МЕДИЦИНА, ПЕДАГОГИКА И ТЕХНОЛОГИЯ: ТЕОРИЯ И ПРАКТИКА
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STATE OF THE ART OF FORECASTING THE DEVELOPMENT AND COURSE OF EARLY POSTINFARCTION ANGINA

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The most urgent medical and socially significant problem of modern society is ischemic heart disease (IHD). Despite the success achieved in the prevention and treatment of IHD, it still dominates in the structure of morbidity, disability, mortality of the working-age population, and financial costs for treatment and rehabilitation [18,30,35]. Acute myocardial infarction (AMI), being an acute manifestation of IHD, remains one of the prognostic unfavorable diseases of the cardiovascular system. In patients with AMI, the risk of sudden death is 4-6 times higher than in the general population, and 25% of men and 38% of women die within the next year after they are diagnosed (Heart Disease and Stroke Statistics-2007 Update). The prognosis of AMI patients is determined both by age, the presence of diabetes mellitus (DM), a history of coronary heart disease, and the localization of AMI, acute complications, and therapy performed during this period. It is fundamentally important for patients with AMI to determine the possible risk of future cardiovascular complications already on the first day of the disease [15,16,17,20]. Early post-infarction angina (PIA) remains one of the most common complications of AMI and is characterized by the recovery of angina attacks within 3 to 28 days of AMI. The relevance of the issue of early PIA can be explained by its unfavorable course: the frequent development of repeated AMI, an increase in cardiac displacement. This makes it important study the pathogenesis of early PIA and its risk factors [12,13,18]. Early PIA is one of the variants of unstable angina (UA) and is characterized by the appearance of angina attacks in the period from the beginning of the 3rd day of AMI and up to 2 weeks after the development of AMI, and is called this regardless of whether the attacks occur angina pectoris in the early post-infarction period for the first time in life (denovo) recurs in patients who have suffered from angina for a long time before AMI [24,25]. Early PIA is characterized by intensely recurring angina pain that occurs at rest or with little physical exertion. A pain attack relieved by nitroglycerin is accompanied or not accompanied by new transient elevation or depression of the ST segment from the

baseline by 1.0 mm or more and changes in the T wave on the electrocardiogram, which appear in the next 30 days after the onset of AMI [29]. Morran T. J. etal. (1982) limits the time of occurrence of early PIA to three weeks. According to D. G. Ioseliani (1984), all patients whose angina attacks resume in the next two months after the onset of AMI can be attributed to the group of post-infarction angina pectoris. Braunwald E. (1989) and other authors indicate the time of occurrence or frequency of angina attacks in 24 hours and up to 8 weeks after the development of AMI[13,31,24]. A serious complication of early PIA is repeated AMI, which is more unfavorable than in primary infarction [19].The clinical course of recurrent AMI is characterized by more frequent and early development of heart failure (HF) and cardiogenic shock, pulmonary edema, and fatal arrhythmias, which in 40% of cases is the cause of death of patients with repeated AMI. The mortality rate for repeated AMI ranges from 23.2% to 60.7%. If the interval between primary and repeated infarction is less than 6 months, the mortality rate in this case reaches 80%, and if the interval is more than 6 months - 50% [8].According to modern concepts, 20-60% of patients with AMI with ST-segment elevation develop PIA, which increases the risk of AMI recurrence, fatal rhythm disorders, acute left ventricular failure, and sudden death [9]. The development of PIA is associated with recurrent myocardial ischemia [11]. Recent studies have shown, that patients with AMI with ST-segment elevation complicated by the development of PIA are characterized by multi - vascular damage to the coronary arteries, the development of re occlusion of the coronary artery infarction, distal embolization by thrombotic masses with the development of collateral blood flow occlusion and microcirculation disorders [37].Some researchers have shown the presence of damage to the intima of the coronary arteries in patients with PIA, and data on the role of the inflammatory response in their genesis are contradictory [22]. Despite the reported adverse outcome, risk factors and predictors of early PIA have not yet been identified in patients with ST-segment elevation AMI [10].Thus, some issues of development, course, and prognosis in patients with AMI with ST-segment elevation in the development of early PIA require further study. Attempts to influence the prognosis with intensive drug therapy did not lead to the desired result, so the idea was expressed about the need for a more active approach to the treatment of these patients, including early diagnostic coronary angiography followed by myocardial revascularization. Inflammation plays a leading role in the initiation and progression

of atherosclerosis and the destabilization of atherosclerotic plaque [2, 10, 14, 29]. It has been shown that US increases the level of fibrinogen, the number of white blood cells, ESR, interleukin (IL-1), and interleukin (IL-6) [25]. One of the prerequisites for the development of atherosclerosis and atherothrombosis is endothelial dysfunction (ED), which is present in the links of homeostasis. The pathogenic role of ED has been proven in arterial hypertension (AH), diabetes mellitus (DM), IHD, and AMI [1,4,30,34]. However, it has not been studied in patients with early PIA. Due to the high risk of developing acute heart failure(AHF), life-threatening arrhythmias, most authors believe that early PIA has an important prognostic value and allows us to divide patients who have undergone AMI into 2 groups: with a high and low risk of mortality in the first year after myocardial infarction [1,6].Thus, early PIA refers to the most severe form of US, primarily due to a recent AMI, the morphological basis of which is the presence of fresh foci of necrosis and, as a result, an acute violation of the contractility of the myocardium, a high risk of developing AMI, life-threatening arrhythmias, as a reflection of electrical instability of the myocardium and high mortality in long-term follow-up [20]. Invasive studies of the electrical function of the myocardium are neither widely available no sufficiently safe for patient with IHD, especially in the early stages of AMI, so new non-invasive methods are proposed as routine examination methods, such as assessing heart rate variability, recording late ventricular potentials, and determining the duration and variance of the Q-T interval[32, 33]. Based on the above, the development of applying optimal risk markers and predicting the course of early PIA is relevant. However, it is not fully established whether early PIA is the mechanism responsible for the increase in post-infarction mortality, or whether it is simply a marker of an unfavorable prognosis [22,38]. In addition, it is not definitively known what factors affect the indicators of HU in AMI, and their relationship with other predictors indicating an unfavorable prognosis, in particular, the development of early PIA, is not completely clear. Thus, considering early PIA as a complication of AMI, it is necessary to study the change in MS parameters, as well as the effect of these markers on the development of cardiovascular complications in patients who have under gone AMI, but also to assess the dynamics of these markers against the background of early therapy in patients with AMI complicated by the development of early PIA in order to optimize the management of this category of patients.

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