

EARLY COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION WITH ST SEGMENT ELEVATION**Ibragimova N.U.**

Tashkent Medical Academy

Mavlonberdiyev S.S.

Tashkent Regional Specialized Somatic Hospital

Aripov Sh. M.

Samarkand State Medical University

Tuyimova G. O'.

Samarkand State Medical University

Received: Apr 04, 2024; Accepted: May 03, 2024; Published: Jun 04, 2024;

Abstract: Myocardial infarction is a dangerous disease that causes irreversible changes in the heart. The disease mainly occurs in people over 50 years of age. According to statistics, in general, about 60% of people over the age of 65 have experienced a heart attack. However, younger people are also at risk of the disease. Assess the true natural history of acute myocardial infarction is difficult for a number of reasons: the possibility of hidden infarction, the high frequency of acute coronary death outside the hospital and the difference methods for diagnosing this condition.

Keywords: myocardial infarction, ST elevation, ischemia

This is an open-access article under the [CC-BY 4.0](https://creativecommons.org/licenses/by/4.0/) license

Introduction

A heart attack is tissue necrosis due to disruption of local blood circulation in an organ (ischemia). Myocardium is the muscle tissue of the heart. It consistently contracts and relaxes in various parts - the ventricles and atria, causing the heart to beat and pump blood.

Myocardial infarction is an acute form of coronary heart disease, in which the blood supply to the myocardium (the main part of the heart mass) is completely or partially stopped. The ECG may show signs of ischemia myocardium (ST-T changes) and necrosis (changes QRS complex). Myocardial infarction can be diagnosed with an increase in the level of biomarkers in the blood serum against the background of clinical signs of acute myocardial ischemia.

Etiology and Pathogenesis

The cause of acute coronary syndrome is almost always a sudden deterioration in coronary blood flow as a result of thrombosis against the background of atherosclerosis, which may or may not be accompanied by vasospasm [1].

Rupture of an atherosclerotic plaque can occur for the following reasons:

- a sharp increase in blood pressure;
- high physical activity;
- high content of oxidized lipoproteins having low density.

As a result of the rupture, a blockage of the artery occurs, since the resulting thrombus fills not only the internal space of the damaged plaque, but also the lumen of the coronary artery. In turn, cessation of blood flow leads to necrosis of myocardial tissue and a heart attack.

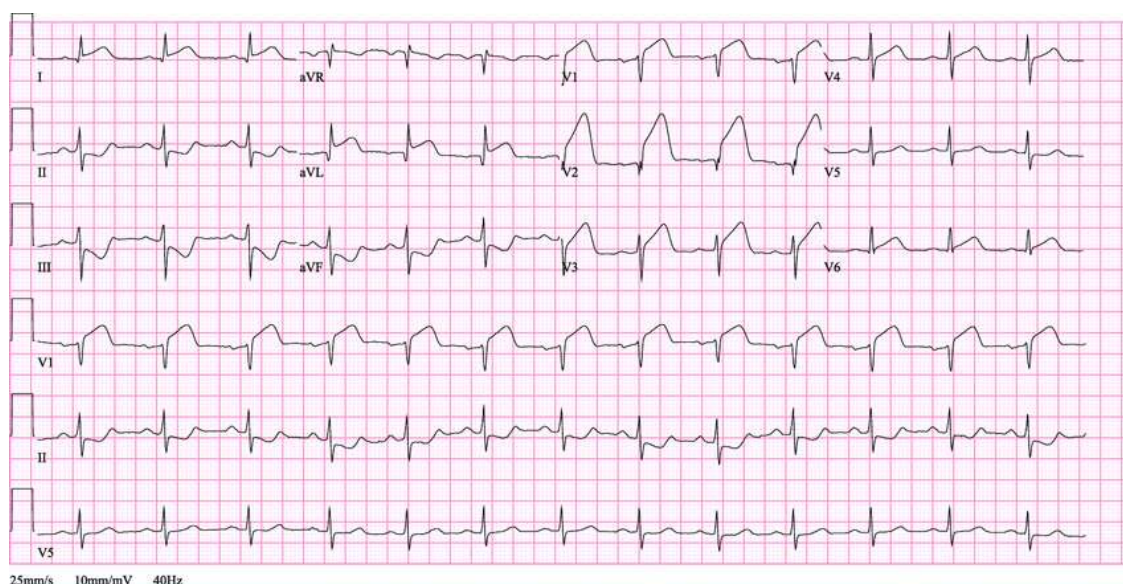
Most patients with STEMI, even with timely treatment, develop myocardial necrosis of varying sizes. The amount of dying and dying myocardium is almost linearly related to the threat of circulatory arrest caused by cardiac asystole or fatal ventricular arrhythmias, primarily ventricular fibrillation. The size and transmural extent of necrosis determine the risk of developing another potentially fatal complication of myocardial rupture. A consequence of the loss of a significant part of the active myocardium is the process of cardiac remodeling. The formation of necrosis in the myocardium is accompanied by changes in the size, shape and thickness of the wall of the left ventricle (LV), and the remaining myocardium experiences increased stress and undergoes hypertrophy. The pumping function of the reshaped LV deteriorates, which leads to the appearance of heart failure (HF).

Symptoms

1. Typical - with pain in the heart, behind the sternum, cold sweat, increased anxiety and an inexplicable fear of death;
2. Atypical - with unusual pain manifestations.
 - peripheral - with pain in various parts of the body: arm, back, lower jaw;
 - abdominal - pain in the stomach area, radiating into the chest, sometimes accompanied by nausea and vomiting;
3. Atypical myocardial infarction without pain:
 - asthmatic - symptoms similar to an asthma attack with shortness of breath;
 - arrhythmic - manifested by disturbances in heart rhythm;
 - cerebral - associated with impaired cerebral circulation, may be similar to a stroke. Nausea, dizziness, and loss of consciousness may also be present;
 - low-symptomatic - a dangerous and unfavorable form, since according to the patient's feelings, the signs of a heart attack are indistinguishable or completely absent, which makes self-diagnosis and timely calling an ambulance difficult.

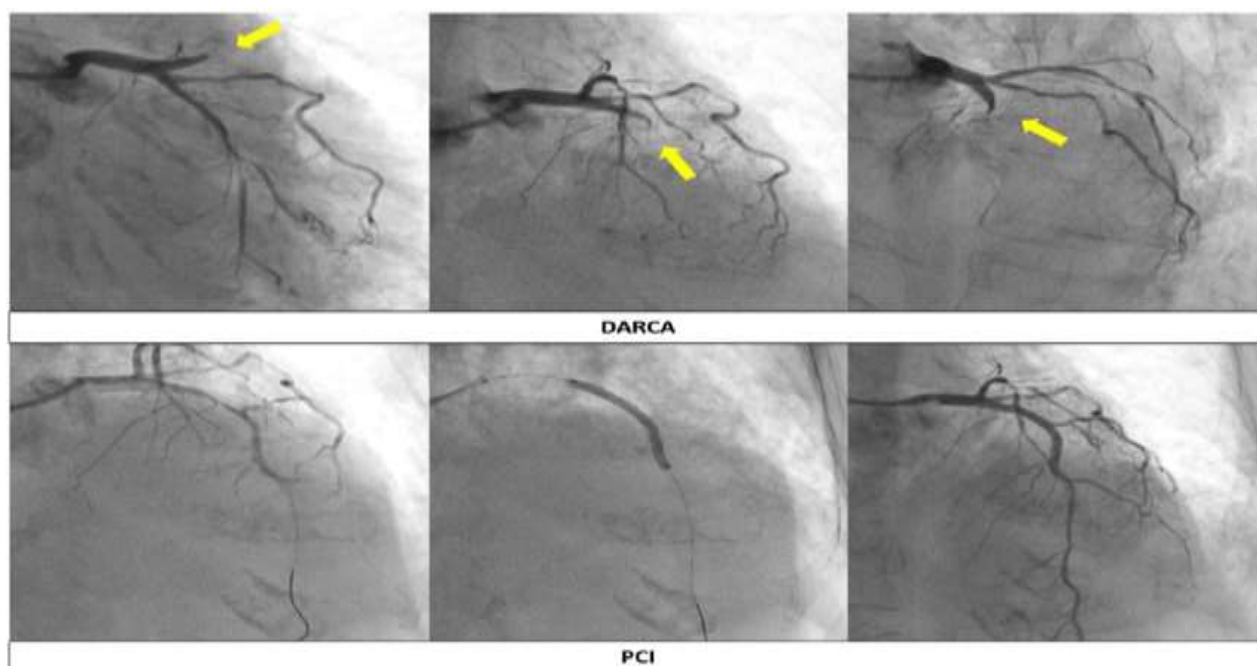
Diagnosis

If there are signs of myocardial infarction, compares electrocardiography readings obtained during the attack and previously recorded ECGs. The ECG may show signs of ischemia myocardium (ST-T changes) and necrosis (changes QRS complex). Working criteria for developing myocardial infarction in the presence of corresponding clinical symptoms have been identified: (1) for the first time detected ST segment elevation (at point J ≥ 0.2 mV in leads V1-3 and ≥ 0.1 mm in other leads) and (2) ST segment depression or T wave changes. Signs of a mature myocardial infarction any Q wave in leads V1-3 or a wide Q wave ≥ 0.03 s in leads I, II, aVL, aVF, V4-6 serve.



Also an integral part of the diagnosis are a general blood test and a biochemical analysis of blood serum. In patients with MI, to confirm the diagnosis, it is recommended to determine the dynamics of the level of biochemical markers of cardiomyocyte damage in the blood, preferably a study of the level of cardiac troponin I or T.

Carrying out echocardiography in case of suspected myocardial infarction helps to timely determine disturbances in myocardial contractility and the presence of complications that have developed as a result of the infarction. If it is difficult to establish a diagnosis, the ECG and tests have low information content, the cardiologist prescribes emergency coronary angiography, which allows to determine the presence of blood clots in the coronary artery that caused myocardial infarction, and the degree of ventricular contractility, as well as the possibility of bypass surgery or angioplasty.



Result and Discussion

Treatment

There are 4 phases of treatment.

1. Emergency treatment, when it is necessary to quickly establish a diagnosis and assess the risk, stop

pain and prevent cardiac arrest.

2. Acute phase - reperfusion should be carried out as quickly as possible to limit the spread of myocardial infarction and combat acute complications such as acute heart failure, shock and life-threatening arrhythmias.

3. Subacute phase - treatment is carried out for later complications.

4. Risk assessment and measures to prevent the progression of coronary atherosclerosis, the development of recurrent infarction, heart failure and of death.

Complications

During the period of developing MI (from 0 to 6 hours), severe rhythm disturbances and acute cardiovascular failure may develop.

Complications of the acute period of MI (from 6 hours to 7 days) include:

- ❖ rhythm and conduction disturbances,
- ❖ shock (cardiogenic, reflex and arrhythmic),
- ❖ cardiac asthma and pulmonary edema (manifestations of acute left ventricular failure),
- ❖ acute cardiac aneurysm,
- ❖ heart breaks,
- ❖ thromboembolic complications in BCC and MCC,
- ❖ paresis of the stomach and intestines, erosive gastritis with gastric bleeding, pancreatitis.

During the period of healing (scarring) MI (from 7 to 28 days), the following complications may develop:

- ❖ thromboendocarditis with thromboembolic syndrome;
- ❖ pneumonia;
- ❖ post-infarction syndrome - Dressler's syndrome (pericarditis, pleurisy, pneumonitis), anterior chest wall syndrome, shoulder syndrome;
- ❖ chronic left ventricular heart failure;
- ❖ the beginning of the formation of a chronic cardiac aneurysm;
- ❖ mental disorders (usually neurosis-like syndromes).

In the post-infarction period (starting from day 29), the formation of a chronic aneurysm ends, and manifestations of Dressler's syndrome and symptoms of CHF may remain.

- ✓ Cardiogenic shock. Cardiogenic shock is a life-threatening condition caused by a sharp decrease in cardiac output (CO) and manifested by severe hypoperfusion of organs and tissues and hypoxemia. The main clinical sign of cardiogenic shock is persistent hypotension ($SBP \leq 90$ mm Hg), refractory to fluid resuscitation and accompanied by signs of acute multiple organ failure as a result of hypoperfusion.
- ✓ Myocardial dysfunction. Myocardial dysfunction is the most common complication of MI. The cause of dysfunction is the death of cardiomyocytes and their stunning (stunning) due to myocardial ischemia. LV dysfunction may be asymptomatic. Severe impairment of systolic function leads to clinical manifestations of HF.
- ✓ LV aneurysm develops in less than 5% of patients with large transmural MI as a result of unfavorable myocardial remodeling. Its manifestation is usually HF, although HF is a frequent complication even with anterior localization of MI even without the formation of an LV aneurysm. Mitral valve insufficiency (MV) with regurgitation can develop due to LV remodeling with lateral-apical displacement of the papillary muscles, which limits the mobility of the chordae and, accordingly, the valve leaflets and dilatation of the valve annulus. This complication is most often late, but can also occur in the subacute period with extensive

LV MI, most often of posterolateral localization (with dysfunction of the posteromedial papillary muscle). For patients with complicated right ventricular infarction, the typical presentation is the triad of hypotension, normal pulmonary chest radiographs, and elevated jugular venous pressure.

- ✓ LV free wall rupture occurs in less than one percent of patients during the first week after transmural infarction and may be accompanied by sudden pain and/or cardiovascular collapse, with or without arrhythmias. Old age, lack of reperfusion or late fibrinolysis increases the likelihood of its development. The development of hemopericardium and tamponade usually quickly leads to death.
- ✓ Ventricular septal rupture usually presents subacutely as a sudden onset condition with acute HF or cardiogenic shock and is manifested by the appearance of a loud systolic murmur on auscultation.

Rupture of the papillary muscle with acute mitral regurgitation may occur 2-7 days after MI due to rupture of the papillary muscle or chordae tendineae. Clinically, it usually manifests as a sudden deterioration in hemodynamics with acute dyspnea, pulmonary edema, and/or cardiogenic shock. On auscultation, the appearance of a systolic murmur of regurgitation is typical

Conclusion

Myocardial infarction is a disease that develops acutely and causes a number of early and late complications. Early and late complications that develop after myocardial infarction differ, and they develop differently depending on the localization of the infarction zone and the size of the affected area. Each of the above complications is dangerous for the patient's life. Correctly conducted treatment tactics improve the patient's quality of life after the disease and prevent the development of complications..

References

- [1]. „ЛЕЧЕНИЕ ОСТРОГО ИНФАРКТА МИОКАРДА С ПОДЪЕМОМ СЕГМЕНТА ST” F. Van de Werf (председатель), D. Ardissino, A. Betriu, D. Cokkinos, E. Falk, K. Fox, D. Julian, M. Lengyel, F.J. Neumann, W. Ruzyllo, C. Thygesen, R. Underwood, A. Vahanian, F. Verheugt, W. Wijns
- [2]. Gurevich MA, Mravyan SR, Grigorieva NM. Coronary heart disease in women. Difficult Patient. 2006;12:1-13. (In Russ.) Гуревич М.А., Мравян С.Р., Григорьева Н.М. Ишемическая болезнь сердца у женщин. Трудный пациент. 2006;12:1-13.
- [3]. Изменения структуры осложнений при остром инфаркте миокарда в течение десяти лет наблюдения: гендерные особенности
- [4]. Округин С.А., Репин А.Н. Кардиоваскулярная терапия и профилактика. 2020;19(3)
- [5]. „ОСЛОЖНЕНИЯ ОСТРОГО КОРОНАРНОГО СИНДРОМА. ДИАГНОЗ, ДИФФЕРЕНЦИАЛЬНЫЙ ДИАГНОЗ, ЛЕЧЕНИЕ.” Е. С. ЕНИСЕЕВА Учебное пособие.
- [6]. Гуландом Зикрияевна Шодикулова, Алишер Шавкатович Нажмиддинов. СОВРЕМЕННЫЙ ВЗГЛЯД НА ПРОБЛЕМУ НАРУШЕНИЯ СЕРДЕЧНОГО РИТМА У ПАЦИЕНТОВ С ИНТЕРСТИЦИАЛЬНОЙ ПНЕВМОНИЕЙ (ОБЗОР ЛИТЕРАТУРЫ) the theory of recent scientific research in the field of pedagogy. 2023/3/3.
- [7]. Najmiddinov Alisher Shavkat o‘g‘li. POTENTIAL OF IMIDAZOLINE RECEPTOR AGONISTS IN THE TREATMENT OF ARTERIAL HYPERTENSION. World Bulletin of

Public Health (WBPH) t Volume-28, November 2023. ISSN: 2749-3644. Available Online at: <https://www.scholarexpress.ne>

- [8]. Д. Р. Зокирова, Г. Ш. Норова, & Э. З. Рахимов (2012). Влияние L-лизина эсцината на клиническое состояние больных острой гипертонической энцефалопатией. Вестник экстренной медицины, (3), 38-39.
- [9]. Norova Gulnoz Sharifovna. (2023). FEATURES OF THE QUALITY OF LIFE OF PATIENTS WITH RESPIRATORY DISEASES AND THEIR DYNAMICS DURING STATIONARY TREATMENT. International Journal of Integrative and Modern Medicine, 1(1), 66–73. Retrieved from <http://medicaljournals.eu/index.php/IJIMM/article/view/25>
- [10]. Норова, Г. Ш., & Фаттоева, Н. Д. (2023). ГИПЕРТОНИЯ КАСАЛЛИГИДА УЧРАЙДИГАН ПСИХО-ЭМОЦИОНАЛ БУЗИЛИШЛАРНИ БАХОЛАШ ВА ДАВОЛАШ. Solution of social problems in management and economy, 2(8), 30-31.
- [11]. Рахимов, Э. З., Норова, Г. Ш., Тилавова, М. М., & Фаттоева, Н. Д. (2023, July). ИЗУЧЕНИЕ ЭФФЕКТИВНОСТИ И БЕЗОПАСНОСТИ ПРЕПАРАТА ИЗ ГРУППЫ АРНИ У БОЛЬНЫХ С ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ. In Past and Future of Medicine: International Scientific and Practical Conference (Vol. 2, pp. 13-19).
- [12]. Зиедуллаев, А. Э., Каюмова, Г. К., Норова, Г. Ш., Тилавова, М. М., & Рахимов, Э. З. (2019). ПРИМЕНЕНИЕ КАРВЕДИЛОЛА У БОЛЬНЫХ С ДИЛАТАЦИОННОЙ КАРДИОМИОПАТИЕЙ. In Молодежь, наука, медицина (pp. 399-401).
- [13]. Кенжаев, М. Л., Рахимов, Э. З., Норова, Г. Ш., Акрамова, Ш. Р., & Закирова, Д. Р. (2018). Применение кораксана у больных с кардиоваскулярной патологией в сочетании с хронической обструктивной болезнью легких. Достижения науки и образования, (13 (35)), 60-62.
- [14]. Рахимов, Э. З., Норова, Г. Ш., & Акрамова, Ш. Р. (2013). Оценка влияния терапии кораксаном на клинико-функциональные показатели у больных с кардиоваскулярной патологией и хронической обструктивной болезнью легких. Вестник экстренной медицины, (3), 233-233.