

## Determine The Risk Of Long-Term Complications In Patients With Acute Coronary Syndrome

Maksudova M.X.1, Qodirova G.R.2, Nadzhmiddinov A.Sh. 3  
Tashkent medical academy, Tashkent, Uzbekistan.

*Received: Apr 02, 2024; Accepted: May 02, 2024; Published: Jun 03, 2024;*

**Abstract:** The article is devoted to one of the current topics of modern healthcare - acute coronary syndrome (ACS). Most patients had associated and comorbid diseases, primarily such as stable angina, chronic heart failure, broncho-obstructive diseases and diabetes mellitus, which in some cases required treatment adjustments [1]. The article provides information on the etiology of acute coronary syndrome, clinical symptoms and risk factors that may develop in patients.

**Keywords:** Acute Coronary Syndrome, Unstable Angina, Myocardial Infarction.



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

### Introduction

Acute coronary syndrome (ACS) is the most dangerous variant of the clinical course of coronary heart disease (CHD). The danger of the situation is that ACS, unlike chronic ischemic heart disease, is characterized by a rapid (hours) and sometimes rapid (minutes) course of the disease, a high risk of adverse outcomes (sudden coronary death, myocardial infarction) and requires immediate measures to save the lives of patients. The term “acute coronary syndrome” combines such clinical conditions as unstable angina and myocardial infarction. The term “ACS” is used when diagnostic information is not yet sufficient for a final judgment about the presence or absence of foci of necrosis in the myocardium and, therefore, represents a preliminary diagnosis in the first hours and days of the disease, while the terms “MI” and “unstable angina” used in formulating the final diagnosis. Accordingly, the term “ACS” can be used at the prehospital or early hospital stages and is subsequently transformed into the diagnosis of “acute MI”, “UA” or, according to the results of differential diagnosis, into any other diagnosis, including non-cardiological.

Patients with acute chest pain but no ST-segment elevation on the ECG are classified as non-ST-segment elevation ACS, reflecting the presence of transient partial coronary artery occlusion or distal embolization from thrombus fragments or ruptured plaque. ECG changes may include transient ST segment elevation (<20 min), persistent or transient ST segment depression, T wave inversion, flattening or the ECG may be normal. The main thing in the management of patients with NSTEMI-ACS at all stages of care is, along with diagnosis, constant stratification of the risk of developing cardiac complications.

Acute coronary syndromes include:

1. Unstable angina

2. Non-ST segment elevation myocardial infarction (NSTEMI)
3. ST-segment elevation myocardial infarction (STEMI)

Unstable angina (acute coronary insufficiency, pre-infarction angina, intermediate coronary syndrome) is defined as one or more of the following conditions in patients whose cardiac markers do not meet criteria for myocardial infarction (MI):

1. Angina at rest that lasts for a long time (usually > 20 minutes)
2. New-onset angina
3. Increased angina

ECG changes such as ST segment depression or elevation or T wave inversion may occur with unstable angina but are transient. As for cardiac markers, creatine kinase is not increased, but cardiac troponin levels may be slightly increased, especially when measured with high-sensitivity troponin (hs-cTn) tests. Unstable angina is a transient clinical condition and often precedes myocardial infarction, arrhythmias, or sudden death.

Non-ST segment elevation myocardial infarction (NSTEMI, subendocardial myocardial infarction) is necrosis of the heart muscle (confirmed by analysis of cardiac markers in the blood: elevated levels of troponin T or troponin I and creatine kinase) that is not accompanied by acute ST segment elevation on the ECG. NSTEMI is characterized by ECG changes such as ST segment depression, T wave inversion, or a combination of both.

Myocardial infarction with ST segment elevation (STEMI, transmural myocardial infarction) is necrosis of cardiomyocytes, accompanied by persistent ST segment elevation on the ECG, which does not disappear after taking nitroglycerin or the first appearance of left bundle branch block. Troponin I or troponin T and creatine kinase (CK) are elevated.

#### Prognosis for acute coronary syndromes

Global risk should be assessed using formal clinical risk scores (thrombolysis in myocardial infarction [TIMI], Global Registry of Acute Coronary Events [GRACE], platelet glycoprotein IIb/IIIa for unstable angina: receptor inhibition by use integrilin [PURSUIT]—1) or a combination of the following high-risk factors:

1. Intermittent angina/ischaemia at rest or during low activity
2. Heart failure
3. Worsening mitral regurgitation
4. High risk stress test result (test stopped after  $\leq 5$  minutes due to onset of symptoms, significant ECG abnormalities, hypotension, or complex ventricular arrhythmias)
5. Hemodynamic instability
6. Sustained ventricular tachycardia
7. Diabetes
8. PCI within the past 6 months
9. Previous CABG
10. LVEF <0.40-spasi-

## Results and Discussion

### Etiology and Pathophysiology

- Formation of a thrombus in an atherosclerotic coronary artery
- Coronary artery embolism
- Coronary spasm
- Spontaneous coronary artery dissection

The atherosclerotic plaque sometimes becomes unstable or inflamed, causing it to rupture or cleave, exposing thrombogenic material that activates platelets and the coagulation cascade and produces an acute thrombus. Platelet activation involves conformational changes in platelet membrane glycoprotein (GP) IIb/IIIa receptors, leading to cross-linking (and thus aggregation) of platelets. Even if the atheroma causes minimal obstruction, its rupture can lead to thrombosis; in >50% of cases the degree of previous stenosis is <40%.

Coronary artery embolism can occur with mitral stenosis, aortic stenosis, infective endocarditis, arrowroot endocarditis, or atrial fibrillation.

When using cocaine and other drugs, coronary artery spasm and myocardial infarction may develop. The development of spasm responsible for myocardial infarction is possible in both intact and atherosclerotic coronary arteries.

Spontaneous coronary artery dissection is a non-traumatic rupture of the intima of a coronary artery with the formation of a false lumen. Blood flowing through such a false lumen expands it, which restricts blood flow in the true lumen, sometimes causing cardiac ischemia or infarction.

Wall dissection can occur in both intact and atherosclerotic coronary arteries. Nonatherosclerotic dissection is more likely in women during pregnancy or the puerperium and in patients with fibromuscular dysplasia or other connective tissue disorders.

### Symptoms and signs

Clinical manifestations of acute coronary syndrome depend on the location and severity of changes in the coronary artery and are quite variable. Painful stimuli originating from the chest organs, including the heart, can cause discomfort described as pressure, tearing, gas with belching, dyspepsia, burning, aching or stabbing pain, and sometimes as needle-sharp pain. Many patients deny that they have pain and insist that it is more of a “discomfort.” Except for those cases when the MI is widespread, large-focal, it is difficult to judge the volume of ischemic myocardium only from clinical data.

## Diagnostics

- ECG in dynamics
- Measuring the level of cardiac markers over time
- Coronary angiography for emergency indications in patients with STEMI or its complications (persisting anginal pain, hypotension, significant increase in marker levels, rhythm disturbances)
- Delayed angiography (24–48 hours) for patients with NSTEMI or unstable angina without the above complications

In STEMI, an ECG is a diagnostic method that detects ST segment elevation  $\geq 1$  mm in 2 or more adjacent leads, reflecting the activity of the damaged myocardial area. Carrying out an ECG diagnosis of myocardial infarction is difficult if the patient has a left bundle branch block, since the ECG changes resemble those caused by STEMI.

Markers of myocardial damage (serum markers of myocardial cell damage) are

- Cardiac enzymes (eg, CK-MB [creatine kinase-MB])
- Intracellular contents (eg, troponin I, troponin T, myoglobin)

These markers are released into the bloodstream after myocardial cell necrosis. Markers are detected in the blood at different times from the onset of damage, and their levels increase at different rates.

Coronary angiography typically combines diagnostic testing with percutaneous coronary intervention (PCI—ie, angioplasty, stent placement). If possible, emergency coronary angiography and PCI are performed as soon as possible after the onset of acute MI (primary PCI). In many tertiary centers, this approach has significantly reduced morbidity and mortality and improved long-term outcomes. Often, the development of a heart attack is actually interrupted if a short time has passed from the onset of pain to PCI (< 3-4 hours).

## Complications

After the completion of acute manifestations of coronary obstruction, complications of the disease may develop. They usually include

- Electrical dysfunction (eg, conduction defects, arrhythmias)
- Myocardial dysfunction (eg, heart failure, ventricular free wall or interventricular septal rupture, ventricular aneurysm, pseudoaneurysm, ventricular thrombus formation, cardiogenic shock)
- Valvular dysfunction (usually mitral regurgitation)

Violation of the electrical properties of the myocardium is possible in any form of ACS; the appearance of myocardial dysfunction usually indicates a large volume of ischemic myocardium. Other complications of ACS include recurrent myocardial ischemia and the development of pericarditis. Pericarditis that develops 2 to 10 weeks after the onset of myocardial infarction is known as post-infarction syndrome or Dressler's syndrome.

### Conclusion

Modern treatment technologies, rehabilitation therapy, rational employment and strict adherence to preventive protective measures make it possible to stabilize the course of the disease, avoid complications, and also improve the prognosis and quality of life of patients with acute coronary syndrome.

### References

- [1] „Обзор острых коронарных синдромов (ОКС) (Overview of Acute Coronary Syndromes (ACS))” Ranya N. Sweis, MD, MS, Northwestern University Feinberg School of Medicine;
- [2] Arif Jivan, MD, PhD, Northwestern University Feinberg School of Medicine
- [3] „Acute Coronary Syndrome” <https://www.heart.org/en/about-us/heart-attack-and-stroke-symptoms>
- [4] ОКС : Тактика на догоспитальном этапе. А.В. Мелехов; К.И.Островская 2018г.
- [5] Аляви, А. Л., Турсунова, Л. Д., Бувамухамедова, Н. Т., & Жуманазаров, С. Б. (2021). Течение сердечно-сосудистых заболеваний у больных COVID-19. Студенческий вестник, (17-4), 38-41.
- [6] Косимбаев, С., Аляви, А., Турсунова, Л., & Жуманазаров, С. (2021). Агрегатное состояние крови и коагуляционный гемостаз у больных с ишемической болезнью сердца перенесших COVID-19.
- [7] Jabbarov, O. O., Daminov, V. T., & Tursunova, L. D. (2019). Роль метаболических факторов в прогрессировании диабетической нефропатии. Вестник Ташкентской медицинской академии, (4), 25-29.
- [8] Tursunova, L. D., & Jabbarov, O. O. (2021). APPLICATION OF SAKABUTRIL/VALSARTAN IN PATIENTS WITH CHRONIC KIDNEY DISEASE WITH TYPE 2 DIABETES MELLITUS. Art of Medicine. International Medical Scientific Journal, 1(1).
- [9] «O'tkir koronar sindrom S-T segment ko'tarilishi bilan kechgan bemorlarda koronaroangioplastika va trombolitik davoning samaradorligini baholash» <http://repository.tma.uz/xmlui/handle/1/2439> Kurbanova Dilnoza Husanbayevna, Saydaliyev Rustam Saydaliyevich, Tursunova Laylo Dilshatovna.
- [10] „Acute Coronary Syndrome: Current Treatment” TIMOTHY L. SWITAJ, MD, SCOTT R. Am Fam Physician. 2017;95(4):232-240
- [11] Acute coronary syndrome in cancer patients. Part I: pathophysiology, clinical presentation and diagnosis  
AUTHORS Grzegorz Piotrowski\* 1, 2
- [12] Мирзаева, Г. П., & Турсунова, Л. Д. (2019). НЕКОТОРЫЕ ПОКАЗАТЕЛИ СУТОЧНОГО МОНИТОРИРОВАНИЯ АРТЕРИАЛЬНОГО ДАВЛЕНИЯ У БОЛЬНЫХ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ. In Противоречия современной кардиологии: спорные и нерешенные вопросы (pp. 40-40).
- [13] 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>

- [14] 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).
- [15] Зиедуллаев, А. Э., Каюмова, Г. К., Норова, Г. Ш., Тилявова, М. М., & Рахимов, Э. З. (2019). ПРИМЕНЕНИЕ КАРВЕДИЛОЛА У БОЛЬНЫХ С ДИЛАТАЦИОННОЙ КАРДИОМИОПАТИЕЙ. In Молодежь, наука, медицина (pp. 399-401).
- [16] Кенжаев, М. Л., Рахимов, Э. З., Норова, Г. Ш., Акрамова, Ш. Р., & Закирова, Д. Р. (2018). Применение кораксана у больных с сердечно-сосудистой патологией в сочетании с хронической обструктивной болезнью легких. Достижения науки и образования, (13 (35)), 60-62.
- [17] Рахимов, Э. З., Норова, Г. Ш., & Акрамова, Ш. Р. (2013). Оценка влияния терапии кораксаном на клинико-функциональные показатели у больных с сердечно-сосудистой патологией и хронической обструктивной болезнью легких. Вестник экстренной медицины, (3), 233-233.