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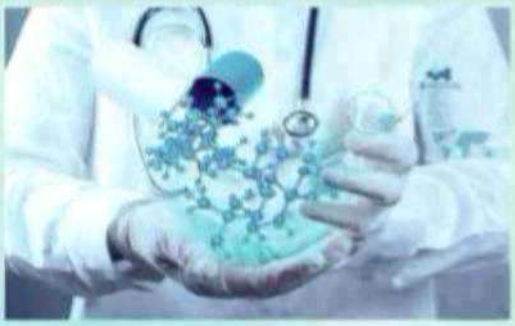

AVICENNA-MED.UZ

ISSN 2181-712X.
EiSSN 2181-2187

2 (88) 2026

ТИББИЁТДА ЯНГИ КУН
НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
Научно-реферативный,
духовно-просветительский журнал

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)

2 (88)

2026
февраль

www.bsmi.uz
https://newdaymedicine.com
E: ndmuz@mail.ru
Тел: +99890 8061882

Received: 20.01.2026, Accepted: 06.02.2026, Published: 10.02.2026

УДК 612.017.1+616.132.2-089

УСОВЕРШЕНСТВОВАНИЕ МЕТОДОВ ПРОФИЛАКТИКИ РЕСТЕНОЗОВ ПОСЛЕ СТЕНТИРОВАНИЯ КОРОНАРНЫХ АРТЕРИЙ

Абдурахманов Санжар Мухамеджанович https://orcid.org/0009-0004-4126-2573
e-mail: sanjarjon_2607@bk.ru
Хамдамов Бахтиёр Зарифович https://orcid.org/0000-0003-3569-6688
e-mail: xamdamov.baxtiyor@bsmi.uz

Бухарский государственный медицинский институт имени Абу Али ибн Сины, Узбекистан, г. Бухара, ул. А. Навои. 1 Тел: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Резюме***

Мероприятия, направленные на прогнозирование и профилактику рестеноза после стентирования коронарных артерий позволили достоверно снизить не только саму его частоту, но и его тяжесть, срочность манифестации и нагрузку на систему здравоохранения.

Ключевые слова: Ишемическая болезнь сердца, коронарные артерии, стентирование, рестеноз

KORONAR ARTERIYALARNI STENTLASHDAN KEYIN RESTENOZNING OLDINI OLISH USULLARINI TAKOMILLASHTIRISH

Abdurahmonov Sanjar Mukhamedjanovich https://orcid.org/0009-0004-4126-2573
e-mail: sanjarjon_2607@bk.ru
Xamdamov Baxtiyor Zarifovich https://orcid.org/0000-0003-3569-6688
e-mail: xamdamov.baxtiyor@bsmi.uz

Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti, O‘zbekiston, Buxoro sh. A. Navoiy kochasi 1 Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Rezyume***

Koronar arteriya stentlashdan keyin restenozni bashorat qilish va oldini olishga qaratilgan chora-tadbirlar nafaqat uning paydo bo'lishini, balki uning og'irligini, namoyon bo'lish shoshilinchligini va sog'liqni saqlash tizimiga yukni sezilarli darajada kamaytirdi.

Kalit so'zlar: Yurak ishemik kasalligi, koronar arteriyalar, stentlash, restenoz

IMPROVEMENT OF METHODS FOR PREVENTING RESTENOSIS AFTER CORONARY ARTERY STENTING

Abdurahmonov Sanjar Mukhamedjanovich https://orcid.org/0009-0004-4126-2573 *e-mail:* sanjarjon_2607@bk.ru
Khamdamov Baxtiyor Zarifovich https://orcid.org/0000-0003-3569-6688 *e-mail:* xamdamov.baxtiyor@bsmi.uz

Bukhara State Medical Institute named after Abu Ali ibn Sina, Uzbekistan, Bukhara, st. A. Navoi. 1 Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Resume***

Measures aimed at predicting and preventing restenosis after coronary artery stenting have significantly reduced not only its incidence, but also its severity, the urgency of its manifestation, and the burden on the healthcare system.

Keywords: Coronary heart disease, coronary arteries, stenting, restenosis

Relevance

Stenting has become the gold standard for the treatment of stenotic forms of coronary artery disease, 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 [1, 3, 5, 7]. 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 [7, 8, 9].

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.

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

Materials and methods

This study was conducted at the Jizzakh branch of the Republican Specialized Scientific and Practical Center of Cardiology. The study included 198 patients with coronary artery disease who had undergone SCA, as well as 20 healthy individuals deemed completely healthy by a medical commission and without signs of cardiac pathology, who constituted the reference group.

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 undergoing PCI. The second stage involved an in-depth immunological assessment of patients with various clinical outcomes after PCI. The third stage analyzed the relationships between clinical and immunological characteristics. The fourth stage focused on developing a clinical and immunological prognostic model. The fifth stage implemented an individualized restenosis prevention strategy in the study group based on the calculated risk using the RISCREST scale. The sixth and final stage aimed to evaluate the effectiveness of the prognostic and preventive model we developed.

Results and discussion

Among the anatomical factors that demonstrated the greatest prognostic power was a lesion length of more than 20 mm (OR=3.64 units (95% CI [1.76; 7.55]); $p<0.001$). A lesion diameter <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 the greater sensitivity of a narrow lumen to even moderate neointimal growth. Multifocal vascular lesions turned out to be another anatomical factor, statistically confirmed (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 in unfavorable vascular remodeling.

Based on logistic analysis and clinical and immunological data, a The RISCREST program (a prognostic model for assessing the likelihood and timing of restenosis development after coronary artery stenting). 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 the inclusion criteria were 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-functional, and immunological. Each variable was assigned a conditional weight, which formed the basis of a scoring model. Each risk factor was assigned a point weight proportional to its prognostic significance. Depending on the final score, 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 timing of restenosis manifestation.

A score of 0-3 indicates no risk of restenosis. A score of 4-6 indicates a low risk of restenosis. A score of 7-10 indicates a moderate risk. A score of 15 or more indicates a very high risk.

When analyzing the predicted timing of restenosis manifestation, the distribution between the groups was also similar. 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, a difference of less than 5%. In the interval of 7-12 months, the indicator 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 significance is that the distribution of the predicted risk by time in the control group almost exactly reproduces the structure of the actual onset of restenosis. Thus, the number of patients with a prognosis of 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 it actually developed in 12 patients. This was most likely due to late autoimmune forms of restenosis, which developed under the influence of mechanisms partially beyond the standard inflammatory predictors more characteristic of the early phenotype.

In addition to the development of a traditional scoring system, an alternative prognostic model was implemented, based on algorithms utilizing 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 prediction. The AI-based model proved particularly useful for interpreting combined effects, such as the combination of moderately elevated miR-21 levels, the presence of calcification, and a subcritical vessel diameter in the absence of T2DM. While this may not significantly affect the scoring system, the AI-based interpretation model interprets it as a combined risk approaching critical. In accordance with the developed risk stratification model using the RISCREST scale, the tactics of preventive monitoring and therapy of patients in the main group who underwent SCA were differentiated depending on the score and the corresponding level of risk of restenosis development.

In patients with a prognostic assessment showing no risk of restenosis (score of 0-3), preventive measures beyond standard cardiac care were not administered. Cardiovascular angiography (CAG) was performed solely when clinically indicated, typically no earlier than 12 months after the procedure. Background therapy was administered, including aspirin 75-100 mg daily, a statin (atorvastatin 20 mg or rosuvastatin 10 mg once daily), an ACE inhibitor/ARB, and a beta-blocker, if indicated.

In patients with a low prognostic risk (score of 4-6), when the risk of restenosis was assessed as minimal and primarily long-term (12 months or later), more intensive clinical follow-up was performed. Patients with a moderate risk of restenosis (score 7-10), in whom restenosis was predicted to occur within 7 to 12 months, underwent dynamic observation with control coronary angiography at 6-9 months. In the high-risk group of patients (score 11-14), in whom restenosis most frequently developed within 6 months, a more aggressive prophylactic strategy was used. In the very high-risk subgroup (score ≥15), in whom most patients were expected to develop restenosis within 3-4 months, a more intensive prophylactic regimen was used.

In addition to the scoring scale for individual prognosis, an important element of the personalized approach was the assessment of the immune phenotype of restenosis. As we have previously described, restenosis developed through various pathogenetic mechanisms, reflecting the dominance of one or another component of the immune response. Elevated inflammatory markers or the appearance of symptoms allow for the patient to be transferred to a higher level of care, whereas persistent normalization of the immune profile and a stable disease course may warrant a reduction in the intensity of follow-up. Accordingly, the RISCREST prognostic model and preventive algorithm we developed can function as an adaptive clinical management tool.

The effectiveness of the developed preventive algorithm was assessed based on the difference between the baseline prognostic level of restenosis development in patients after SCA and the follow-up period.

Actual data on the development of restenosis in the study group after the implementation of the individualized prognostic and preventive strategy developed by us showed that the overall frequency of an adverse outcome was 25%, which is significantly lower than the similar indicator in the control group, where restenosis was recorded in 47.9%. Thus, the implementation of an individualized intervention based

on a clinical and immunological prognosis ensured a reliable reduction in the frequency of complications by 22.9 percentage points, or almost 2-fold.

In general, the main causes leading to the development of restenosis, even with the use of the methods we developed for the prediction and prevention of this outcome of SCA, are: mechanical overstrain of the stent with an extended lesion and multiple implantations; hemodynamically unstable zones (bends, bifurcations) with increased local turbulence; calcified walls that reduce elasticity and adequate deployment of the stent; small vessel diameter, where even minimal neointimal growth can become clinically significant; Situations requiring emergency PCI, in which post-dilation and precise technique correction are impossible; a combination of several unfavorable features, in which even effective prevention is unable to suppress the cumulative negative effect. The summarized comparative data showed that the incidence of restenosis in the study group corresponded to a 1.9-fold decrease as a result of the use of our developed methods for predicting and preventing this outcome. Moreover, a particularly pronounced difference was observed in the early stages. Thus, at ≤6 months, restenosis developed in 18 patients out of 47 (38.3%) in the control group, and in only 13 out of 100 patients in the study group.

Thus, an integrated analysis of clinical outcomes between groups showed that the implementation of measures aimed at predicting and preventing restenosis after SCA 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.

Conclusions:

1. The RISCREST personalized restenosis prognosis model, based on the integration of clinical, anatomical, functional, and immunological parameters (IL 6, TLR4, miR 21, SII), enables 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 based on three principles was developed and implemented among patients in the main group: cardiac and immunological correction, as well as individualized monitoring timeframes.

3. The use of a clinical-immunological risk stratification model and personalized prevention significantly reduces both the incidence of restenosis and its clinical severity.

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Entered 20.01.2026