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Patients with Acute Coronary Syndrome Complicated by Arrhythmias at the Stages of Treatment

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Relevance. Coronary heart disease is one of the most widespread diseases of our time, which in all countries of the world tend to rejuvenate, increase the number of patients and involve different segments of the population in the pathological process. Coronary heart disease occupies the first place in the structure of forensic medical research of 17 sudden deaths. According to the Flemingham study, VCS and AMI are the first manifestation of coronary heart disease in 59.3% of men and 43.5% of women. The probability of sudden death in the first hours of AMI is especially high. Diagnosis of this disease is significantly difficult, since there are no clinical manifestations in its early stages, while acute focal lesions of muscle fibers occur in the myocardium, which leads to cardiac arrest. Ischemic disease is characterized by alternating periods of exacerbation and remission. During the period of exacerbation, patients develop unstable angina (NS), myocardial infarction (MI) without a Q wave, myocardial infarction with a Q wave, and sudden cardiac death develops (V. S. Botasheva, F.A. Barotov, 2015). Acute coronary syndrome is a concept that includes different clinical types of exacerbation of coronary heart disease. Acute coronary syndrome is a set of symptoms and clinical manifestations of the period of exacerbation of coronary heart disease on the basis of which it is possible to recognize the beginning of NS or MI.

Unstable angina and myocardial infarction are based on coronary artery thrombosis. A blood clot forms at the site of damage to the integrity of the atherosclerotic plaque. The severity of thrombosis varies. ACS remains one of the leading causes of hospitalization and mortality of patients in industrialized countries. ACS includes the early stages of MI, in which the highest risk of death is observed. In this regard, it is important to have the correct and adequate treatment tactics on which the outcome of the disease depends. In this regard, the allocation of ACS as a group concept is of great practical importance.

With unstable angina pectoris, platelet masses (parietal "white" trom) are formed in the lumen of the coronary artery, which, as a result of proper treatment, or spontaneously dissolve, i.e., thrombolysis is observed. Unstable angina accounts for 75-80% of all cases of ACS. Nontransmural myocardial infarction without a Q wave is observed with the formation of an obstructing loose thrombus, a significant decrease in blood flow and prolonged occlusion (up to 1 hour) of the coronary artery. With the formation of a strong (mixed) thrombus, which is well fixed to the wall of the coronary artery and complete occlusion of the vessel lumen, a transmural myocardial infarction is observed. There are two forms of ACS depending on the presence or absence of ST segment elevation on the ECG (classification by E. Braunwald et al.). In the absence of ST segment elevation, ACS may manifest in the form of HC or MI; in the presence of elevation, in the form of MI. An in-depth study of the dynamics of ACS development and determination of the amount of troponins in blood serum revealed the presence of two varieties of ACS. Analyzing the dynamics of the quantitative content of troponin, it becomes possible to determine the different stages of myocardial ischemia.

That is why troponinegative (Tn -) and troponinpositive (Tn+) forms/stages of ACS (ACS-Tni ACS-Tn+) were distinguished. Myocardial ischemia is observed in NS, and a heart attack within ACS indicates the early stages of its necrosis (F.A. Barotov, 2013; V.S. Botasheva, F.A. Barotov, 2016). With an increase in the ST segment, we should talk about the beginning of the transition phase from ischemia to myocardial necrosis; the appearance of the Q wave indicates its necrosis. In patients with clinical symptoms of angina pectoris and ischemic ECG manifestations induced by stress tests, unchanged coronary arteries (CA) are diagnosed with a frequency of up to 37% according to coronary angiography. This condition is defined in the cardiological literature as cardiac (coronary) syndrome X (CSX). In recent years, there have been increasing reports of cases of AMI and SCD in persons with CSH. Autopsy data show that myocardial ischemia in such patients 19 is caused, in particular, by disorders at the microcirculatory level. Specialists of the working group of the European Society of Cardiology proposed to consider CSH as a coronary microvascular heart disease (CMSD).

The risk factors of VCS are well studied. These include: males aged 40 to 60 years, consumption of psychoactive substances, diabetes mellitus, heredity, obesity, smoking, hypertension, psychoemotional stress, hypercholesterolemia, sudden changes in meteorological conditions, violation of the regulation of immune processes, insufficient magnesium content in the body. Pain-free forms of myocardial ischemia lead to sudden death in patients with coronary artery disease (silent ischemia). A burdened family history regarding the occurrence of coronary heart disease and the risk of VCS is not a direct proof of the presence of genetic pathology, since it is impossible to exclude the influence of lifestyle, bad habits, psychological factors. The presence of a family history increases the risk of VCS by almost 2 times. The relative risk of VCS increases sharply (almost 2 times) in families in which cases of VCS on the maternal and paternal lines were observed. It is assumed that the genetic predisposition to VCS may be based on a genetically determined violation of the biological function of proteins. Their significant influence in a patient with coronary heart disease is formed, apparently, as a result of a combination of a certain number of altered nucleotides (Single nucleotide polymorphism - SNP). Mutations in several genes were identified: ALOX5AP, ApoE, LTA, FAM5C. 20 An increase in the concentration of apolipoprotein B (Apo-B) in the blood indicates an accelerated development of atherosclerosis and coronary heart disease. An increased risk of developing cardiovascular diseases is also indicated by an increase in the level of fibrinogen.

The presence of AG polymorphism of the SURZA4 gene – the most significant enzyme of the cytochrome P450 system – is also associated with a predisposition to an unfavorable course of coronary heart disease. Currently, numerous risk factors for the development of coronary heart disease have been identified: left ventricular dysfunction with an ejection fraction of less than 35-40%, an increase in the wall thickness of the left ventricle, an increase in the number of heart contractions, an imbalance of the sympathetic parasympathetic nervous system, violations of the end part of the ventricular ECG complex, there is a dispersion and an increase in the length of the QT interval. An important prognostic value is extrasystole and unstable ventricular tachycardia in people with coronary heart disease, as well as in those who have already suffered MI. Unique data were obtained by Kalra L., showing that atrial fibrillation (AF) is an independent predictor of SCD. According to a number of authors, AF increases mortality rates by 1.8-2 times.

Adverse risk factors are alcohol abuse and excessive physical exertion. Electrical instability of the myocardium, decreased contractile function of the left ventricle and myocardial ischemia constitute a triad of the most important thanatogenetic risk factors for SCD. Among other factors that cause SCD, there are also disorders in the conduction system of the heart, disorders of the hypothalamus structure, pronounced changes in the metabolism of cardiomyocytes, as well as the presence of coronary artery ectasia (CA). CA ectasia, leading to changes in blood flow velocity and pressure gradient, create conditions for the syndrome of intercoronary theft and transmyocardial blood discharge, which leads to pronounced hemodynamic disruptions. The latter, due to zonal collateral blockade, as a rule, form foci of ischemia "at a distance" (ischemia at a distance from the "interested vessel). Local myocardial ischemia due to CA ectasia leads to its electrical instability due to concentration gradients of different metabolites between perfused and non-perfused areas.

This causes a distortion of the shape of the action potential and an increase in the rate of diastolic depolarization. At this stage, disturbances of the rhythmic activity of the heart are recorded, preceding and contributing to ventricular fibrillation, which is the main mechanism of the onset of SCD in persons with CA ectasia. The causes of rupture or erosion of the atherosclerotic plaque, which should be considered the initial moments in the development of ACS, include: exacerbation of the inflammatory reaction in the plaque, vasa vasorum rupture and massive hemorrhage into the plaque, a significant increase in the amount of lipids in the plaque, thinning of the plaque cap, platelet adhesion and aggregation, thrombus formation, stenosis and occlusion of the coronary artery. Particular attention is drawn to the immunological aspects of the pathogenesis of ACS. Acute episodes of dyslipidemia are one of the possible prerequisites for atheromatous plaque damage and the occurrence of an intravascular thrombus. The mechanism of atherosclerosis development is associated with the influence of a number of factors. In the pathogenesis of the atherosclerotic process, an important role is played by endothelial dysfunction, arterial hypertension, impaired lipid metabolism, oxidative stress, autoimmune inflammation, and chronic infection.

The peroxide concept of the pathogenesis of atherosclerosis suggests that the key stage in the occurrence and progression of this disease is the activation of free radical oxidation processes in the body and the inability of endogenous antioxidant systems to cope with the disposal of emerging products. In this situation, the main role of defenders against free radicals is assumed by lipids, the modification of which and the corresponding increase in the concentration of their peroxidation products can serve as an index of free radical oxidation. The inadequacy in the work of antioxidant systems and the increase in the content of POL products indicate the development of chronic stress of antioxidant protection and depletion of adaptive potential. The potential of the antioxidant system, which has a protective effect on the growth of POL products, is of great importance.

In this regard, the study of nitrogen (NO) and superoxide dismutase (COD) indicators is of great importance. Anti-inflammatory and anticoagulant indicators of endothelial cells are closely interrelated with the NO molecule. With a decrease in the content or bioavailability of nitric oxide, early signs of atherosclerosis appear in conditions of impaired hemodynamics. A decrease in the bioavailability of nitric oxide occurs for two reasons: a decrease in the synthesis of nitric oxide or an increase in its deactivation, which is due to endothelial dysfunction. Among the factors contributing to the thinning and subsequent rupture of the plaque tire is an increase in the inflammatory reaction in the plaque. An active inflammatory reaction in an atherosclerotic plaque is one of the leading factors weakening the fibrous capsule of the plaque and contributing to its rupture.

Migration of monocytes from the vascular bed is provided by local changes in the spectrum of adhesive molecules. In vulnerable atheromatous plaques, 6-9 times more macrophages are found than in stable plaques. A number of factors inhibit the proliferation of smooth myocytes, activate their apoptosis, enhance the proteolytic ability of macrophages, and reduce collagen synthesis by smooth myocytes. Such factors include cytokines, namely, interleukin 1, interleukin 6, interleukin 12, Y-interferon and tumor necrosis factor a. These factors synthesize macrophages. Literature data indicate that inflammatory reactions in patients with coronary heart disease are common with an increase in blood levels of markers and mediators of inflammation. Recent studies have shown that in complicated plaques, compared with stable plaques, the content of RARPA-A is significantly increased. It is a protein that circulates in the blood and is part of zinc-containing metalloproteinases. RARPA-A is a local regulator of the content of insulin–like growth factor - 1 and controls local cell proliferation.

An increase in plasma protein A (RARPA-a) is an indicator of an unfavorable prognosis for coronary heart disease, since this protein indicates the destruction of atherosclerotic plaque. The results of experimental and clinical studies have shown that the placental growth factor (PLGF) molecule is similar to the endothelial growth factor molecule and an increase in PLGF content is an indicator of the progression of atherosclerosis and a sign of instability of atherosclerotic plaque. With tears or ruptures of the atherosclerotic plaque, thrombogenic substances from the lipid core of the plaque penetrate into the blood, which contribute to the formation of platelet aggregates. Platelet aggregates form the basis of the primary parietal thrombus. Increased platelet activity in different forms of ACS

has been recorded in many studies using various methodological approaches. Membrane glycoprotein proteins (glycoprotein complex IIb – IIIa) are involved in two main platelet reactions, namely adhesion and aggregation. In patients with acute coronary syndrome, there is an increased tendency to thrombosis, which is due to violations of the carbohydrate composition and the spatial structure of the complex of glycoproteins that form platelet membrane receptors. In 90% of cases, ventricular fibrillation is the cause of cardiac arrest in VCS. Ventricular fibrillation after the development of acute coronary syndrome usually occurs within the first hours. Accordingly, an important task is to find out the trigger mechanism that stimulates its occurrence.

In addition, myocardial ischemia is accompanied by activation of free radical processes, which also leads to disruption of the functioning of ion channels, changes in the transmembrane potential and excitability of cells of the conducting system and cardiomyocytes, the formation of zones with impaired electrophysiological properties in the ischemic myocardium. Patients with ischemic heart disease have changes in the autonomic nervous system of the heart, which are caused by structural and functional disorders. At the same time, sympathetic and parasympathetic regulatory mechanisms are violated. Activation of the sympatho-adrenal system and inhibition of the parasympathetic system is observed. These changes are due to a pronounced morphological restructuring of the myocardium, an increase in electrical instability and a tendency to fatal changes in heart rhythm.

It has been established that endothelin –1 (ET-1) has an arrhythmogenic effect and is capable of causing vasospasm, and reperfusion of ischemic myocardium is a trigger mechanism that induces ventricular fibrillation in VCS. During reperfusion, arrhythmogenic substances are washed out of the ischemic zone, which damage the membranes of cardiomyocytes, which leads to the occurrence of electrical instability of the myocardium and ventricular fibrillation.

LITERATURE USED

1. Абрамкин, Д. В. Неинвазивные сердечно-сосудистые рефлекторные тесты и прогноз внезапной сердечной смерти после перенесенного инфаркта миокарда: какой метод предпочесть? / Д. В. Абрамкин, И. С. Явелов, Н. А. Грацианский // Кардиология. – 2004. – № 10. – С. 4-12.
2. Абрамкин, Д. В. Сердечно-сосудистые рефлекторные тесты после недавно перенесенного инфаркта миокарда: связь с прогнозом заболевания / Д. В. Абрамкин, И. С. Явелов, Н. А. Грацианский // Кардиология. – 2004. – № – С. 37-46.
3. Акимова, Е. В. Динамика распространенности ишемической болезни сердца и риск сердечно-сосудистой смерти в открытой популяции Тюмени / Е. В. Акимова, В. А. Кузнецов, В. В. Гафаров // Кардиология. – 2006. – № 1. – С. 14-18.
4. Аль-Обади, И. С. Генные маркеры как предикторы внезапной сердечной смерти в спорте / И. С. Аль-Обади, А. В. Смоленский // Рос. кардиол. журн. – 2007. – № 1. – С. 57-61.
5. Антонова, И. С. Предикторы внезапной смерти у больных сердечной недостаточностью различного генеза / И. С. Антонова, О. М. Моисеева, В. С. Морошкин // Артериальная гипертензия. – 2009. – № 4. – С. 462-465.
6. Аритмогенная кардиомиопатия/дисплазия правого желудочка как причина внезапной сердечной смерти молодых людей / М. В. Гордеева, Л. Б. Митрофанова, А. В. Пахомов [и др.] // Вестн. аритмологии. – 2012. – № 69. – С. 38-47.
7. Ассоциированный с беременностью протеин плазмы – новый биохимический маркер острого коронарного синдрома и предиктор неблагоприятного прогноза у больных ишемической болезнью сердца / О. П. Шевченко, А. О. Шевченко, Е. В. Кочетова [и др.] // Кардиоваскуляр. терапия и профилактика. – 2006. – № 4. – С. 110-116. 114
8. Асташкина, О. Г. Внезапная сердечная смерть: судебно-медицинские аспекты / О. Г. Асташкина, Н. В. Власова, Г. А. Пашинян // Рос. мед. журн. – 2010. – № 6. – С. 30-35.
9. Афанасьева, Ю. И. Гистология, цитология и эмбриология / Ю. И. Афанасьева, С. Л. Кузнецова, Н. А. Юрина. – Москва : Медицина, 2004. – 765 с.

10. Баротов, Ф. А. Морфологическая характеристика нестабильной стенокардии / Ф. А. Баротов // Фундаментальные исследования в биологии и медицине : сб. тр. – Ставрополь, 2013. – Вып. 10. – С. 27-30.
11. Баротов, Ф. А. Патоморфологическая характеристика инфаркта миокарда при внезапной сердечной смерти / Ф. А. Баротов // Фундаментальные исследования в биологии и медицине : сб. тр. – Ставрополь, 2013. – Вып. 10. – С. 24-27.
12. Берестовская, В. С. Кардиальные маркеры в перикардиальной жидкости при внезапной коронарной смерти / В. С. Берестовская, М. В. Еремина, А. В. Козлов // Клинич. лаб. диагностика. – 2003. – № 5. – С. 8-10.
13. Бойцов, С. А. Актуальные вопросы терминологии, классификации и статистического учета острых форм ишемической болезни сердца / С. А. Бойцов, С. С. Якушин, Н. Н. Никулина // Терапевт. архив. – 2010. – № 9. – С. 5- 13.
14. Бокарев, И. Н. Острый коронарный синдром и его лечение / И. Н. Бокарев // Consilium medicum. – 2006. – Т. 8, № 5. – С. 72-81.
15. Бокарев, И. Н. Углеводный обмен у больных острым коронарным синдромом / И. Н. Бокарев, Т. В. Хлевчук // Клинич. медицина. – 2007. – № 6. – С.14-18.
16. Бокерия, Л. А. Внезапная аритмогенная смерть на госпитальном этапе после операции на сердце / Л. А. Бокерия, Р. А. Серов, А. А. Можина // Арх. патологии. – 2001. – № 2. – С. 30-36. 115
17. Бокерия, Л. А. Риск внезапной сердечной смерти у больных гипертрофической кардиомиопатией / Л. А. Бокерия, К. В. Борисов, Е. Д. Савченко // Грудная и сердечно-сосудистая хирургия. – 2001. – № 1. – С. 62- 66.
18. Боташева, В. С. Анализ летальности при остром коронарном синдроме / В. С. Боташева, А.В. Копылов, Ф. А. Баротов // Инновационные технологии в медицине: материалы 11-ой науч.-практ. конф. врачей Карачаево-Черкес. респ. с междунар. участием. – Черкесск, 2014. – С. 144-146.
19. Боташева, В. С. Иммуногистохимическая и гистологическая диагностика острого коронарного синдрома / В. С. Боташева, Ф. А. Баротов // Мед. алфавит (Кардиология). – 2017. – Т. 1, № 13. – С. 25-27.
20. Боташева, В. С. Морфологические критерии диагностики острого коронарного синдрома / В. С. Боташева, Ф. А. Баротов // Здравоохранение Таджикистана. – 2015. – № 3. – С. 12-17.
21. Боташева, В. С. Морфология нестабильной бляшки при остром коронарном синдроме / В. С. Боташева, Ф. А. Баротов // Актуальные вопросы современной медицины : материалы науч.-практ. конф. Форума. – Ставрополь, 2015. – Ч. I. – С. 92-95.
22. Боташева, В. С. Морфология острого коронарного синдрома / В. С. Боташева, Ф. А. Баротов // Функциональные проблемы адаптации: сб. науч. тр. Междунар. конф. – Ставрополь, 2015 – С. 36-37.
23. Боташева, В. С. Некоронарогенные некрозы миокарда / В. С. Боташева, Ф. А. Баротов // Фундаментальные исследования в биологии и медицине: сб. тр. – Ставрополь. – 2014. – Вып. 11. – С. 43-46.
24. Боташева, В. С. Патологическая анатомия острой ишемии миокарда / В. С. Боташева, Л. Д. Эркенова, Ф. А. Баротов // Мед. алфавит (Кардиология). – 2016. – Т. 2, № 28. – С. 22-24. 116
25. Боташева, В. С. Характер структурных изменений в миокарде при внезапной сердечной смерти / В. С. Боташева, Ф. А. Баротов // Наука. Инновации. Технологии. – 2015. – № 1. – С. 107-111.
26. Боташева, В. С. Характер структурных изменений в нестабильной бляшке при остром

коронарном синдроме / В. С. Боташева, Ф. А. Баротов // Здравоохранение Таджикистана. – 2015. – № 4. – С. 5-9.

27. Васина, Л. В. Аннексин А5 и антитела к аннексину А5 при остром коронарном синдроме с подъемом и без подъема сегмента ST / Л. В. Васина // Регионар. кровообращение и микроциркуляция. – 2009. – № 2. – С. 22-25.
28. Васина, Л. В. Клеточные и гуморальные маркеры апоптоза при остром коронарном синдроме в сочетании с гипертонической болезнью / Л. В. Васина // Артериальная гипертензия. – 2008. – № 4. – С. 332-335.
29. Васина, Л. В. Участие циркулирующих антител к аннексину А5 в регуляции процесса фибринолиза при остром коронарном синдроме / Л. В. Васина // Регионар. кровообращение и микроциркуляция. – 2007. – № 4. – С. 14-20.
30. Взаимосвязь между показателями нарушения перфузии миокарда, динамикой течения хронической сердечной недостаточности и развитием пароксизмов фибрилляции предсердий у больных с острым коронарным синдромом / С. П. Алферов, А. С. Свистов, А. Э. Никитин [и др.] // Вестн. аритмологии. – 2009. – № 55. – С. 34-37.
31. Плавинская, С. И. Прогностическая значимость основных факторов риска у женщин по данным проспективного популяционного исследования и шкала риска смерти от ССЗ / С. И. Плавинская, С. Л. Плавинский, Д. Б. Шестов // Рос. семейный врач. – 2006. – № 4. – С. 4-9