

## SPECIFIC CLINICAL COURSE OF ACUTE CORONARY SYNDROME IN YOUNG PEOPLE

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### ABSTRACT

Acute coronary syndrome is a serious life-threatening condition that often serves as the clinical debut of ischemic heart disease. In 20-25% of cases, angina pectoris begins with acute myocardial infarction and painful angina after acute myocardial infarction. The high incidence of acute coronary syndrome in young people in recent years indicates that the disease is getting younger. From the point of view of modern knowledge, which is the gold standard in the treatment of acute coronary syndrome - modern pharmacological support is very important both before endovascular surgery and during percutaneous intervention.

**Keywords:** decompensation, hypoperfusion, levosimendan, cardiovascular thrombosis

Acute heart failure is a medical emergency characterized by the rapid onset or worsening of symptoms of heart failure. It can be newly developed or a manifestation of chronic heart failure decompensation. Cardiac dysfunction may result from myocardial ischemia, rhythm disturbances, valve dysfunction, pericardial damage, increased filling pressure or systemic resistance. Newly developed acute heart failure is most often observed in patients with acute coronary syndrome. The development of decompensation of chronic heart failure is facilitated by low adherence to treatment, fluid overload, infections, alcohol consumption, certain drugs (for example, non-steroidal anti-inflammatory drugs or thiazolidinediones, which cause fluid retention), etc. Acute heart failure is usually accompanied by congestion of blood in the lungs, although in some patients the clinical picture is dominated by signs of decreased cardiac output and tissue hypoperfusion.

Elimination of its cause is important in the treatment of acute heart failure, in particular, lowering blood pressure, arresting arrhythmias, prosthetics of the heart valve, and myocardial revascularization in acute coronary syndrome. The last two methods can prevent the development of new episodes of acute heart failure and improve long-term prognosis. However, in many cases, eliminating the cause of acute heart failure is not possible. This primarily concerns patients with decompensation of chronic heart failure against the background of severe myocardial damage. For the treatment of acute heart failure, various drugs are used, including diuretics, vasodilators (nitrates, nitroprusside, nesiritide), inotropic (Pj-adrenergic receptor agonists, levosimendan, phosphodiesterase inhibitors) and vasopressor (norepinephrine) drugs. The use of drugs that increase the inotropic function of the heart is justified in case of low cardiac output, persistence of signs of tissue hypoperfusion or blood stagnation despite the introduction of vasodilators and / or diuretics. Vasopressor agents (norepinephrine) are recommended for use only in cardiogenic shock, when the administration of inotropic agents and fluids does not lead to an increase in systolic blood pressure of more than 90 mm Hg. Art., and the perfusion of internal organs remains inadequate despite the increase in cardiac output.

At present, reperfusion of cardiovascular thrombosis is accomplished through fibrinolytic drugs and percutaneous intervention [5,6]. The main disadvantages of systemic thrombolysis are the risk of serious hemorrhagic complications and low recovery of blood flow in the injured vessel. However, the maximum benefit of endovascular intervention should only be discussed provided that well-qualified medical personnel are available, as well as equipped with a high-tech X-ray machine. [4]. At present, there are no clear risk factors for the development of the disease in patients with this diagnosis.

In addition to the great interest in the development of new drugs to treat acute heart failure, past experiences such as diuretics, vasodilators, and inotropic drugs, and the potential for current and future use, remain a topic of discussion and anticipation [1]. The greatest controversy in considering these three groups of drugs is caused by inotropes [2]. There are no promising new drugs that require an important role in this area [3], this article discusses levosimene, the latest addition to the arsenal of inotropes and inodilators.

In the treatment of acute congestion and pulmonary edema, a combination of oxygen therapy with intravenous morphine, diuretics and vasodilators (more often nitroglycerin) is used. The need for the use of cardiotoxic drugs usually arises with insufficient effectiveness of these interventions, as well as arterial hypotension [11]. However, along with the improvement of hemodynamic parameters, the use of widespread positive inotropic agents (beta-adrenostimulants, phosphodiesterase III inhibitors) is accompanied by frequent side effects and may increase mortality even with short-term intravenous administration [7]

The reason for this effect is seen in the fact that existing cardiotoxic drugs have arrhythmogenic potential, increase myocardial oxygen demand and aggravate damage to myocardial cells due to their calcium overload [5].

Peculiarities of action of levosimendan and dobutamine.

Levosimendan increases the contractile proteins of cardiomyocytes to calcium. Positive inotropic (cardiotonic) action dose-dependent levosimendan has been demonstrated experimentally [8], in healthy individuals, with left ventricular contractile dysfunction [9], after coronary angioplasty and coronary artery bypass grafting [10]. To realize the cardiotonic effect of this drug, an increase in the calcium content inside the cell and a noticeable increase in myocardial oxygen demand are not required, which has been demonstrated both in experiment and in a clinical study of the drug. The effect of levosimendan is reversible - in diastole at lower calcium concentrations, it dissociates from cardiac troponin and, as a result, does not violate myocardial relaxation [4]. The effect of levosimendan is manifested at the available calcium concentrations in the cell and is fundamentally different from widely used positive inotropic agents, leading to an increase in the content of cAMP and calcium inside the cell due to the stimulation of beta-adrenergic receptors (dobutamine, dopamine) or inhibition of phosphodiesterase (milrinone) [8].

Another, no less important mechanism of action of levosimendan is its ability to open ATP-dependent potassium channels in the smooth muscle of the vascular wall and mitochondria. As a result, there is an expansion of veins and arteries, including coronary arteries, which is the basis for a decrease in pre- and afterload, a decrease in pressure in the pulmonary circulation, as well as anti-ischemic action [12].

Accordingly, levosimendan, on the one hand, belongs to non-glycoside cardiotonic (inotropic) agents and is a member of a class of drugs called "calcium sensitizers", on the other hand, it provides vasodilation. Therefore, it can be attributed to the so-called "vasodilators".

Levosimendan has an active metabolite, which leads to the persistence of the effect for several days after discontinuation of drug administration [11]. This property distinguishes it from dobutamine, to the hemodynamic action of which tolerance develops rather quickly.

Experimental and clinical data indicate an improvement in the contractility of the "stunned" myocardium under the influence of levosimendan (after an episode of ischemia that does not lead to necrosis, followed by reperfusion) [7].

According to the accumulated evidence, the use of levosimendan in the recommended dose range in patients with severe CHF does not lead to additional adrenergic stimulation and an increase in the level of catecholamines in the blood. However, with less pronounced manifestations of the disease, in particular, in stable patients with a pulmonary artery wedge pressure (PAWP) of about 15 mm Hg, the intervention can lead to activation of the sympathetic division of the autonomic nervous system with an increase in norepinephrine levels [1]. According to the results of electrophysiological studies, levosimendan does not significantly affect the duration of the QT interval and an increase in the value of this indicator against the background of drug infusion is associated almost exclusively with an increase in heart rate (HR) [13]. All this testifies in favor of the absence of a pronounced arrhythmogenic potential in levosimendan with proper use of the drug.

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