

# The Frequency of Cardiovascular Events in Patients of Working Age with Unfavorable Remodeling of the Left Ventricle after Myocardial Infarction

Saidov M. A., Nizamov H.Sh., Akhmadov M. A.

*Samarkand regional branch of the Republican Specialized Scientific and Practical Medical Center of Cardiology*

Khasanjanova F. O.

*Samarkand State Medical University, Samarkand, Uzbekistan*

**Abstract:** In this scientific work, the frequency of cardiovascular events in patients of working age with unfavorable remodeling of the left ventricle after myocardial infarction was analyzed. The study included 130 patients with STEMI (age  $51.2 \pm 18.8$  years) hospitalized in the departments of acute coronary syndrome and coronary heart disease and cardiac resuscitation of the Samarkand regional branch of the Republican Specialized Scientific and Practical Medical Center of Cardiology (SRF RSNPMC) in the first 24-96 hours from the onset of the disease. A prerequisite was the presence of hemodynamically significant stenosis of only one coronary artery according to the results of coronary angiography. Initially, on days 7-9 from the onset of the disease and after 24 weeks of follow-up, patients underwent standard echocardiography to determine the index of final diastolic volume (iCDO), the index of final systolic volume (ICso). After 24 weeks, the patients were divided into two groups. Group 1 included 68 patients with unfavorable LV remodeling (iCDO increase  $>20\%$  and/or ICso  $>15\%$ ). Group 2 consisted of 62 patients without echocardiographic signs of cardiac remodeling. The development of unfavorable left ventricular remodeling after STEMI is associated with an 8.5-fold increase in the risk of cardiovascular events within 48 weeks after the index event.

**Keywords:** myocardial infarction, remodeling, working age, echocardiography, etc.

## Relevance

Coronary heart disease and its complications, in particular, myocardial infarction (MI), occupy a leading position among the causes of mortality and disability of the population of developed countries. Patients with MI, despite the widespread use of reperfusion therapy as a therapeutic technique that improves the early prognosis of the disease, are characterized by a high risk of complications both in the early (24-48 hours) and in the long-term periods of the disease [1, 5, 9, 15]. In this regard, the search for new specific and easy-to-use approaches to assessing the risk of developing MI complications, as well as the identification of promising molecular objects as targets for therapeutic effects in patients with MI continue to be relevant [2, 6, 10, 16].

The most common cause of MI is thrombotic occlusion of atherosclerotically altered coronary arteries with the development of coronary blood flow insufficiency (90-95% of all cases). Most of the hospital mortality occurs in the first two days from the moment of MI development, therefore, the main therapeutic measures are carried out during this period [3, 7, 11, 17]. Timely restoration of blood flow through the infarct-related artery leads to a reduction in the zone of myocardial necrosis up to complete functional restoration of the myocardium, which in turn determines the clinical prognosis and survival. Successful treatment of AMI depends on the degree of restoration of the lumen of the occluded artery and, accordingly, blood flow in the ischemic myocardial tissue, as well as the time elapsed between

occlusion and restoration of the vessel lumen [4, 12, 18]. That is why the earliest myocardial reperfusion is the pathophysiological basis for the treatment of acute myocardial infarction [5, 13, 19].

As is known, myocardial infarction causes noticeable changes in the cardiac structure, the most obvious of which is scarring of the infarction [15, 20]. Post-infarction changes are usually combined under the name "remodeling", which is a set of structural and geometric changes of the heart. The degree of cardiac remodeling is considered to be the main factor determining the development of heart failure [3, 17, 19]. Remodeling is associated with an inflammatory reaction and subsequent scar formation at the site of a heart attack [10, 14, 19]. Structural changes after a heart attack also extend to a viable myocardium and include cardiomyocyte hypertrophy, capillary network growth and an increase in interstitial collagen [12, 16, 18].

After a myocardial infarction, both short-term and long-term compensation mechanisms are activated. Although their activation is necessary early after a heart attack, they can have an adverse effect if they last for a longer time [15, 20]. Replacement of dead cardiomyocytes with fibrous tissue is a necessary stage of myocardial repair, since it helps to preserve its structural integrity. However, reconstruction of a viable myocardium can have negative consequences for cardiac function [10, 16].

Cardiac dysfunction after a heart attack is associated with two main problems: massive loss of cardiomyocytes, which form the basis of contractile function, and consequently changes in tissue structure as a result of scar tissue formation [15, 18]. It is obvious that cardiac function and structure are inextricably linked, therefore, intervention in post-infarction myocardial repair undoubtedly affects heart function [8, 11, 17]. The severity of tissue damage and death of cardiomyocytes caused by ischemia depends on several factors: the initial size of the infarction, the duration of ischemia, and the effectiveness of reperfusion [14].

**The purpose of the study.** To study the incidence of cardiovascular events in patients of working age after undergoing ST-segment elevation myocardial infarction (STeMI) with various cardiac remodeling options during 48 weeks of follow-up.

**Material and methods.** The study included 130 patients with STEMI (age  $51.2 \pm 18.8$  years) hospitalized in the departments of acute coronary syndrome and coronary heart disease and cardiac resuscitation of the Samarkand regional branch of the Republican Specialized Scientific and Practical Medical Center of Cardiology (SRF RSNPMC) in the first 24-96 hours from the onset of the disease. A prerequisite was the presence of hemodynamically significant stenosis of only one coronary artery according to the results of coronary angiography. Initially, on days 7-9 from the onset of the disease and after 24 weeks of follow-up, patients underwent standard echocardiography to determine the index of final diastolic volume (iCDO), the index of final systolic volume (ICso). After 24 weeks, the patients were divided into two groups. Group 1 included 68 patients with unfavorable LV remodeling (iCDO increase  $>20\%$  and/or ICso  $>15\%$ ). Group 2 consisted of 62 patients without echocardiographic signs of cardiac remodeling: the dynamics of iCDO after STEMI was less than 20%, ICO less than 15%. 48 weeks after STEMI, the frequency of endpoints was analyzed in the comparison groups: repeated myocardial infarction, cardiac surgery, unstable angina pectoris, hospitalization for decompensation of chronic heart failure, life-threatening rhythm disturbances.

**Results.** Endpoints in group 1 were identified in 30.2% (n=19): 17 patients were hospitalized for unstable angina (21.1%); 1 patient (1.6%) was diagnosed with recurrent AMI; 2 people (3.2%) with decompensation of chronic heart failure (CHF). Cardiac surgery was performed in 17 (31.1%) patients. Life-threatening rhythm disorders were diagnosed in 12 (23.2%) patients. In group 2, endpoints were diagnosed in 13 (14.8%) people. 11 (2.6%) patients underwent cardiac surgery. 2 (3.2%) patients were hospitalized for unstable angina pectoris. The ratio of the chances of developing an unfavorable outcome in group 1 compared with group 2 was 8.5 (95% CI 2.4—30.5) (p=0.0004). To analyze the relationship between the processes of cardiac and vascular remodeling, patients were divided into 2 groups: group 1 – with carotid artery TIM less than 0.9 mm and group 2 – with carotid artery TIM equal to or more than 0.9 mm. The results of the echocardiography study demonstrated that in group 2 individuals, more pronounced processes of remodeling of the left ventricular myocardium (LV) and

violations of its diastolic function were revealed. This is supported by high values (CDR) and diastolic thickness of the posterior LV wall (DT LVL), and a significant increase in LVL in comparison with group 1, combined with relative wall thickness (OTC), suggests the formation of concentric LV hypertrophy in individuals with increased TIM, in parallel with vascular remodeling. In patients of the 2nd group, significantly high indicators were found that characterize the contractility of the myocardium (ejection fraction - PV and shock volume - UO), but the detection of large left atrium (LP) and E/A index in them indicates a tendency, in comparison with the 1st group, to the formation of diastolic LV dysfunction.

**Conclusions:** thus, the development of unfavorable left ventricular remodeling after STEMI is associated with an 8.5-fold increase in the risk of cardiovascular events within 48 weeks after the index event.

#### List of literature:

1. Thygesen, Kristian, et al. "Четвертое универсальное определение инфаркта миокарда (2018)." *Российский кардиологический журнал* 24.3 (2019): 107-138.
2. Аскарлов И. К. и др. Предикторы Кардиопротекции Пациентов Хронической Сердечной Недостаточности, Как Последствие Инфаркта Миокарда //Periodica Journal of Modern Philosophy, Social Sciences and Humanities. – 2023. – Т. 17. – С. 137-140.
3. Жураева, Х. И., Каюмов, Л. Х., Убайдова, Д. С., & Джабборов, Ж. Ж. (2019). Взаимосвязь инфаркта миокарда с метаболическим синдромом. *Биология и интегративная медицина*, (4 (32)), 66-77.
4. Кодирова Г. И. и др. Нарушения процессов пероксидации и иммунной системы у больных инфарктом миокарда //Евразийский кардиологический журнал. – 2019. – №. S1. – С. 212.
5. Кузьмичев, Денис Евгеньевич, et al. "Инфаркт миокарда в клинике." *Проблемы экспертизы в медицине* 15.1-2 (57-58) (2015): 49-51.
6. Мадалиев А. У., Байкузиев У. К., Махмудов Н. И. Наблюдение идентичной локализации случаев инфаркта миокарда в определенный промежуток //Евразийский кардиологический журнал. – 2019. – №. S1. – С. 215.
7. Миронова О. Ю. Инфаркт миокарда типа 4а //Терапевтический архив. – 2014. – Т. 86. – №. 9. – С. 102-107.
8. Пулатов, Шухрат Шуропович, Амира Асроровна Рузиева, and Фарида Одыловна Хасанжанова. "Аспекты Кардиопротекции Пациентов Хронической Сердечной Недостаточности, Как Последствие Инфаркта Миокарда." *Periodica Journal of Modern Philosophy, Social Sciences and Humanities* 17 (2023): 133-136.
9. Ризаев Ж. А., Саидов М. А., Хасанжанова Ф. О. Современные тенденции распространенности и исхода сердечно-сосудистых заболеваний среди населения республики Узбекистан //Journal of cardiorespiratory research. – 2023. – Т. 1. – №. 1. – С. 18-23.
10. Ризаев Ж. А., Саидов М. А., Хасанжанова Ф. О. Статистический анализ информированности кардиологических и кардиохирургических пациентов о высокотехнологичной медицинской помощи в Самаркандской области //Вестник науки. – 2023. – Т. 1. – №. 11 (68). – С. 992-1006.
11. Ташкенбаева Э. Н. и др. Предикторы развития сердечно-сосудистых осложнений у больных с острым инфарктом миокарда с подъемом сегмента ST //Наука и общество в эпоху перемен. – 2018. – №. 1. – С. 12-15.

12. Хасанжанова Ф. О. и др. Клиническое течение хронической сердечной недостаточности от локализации острого инфаркта миокарда //Евразийский кардиологический журнал. – 2019. – №. S1. – С. 221.
13. Хасанжанова Ф. О. и др. Оценка влияния рамиприла и пириндоприла на систолическую функцию левого желудочка у больных с острым инфарктом миокарда без элевации сегмента ST //Central Asian Journal of Medical and Natural Science. – 2023. – Т. 4. – №. 3. – С. 461-465.
14. Хасанжанова Ф. О. и др. Различия в частоте развития основных осложнений у больных с острым инфарктом миокарда //Актуальные научные исследования в современном мире. – 2018. – №. 10-6. – С. 39-41.
15. Хасанжанова Ф. О. и др. Роль изменения маркеров некроза кардиомиоцитов у больных инфарктом миокарда в зависимости от возраста //Актуальные научные исследования в современном мире. – 2018. – №. 10-6. – С. 42-45.
16. Хасанжанова Ф. О., Авазова Х. А. Особенности Клинического Течения Инфаркта Миокарда С Хронической Сердечной Недостаточностью У Больных В Молодом Возрасте //Central Asian Journal of Medical and Natural Science. – 2023. – Т. 4. – №. 2. – С. 637-640.
17. Хасанжанова, Ф. О., et al. "Экспериментально-Клинические Исследования Хронической Сердечной Недостаточности С Острым Инфарктом Миокарда У Мужчин Молодого Возраста." *Central Asian Journal of Medical and Natural Science* 4.3 (2023): 1021-1024.
18. Хасанжанова, Фарида Одыловна, and Мумин Шамсиевич Рофеев. "Часто встречаемые факторы риска при инфаркте миокарда у мужчин молодого возраста при разных исходах заболевания." *Актуальные научные исследования в современном мире* 10-7 (2019): 87-90.
19. Чаулин А. М. и др. Способ неинвазивной диагностики острого инфаркта миокарда. 2020 //Номер патента: RU. – 2020. – Т. 2736001. – С. С1.
20. Чаулин А. М., Дупляков Д. В. Биомаркеры острого инфаркта миокарда: диагностическая и прогностическая ценность. Часть 2 (обзор литературы) //Клиническая практика. – 2020. – Т. 11. – №. 4. – С. 70-82.