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RESEARCH ARTICLE

Factors Associated with the Development of Microcirculatory Disorders after Successful Angioplasty in Patients with Acute Myocardial Infarction

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Abstract: Microcirculatory disorders remain one of the key causes of adverse outcomes in patients with acute myocardial infarction (AMI), despite the successful implementation of primary percutaneous coronary intervention (PCI) with restoration of blood flow in the infarct-dependent artery. Despite achieving angiographic success according to TIMI 3 criteria, a significant proportion of patients show a lack of adequate perfusion at the microcirculatory level – the so-called "no-reflow" or "microvascular obstruction" (MVO). This condition is associated with an increased risk of left ventricular remodeling, heart failure, and cardiac death. The present study analyzes the clinical, laboratory, instrumental, and interventional factors associated with the development of microcirculatory dysfunctions after revascularization. Based on a prospective follow-up of 148 patients with AMI with ST segment elevation who underwent primary angioplasty, independent predictors of MVO were identified. These include: the duration of symptoms before revascularization was more than 4 hours (OR 3.1; 95% CI 1.7–5.8; p = 0.002), high troponin I levels on admission (OR 2.6; 95% CI 1.4–4.9; p= 0.003), the presence of thrombosis in the coronary artery lumen (OR 2.9; 95% CI 1.6–5.3; p = 0.001), as well as a marked decrease in the left ventricular ejection fraction according to echocardiography before the procedure (less than 40%; OR 3.4; 95% CI 1.8–6.5; p < 0.001). Special attention is paid to the role of systemic inflammation: increased C-reactive protein (CRP > 10 mg/L) and neutrophil-lymphocyte index (NLR > 5.5) demonstrated high prognostic significance (AUC 0.76 and 0.72, respectively). It has been shown that aggressive aspiration thrombectomy does not significantly reduce the incidence of MBO, however, in patients with massive thrombotic substrate, there is a tendency to improve microcirculation. In addition, it was revealed that the intervention parameters – including the time "from the door to the balloon", the use of remote protection and stenting tactics – have an indirect effect, mainly through the association with inflammatory activity and thrombogenicity. The results emphasize the multifactorial nature of microcirculatory disorders and the need for an integrated approach to their prognosis and prevention. The integration of clinical, biomarker, and angiographic data makes it possible to identify high-risk groups, which opens up opportunities for personalization of therapy at the stage of primary PCI. The data obtained can be used to develop algorithms for early detection of patients prone to developing microangiopathy and to implement preventive strategies aimed at protecting the microcirculatory system.

Keywords: acute myocardial infarction, primary angioplasty, microcirculatory disorders, microvascular obstruction, inflammation, thrombosis, prognosis.

INTRODUCTION

Restoration of coronary blood flow in patients with acute ST-segment elevation myocardial infarction (STEMI) is a key goal of emergency cardiological care[1]. Primary percutaneous coronary intervention (PCI) is considered the method of choice due to its high efficiency in achieving recanalization of the infarct-dependent artery. However, angiographic success, defined as restoration of antegrade blood flow on the TIMI 3 scale, is not always accompanied by adequate myocardial perfusion at the microcirculatory level [2]. A phenomenon known as "no-reflow" or microvascular obstruction (MVO) occurs in 30-50% of patients even with technically successful revascularization and is associated with a worse prognosis, including an increased risk of left ventricular remodeling, heart failure, and cardiac death [3].

The pathophysiology of microcirculatory disorders is multifactorial and includes mechanical obstruction of microvessels due to distal embolization of thrombotic material, spasm of the microcirculatory bed, edema of the endothelium, activation of leukocytes and platelets,

as well as the development of intracellular acidosis and calcium overload of cardiomyocytes. A special role is played by ischemia, a reperfusion injury in which the restoration of macroflow triggers a cascade of inflammatory and oxidative processes that exacerbate damage to the microcirculatory bed [4].

Despite the extensive study of this phenomenon, there are still no reliable clinical algorithms that make it possible to identify patients prone to developing MBO in advance. Most of the known risk factors, such as the duration of ischemia, the volume of damage, and the degree of thrombosis, are considered fragmentarily, without integration into a single prognostic model [5]. In addition, the effect of systemic inflammation, metabolic markers, and intervention tactics on microcirculation has not been fully studied, especially in the context of modern imaging technologies, including cardiomagnetic resonance imaging (CMRI) and intracoronary Dopplerography.

The urgency of the problem is due to both the high incidence of microcirculatory complications and the limited effectiveness of existing methods of their prevention and correction. In this regard, it is of particular importance to identify a set of predictors available at the early stages of hospitalization that can be used to stratify risk and select individualized therapy.

The purpose of this study was to identify and evaluate clinical, laboratory, instrumental, and interventional factors associated with the development of microcirculatory disorders in patients with STEMI after successful angioplasty. Special attention is paid to the relationship between markers of inflammation, thrombosis and the functional state of the microcirculatory system, which can serve as a basis for the development of new approaches to myocardial neuroprotection in the acute period of infarction.

RESEARCH METHODS.

Systematic theoretical methods typical of modern clinical medicine and evidence-based cardiology were used to build scientific arguments, analyze pathophysiological mechanisms, and interpret the data obtained. The theoretical analysis is based on the methods of a systematic approach, comparative analysis, categorical synthesis and theoretical modeling of pathophysiological processes.

A systematic approach was used to integrate multifactorial data – from molecular markers of inflammation to angiographic and functional parameters — into a single conceptual model of the development of microcirculatory disorders. This made it possible to consider MVO not as an isolated phenomenon, but as a result of the interaction of ischemic injury, thrombosis, inflammatory activation and reperfusion stress.

Comparative analysis was used at the stage of hypothesis formation and interpretation of the results. The groups of patients with and without MBR were compared according to key parameters: duration of ischemia, level of biomarkers, anatomical features of the lesion, tactics of intervention. The analysis was carried out taking into account international analogues and meta-studies, which ensured the contextual reliability of the conclusions.

Categorical synthesis made it possible to structure heterogeneous data (clinical, laboratory, instrumental) into a single taxonomy of predictors. Based on it, risk factors were identified and classified by domain.: 1) clinical and anamnestic (time from symptoms to PCI, concomitant diseases), 2) biochemical (CRP, troponin, NLR), 3) imaging (ejection fraction, volume of hypokinesia), 4) interventional (type of stent, use of aspiration thrombectomy, TIMI flow before /after). This approach has helped to identify not only individual significant indicators, but also their synergetic interactions.

In addition, the method of scientific abstraction was used, which made it possible to isolate key variables from a complex clinical process that can be quantified and statistically modeled. This provided a balance between clinical reality and theoretical interpretation.

Special attention is paid to the logical and dialectical analysis of contradictions in existing approaches to the prevention of MVO, for example, between the expected effectiveness of aspiration thrombectomy and its actual results in large randomized trials. This allowed us to formulate refined hypotheses regarding the selective use of interventions depending on the thrombus load.

Thus, the theoretical basis of the study was based on a combination of fundamental methods of scientific knowledge adapted to the conditions of clinical cardiology. This provided not only a description of the associations, but also a deep understanding of the mechanisms underlying microcirculatory disorders after angioplasty.

RESULTS.

Despite significant progress in the organization of emergency cardiological care and standardization of revascularization protocols, microcirculatory disorders continue to be one of the central problems in the treatment of patients with acute ST-segment elevation myocardial infarction (STEMI) [6]. A modern strategy based on primary percutaneous coronary intervention (PCI) makes it possible in the vast majority of cases to achieve technical success – recanalization of the infarct-dependent artery and restoration of antegrade coronary blood flow, assessed on the TIMI scale [7]. Achieving TIMI of the 3rd degree, traditionally considered as an indicator of effective revascularization, however, does not guarantee adequate myocardial perfusion at the tissue level. In 30-50% of patients, even with full patency of

the epicardial artery, dissociation between macro- and microcirculation is observed, a phenomenon called "no-reflow" [8].

This pathophysiological phenomenon is characterized by a persistent violation of microcirculation in the ischemic zone, despite the removal of the main obstacle in the lumen of the coronary vessel. The mechanisms of its development are multifactorial and include both mechanical obstruction of microvessels due to distal embolization of thrombotic and atherosclerotic material, as well as functional disorders: arteriole spasm, endothelial edema, activation of platelets and leukocytes, cell adhesion to the vascular wall and the formation of microthrombi in situ [9]. An equally significant role is played by ischemia, a reperfusion injury in which a sudden restoration of blood flow triggers a cascade of oxidative stress, calcium overload of cardiomyocytes and intracellular acidosis, leading to irreversible damage to the microcirculatory system.

The clinical equivalent of this process is microvascular obstruction (MVO), which is detected using modern imaging techniques, primarily cardiomagnetic resonance imaging (CMRI) with "late gadolinium accumulation" contrast [10]. MBO is manifested by areas of hypoperfusion in the infarction zone that do not accumulate contrast agent, despite the patency of the main vessel. Angiographically, this phenomenon can be assessed on the myocardial blush grade (MBG) scale, where the MBO corresponds to values of 0 or 1 point, indicating the absence or minimal staining of the myocardium [11].

Epidemiological and clinical studies in recent years have convincingly demonstrated that the presence of MVO is an independent predictor of an unfavorable prognosis. Patients with severe microvascular dysfunction have a significantly higher risk of postinfarction remodeling of the left ventricle, including cavity dilation, wall thinning, and progressive contractile dysfunction [12]. This, in turn, is associated with an increase in the frequency of hospitalizations for acute and chronic heart failure. In addition, the registry data show a direct correlation between the degree of MVO and the risk of cardiac death in the immediate and long-term periods after a heart attack.

Thus, the success of PCI cannot be assessed solely by angiographic criteria. The true effectiveness of revascularization is determined not only by the patency of the epicardial vessel, but also by the functional state of the microcirculatory bed, which provides oxygen and nutrients at the myocardiocyte level [13]. In this context, microcirculatory disorders cease to be a minor complication and turn into a key pathophysiological node that determines the clinical fate of a patient after an acute heart attack. This necessitates the transition from the assessment of the "vessel" to the assessment of the "myocardium", as well as the development of strategies aimed at protecting microcirculation at all stages of treatment – from the prehospital stage to interventional intervention and subsequent drug therapy [14].

One of the central tasks of modern interventional cardiology is not only to restore patency of the infarct-dependent artery, but also to ensure adequate tissue perfusion in the area of injury [15]. However, as practice shows, even with technically successful primary angioplasty, a significant number of patients experience dissociation between macro- and microcirculation. To understand the mechanisms and predict such complications, a prospective study was analyzed involving patients who were urgently hospitalized with acute ST-segment elevation myocardial infarction and underwent primary percutaneous coronary intervention within the first 12 hours of the onset of symptoms [16]. All study participants underwent a comprehensive assessment, which included a clinical examination, laboratory diagnostics, instrumental imaging techniques, and a detailed analysis of the parameters of the intervention. The focal point for assessing microcirculatory dysfunction was microvascular obstruction (MVO), verified by cardiomagnetic resonance imaging (CMRI) in the late period (5-7 days after a heart attack), which allowed minimizing the effect of time fluctuations and increasing the reliability of diagnosis.

The analysis of multifactorial dependence revealed a number of clinically significant and statistically reliable predictors of the development of MVO. The strongest associated factor was the duration of ischemia before revascularization. In patients with an interval from the onset of symptoms to the onset of PCI of more than 4 hours, the probability of MVO formation increased 3.1–fold (OR 3.1; 95% CI 1.7-5.8; p = 0.002). This result is consistent with the known data on the progressive nature of ischemic damage: the longer the myocardium remains in hypoxia, the higher the degree of irreversible changes in the microcirculatory system, including endothelial dysfunction, edema of perivascular structures and activation of profibrous mechanisms [17]. Prolonged ischemia also contributes to the accumulation of metabolites, a reduction in reserves of energy substrates, and a decrease in the resistance of the vascular endothelium to reperfusion injury [18].

Equally important was the level of cardiospecific troponin I at the time of admission. Patients with high biomarker values (exceeding the 99th percentile of the upper limit of the norm by more than 15 times) had an increased risk of MVO (OR 2.6; 95% CI 1.4–4.9; p = 0.003). Although troponin is traditionally considered as a marker of necrosis volume, in this context, its early increase may reflect not only the size of the infarct zone, but also the degree of microvascular damage at the time of admission. This assumption is supported by data indicating a correlation between peak troponin values and MVO volume according to CMRI data [19].

Special attention in the study is paid to the angiographic characteristics of the lesion. The presence of a significant thrombotic substrate in the coronary artery lumen, assessed visually and using an occlusive index, was associated with an increased risk of microvascular obstruction (OR 2.9; 95% CI 1.6–5.3; p = 0.001). The mechanism of this phenomenon is associated with thrombus fragmentation during balloon dilation and stenting, which leads to distal embolization of thrombotic particles and subsequent obliteration of the terminal branches of the microcirculatory bed [20]. This factor highlights the importance of visual assessment of the nature of occlusion before the intervention and the potential expediency of adapting tactics (for example, more careful catheter manipulation, targeted thrombolysis or selective aspiration) in patients with high thrombus load.

Among the instrumental indicators, the most significant was the functional status of the left ventricle before the intervention, assessed by transthoracic echocardiography. In patients with an ejection fraction (EEF) of less than 40%, the risk of developing MBO was almost 3.4 times higher than in those with preserved systolic reserve (OR 3.4; 95% CI 1.8–6.5; p < 0.001). A decrease in LV can be considered as an integral marker of both the severity of ischemia and the presence of chronic microangiopathy, including concomitant ischemic cardiomyopathy [21]. In addition, myocardial dysfunction in the acute period of infarction is accompanied by increased intracavitary pressure and impaired diastolic relaxation, which creates an additional obstacle to effective perfusion in the microcirculatory system [22].

Of particular interest is the role of the systemic inflammatory response in the pathogenesis of microcirculatory disorders. The study assessed inflammatory markers, including C-reactive protein (CRP) and the neutrophil-lymphocyte index (NLR), a simple but informative indicator of systemic inflammatory activity calculated as the ratio of the absolute number of neutrophils to lymphocytes. Patients with CRP > 10 mg/L and NLR > 5.5 had a significantly higher incidence of MVO. An analysis under the ROC curve showed that NLR has a predictive accuracy of AUC = 0.72, and CRP has an AUC = 0.76, which indicates a moderate but clinically significant ability of these markers to differentiate patients with and without MVO [23].

The increased level of inflammation, apparently, enhances endothelial dysfunction, promotes the adhesion of leukocytes to the vascular wall, activates the coagulation link and reduces the ability of the microcirculatory bed to vasodilation in response to metabolic needs. Thus, the inflammatory status of the patient at the time of admission can serve not only as a reflection of the severity of the process, but also as a potential regulated risk factor, which opens up prospects for early intervention (for example, using anti-inflammatory strategies, including colchicine or IL-1b-inhibitors, in future studies).

DISCUSSION.

Mechanical removal of the thrombotic substrate prior to stent opening, the so–called aspiration thrombectomy, remains one of the key directions in an attempt to prevent the development of microvascular obstruction (MVO) during primary percutaneous coronary intervention (PCI) [25]. In theory, removal of the thrombus before dilation should reduce the risk of distal embolization, thereby preventing blockage of the terminal branches of the microcirculatory bed and maintaining adequate tissue perfusion [26]. However, the results of large randomized trials, including TASTE and TOTAL, have demonstrated the lack of a reliable clinical benefit from the routine use of aspiration techniques in the general population of patients with STEMI. The study reviewed confirms these data: when comparing groups with and without aspiration, there was no statistically significant decrease in the frequency of MBR (32.7% vs. 38.4%; p = 0.21), which indicates the limited effectiveness of this approach as a universal strategy.

Nevertheless, a detailed stratified analysis revealed a tendency to improve microcirculation in a subgroup of patients with severe thrombosis, determined by angiographic criteria (thrombosis > 70% of the lumen, visual assessment, TIMI Thrombus Grade 5 score). In this category, when aspiration catheterization was used, the incidence of MVO decreased from 46.2% to 35.1% (p = 0.07), and the median volume of obstruction according to CMRI decreased by 18%. Although the difference has not reached the level of statistical significance, the clinical difference seems biologically relevant. This suggests that aspiration thrombectomy may be justified not as a standard procedure, but as a selective measure used in the presence of a high thrombus load [27]. It is probably in such cases that mechanical removal of a blood clot has a protective effect by minimizing the number of embolized particles, which is critically important for maintaining microcirculatory integrity.

As for other parameters of interventional intervention, their influence on the development of MVO turned out to be indirect and indirect. For example, the time from the patient's admission to the hospital to the start of balloon dilation is traditionally considered as one of the key indicators of the quality of emergency care. In the study, a short interval (<90 minutes) was associated with a lower frequency of MVO (31% versus 44% at >120 minutes), however, with multifactorial regression, this relationship lost its significance. The analysis showed that the effect of time is realized not directly, but through the relationship with the duration of ischemia and the degree of inflammatory activation: patients with a long delay in intervention showed higher levels of CRP and

NLR, which, in turn, correlated with damage to the microcirculatory endothelium.

A similar pattern was observed with regard to stenting tactics. The use of modern drugs, elution stents (DES) with thin struts and biodegradable polymers, was associated with a lower frequency of MVO compared with older generations of stents (33% versus 42%) [28], but this difference was explained not so much by the type of implant itself as by the tactics of deployment: high-pressure postdilation was more often used in the DES group and intravascular imaging (OCT or SCD), which reduced the risk of dissection and incomplete attachment of struts, factors contributing to local thrombogenicity and subsequent inflammation.

The issue of using remote protection devices (RPD) deserves special attention. In theory, such devices should prevent distal embolization by intercepting thrombus fragments [29]. However, in real clinical practice, their effectiveness is limited by technical difficulties (positioning difficulties, the risk of collateral occlusion) and narrow indications. In the reviewed study, the use of RPD was rare (less than 8% of cases), which did not allow for a full-fledged statistical analysis. However, patients who had protective filters installed tended to have a better myocardial blush grade (MBG ≥2 in 70% versus 52% in the control group), which may indicate a potential, although clinically unrealized, effect.

It is important to note that most interventional parameters do not directly affect microcirculation, but through the activation of pro-inflammatory and procoagulant cascades[30]. For example, aggressive catheter manipulation in conditions of a large blood clot can cause massive platelet activation and release of pro-inflammatory cytokines, which exacerbates endothelial dysfunction. On the contrary, sparing tactics, including low-pressure predilation, targeted aspiration, and imaging monitoring, were associated with lower levels of post-interventional CRP and NLR, which probably determines a better microcirculatory outcome [31].

Thus, the effectiveness of an interventional strategy in the context of MVO prevention is determined not so much by individual techniques as by a holistic approach that takes into account the anatomical features of the lesion, the thrombo-inflammatory status of the patient and the individualization of tactics. This highlights the need to move from standardized algorithms to personalized intervention, where the choice of method depends on a comprehensive risk assessment at the pre-coronary stage [32].

The development of microcirculatory disorders in the acute period of myocardial infarction is a complex, polymorphic process, the formation of which involves interrelated and mutually reinforcing pathophysiological mechanisms [33]. The analysis of the data obtained convincingly demonstrates that microvascular obstruction (MVO) cannot be reduced to a single trigger or isolated event – it is the result of the convergence of many factors covering both the pre-existing condition of the patient and the dynamics of acute coronary syndrome, as well as the features of the interventional intervention [34]. Such a multifactorial nature of CBM determines the limited effectiveness of any unified prevention strategies and requires a transition to a more differentiated, pathogenetically based approach.

Predisposing conditions, including chronic endothelial dysfunction, concomitant diseases (hypertension, type 2 diabetes mellitus, CKD), as well as underlying inflammatory activity, play a key role in the initial stages of MBO formation [35]. These factors create a "vulnerable" microcirculatory bed, less capable of vasodilation, more prone to thrombosis, and less resistant to ischemic reperfusion injury. In such patients, even moderate thrombosis or short-term ischemia can lead to a pronounced violation of perfusion at the tissue level, whereas in individuals with preserved vascular reserve, a similar intervention may result in a favorable microcirculatory outcome.

Against these background, an acute coronary episode triggers a cascade of events, including massive platelet activation, release of pro-inflammatory cytokines (IL-6, TNF-α), neutrophil infiltration, and formation of neutrophil extracellular traps (NETs), which directly damage the endothelium and promote microthrombosis. In this context, systemic inflammation, reflected by such available markers as C-reactive protein (CRP) and the neutrophil-lymphocytic index (NLI), acts not just as a concomitant phenomenon, but as an integral indicator of the biological aggressiveness of the process, which determines the severity of microcirculatory damage [36].

At the same time, the anatomical and functional characteristics of the lesion - the volume of the thrombus, the localization of occlusion, and the degree of collateral blood flow – determine the mechanical risks, primarily the likelihood of distal embolization during manipulation of the coronary artery. The duration of ischemia, in turn, directly correlates with the depth of damage to the myocardium and microcirculatory endothelium, reducing the ability of blood vessels to adequately vasodilate even after the restoration of macroflow [37].

It is the combination of these components – clinical, laboratory, imaging, and interventional – that forms the ultimate risk of developing MBO. This makes it impossible to predict based on one or two parameters and necessitates a comprehensive, integrated approach to patient assessment at the earliest stages of hospitalization [38]. For example, a patient with prolonged pain (>4 hours), high NLR (>5.5), decreased EF on EchoCG and massive thrombosis on angiography has a multifactorial predisposition to MVO, and his tactics should differ from those of a patient with short ischemia, low inflammation and moderate thrombosis.

The integration of these data makes it possible to identify high-risk groups and move from a reactive to a preventive management style. In such patients, modification of the standard protocol may be justified already at the stage of primary PCI: gentle stenting tactics, targeted thrombus aspiration, the use of antiplatelet agents with rapid onset (for example, ticagrelor), and in the future, the use of adjuvant therapies aimed at protecting microcirculation (colchicine, IL–1b inhibitors, inhaled NO). In addition, the presence of a high risk may serve as a basis for earlier and expanded use of CMRI in order to monitor remodeling and correct therapy.

Thus, effective prevention of microcirculatory disorders is impossible without personalizing the approach based on a comprehensive assessment of the patient's pathophysiological profile. The future of interventional cardiology lies not in unification, but in adaptation: the choice of tactics should be determined not only by the anatomy of the vessel, but also by the state of the myocardium, inflammatory status, thrombogenicity and functional reserve of the microcirculatory bed. Only this approach can bridge the gap between angiographic success and true clinical outcome.

CONCLUSIONS.

The data obtained during the study confirm that microcirculatory disorders after successful primary percutaneous coronary intervention in patients with acute myocardial infarction are not a technical complication, but a complex pathophysiological phenomenon caused by the interaction of clinical, biological, anatomical and interventional factors. Despite the achievement of angiographic success – the restoration of blood flow through the infarct-dependent artery in accordance with the TIMI 3 criteria – a significant proportion of patients still have microcirculatory dysfunction, manifested as microvascular obstruction (MVO) or the phenomenon of "no-reflow". This condition is not an epiphenomenon, but directly determines the further clinical fate of the patient, associated with an increased risk of postinfarction remodeling of the left ventricle, progression of heart failure and increased cardiac mortality.

The analysis showed that the development of MVO is polymorphic and multifactorial in nature, where both pre—existing conditions (endothelial dysfunction, chronic inflammation, concomitant diseases) and dynamic parameters of the acute period play a key role - the duration of ischemia, the volume of the thrombotic substrate, the degree of activation of inflammatory and coagulation cascades. Independent predictors were identified: delayed revascularization of more than 4 hours, high troponin I levels on admission, a decrease in the left ventricular ejection fraction to 40%, massive thrombosis according to coronary angiography, as well as markers of systemic inflammation – CRP > 10 mg/l and NLR > 5.5. These indicators, considered together, form the pathophysiological profile of a patient at high risk of MVO, which opens up opportunities for early stratification and targeted intervention.

Importantly, traditional interventional strategies such as routine aspiration thrombectomy show limited effectiveness in the general population. However, a detailed analysis reveals a selective benefit: in patients with high thrombus load, aspiration is associated with a tendency to improve microcirculation, and early intervention has an indirect, indirect effect by reducing the duration of ischemia and, as a result, reducing inflammatory activation. This indicates that the effectiveness of intervention is determined not so much by individual techniques as by the adequacy of tactics to a specific pathophysiological context.

Thus, the key conclusion of this study is the need to move from unified protocols to a personalized management strategy for patients with STEMI. The success of revascularization should be assessed not only by the patency of the epicardial vessel, but also by the state of the microcirculatory bed, which is a true determinant of tissue perfusion and myocardial viability. This requires the integration of data obtained at the prehospital and early inpatient stages: the clinical picture, laboratory markers (including NLR and CRP), EchoCG and angiography results. This approach makes it possible to form a risk profile and adapt tactics before the start of PCI, from choosing antiplatelet therapy to deciding on gentle dilation, targeted aspiration, or planning early use of cardioprotective agents.

The prospects for further development are related to the development of multimodal forecasting algorithms that include both objective biomarkers and imaging technologies (CMRI, OCT, florent reserve microcirculation). The introduction of such models into clinical practice can become the basis for creating decision support systems at the intervention stage, which will improve the quality of medical care and improve long-term outcomes. The problem of microcirculatory disorders is not only a challenge for an interventional cardiologist, but also a call for a systemic rethinking of the goals of revascularization.

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