0,52±0,06	1718,54±61,14	16,08±0,32	34,58±1,24
Group 1 (n=35) Baseline Conventional therapy	1,24±0,20**	1425,23±58,42**	12,86±0,40***	18,44±1,24***
Group 1 (n=40) Baseline — Conventional +	1,28±0,22**	1445,23±34,44***	13,42±0,32***	20,46±1,32***

Calcium Dobesilate				

Note: * - differences are significant compared to control group indicators (* - p<0.05, ** - p<0.01, *** - p<0.001); ^ - differences are significant compared to baseline indicators (^ - p<0.05, ^^ - p<0.01, ^^^ - p<0.001); # - differences are significant compared to indicators at different time points between post-treatment groups (# - p<0.05, ## - p<0.01, ### - p<0.001).

In patients of Group 2, consisting of those with chronic kidney disease of diabetic etiology, malondialdehyde was similarly elevated in both subgroups compared to the control group. Superoxide dismutase and catalase indicators were also uniformly and significantly decreased compared to the control group, as clearly demonstrated in the table. (Table 1.2)

Free metabolites of nitric oxide were likewise found to be uniformly and significantly decreased, as noted above.

Table 1.

Changes in LPO/AOS and Endothelial Function Markers Against the Background of Treatment in Patients with Chronic Kidney Disease of Diabetic Genesis

Parameters	MDA, nmol/ml	SOD, U/g Hb	CAT, act. U	NO free metabolites, µmol/L
Control group (n=20)	0,52±0,06	1718,54±61,14	16,08±0,32	34,58±1,24
Group 2 (n=20) Baseline — Conventional therapy	1,25±0,2**	1423,19±57,8 **	12,78±0,71***	17,96±1,24***
Group 2 (n=25) Baseline — Conventional + Calcium Dobesilate	1,29±0,22**	1439,45±33,97***	13,39±0,32***	18,89±1,32***

Note: * - differences are significant compared to control group indicators (* - p<0.05, ** - p<0.01, *** - p<0.001); ^ - differences are significant compared to baseline indicators (^ - p<0.05, ^^ - p<0.01, ^^^ - p<0.001); # - differences are significant compared to indicators at different time points between post-treatment groups (# - p<0.05, ## - p<0.01, ### - p<0.001).

DISCUSSION

Under the influence of lipid peroxidation products, significant changes occur in cell membranes, phospholipases, proteinases, complement, and cytotoxic factors. On the one hand, activation of lipid peroxidation and, on the other hand, sufficient activity of the antioxidant system promote the release of proteolytic enzymes by leukocytes, leading to damage of the glomerular basement membrane and protein-lipid complexes of cytomembranes, inhibition of cellular immunity, increased collagen formation, and development of renal parenchymal ischemia. Lipid peroxidation of endotheliocyte membranes leads to the development of endothelial dysfunction and a decrease in endothelial NO synthase activity. In addition, basal expression of nitric oxide decreases, and the endothelial response to shear stress is also reduced.

Taking into account the role of free radical processes in regulating cell-humoral interactions, their effect on the hemostasis system as a whole and its individual components in patients with CKD is of considerable interest. A number of studies have shown that increased intensity of LPO processes in platelets and decreased antioxidant defense activity occur in parallel with increased vascular-platelet hemostasis activity. Therefore, studying the changes in the lipid peroxidation system in the dynamics of treatment in patients with CKD is of great interest for further investigation.

In this regard, we examined the main characteristics of the LPO/AOS system and endothelial function. Accordingly, in patients with CKD of various genesis, the lipid peroxidation/antioxidant system and endothelial function indicators revealed the following picture in our study.

Malondialdehyde, which accumulates as a result of lipid peroxidation and has a damaging effect on the glomerular basement membrane, was significantly

elevated in both groups compared to the control group at baseline. After 1 month of treatment, it decreased insignificantly compared to baseline in both the conventional therapy group and the group receiving additional calcium dobesilate. By the end of treatment, malondialdehyde decreased insignificantly in those receiving conventional therapy alone, while in both groups receiving calcium dobesilate in addition to conventional therapy, a marginally significant decrease was observed. Comparing end-of-study results with day 30, a significant change in values was found in patients with non-diabetic CKD, while marginally significant changes were observed in patients with diabetic CKD (Tables 5.9–5.10).

Superoxide dismutase was significantly decreased in both groups compared to the control group at baseline. After 1 month of treatment, it changed insignificantly compared to baseline in patients receiving conventional therapy, while it increased significantly in Group 1 and marginally significantly in Group 2 among those receiving additional calcium dobesilate. By the end of treatment, superoxide dismutase increased marginally significantly in both groups receiving conventional therapy alone, while a significant increase was observed in both groups receiving calcium dobesilate in addition to conventional therapy. Comparing end-of-study results with day 30, values changed insignificantly in patients with CKD of both genesis (Table 1.3).

Table 1.3

Changes in LPO/AOS and Endothelial Function Markers Against the Background of Treatment in Patients with Chronic Kidney Disease of Non-Diabetic Genesis

Parameters	MDA, nmol/ml	SOD, U/g Hb	CAT, act. U	NO free metabolites, µmol/L
Control group (n=20)	0.52±0.06	1718.54±61.14	16.08±0.32	34.58±1.24
Group 1 (n=35) Baseline — Conventional therapy	1.24±0.20**	1425.23±58.42**	12.86±0.40***	18.44±1.24***
Group 1 (n=35) After 1 month — Conventional therapy	1.18±0.14**	1554.44±52.35*	14.26±0.34*^	22.32±1.46***
Group 1 (n=35) After 6 months — Conventional therapy	1.10±0.09*	1586.42±41.22^	14.82±0.42*^^	28.46±2.54**^^#
Group 1 (n=40) Baseline — Conventional + Calcium Dobesilate	1.28±0.22**	1445.23±34.44***	13.42±0.32***	20.46±1.32***
Group 1 (n=40) After 1 month — Conventional + Calcium Dobesilate	1.02±0.06***	1645.23±34.44^^	15.32±0.52^^	26.31±1.62*^^
Group 1 (n=40) After 6 months — Conventional + Calcium Dobesilate	0.68±0.06^##	1687.43±54.12^^^	16.49±0.24^^^#	32.66±1.24^^^##

Note: * — differences are significant compared to control group indicators (* — p<0.05, ** — p<0.01, *** — p<0.001); ^ — differences are significant compared to baseline indicators (^ — p<0.05, ^^ — p<0.01, ^^^ — p<0.001); # — differences are significant compared to indicators at different time points between post-treatment groups (# — p<0.05, ## — p<0.01, ### — p<0.001).

Catalase was significantly decreased in both groups compared to the control group at baseline. After 1 month of treatment, compared to baseline, it changed marginally significantly in Group 1 receiving conventional therapy and insignificantly in Group 2, while it increased significantly in Group 1 and marginally significantly in Group 2 among those receiving additional calcium dobesilate. By the end of the study, catalase increased significantly in Group 1 and marginally significantly in Group 2 receiving conventional therapy alone, while a significant increase was observed in both groups receiving calcium

dobesilate in addition to conventional therapy — with values in patients with non-diabetic CKD even exceeding those of the control group. Comparing end-of-study results with day 30, a marginally significant increase was found in patients with non-diabetic CKD, while an insignificant increase was observed in the diabetic genesis group, which was also reflected in the diagram illustrations (Table 1.4).

Free metabolites of nitric oxide (NO), as markers of endothelial function, were significantly decreased in both groups compared to the control group at baseline. After 1 month of treatment, compared to baseline, they changed insignificantly in both groups receiving conventional therapy, while a significant increase was observed in Group 1 and a marginally significant increase in Group 2 among those receiving additional calcium dobesilate. By the end of the study, free NO metabolites increased significantly in Group 1 and marginally significantly in Group 2 receiving conventional therapy alone, while a significant increase was observed in both groups receiving calcium dobesilate in addition to conventional therapy. Comparing end-of-study results with day 30, a marginally significant increase was found in patients with non-diabetic CKD receiving conventional therapy alone, while a significant increase was observed in those additionally receiving calcium dobesilate; in the diabetic genesis groups, free NO metabolites changed insignificantly (Tables 5.9–5.10).

Table 1.4

Changes in LPO/AOS and Endothelial Function Markers Against the Background of Treatment in Patients with Chronic Kidney Disease of Diabetic Genesis

Parameters	MDA, nmol/ml	SOD, U/g Hb	CAT, act. U	NO free metabolites, µmol/L
Control group (n=20)	0.52±0.06	1718.54±61.14	16.08±0.32	34.58±1.24
Group 2 (n=20) Baseline — Conventional therapy	1.25±0.20**	1423.19±57.80**	12.78±0.71***	17.96±1.24***
Group 2 (n=20) After 1 month — Conventional therapy	1.21±0.14**	1549.32±51.98*	14.26±0.34*	21.77±1.46***
Group 2 (n=20) After 6 months — Conventional therapy	1.12±0.09*	1584.26±42.10^	14.91±0.52*^	24.84±2.54**^
Group 2 (n=25) Baseline — Conventional + Calcium Dobesilate	1.29±0.22**	1439.45±33.97***	13.39±0.32***	18.89±1.32***
Group 2 (n=25) After 1 month — Conventional + Calcium Dobesilate	1.08±0.13***	1603.45±33.90^	14.98±0.52^	24.53±1.62**^
Group 2 (n=25) After 6 months — Conventional + Calcium Dobesilate	0.71±0.06^#	1631.87±55.10^^	15.42±0.51^^	29.17±2.46^^

Note: * — differences are significant compared to control group indicators (* — p<0.05, ** — p<0.01, *** — p<0.001); ^ — differences are significant compared to baseline indicators (^ — p<0.05, ^^ — p<0.01, ^^^ — p<0.001); # — differences are significant compared to indicators at different time points between post-treatment groups (# — p<0.05, ## — p<0.01, ### — p<0.001).

CONCLUSION

Thus, the addition of calcium dobesilate to conventional therapy in the treatment of CKD patients has a positive effect on lipid peroxidation/antioxidant system and endothelial function indicators. This efficacy is particularly more pronounced and statistically more significant in patients with CKD of

non-diabetic genesis compared to those with diabetic renal failure. It is well known that all kidney diseases are, in one way or another, associated with pathology of the basal or cell membranes.

Thus, the addition of calcium dobesilate to conventional therapy in the treatment of CKD patients has a positive effect on lipid peroxidation/antioxidant system and endothelial function indicators. This efficacy is particularly more pronounced and statistically more significant in patients with CKD of non-diabetic genesis compared to those with diabetic renal failure.

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