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Prediction and Prevention of Restenosis After Coronary Artery Stenting

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Abstract An integrated analysis of clinical outcomes between groups showed that the implementation of measures aimed at predicting and preventing restenosis after coronary artery stenting significantly reduced not only its incidence but also its severity, urgency of manifestation, and the burden on the healthcare system, emphasizing its high practical value and clinical effectiveness.

Keywords Coronary artery disease, Coronary arteries, Stenting, Restenosis

1. Introduction

Coronary artery disease (CAD) remains a leading cause of death and disability among adults in both developed and developing countries [1,3,5]. Stenting has become the gold standard for the treatment of stenotic forms of CAD, particularly in non-ST-segment-elevation ACS. However, despite the high efficacy of percutaneous coronary interventions (PCI) and the constant improvement of technologies, including the transition to drug-eluting stents, the problem of restenosis, the development of recurrent narrowing of the arterial lumen at the site of stent implantation, remains clinically significant. Furthermore, repeat interventions associated with restenosis increase the risk of adverse outcomes, highlighting the need to develop reliable methods for predicting and preventing this condition [2,4,6].

The incidence of restenosis after PCI depends significantly on the type of stent used. In the early days of interventional cardiology, drug-free stents, known as "bare-metal stents" (BMS), were used. These provided mechanical dilation of the vessel but did not impede pathological reparative processes in the vascular wall. As a result, the restenosis rate with BMS reached 20-30% within the first year after the procedure, especially in patients with high-risk factors [7,8,10].

Understanding the molecular and cellular mechanisms of the immune response to vascular injury enables the development of new approaches to the prediction and prevention of restenosis. Of particular interest are inflammatory biomarkers, which can not only identify high-risk patients but also serve as potential targets for personalized therapy.

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Study Objective: To develop and compare methods for the prediction and prevention of restenosis after coronary artery stenting.

2. Materials and Methods

This study was conducted at the Jizzakh branch of the Republican Specialized Scientific and Practical Center of Cardiology of the Ministry of Health of the Republic of Uzbekistan. The study included 198 patients with coronary heart disease who had undergone SCA, as well as 20 healthy individuals, deemed completely healthy by a medical commission and with no signs of cardiac pathology, who constituted the reference group.

All patients were divided into two groups: the control group consisted of 98 patients who were retrospectively followed in 2021-2022; the study group consisted of 100 patients examined prospectively in 2023-2024.

The reference group consisted of 10 men and 10 women without verified cardiovascular disease, matched for age to the main sample.

The study was a mixed cohort design, including a retrospective data analysis (control group) and prospective observation (study group). The total patient follow-up period was 12 months, with mandatory follow-up at 6 and 12 months, as well as as clinically indicated.

In the control group, assessment was based on actual clinical and angiographic outcomes.

In the study group, prognosis was performed until the onset of restenosis based on the RISCREST model, after which patients received preventive care based on their individual risk.

The study design included six stages of analysis. The first stage involved a clinical and anatomical study of patients who underwent PCI. The second stage involved an in-depth immunological examination of patients with various

clinical outcomes after PCI. The third stage analyzed the relationships between clinical and immunological characteristics. The fourth stage focused on the development of a clinical and immunological prognostic model. The fifth stage involved the implementation of an individualized restenosis prevention strategy in the study group of patients based on the calculated risk using the RISCREST scale. The sixth and final stage was aimed at assessing the effectiveness of the prognostic and preventive model we developed.

3. Results and Discussion

The most significant predictor, from both clinical and molecular points of view, was increased miR 21 expression. At a level above 2.0 units, the OR was 4.87 units (95% CI [2.12; 11.21]; p<0.001), which means an almost fivefold increase in the likelihood of restenosis. Among the clinical variables, T2DM proved to be the most convincing, with OR=2.91 (p=0.005), which emphasizes its multicomponent pathogenetic involvement from endothelial dysfunction to impaired repair and TLR signaling activation. CKD also demonstrated an independent association with restenosis (OR=2.53 units; p=0.023), confirming the role of uremic inflammation and autoimmune activation in the pathogenesis of vascular remodeling. A history of MI increased the risk of restenosis by 2.77 times (p=0.009), which is associated with the initial instability of the atherosclerotic process and probable post-infarction inflammation.

Among the anatomical factors that demonstrated the greatest prognostic power was the length of the lesion greater than 20 mm (OR=3.64 units (95% CI [1.76; 7.55]); p<0.001). The diameter of the lesion <2.75 mm was also significantly associated with an increased risk of restenosis (OR=2.38 units; 95% CI [1.18; 4.79]; p=0.016), which is explained by a more pronounced sensitivity of a narrow lumen to even moderate neointimal growth. Multifocal vascular disease turned out to be another anatomical factor, confirmed statistically (OR=2.89 units (95% CI [1.42; 5.91]; p=0.003), which is consistent with the concept of anatomical complexity as a factor of unfavorable vascular remodeling.

Based on logistic analysis and clinical and immunological data, the RISCREST (Prognostic Model for Assessing the Probability and Timing of Restenosis Development after Coronary Artery Stenting) program was developed. The model is implemented as a scoring scale and an adaptive algorithm based on artificial intelligence. The RISCREST model is based on variables significantly associated with restenosis, and inclusion criteria included both statistically significant differences between subgroups ($p < 0.05$) and confirmed correlations ($r \geq 0.4$).

All variables were divided into three groups: clinical, anatomical and functional, and immunological. Each variable was assigned a conditional weight, which formed the basis of the scoring model. Each risk factor was assigned a weight in points commensurate with its prognostic significance. Depending on the resulting value, the patient is assigned a risk level and a predicted immune phenotype of restenosis. (inflammatory-proliferative, autoimmune, or mixed). Based on the total score, we identified specific risk levels and predicted restenosis onset times.

With a score of 0-3, there is no risk of restenosis. With a score of 4-6, there is a low risk of restenosis. A score in the range of 7-10 corresponds to a moderate risk level. A score of ≥15 or more indicates a very high risk.

When analyzing the predicted restenosis onset times, the distribution was also similar between the groups. Thus, for a period of up to 6 months, there were 29 patients (29.6%) in the control group, and 31 (31%) in the main group, which differs by less than 5%. In the 7-12 month range, the rate was 21.4% in the control group and 23% in the main group. In the late restenosis category (>12 months), the proportions were 9.2% and 9%, respectively. Of particular importance is the distribution of the predicted risk by The predicted time frame in the control group almost exactly reproduces the pattern of actual restenosis onset. Thus, the number of patients predicted to develop restenosis in the first 6 months was 1.6 times higher (29 patients) than the actual number of patients with early restenosis (18 patients), reflecting the high sensitivity of the model. At the same time, the model slightly underestimated the likelihood of late restenosis, predicting it in 9 patients, while in fact it developed in 12 patients. This was most likely due to late autoimmune forms of restenosis, which were influenced by mechanisms that partially extend beyond the standard inflammatory predictors more characteristic of the early phenotype.

In addition to the development of a traditional scoring scale, an alternative form of the prognostic model was implemented, based on algorithms using artificial intelligence to analyze the entire data set. Standard metrics for determining the area under the receiver operating characteristic (ROC) curve (AUC) were used to assess the quality of the prognosis. The model using artificial intelligence proved particularly useful for interpreting Combined effects, such as moderately elevated miR 21 levels, the presence of calcification, and a subcritical vessel diameter in the absence of T2DM, may not trigger a significant trigger on the scoring scale, but in the artificial intelligence interpretation model, it is perceived as a cumulative risk approaching critical. In accordance with the developed risk stratification model using the RISCREST scale, the tactics of preventive monitoring and treatment of patients in the main group who underwent SCA were differentiated depending on the score and the corresponding risk level of restenosis.

In patients who, according to the prognostic assessment, were unlikely to develop restenosis (score of 0-3), preventive measures beyond standard cardiac management were not administered. CGA was performed solely for clinical indications, typically no earlier than 12 months after the intervention. Background therapy was used, including acetylsalicylic acid at a dose of 75-100 mg per day, one statin (atorvastatin). 20 mg or rosuvastatin 10 mg once daily), as well as an ACE inhibitor/ARB and a beta-blocker, if appropriately indicated.

In the presence of a low prognostic risk level (4-6 points), when the probability of restenosis development was assessed as minimal and predominantly late (after 12 months and later), more intensive clinical observation was carried out. In patients with a moderate risk of restenosis (7-10 points), in whom restenosis was predicted within 7 to 12 months, dynamic observation with control coronary angiography at 6-9 months was carried out. In the group of patients with a high risk (11-14 points), in whom restenosis most often developed within 6 months, a more aggressive preventive strategy was used. In the subgroup with a very high risk (≥15 points), where restenosis could develop in most patients within 3-4 months, the most.

4. Conclusions

1. Based on a clinical and immunological analysis of patients after coronary artery stenting and the identification of pathogenetic relationships, a personalized restenosis prognosis model, RISCREST, was developed. This model integrates clinical (diabetes mellitus, CKD, ischemia severity), anatomical and functional (lesion length and nature), and immunological parameters (IL 6, TLR4, miR 21, SII). The model allows for a quantitative assessment of the risk and timing of restenosis development and is implemented as a scoring scale with the ability to be algorithmically interpreted using artificial intelligence technologies.
2. Depending on the degree of predicted restenosis risk, a preventive algorithm was developed and implemented among patients in the main group. This algorithm is based on three principles: cardiac and immunological correction, as well as individualized monitoring timeframes. This approach has significantly reduced the incidence of restenosis by more than half compared to the baseline prediction, particularly among high- and very-high-risk patients, confirming its clinical efficacy and pathogenetic validity.
3. The use of a clinical-immunological risk stratification model and personalized prevention has significantly reduced both the incidence of restenosis and its clinical severity.

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