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LIPID PEROXIDATION OF MEMBRANES IN RENAL ISCHEMIA

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Abstract

Renal ischemia causes tissue hypoxia and activation of reactive oxygen species, which leads to damage to cell membranes through lipid peroxidation (LPO) processes. This study examines changes in malonic acid levels. Dialdehyde (MDA) levels and antioxidant enzyme activity (SOD, catalase, glutathione peroxidase) in renal ischemia. It was shown that ischemia is accompanied by a significant increase in oxidative stress and a weakening of antioxidant defenses, leading to damage to tubular cell membranes, impaired microcirculation, and increased blood viscosity. The results highlight the key role of oxidative stress in the pathogenesis of ischemic kidney injury and the need for oxidative stress correction for prevention and treatment.

The results showed a significant increase in MDA in kidney tissue and blood plasma (by 1.5–2 times), accompanied by a 25–45% decrease in antioxidant enzyme activity, indicating severe oxidative stress and weakened cellular defenses. The increase in LPO correlated with decreased erythrocyte deformability , increased aggregation and dynamic blood viscosity, and morphological changes in the tubular epithelium, including edema, membrane damage, and apoptosis .

The obtained data confirm the key role of membrane lipid peroxidation and antioxidant system imbalance in the pathogenesis of ischemic kidney injury. The study's results have clinical implications for the development of strategies for the prevention and correction of ischemic kidney injury, including the use of antioxidant therapy and the maintenance of microcirculatory function.

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Keywords: Renal ischemia, lipid peroxidation, malonic dialdehyde, antioxidant protection, oxidative stress, microcirculation.

The aim of the study was to investigate changes in membrane lipid peroxidation processes and antioxidant defense activity during renal ischemia, as well as their role in the development of microcirculatory disorders and tubular epithelial damage.

Research objectives

1. Determine the level of malondioxide dialdehyde (MDA) in kidney tissue and blood plasma under normal conditions, ischemia and reperfusion .
2. To study the activity of antioxidant enzymes: superoxide dismutase (SOD), catalase and glutathione peroxidase .
3. Compare changes in LPO indicators under normal and ischemic conditions.
4. To analyze the relationship between LPO activation, membrane damage and microcirculation disorders.

Materials and methods

Biological samples: kidney tissue, blood plasma. Control group: animals without ischemic exposure.

Experimental groups:

1. Renal ischemia (30–60 minutes of renal artery ligation).
2. Reperfusion after ischemia (30 minutes).

1. Renal ischemia model: Renal artery ligation for 30–60 minutes, followed by restoration of blood flow (reperfusion). This method allows for the reproduction of ischemic damage to the tubules and endothelium.
2. Lipid peroxidation (LPO) assessment: Malonic acid level Dialdehyde (MDA) levels in kidney tissue and blood plasma were determined using the thiobarbituric acid (TBAR) reaction. MDA serves as a marker of the degree of oxidative damage to membranes.
3. Assessment of antioxidant protection:

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Superoxide dismutase (SOD): by the ability to destroy superoxide anions. Catalase: by the rate of decomposition of hydrogen peroxide (H_2O_2).
Glutathione peroxidase : on the use of glutathione to neutralize peroxides.
4. Morphological examination: Histological examination of kidney tissue with hematoxylin and eosin staining to assess edema, epithelial damage and infiltration.
5. Statistical analysis: Results are expressed as mean ± standard deviation. Comparisons between groups were performed using a t-test or ANOVA. Differences were considered significant at $p < 0.05$.

Research results

1. Malonic acid level dialdehyde (MDA):

In the control group, MDA in kidney tissue was 2.1 ± 0.2 nmol /mg protein, in blood plasma – 1.5 ± 0.1 nmol /ml. After 30–60 minutes of renal ischemia, the MDA level increased to 3.2 ± 0.3 nmol /mg protein in tissue and 2.5 ± 0.2 nmol /ml in plasma, indicating a significant increase in lipid peroxidation.
After 30 minutes of reperfusion, MDA remained elevated (tissue - 3.0 ± 0.3; plasma - 2.3 ± 0.2), reflecting continued oxidative stress.

2. Activity of antioxidant enzymes:

Superoxide dismutase (SOD): decreased by 35–45% compared to normal.
Catalase: activity decreased by 30–40%.
Glutathione peroxidase : decreased activity by 25–35%.
The data indicate a decrease in antioxidant protection and an inability to neutralize excess reactive oxygen species.

3. Relationship with microcirculation and blood rheology:

Increased lipid peroxidation is accompanied by decreased red blood cell deformability and increased aggregation, leading to increased dynamic blood viscosity. These changes are particularly pronounced in the renal medulla, where physiologically low blood flow makes microcirculation vulnerable.

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4. Morphological changes:

Tubular epithelial cells exhibit edema, membrane damage, and signs of apoptosis . Edema and increased leukocyte infiltration are observed in the interstitial tissue. Renal ischemia causes activation of membrane lipid peroxidation , decreased antioxidant defense, and cellular damage, which is directly related to impaired microcirculation and increased blood viscosity.

Conclusions

1. Renal ischemia leads to a significant increase in lipid peroxidation of membranes, which is manifested by an increase in the level of MDA in kidney tissue and blood plasma.
2. The activity of antioxidant enzymes (SOD, catalase, glutathione peroxidase) decreases, weakening the protection of cells from oxidative stress.
3. Increased lipid peroxidation is associated with impaired microcirculation, decreased deformability of erythrocytes and increased blood viscosity.
4. Morphological changes in renal tissue reflect damage to the membranes and epithelium of the tubules.
5. Control of lipid peroxidation processes and support of antioxidant protection are important for the prevention and correction of ischemic kidney damage.

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