

DEVELOPMENT OF ACUTE HEART FAILURE IN PATIENTS WITH ACUTE MYOCARDIAL INARCT

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Abstract: Early identification of predictors of acute heart failure (AHF), the fact of myocardial necrosis, high diagnostic accuracy in the first hours of the disease in patients with acute coronary syndrome is the most important task of emergency cardiology. Solving this problem, creating new models for stratifying the risk of the occurrence and course of AHF depending on various factors will allow us to develop new intensive care algorithms, determine individual prognostic parameters, and, therefore, will allow timely initiation of the necessary treatment measures, which will have a positive impact on the prognosis and will contribute to improvement quality of life of patients, reducing mortality, economic costs, reducing the clinical and social consequences of coronary heart disease.

Keywords: acute myocardial infarction, complications of myocardial infarction.

Relevance Epidemiological, clinical and morphological data are quite convincingly expressed that ischemic heart disease, acute heart failure (AHF), acute coronary syndrome (ACS) is most severely manifested by heart rhythm disease and sudden cardiac death (SCD) [20, 27, 32].

SCD and ischemic heart disease have common risk factors; many people who died suddenly have acute myocardial infarction (AMI) at autopsy, and most have large size or large post-infarction scars. In addition, the majority of those who suddenly died, but no such requests for help and correctional doctors were identified, have a significant degree of severity of anginal syndrome [32, 33, 39]. Most often, SCD occurs in the early stages of the onset of an attack of acute coronary insufficiency, which is the main disease of high prehospital mortality in patients [48].

Atrial fibrillation (AF) is the most common cardiac arrhythmia after extrasystole in patients with coronary artery disease. Its prevalence doubles with each decade of life, from 0.5% at the age of 50–59 years to 9% at the age of 80–89 years [27].

The problem of excretion manifests itself with an increased risk of paroxysmal heart rhythms, which often occur with the pathophysiological mechanisms underlying arrhythmogenesis. As mentioned above, arrhythmic complications usually occur in patients who have structural changes in the heart, most often associated with coronary heart disease, therefore, the study of the totality of heart rhythms occurring against the background of coronary artery disease is of particular interest due to their potential danger to the patient [15, 32, 47].

In 1991, Victor Dzau and Eugene Braunwald welcomed the cardiovascular continuum, which represents the development of subsequent pathophysiological events leading to the development of chronic heart failure and disease [17, 18].

It was revealed that cardiovascular risk factors, the development (manifestation) of diseases and associations that cause pathological excitability of events that arise in connection with the identification of a stage from an asymptomatic patient with a risk factor for the development of coronary atherosclerosis in a patient with congestive heart failure. The trigger points of the “fatal cascade” are cardiovascular risk factors, arterial hypertension (AH), diabetes mellitus, etc. d. [17, 18].

It has been shown that the development of almost any cardiovascular disease occurs according to the standard scheme: there is a certain pathological phenomenon (stress, ischemia, overload, etc.), leading to the manifestation of heart function, resulting in activation of neurohormonal systems aimed at the use cardiac activity. Over time, early sensitivity activation reverses itself and increases the impact of negative affect, ultimately leading to the expression of CH. At the center of the cardiovascular continuum is a vascular factor - endothelial dysfunction, anatomical and electrophysiological remodeling of the heart, neurohumoral factors (sympathoadrenal and renin-angiotension-aldosterone systems), which play a key role in all cases of the continuum.

Increased activity of the sympathetic division of the autonomic nervous system, stimulation of the central mechanisms of blood circulation regulation, changes in the sensitivity of baroreceptors, impaired reuptake of norepinephrine from synaptic alkali. The result of these processes is an increase in the growth of cardiovascular diseases (HR) and suspicion of contractile activity of the heart. In the implementation of the hypertensive effect of sympathicotonia, the effects of the mediated sympathetic nervous system are of great importance. The most significant is the adverse effect of catecholamines on the endothelium, manifested in inhibition of NO synthesis and increased formation of endothelin I, leading to an increase in peripheral vascular resistance.

Long-term increased activity of the sympathetic nervous system associated with hyperinsulinemia, impaired cellular transport of electrolytes, the development of cardiac hypertrophy, high metabolic syndrome and increased instability of the cardiovascular system. The special role of autonomic imbalance in the risk of paroxysmal combinations of heart rhythm is emphasized [21, 36, 39, 40, 45].

The nature of cardiac remodeling against the background of continuum development is heterogeneous; on the one hand, it is a response to damaging overload during arterial hypertension; on the other hand, it has been revealed that remodeling is a process associated with a neurohumorally mediated disruption of cellular ion transport. Among the humoral participants involved in remodeling processes, the main role is played by pathological activation of the renin-angiotensin system and hypersympathicotonia [8].

Slower left ventricular relaxation, hypertrophy, and concentric remodeling are associated with greater clinical severity of AF attacks and the risk of SCD.

Organic changes in the myocardium (due to ischemia, post-infarction cardiosclerosis or acute myocardial infarction), inflammation, fibrosis, left ventricular dysfunction significantly increase the risk of paroxysmal arrhythmias and SCD.

Myocardial ischemia, acute or chronic, is a particularly pathological disease of the cardiovascular continuum [32, 35]. Thus, acute cardiac ischemia, if it does not lead to acute coronary death, causes the appearance of a large zone of cardiac asynergy, which is associated with activation of the renin-angiotensin-aldosterone and sympathoadrenal systems and restoration of left ventricular remodeling (it becomes more spherical and thinner).

When analyzing data from daily ECG monitoring, patient income, and especially during the occurrence of episodes of life-threatening arrhythmias, an increase in “silent” ischemia, expressed in ST segment depression, was detected in 12.5% of cases in the occurrence of CV as a result of ventricular fibrillation. As Bayes de Luna established, for the occurrence of fatal cases of heart rhythm, it is not so much the severity of

ischemia that manifests itself in the anatomically vulnerable myocardium that is important, but rather its intensity and persistence [12].

A number of typical changes are described that are present when the threshold of resistance in coronary artery disease decreases. At the cellular-tissue level, this is the heterogeneity of the contractile heart rhythm of alternating cardiomyocytes with oxidoreductase activity, their disseminated damage, hypertrophy, atrophy, apoptosis, interstitial edema and the phenomena of cardiosclerosis that disrupt the consolidation of vascular cells.

Ischemia worsens left ventricular dysfunction directly or indirectly through a pathway of slow cardiac instability.

Cardiac remodeling is a multifactorial process involving bioenergetics and molecular mechanisms.

“Ventricular remodeling is an intensive, targeted process that has a regional and global impact on wall thickness, size and chamber formation, and systolic and diastolic function of the left ventricle as a whole” [3].

In this case, a complex disturbance of the structure and function of the heart occurs, including an increase in body weight, dilation of the cavities and changes in their geometric characteristics [1, 3, 6, 31].

Remodeling concerns components of the two circulatory systems: both skeletal structure and involvement, including the coronary vessels. Although the term "myocardial remodeling" has been used to characterize the process that occurs during and after myocardial infarction, the meaning of the term has subsequently been expanded. Currently, this term is used to assess indicators of disorders that occur in conditions including mechanical overload, hypertensive and valvular cardiopathy, and dilated cardiomyopathy [5].

The anatomical basis of remodeling is a change in the structure of the heart. Remodeling is associated with left ventricular mass, volume, and shape changes [23]. In myocardial infarction, remodeling is a massive process and is associated with expansion of the infarction zone and subsequent hypertrophic changes in the intact region [6].

When remodeling occurs against the background of the general cardiovascular system, such as arterial hypertension, ventricular hypertrophy, it remains symmetrical. Compensated hypertrophy is concentric and is manifested by the presence of thickened ventricular walls and septum. One of the negative consequences of cardiac remodeling is heart failure. Heart failure syndrome is considered a functional disorder caused by damage to the left ventricle of the heart and its inability to perform a full pumping function. The differentiation of systolic and diastolic dysfunction is expressed by a pronounced increase between the abnormalities of recovery and relaxation.

Remodeling of the cardiovascular continuum is a progressive process, even in states with pronounced persistent manifestations of HF. This process of progressive remodeling is not necessarily a consequence of the development of cardiovascular disease or infarction [1, 51]. Cardiac arrhythmias are a consequence of the impact of poor prognosis and high mortality from HF [19, 35, 42, 47].

An analysis of the causes of death in patients with heart failure at various stages [49] revealed that some diseases develop suddenly without previous progression of decompensation.

The incidence of ventricular arrhythmias is high in patients with heart failure (II-IV according to B. Lown's classification). A meta-analysis of studies revealed ventricular arrhythmias in 87% of patients with HF, of which 45% had “jogs” of ventricular tachycardia. An increase in the number and frequency of ventricular arrhythmias occurs simultaneously with a deterioration in hemodynamics as heart failure progresses [49, 50].

Atrial fibrillation is also very common in patients with HF [19, 22, 25, 27, 37, 49]. During the 38-year

Framingham follow-up, 20.6% of men had congestive heart failure at baseline and subsequently developed atrial fibrillation, in contrast to 3.2% of men without signs of heart failure. Similar figures for women are 26.0 and 2.9%, respectively [18].

The above data quite clearly document the continuity of anatomical and morphological changes and changes in its electrophysiological characteristics at all stages of the cardiovascular continuum.

Humoral-metabolic and structural disorders of ion transport in cardiomyocytes, an increase in sodium ions and their content inside the cell, leads to activation of the sympathoadrenal system. As a result of the disease, conditions arise for the formation of the so-called subsequent remodeling of the vessels of the atria and ventricles and the development of individual heart rhythms. Electrophysiological remodeling represents persistent changes in the electrophysiological characteristics of the heart that occur in connection with the emergence of zones of ischemic damage and the formation of a morphological substrate with heterogeneous provocability in an increase in the zone of the heart. Similar processes in the ventricles increase the risk of ventricular fibrillation. Electrophysiological remodeling plays a large role in the recurrence of arrhythmia and maintaining its constant form, and cardiac arrhythmias tend to manifest themselves when their frequency and progression develop slowly, with possible refractoriness to therapy. An example is atrial fibrillation.

In patients with paroxysms of atrial fibrillation/flutter, changes in electrophysiological properties were identified, among which the most significant for the prognosis of recurrence is considered to be the prescription of the time of interatrial and intraatrial excitation, as well as the expansion of the zone of atrial fragmented activity [5]. A similar sequence of events is associated with electrophysiological remodeling of the left ventricle, which in turn significantly increases the risk of malignant ventricular tachyarrhythmias [37, 41]. Extrasystoles in most cases are mainly triggering events, such as ventricular tachycardia and atrial fibrillation. Just as ventricular tachycardia can develop into ventricular fibrillation, other types of ventricular tachycardia can transform into atrial fibrillation (caused by tachycardia) [50].

It is known that cardiomyocytes change their electrophysiological properties during myocardial hypertrophy and heart failure. Changes in the functional activity of various ion influences and receptors may predispose to the development of arrhythmias. Progression of HF leads to the occurrence of atrial fibrillation and ventricular arrhythmias. In this case, they are caused by arrhythmogenesis.

Ivanov G. G. et al. (2003) proposed a specific definition of the concept “electrophysiological remodeling of the myocardium”: this is a complex of molecular, metabolic and ultrastructural changes in cardiomyocytes and extracellular matrix, causing disturbances in electrophysiological properties and characteristic of pathological and electrocardiographic phenomena accompanying structural remodeling of the heart [5]:

- changes in the duration of limited action [34];
- vascular hypertrophy and heart failure, leading to differences in the duration of action [42];
- changes in potassium ion currents [44];
- disturbance of calcium homeostasis [43];
- neurohumoral effects and hypertonicity of the sympathetic nervous system. Experimental studies have revealed an increase in the sympathetic activity of the nervous system during ischemia associated with excitation of the ventricular rhythm, while an increase in the activity of the parasympathetic nervous system has a protective effect [30, 38, 42, 48];
- during myocardial infarction in patients who died suddenly, foci of denervation and changes in the autonomic nerve ganglia were identified [2];
- non-esterified fatty acids (NEFA) - an increase in the concentration of NEFA is associated with high

sympathetic activity: catecholamines enhance endogenous lipolysis and sensitize the myocardium to high concentrations of NEFA [4];

➤ pacemaker current [5].

Various types of remodeling (functional, morphological and electrical) are components of the dynamics of pathological processes in many heart diseases. Obviously, the data that the data are parallel, reveal electrocardiographic phenomena, are ahead of mechanical ones [5].

Electrophysiological remodeling and electrical instability are present to varying degrees at all stages of the cardiovascular continuum.

It has been established that for the occurrence of malignant tachyarrhythmias from structurally altered conditions of the heart, in addition to increased ectopic activity, additional functional disorders are required in the form of transient ischemia or reperfusion, metabolic or electrolyte imbalance, disturbances in the properties of the myocardium, disturbances in the regulation of cardiac activity, identification of endogenous and exogenous toxic substances on myocardium, including the proarrhythmogenic effect of drugs [12, 27, 38, 49].

Thus, arrhythmias arise as a result between structural changes in the heart, targeted disorders of muscle tissue and a specific electrophysiological mechanism of arrhythmogenesis (the so-called Kumel's triangles, or Kumel's triangle) [28].

The probability of sudden cardiac death in each patient is different and with a high degree of probability can be assessed on the basis of a complex of clinical and instrumental studies, including 24-hour ECG monitoring, stress tests, echocardiography, electrophysiological study (EPS), etc. Sustained ventricular response induced during EPS tachycardia is an important predictor of arrhythmic complications, AHF and SCD. However, this method cannot be used as a screening method due to its invasiveness and high cost. In this regard, priority is currently given to the scientific search for opportunities to use non-invasive ECG screening tests.

Reduced left ventricular ejection fraction (EF) is the most important risk factor for overall mortality. To assess increased cardiac instability in patients with coronary artery disease, various non-invasive ECG methods are used: high-resolution electrocardiography, assessment of heart rate variability and baroreflex sensitivity, duration and dispersion of the QT interval, assessment of T wave alternans (TWA), heart rate turbulence (HRT), dispersion mapping, dipole electrocardiography (DECARTO), etc. [5, 28, 30, 47]. Multiple pathogens increase their predictive flora, although positive predictive flora is more than 40% [29, 30, 42].

It has now been shown that the widespread use of beta-blockers, statins and ACE inhibitors, and thrombolytic therapy in patients with coronary artery disease seriously affects mortality and the rate of CV events. Modern approaches to the treatment of coronary artery disease have been noted in patients with a pronounced prognostic significance of a large increase in non-invasive ECG methods for assessing significant infarction instability. It is believed that at present it is impossible to extrapolate the results obtained in pre-thrombolytic external therapy to the current situation, and it is necessary to conduct studies aimed at finding the most significant risk markers [42].

It is not yet known which ECG markers are useful and are used to combine with parameters characterizing heart rate variability, refractoriness, electrocardiographic parameters and high-resolution parameters. He determined the priority of detention, their predictive detention during a comprehensive collection. Also, determine whether to exclude the previous gradations of significant risk when collecting them individually, in a multivariate analysis. The question of which combination of parameters may have the greatest diagnostic and prognostic sensitivity has not yet been answered.

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