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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE**

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IMMUNOGENETIC PREDISPOSITION OF PATIENTS TO NON-INFECTIOUS POSTOPERATIVE COMPLICATIONS OF CORONARY ARTERY BYPASS GRAFTING

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✓ *Resume*

The developed program makes it possible to predict the likelihood of a favorable or unfavorable course of the postoperative period without the need for an extensive and labor-intensive examination.

Keywords: coronary artery bypass grafting, postoperative complications.

НЕИНФЕКЦИОННЫЕ ПОСЛЕОПЕРАЦИОННЫЕ ОСЛОЖНЕНИЯ АОРТОКОРОНАРНОГО ШУНТИРОВАНИЯ

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✓ *Резюме*

Разработанная программа даёт возможность прогнозирования вероятности благоприятного или неблагоприятного течения послеоперационного периода без необходимости расширенного и трудоемкого обследования.

Ключевые слова: аортокоронарное шунтирование, послеоперационные осложнения.

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✓ *Rezyume*

Ishlab chiqilgan dasturiy yechimning amaliy ahamiyati keng qamrovli va ko'p vaqt talab qiladigan tekshiruvsiz operatsiyadan keyingi davrning ijobiy yoki salbiy kechish ehtimolini oldindan aytib berish qobiliyatidadir.

Kalit so'zlar: aortokoronar шунтлаш, operatsiyadan keyingi asoratlar.

Relevance

Coronary artery disease continues to be a leading cause of overall mortality and disability worldwide, remaining one of the key medical and social problems in modern medicine. Despite significant progress in primary and secondary prevention, a significant proportion of patients with severe and widespread forms of coronary atherosclerosis still require surgical myocardial revascularization (1,3,5). In this context, CABG remains the most radical and clinically effective treatment option, improving the

prognosis and survival of patients with multivessel coronary disease and a high risk of adverse outcomes (2,4).

Against the backdrop of a projected increase in the number of coronary artery surgeries and an increasing proportion of high-risk patients, the problem of CABG is becoming persistent and requires a rethinking of existing approaches to its prevention (2,4,6,7,8,9,10). The limited effectiveness of universal prophylactic regimens and the high interindividual variability of clinical outcomes highlight the need to develop more precise and personalized patient management strategies that address not only clinical but also biological characteristics.

Study objective: to develop methods for diagnosing the immunogenetic predisposition of patients to non-infectious postoperative complications of coronary artery bypass grafting.

Materials and methods

The clinical material for the study included observations of 166 patients with coronary artery disease who underwent elective CABG. All examinations and observations were conducted as part of a prospective study.

The control group consisted of 82 patients (49.4%) who underwent surgery in 2023-2024 and received standard postoperative care without the use of our developed LDA. The study group consisted of 84 patients (50.6%) who underwent surgery in 2025-2026 and received the personalized LDA, based on immunogenetic phenotyping, for the prevention of NIPO CABG and the management of PO. Additionally, the study included 20 clinically healthy individuals, recognized by the medical commission as practically healthy based on the results of a comprehensive examination, who were recruited to establish reference values for immunological and immunogenetic parameters. The relationships between laboratory, immunological, and clinical parameters were assessed using correlation analysis with the calculation of the correlation coefficient r . Multivariate logistic regression analysis was used to identify independent factors associated with the development of NIPO CABG. Model results were presented as the regression coefficient (β), standard error (SE), significance level (p), odds ratio (OR), and odds ratio CI (95%). Variables for multivariate analysis were selected based on their clinical significance and the results of preliminary univariate comparisons.

Results and discussion

In the presence of multiple interrelated indicators, simple comparison of mean values or even correlations do not allow one to definitively determine which parameters maintain an independent relationship with the cumulative course of complications when other components of the immune response are taken into account. The final model included IL-6, MCP-1, IL-10, IL-8, the NLR index, tryptase, IL6-174G/C, TNFA-308G/A, FCGR2A H131R polymorphisms, and the sVCAM-1 endothelial activation index, ensuring a balance between the immunological and immunogenetic components while maintaining a clinically relevant vascular link. IL-6 was one of the leading independent predictors of the cumulative course of complications; its increase was associated with an almost 1.8-fold increase in the odds of cumulative complications ($p < 0.001$). Pathogenetically, this change corresponds to the role of IL-6 as a mediator of systemic proinflammatory activation, which is associated not only with the severity of the early response to surgical trauma but also with its prolongation, creating conditions for the transition from functional disorders to more persistent forms of dysregulation.

The significance of IL-6 in the multivariate model remained consistent when other parameters were considered, indicating its independent contribution rather than a reflection of the overall inflammatory background.

The consistency of immunological and genetic levels within the same pathogenetic pathway proved crucial for interpreting the results. Carriage of the IL6-174G/C (GC+CC) variant was associated with a 1.77-fold increase in the odds of cumulative progression ($p=0.010$).

MCP-1 made a significant contribution to the model, with an increase in its levels increasing the odds of cumulative progression by 1.55-fold ($p=0.002$). Pathogenetically, MCP-1 reflects chemokine activity and recruitment of the monocyte-macrophage system, which is associated with the maintenance of the inflammatory process and its transition to a more protracted phase. Within the context of cumulative disease progression, this is particularly significant, as this marker is functionally associated with mechanisms that prolong inflammation and create conditions for recurrent or late complications.

The anti-inflammatory component in the model is reflected by IL-10, which demonstrated an inverse relationship with cumulative disease progression. An increase in IL-10 was associated with a decrease in the odds of cumulative disease progression by approximately 40% (OR=0.59; $p=0.002$).

IL-8 was included in the model as a marker of early neutrophil-chemokine activation; its increase increased the odds of cumulative disease progression by 1.48 times ($p=0.009$). In the context of cumulative disease progression, IL-8 is important not as a general marker of inflammation, but as a reflection of a scenario in which early mediator load reaches a level capable of triggering a longer chain of dysregulation, especially when combined with a genetic predisposition and an insufficient regulatory response. The integral cellular NLR index retained significance in the multivariate model and was associated with a 1.62-fold increase in the odds of cumulative progression ($p=0.003$).

Allergy-like reactivity is reflected by tryptase levels, with increases increasing the odds of cumulative progression by 1.58 times ($p=0.005$). Tryptase is included in the model as a pathogenetically significant factor in early postoperative reactions associated with mast cell activation and vascular mediator effects.

The genetic component of the model includes the TNFA-308G/A polymorphism, whose carriage was associated with a 1.67-fold increase in the odds of cumulative disease ($p=0.014$), consistent with the role of TNF- α as a key proinflammatory mediator involved in initiating and maintaining systemic activation.

The FCGR2A H131R (RR) polymorphism, which is associated with a 1.99-fold increase in the odds of cumulative disease ($p=0.016$), deserves special attention. Its inclusion in the model is justified by the fact that this genetic marker reflects the characteristics of interaction with immune complexes and clearance, and may also be associated with thromboinflammatory and vascular mechanisms.

The vascular-endothelial component of the model is represented by the sVCAM-1 level, an increase in which was associated with a 1.4-fold increase in the OR of cumulative progression ($p=0.028$). It is included as a reflection of endothelial activation and microcirculatory dysregulation, which are pathogenetically associated with the development of organ complications and prolongation of the systemic response. Its presence in the model emphasizes that the cumulative variant of complicated progression is not limited to cytokine mechanisms and includes the vascular component as a functional bridge between the systemic immune response and the clinical manifestations of organ dysfunction.

Based on the correlation and multivariate analysis that identified independent immunological and genetic predictors of an unfavorable course of PO, we developed a diagnostic scale for determining the IGF predisposition to NIPO ("IGF-NIPO"), which combines clinically and pathogenetically significant indicators of the systemic immune response and the hereditary background of patients. The development of this scale was aimed at integrating heterogeneous biological characteristics into a single diagnostic tool, allowing the transition from statistical analysis of risk factors to practical stratification of patients by the nature and severity of immunogenetic vulnerability (Table 1).

A key feature of the developed scale is its differentiated approach to assessing immunological and genetic components. The immunological component of the scale is represented by a multi-level gradation of indicators reflecting the dynamics and amplitude of the SVR.

Table 1

Diagnostic scale for determining the immunogenetic phenotype of patients with a predisposition to NIPO CABG ("IGF-NIPO")

INDICATOR	PHENOTYPE AND SCORE		
	No (0 points)	Low (1 point)	High (3 points)
IL-6, pg/ml	<11	11-22	>22
IL-8, pg/ml	<15	15-25	>25
MCP-1, pg/ml	<200	200-300	>300
IL-10, pg/ml	$\geq 2,0$	1,1-1,9	$\leq 1,0$
NLR, units.	$\leq 3,5$	3,6-5,0	>5,0
Tryptase, $\mu\text{g/L}$	$\leq 5,5$	5,6-7,5	$\geq 7,6$
sVCAM-1, ng/ml	≤ 750	751-900	>900
IL6-174G/C	GG	GC / CC*	
TNFA-308G/A	GG	GA / AA*	
FCGR2A H131R	HH / HR		RR RR*

**Note: The presence of any unfavorable genetic variant, regardless of the degree of clinical implementation, was considered as a high level of genetic predisposition regardless of the table column and was scored as 3 points.*

For each immunological marker, three levels were defined, corresponding to the absence of clinically significant abnormalities, transient activation characteristic of early functional complications, and severe or prolonged activation associated with organ-specific and cumulative forms of complicated disease. Level boundaries were determined based on the maximum recorded values of the corresponding indicators in clinical subgroups, ensuring a direct correlation between the scale and actual data.

The genetic block in the IGF-NIPO scale has a different conceptual load in the form of background risk modifiers and is not subject to quantitative grading. The absence of unfavorable genetic variants is classified as the absence of immunogenetic vulnerability, while the presence of any unfavorable genetic variant, regardless of the degree of clinical manifestation, is considered a high level of genetic predisposition and is assigned a score of 3 points.

The total score on the IGF-NIPO CABG scale was calculated by summing the scores for all included indicators. Based on the obtained value, three IGF levels were distinguished. Values of ≤ 4 points characterized patients without clinically significant immunogenetic vulnerability, in whom the systemic immune response was adaptive and did not result in persistent postoperative complications. A range of 5 to 14 points corresponded to a "low" IGF level and reflected predominantly transient immunological activation, manifested as early functional POPs without signs of cumulative pathological process. A total value of ≥ 15 points characterized a "high" IGF level and was associated with pronounced and/or prolonged immune activation, often combined with an unfavorable genetic background, creating conditions for sequential or repeated implementation of NIPO CABG.

Thus, the developed "IGF-NIPO" CABG scale allows for the systematization of immunological and genetic data and their translation into a form suitable for the practical diagnosis of IGF patients. Moreover, based on the results of retrospective summation of scores on the IGF-NIPO CABG scale, the absence of IGF was detected in 19 (23.2%) patients in the control group. In the remaining 63 (76.8%) patients in the control group, IGF of varying severity was detected according to the IGF-NIPO CABG scale.

Among the patients with identified IGF, the "low" level was diagnosed in 38 (60.3% among patients with the phenotype). This category was characterized predominantly by transient immunological activation and the development of early functional NIPO CABG without signs of their prolongation or repeated implementation. "High" IGF levels were established in 25 patients (39.7% among patients with the phenotype). This category of patients was characterized by pronounced and/or prolonged immune activation, often in combination with an unfavorable genetic background, which was clinically manifested by sequential or repeated implementation of NIPO CABG. In general, based on the formed array of retrospective data from the control group, we created a structured database designed for the diagnosis of IGF NIPO CABG. This database includes the results of clinical observation, immunological profile indicators and information on the genetic background of patients who underwent CABG, with subsequent determination of the actual nature of the course of PO. The database was formed after the completion of a full cycle of examinations, which made it possible to compare the quantitative values of the indicators with the actual realized variants of the postoperative response and to create a reference array for further diagnostic interpretation.

The use of this dataset allowed us to move from a static scale assessment to the development of a software diagnostic framework based on AI technologies. The IGF-NIPO-AI program is designed to minimize the volume of required input data while maintaining diagnostic information. In practical use, the physician only needs to enter information about the presence or absence of unfavorable genetic variants and a limited set of key immunological parameters obtained during standard laboratory testing. The remaining parameters, including the probable degree of immunological activation and the total score on the IGF-NIPO CABG scale, are calculated automatically based on a comparison of the entered data with the accumulated patterns of the control group. The practical significance of the developed software solution lies in the ability to predict the likelihood of a favorable or unfavorable course of POP without the need for extensive and time-consuming examination.

Conclusions

1. The developed "IGF-NIPO" scale allows for the systematization of immunological and genetic data and their translation into a form suitable for practical diagnostics of IGF patients.

2. The practical significance of the developed software solution lies in the ability to predict the likelihood of a favorable or unfavorable course of POP without the need for extensive and time-consuming examination.

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