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# APPLICATION OF MYOCARDIAL CYTOPROTECTORS IN ISCHEMIC HEART DISEASES

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**Abstarct:** Ischemic heart disease remains a leading cause of morbidity and mortality in all industrialized nations, resulting from blockage of one or more coronary arteries. Although the treatment of myocardial ischemia continues to improve with more timely and effective reperfusion, no effective therapy exists to prevent or attenuate the resulting myocardial ischemia–reperfusion injury. Moreover, the major determinant of the long-term outcome is the amount of myocardium that is destroyed as a result of ischemic injury (i.e., the size of infarction). Thus, it is believed that a significant reduction in myocardial infarct size will decrease subsequent morbidity and mortality. As such, interventions designed to effectively reduce myocardial infarction are still needed.

**Keywords:** Ventricular hypertrophy, Coronary heart disease, Metabolic syndrome, Acute myocardial infarction, Cardiovascular system, Stable angina

Modern strategies for providing care to patients with coronary artery disease (CHD) include modification of risk factors, drug therapy and revascularization interventions, which play a key role in the treatment of these patients. Pharmacological treatment of stable angina pectoris involves, first of all, the use of drugs that improve the prognosis of patients with angina (antiplatelet agentsA, antihypertensive drugsA, beta-blockersA, angiotensin-converting enzyme inhibitorsA).

However, an important direction in the treatment of patients is optimal antianginal therapy aimed at improving the quality of life (short-acting and long-acting nitrates, beta-blockersA, calcium antagonistsA, inhibitor of sinus node If-channelsB 1)

However, the use of hemodynamic drugs (beta-blockers, calcium antagonists, nitrates), which optimize the relationship between the oxygen demand of the heart muscle and its delivery, does not always effectively control the symptoms of the disease, even when using two or more drugs of this group. Moreover, the results of comparative studies indicate the absence of clear advantages of drug combinations over monotherapy and an increase in the frequency of side effects when using two or more drugs in this group. Apparently, this is due to the fact that the possibilities of therapy in this area are limited by the conditions of myocardial functioning during ischemia, the presence of other adaptive and maladaptive processes that have a significant impact on cardiomyocytes (CMC) and the myocardium as a whole. In addition, these drugs

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are characterized by numerous side effects, which are, in fact, a continuation of their main therapeutically desirable effects on the cardiovascular system and, in some cases, significantly limit their use.

Myocardial cytoprotectors are drugs of various chemical classes, the action of which is not associated with a hemodynamic effect, but is mediated by optimization of the processes of formation and energy consumption, correction of the function of the respiratory chain, normalization of the balance between the intensity of free radical oxidation processes and antioxidant protection, and a direct effect on cardiomyocytes, which contributes to their survival under ischemic conditions, prevents the formation of "myocardial metabolic remodeling" (the term was proposed by M. van Bilsen et al. in 2004 to designate pathological changes mediated by excess activation of fatty acids in the cardiomyocyte).

Classification of cardiocytoprotectors by localization of pharmacological effect:

Intramitochondrial cytoprotectors.

1. 1. Inhibition of fatty acid oxidation:

- suppression of beta-oxidation of fatty acids (trimetazidine);
- suppression of the transport of fatty acids into mitochondria (meldonium).

1. 2. Direct stimulation of glucose oxidation (2-ethyl-6-methyl-3-hydroxypyridine succinate).

1. 3. Stimulation of the cytochrome chain (coenzyme Q10).

Transport of energy substrate into mitochondria (phosphocreatine, glucose-insulin mixture (ineffective), succinic acid).

Stimulation of anaerobic glycolysis (thiatriazoline) - poorly developed and ineffective.

Antioxidants and mitochondrial cytoprotectors with antioxidant properties.

Optimizing myocardial energy metabolism provides great opportunities in terms of protecting the heart muscle from ischemic damage [9]. However, when choosing a drug, it should be taken into account that the oxidation of glucose in the metabolic chain of ATP synthesis requires 35–40% less oxygen per ATP molecule than the oxidation of fatty acids (FA), while oxygen savings are ensured not only due to the peculiarities of the metabolic oxidation cycle glucose (up to 20%), but also due to the absence of the need for fatty acids to enter the mitochondria (about 15–20%), the active transport of which requires ATP, which is deficient under hypoxic conditions. It has been established that myocardial ischemia at the cellular level is characterized by a decrease in glucose oxidation and an increase in the use of fatty acids. A sharp imbalance between the oxidation of glucose and FA towards the latter, as well as an increased concentration of FA in the ischemic zone, are the main factors of reperfusion damage and myocardial dysfunction, and the development of dangerous cardiac arrhythmias [10]. Therefore, the use of drugs that block FA beta-oxidation in different

ways and stimulate glucose oxidation on an alternative basis is considered as the most promising direction of myocardial cytoprotection. The most studied drugs that block FA oxidation are p-FOX inhibitors (partial fatty and oxidation inhibitors), which include trimetazidine and meldonium used in Russia. Each of these drugs has its own mechanism for blocking FA oxidation.

In conclusion, I would like to note another important aspect of the additional opportunities associated with the use of MC. In light of the data obtained to date on their positive effect on the functional state of the kidneys, carbohydrate and lipid metabolism, and insulin resistance [38–41], it seems very relevant to continue studying their influence not only on the pathogenetic factors of the ischemic process, but also on various organs and systems.

Thus, today myocardial cytoprotection is a new, evidence-based direction in the treatment of cardiovascular diseases. Due to the short time spent studying them, there is no serious evidence base for their effect on survival and mortality in patients with CVD. However, the clinical data obtained to date, indicating an increase in the effectiveness of treatment of patients with coronary heart disease, indicate the prospects of their use in combination therapy of patients with cardiovascular pathology.

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