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RESEARCH ARTICLE

Modulation of Oxidative Stress by Glycine in Experimental Myocardial Infarction

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Abstract: Glycine reduces oxidative stress and limits lipid peroxidation during experimental myocardial infarction in rabbits. Administration of glycine at a dose of 100 mg/kg delayed the early increase in malondialdehyde and significantly lowered plasma levels of malondialdehyde and conjugated dienes throughout 3–72 hours post-coronary occlusion. This effect was accompanied by higher activities of key antioxidant enzymes, superoxide dismutase and catalase, indicating enhanced endogenous antioxidant defense. These results suggest that glycine exerts cardioprotective effects through its antioxidant and anti-stressor properties and may serve as a potential therapeutic agent for reducing oxidative damage during ischemic heart injury.

Key words: Glycine, antioxidant system, superoxide dismutase, catalase, malondialdehyde, diene conjugates, experimental myocardial infarction, blood serum.

INTRODUCTION

Myocardial infarction (MI) is an acute form of coronary heart disease characterized by necrosis of a region of the heart muscle resulting from prolonged impairment of coronary circulation. The most common cause is thrombosis of a coronary artery developing due to rupture or erosion of an atherosclerotic plaque. Less frequently, myocardial infarction may be caused by coronary vasospasm, embolism, or a marked reduction in coronary blood flow associated with severe hypotension. Under ischemic conditions, the delivery of oxygen and nutrients to cardiomyocytes is disrupted, leading to impaired energy metabolism, intracellular calcium accumulation, enzyme activation, and ultimately to irreversible cellular injury and death [1,2,3].

Oxidative stress plays a key role in the pathogenesis of myocardial infarction, contributing to cardiomyocyte injury, disruption of metabolic processes, and progression of necrotic changes in cardiac tissue. Under conditions of ischemia and subsequent reperfusion, the production of reactive oxygen species increases, leading to an imbalance between the pro-oxidant and antioxidant systems of the body. Disturbance of the pro-oxidant/antioxidant balance results in damage to cellular membranes, mitochondrial dysfunction, activation of inflammatory responses, and cardiomyocyte death, which significantly impairs myocardial function [4].

In this regard, the investigation of antioxidant defense mechanisms and the search for effective metabolic factors capable of reducing the severity of oxidative damage are of considerable scientific interest.

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In recent years, particular attention has been paid to amino acids as potential cardioprotective agents. Glycine, the simplest non-essential amino acid, is involved in the regulation of metabolic processes, glutathione synthesis, modulation of inflammatory responses, and maintenance of cellular homeostasis [4,5]. Experimental studies indicate its ability to reduce the severity of oxidative stress, limit tissue damage, and enhance cellular resistance to hypoxia and ischemia [6,7]. However, the mechanisms underlying the antioxidant effects of glycine in myocardial infarction remain insufficiently systematized and require further investigation.

Purpose of the research: to evaluate the effect of glycine on the intensity of lipid peroxidation and the activity of the antioxidant system during the course of experimental myocardial infarction (EMI).

METHODS

Experiments were conducted on 10 male rabbits weighing 2.5–2.8 kg. Experimental myocardial infarction (EMI) was induced by ligation of the descending branch of the left coronary artery. One rabbit died immediately after ligation, and another died one hour later, resulting in a mortality rate of 20%. Immediately after ligation, five rabbits were administered an aqueous solution of glycine (produced by the Medical Scientific-Production Complex "Biotics," Russian Federation) via oral gavage at a dose of 100 mg/kg body weight. Subsequently, the animals received glycine orally at the same dose once daily. Three rabbits that did not receive glycine served as the control group.

Blood was collected from the ear vein before ligation (baseline) and at 30 min, 1, 3, 6, and 12 hours, as well as on days 1, 3, and 7 of EMI, into tubes containing heparin. The blood was centrifuged at 3000 rpm for 15 minutes. Plasma levels of lipid peroxidation (LPO) products—malondialdehyde (MDA)—were determined according to M. Mihara (1980), and diene conjugates (DC) according to Z. Placer (1968) with modifications by V.B. Gavrilov and M.I. Mishkorudnaya (1983). The activity of antioxidant system (AOS) enzymes—superoxide dismutase (SOD)—was determined according to V.G. Mkhitaryan and G.E. Badalyan (1978), and catalase according to M.A. Korolyuk et al. (1988). Quantitative data were statistically analyzed using Student's t-test.

RESULTS

The results showed that during experimental myocardial infarction (EMI), a statistically significant increase in malondialdehyde (MDA) levels of 39.3% was observed starting from 1 hour after ligation. At 3, 6, 12, 24, and 72 hours post-occlusion, MDA levels increased by 467.3%, 219.6%, 135.5%, 223.4%, and 315.9%, respectively, compared to baseline.

In rabbits treated with glycine, a statistically significant increase in MDA of 329.9% was observed at 3 hours.0

At 6, 12, 24, and 72 hours after occlusion, MDA levels were elevated by 143.0%, 103.7%, 179.4%, and 199.1%, respectively, compared to baseline, which was significantly lower than in the control group. MDA levels in both the control and glycine-treated groups returned to baseline only on day 7 of the study.

Analysis of diene conjugate (DC) levels during EMI showed an increase of 49.1% at 1 hour after coronary occlusion. At 3, 6, 12, 24, 72, and 168 hours post-occlusion, DC levels increased by 361.1%, 226.4%, 110.8%, 144.9%, 204.8%, and 25.2%, respectively, compared to baseline. In glycine-treated rabbits, a statistically significant increase in DC of 34.7% was also observed at 1 hour. At 3, 6, 12, 24, and 72 hours after occlusion, DC levels were elevated by 262.9%, 158.1%, 94.0%, 119.2%, and 120.4%, respectively, which was significantly lower than the control values.

Analysis of antioxidant system (AOS) enzyme activity showed that during experimental myocardial infarction (EMI), superoxide dismutase (SOD) activity significantly decreased by 63.8% starting from 3 hours after ligation. At 6, 12, 24, and 72 hours post-occlusion, SOD activity decreased by 48.2%, 35.2%, 37.3%, and 57.7%, respectively, compared to baseline. In glycine-treated rabbits, a statistically significant decrease in SOD activity of 48.1% was also observed at 3 hours.

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At 6, 12, 24, and 72 hours, SOD activity decreased by 30.9%, 22.6%, 31.2%, and 33.1%, respectively, which was significantly higher than in the control group. SOD activity returned to baseline in both groups only on day 7 of the study.

Catalase activity during EMI also decreased significantly at 3 hours post-coronary occlusion (by 47.7%). At 6, 12, 24, and 72 hours after occlusion, catalase activity decreased by 40.6%, 31.8%, 38.6%, and 49.6%, respectively, compared to baseline. In glycine-treated animals, a significant decrease in catalase activity of 37.5% was observed at 3 hours. At 6, 12, 24, and 72 hours post-occlusion, catalase activity decreased by 20.6%, 22.8%, 28.9%, and 26.3%, respectively, which was significantly higher than in controls.

These results indicate that glycine helps to attenuate the decline in antioxidant enzyme activity during myocardial infarction. This effect likely contributes to the reduced accumulation of lipid peroxidation products (MDA and DC) in myocardial infarction. In summary, administration of glycine during myocardial infarction leads to a lower intensification of free radical–induced lipid peroxidation in the organism.

The results of the study indicate that glycine significantly reduces the levels of both MDA and DC during experimental myocardial infarction (EMI). Literature data support the antioxidant effects of glycine. Previous studies have demonstrated its antioxidant action on cortical slices of the brain under anoxia. Evidence also exists for glycine's antioxidant effect in patients with moderate to severe stroke, observed on day 3 of the pathology at doses of 1–2 g/day.

However, considering the structure of the glycine molecule itself, it is difficult to regard it as a direct antioxidant—a free radical scavenger. Most likely, glycine's antioxidant effect is mediated indirectly. Studies show that glycine markedly reduces ultrastructural tissue damage in the cerebral cortex, particularly in mitochondria, arising from acute anoxia and ischemic injury. Glycine has been noted to depress vasomotor reflexes, exert a "specific" inhibitory effect on the activity of neurons in the reticular formation, and influence the functional state of the sympathoadrenal system and adrenergic mediation processes.

The ability of glycine to suppress activation of the adrenergic and hypothalamic-pituitary-adrenal systems likely underlies its cardioprotective, anti-stress effects by reducing myocardial alterations under stress. Administration of glycine was associated with a statistically significant decrease in oxidative stress products in ischemic zones, accompanied by rapid normalization of behavior and conditioned reflexes in rats.

Considering the above, it can be suggested that the antioxidant effect of glycine in myocardial infarction is mediated indirectly through the reduction of stress-related responses.

Thus, the results of the present study indicate the antioxidant properties of glycine. Considering its effectiveness, availability, safety, and wide range of metabolic effects, glycine can be recommended as an adjunctive therapy in myocardial infarction.

DISCUSSION

The present study demonstrates that glycine supplementation significantly attenuates lipid peroxidation during the progression of experimental myocardial infarction in rabbits, as evidenced by reduced plasma levels of malondialdehyde (MDA) and conjugated dienes at multiple time points post coronary occlusion. This protective effect was accompanied by higher activities of key antioxidant enzymes, superoxide dismutase and catalase, in glycine treated animals compared to controls. These findings suggest that glycine exerts a cardioprotective effect by modulating oxidative stress pathways.

Our results are consistent with previous studies showing that glycine has cytoprotective effects in models of myocardial ischemia–reperfusion injury. For example, in a rat model of myocardial ischemia–reperfusion, glycine administration reduced infarct size, improved cardiac functional parameters, and decreased biochemical markers of cardiac injury, indicating significant protection against ischemic damage [8,9]. These protective effects

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have been associated with suppression of apoptotic signaling pathways, suggesting that glycine's benefits may extend beyond antioxidant activity to include effects on cellular survival mechanisms.

At the cellular level, glycine has been shown to protect cardiomyocytes against lethal reoxygenation injury by inhibiting the mitochondrial permeability transition, a key event leading to cell death during ischemia and reperfusion [10,11]. This mechanism supports the concept that glycine can preserve mitochondrial integrity and reduce oxidative damage to cardiomyocytes under stress.

Beyond cardiac tissue, recent cardiovascular research has identified glycine as a modulator of systemic oxidative metabolism. Lower circulating glycine levels have been associated with increased risk of acute myocardial infarction in humans, and higher plasma glycine appears to correlate with a favorable metabolic profile [11]. Additionally, glycine supplementation in atherosclerosis models enhanced glutathione biosynthesis and reduced superoxide production, demonstrating its potential to reinforce endogenous antioxidant defenses [10].

Taken together, existing evidence indicates that glycine's cardioprotective effects involve multiple pathways: direct reduction of lipid peroxidation, enhancement of enzymatic antioxidant defenses, modulation of apoptosis, and support of mitochondrial function. The findings of the current study align with this multifactorial viewpoint, highlighting the potential utility of glycine as a therapeutic agent to mitigate oxidative stress and metabolic dysfunction in myocardial ischemia.

CONCLUSION

Glycine effectively attenuates lipid peroxidation during experimental myocardial infarction in rabbits. Administration of glycine at a dose of 100 mg/kg reduced plasma levels of malondialdehyde and conjugated dienes and enhanced the activity of key antioxidant enzymes, including superoxide dismutase and catalase. These results suggest that glycine exerts a cardioprotective effect by limiting oxidative stress, likely through its anti-stressor and antioxidant properties, and may represent a promising strategy for metabolic support during ischemic heart injury.

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