



Original study article | Оригинальное исследование
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Early prediction of acute myocardial infarction in patients with new coronavirus infection and acute coronary syndrome

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Abstract

Background. Considering the wide prevalence of COVID-19 (SARS-CoV-2) worldwide and in the Russian Federation, high frequency of its mutations and non-persistent post-infection and post-vaccination immunity, the epidemic potential of COVID-19 persists. The experience of the pandemic demonstrated high mortality among individuals with coronavirus and ACS (acute coronary syndrome), specifically, from myocardial infarction (MI).

Aim: to create a multifactorial model for prediction of myocardial infarction using laboratory and instrumental data of progression of COVID-19 in ACS patients.

Material and methods. The open prospective non-randomized study included 104 patients with ACS due to severe COVID-19 hospitalized in 2022. To solve the problem of early prediction of MI among patients, observation groups were formed: Group 1 (n=35), patients with unstable angina and Group 2 (n=69) with MI. All patients underwent round-the-clock monitoring of vital functions using a dynamic bedside monitor. Laboratory parameters (general clinical and biochemical), cytokine levels (1b, 2, 4, 6, 10, interleukins, γ interferon, tumor necrosis factor α) and instrumental parameters (CT of chest organs, EchoCG) were studied. Scores were calculated using the SOFA (Sepsis-related Organ Failure) and SAPS II (Simplified Acute Physiology Score) scales. Statistical data processing was performed in the SPSS 25.0 software suite. Mathematical

modeling was performed using multidimensional logistic regression. An analysis of the characteristic curves (ROC curves) in the predicted probability of developing MI in the multidimensional model was performed. The results were considered statistically significant at $p < 0.05$. Based on the data obtained, a multidimensional logistic regression model was constructed with step-by-step inclusion or exclusion of predictors using the Wald algorithm.

Results. The prognostic model included SAPS II scores, cytokines (γ interferon, TNF α), and CT scans. The analysis revealed that the developed mathematical model for assessing the risk of MI in patients with ACS on the background of severe COVID-19, created by the method of multidimensional logistic regression based on cytokine profile, lung CT and SAPS II scale, has a sensitivity of 98.6% and a specificity of 85.7%.

Conclusion. Early predictors of MI development have been established in COVID-19 patients with ACS: the degree of lung damage according to CT data, the number of points on the SAPS II scale, levels of interferon and tumor necrosis factor, on the basis of which a mathematical model has been built that allows predicting MI in patients with severe COVID-19.

Keywords: COVID-19, myocardial infarction, cytokines, model, prediction.

Conflict of interest: nothing to disclose.

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Возможности прогнозирования развития инфаркта миокарда у больных новой коронавирусной инфекцией с острым коронарным синдромом

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Аннотация

Обоснование. В связи с широким распространением вируса SARS-CoV-2 в мире и в России, его постоянными мутациями, а также нестойким поствакцинальным и постинфекционным иммунитетом у населения, в настоящее время сохраняется эпидемический потенциал новой коронавирусной инфекции (COVID-19). Опыт пандемии продемонстрировал высокую летальность среди больных COVID-19 с острым коронарным синдромом (ОКС), в частности, от инфаркта миокарда (ИМ).

Цель: на основании лабораторно-инструментальных особенностей течения COVID-19 у больных с ОКС разработать модель прогнозирования развития ИМ у данной категории больных.

Материал и методы. В открытое проспективное нерандомизированное исследование были включены 104 пациента с ОКС на фоне тяжелого течения COVID-19, госпитализированных в 2022 году. Для решения задачи прогнозирования ИМ среди пациентов были сформированы группы наблюдения: группа 1 (n=35) – пациенты с нестабильной стенокардией (НС) и группа 2 (n=69) – с ИМ. Всем пациентам проводился круглосуточный мониторинг витальных функций с помощью прикроватного монитора в динамике. Исследовались лабораторные (общеклинические, биохимические) показатели, уровень цитокинов (интерлейкины: 1b, 2, 4, 6, 10, интерферон γ , фактор некроза опухоли α); инструментальные (КТ

органов грудной клетки, ЭхоКГ); рассчитывали баллы по шкалам SOFA (Sepsis-related Organ Failure) и SAPS II (Simplified Acute Physiology Score). Статистическая обработка данных выполнена в среде пакета SPSS 25.0. Выполняли математическое моделирование с помощью многомерной логистической регрессии. Проводили анализ характеристических кривых (ROC-кривых) в предсказанной в многомерной модели вероятности развития ИМ. Результаты считали статистически значимыми при $p < 0,05$. По полученным данным была построена многомерная модель методом логистической регрессии с пошаговым включением или исключением предикторов по алгоритму Вальда.

Результаты. В прогностическую модель вошли баллы по шкале SAPS II, цитокины (интерферон гамма, фактор некроза опухоли альфа) и степень поражения легких по данным компьютерной томографии. В ходе прове-

денного анализа установлено, что разработанная математическая модель оценки риска развития ИМ у больных с ОКС на фоне тяжелой формы COVID-19, созданная методом многомерной логистической регрессии, обладает чувствительностью 98,6% и специфичностью 85,7%.

Выводы. Установлены ранние предикторы развития ИМ у пациентов с ОКС на фоне COVID-19: степень поражения легких по данным КТ, количество баллов по шкале SAPS II, уровни цитокинов (интерферона и фактора некроза опухоли). На основании данных предикторов разработана математическая модель, позволяющая прогнозировать ИМ на фоне тяжелой формы COVID-19.

Ключевые слова: COVID-19, инфаркт миокарда, цитокины, модель, прогнозирование.

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Список сокращений

COVID-19 – новая коронавирусная инфекция; ОКС – острый коронарный синдром; НС – нестабильная стенокардия; ИМ – инфаркт миокарда; СРБ – С-реактивный белок; КФК – креатинфосфокиназа; ИЛ – интерлейкин; ФНО- α – фактор некроза опухоли- α ; SOFA – Sepsis-related Organ Failure; SAPS II – Simplified Acute Physiology Score.

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INTRODUCTION

Due to the wide prevalence of the SARS-CoV-2 virus worldwide and in Russia, its continued mutations and the unstable post-vaccination and post-infection immunity among the population, the epidemic potential of the new coronavirus infection (COVID-19) still persists. The experience of the pandemic showed a high level of lethality (up to 40%) from cardiovascular complications among COVID-19 patients [1]. N.R. Smilowitz et al. (2020) report that a third of patients hospitalized with COVID-19, already had signs of myocardial damage as early as on admission; follow-ups showed that the share of such patients reached 47%, which resulted in a quadruple increase of intra-hospital mortality, from 9.7% to 39.1% [2]. A 3.3-fold increase of mortality in the event of an acute coronary syndrome (ACS) in COVID-19 patients is reported by Turkish researchers T. Çınar et al. (2022) [3]. Currently, a number of direct and indirect mechanisms are studied by which SARS-CoV-2 influences the development of cardiovascular complications: from tissue penetration to induction of a massive systemic inflammatory reaction [4]. Cytokine-mediated myocardial damage, as the leading cause of cardiac damage under COVID-19, is reported in a study of American authors [5]. SARS-CoV-2 disrupts the interaction of the angiotensin converting enzyme 2 (ACE2), renin-aldosterone and kinin-kallikrein systems that balance the inflammation, cell proliferation and platelet aggregation resulting in the hemostasis disorder and myocardial damage [6]. The work of Russian researchers shows a multi-component mechanism of ischemia/infarction of the myocardium under COVID-19 that includes the dysfunction of the renin-angiotensin-aldosterone system, hyperinflammation and 'cytokine storm', endothelial dysfunction and coagulopathy, hypoxemia and hypoxia [1]. The clinical diagnostics of the

ACS against the background of an acute infectious disease may be complicated: pains in the chest, respiratory and cardiovascular failure are characteristic both for a severe form of COVID-19 and for ACS including unstable angina (UA) and myocardial infarction (MI). According to cardiologists' recommendations, diagnostics of the ACS shall include ECG and blood serum troponin test; however, some authors point out that the test is not always specific, especially in the event of an acute infectious disease [7, 8]. Therefore, the search for laboratory indicators and data of instrumental analysis of the predictors to predict MI, that would be available in practical healthcare, still remains a vital question.

AIM

To create a model for prediction of myocardial infarction using laboratory and instrumental data of progression of COVID-19 in ACS patients.

MATERIAL AND METHODS

The open prospective non-randomized study included 104 patients with ACS on the background of severe progression of COVID-19 (predominantly, Omicron strain), hospitalized throughout the year 2022. The diagnosis of COVID-19 and ACS, their verification, and patient treatment was performed according to the effective provisional methodological recommendations "Prevention, diagnostics and treatment of the new coronavirus infection (COVID-19)" and clinical recommendations of the Society of Cardiology¹.

Inclusion criteria: men and women aged 50 to 80 years with severe form of COVID-19 combined with ACS who received no glucocorticosteroids and anticoagulants on the pre-hospitalization stage, availability of a signed informed consent.

¹ Provisional methodological recommendations "Prevention, diagnostics and treatment of the new coronavirus infection (COVID-19)", version 16, 2022; Clinical recommendations "Acute coronary syndrome without ST segment elevation of the cardiogram" 2020; Clinical recommendations "Acute coronary syndrome with ST segment elevation of the cardiogram" 2020.

Predictors	Group I, UA (n=35) Me (Q1; Q3)	Group II, MI (n=69) Me (Q1; Q3)	p-value
Age	63.00 (54.00; 68.00)	64.00 (54.50; 76.50)	0.121
CT of lung involvement, %	40.00 (35.00; 45.00)	45.00 (40.00; 60.00)	<0.001
SAPS II score	16.00 (15.00; 18.00)	24.00 (21.00; 25.50)	<0.001
SOFA score	16.00 (14.00; 17.00)	16.00 (14.00; 18.00)	0.462
Thickness of the pericardium cavity, mm	5.00 (2.00; 7.00)	5.00 (3.00; 7.00)	0.912
Systolic pressure in the PA (mm Hg)	49.90 (35.00; 54.60)	51.60 (46.00; 58.50)	0.118
Pulmonary acceleration time, ms	61.00 (45.00; 80.00)	47.00 (36.00; 69.00)	0.004
Banded neutrophils, %	2.00 (1.00; 4.00)	8.00 (6.00; 10.00)	<0.001
Segmentonuclear neutrophils, %	55.00 (49.00; 70.00)	72.00 (64.55; 78.50)	<0.001
Lymphocytes, %	30.00 (19.00; 36.00)	11.40 (7.80; 20.00)	<0.001
Platelets, 10 ⁹ /L	322.00 (224.00; 416.00)	175.00 (126.00; 234.00)	<0.001
Total protein, g/L	63.80 (59.40; 69.90)	54.10 (48.95; 61.35)	<0.001
C-reactive protein, mg/L	76.00 (35.20; 129.70)	157.80 (110.95; 252.00)	<0.001
CPK, U/L	1179.00 (765.00; 1504.00)	2088.00 (1974.50; 2394.50)	<0.001

Table 1. Characteristics of potential predictors (instrumental and laboratory) of MI in ACS patients with severe COVID-19

Таблица 1. Характеристика потенциальных предикторов (инструментальных и лабораторных) ИМ у пациентов с ОКС на фоне тяжелой формы COVID-19

Exclusion criteria: patients with severe and terminal concomitant pathologies, oncological, autoimmune and allergic diseases, patients with co-infections (viral hepatitis, type B, C, and HIV infection), mental disorders, pregnancy, refusal from examination. To address the task of IM prediction among COVID-19 patients admitted for hospitalization, two groups were formed according to ACS progression type: Group I (n=35), patients with unstable angina, and Group II (n=69), patients with myocardial infarction.

All patients underwent 24-hour monitoring of vital functions using the bedside monitor Nihon Kohden PVM-2703 (Japan) taking the electrocardiogram (ECG), heart rate (HR), arterial blood pressure (BP), respiratory rate (RR), saturation (SpO₂), body temperature (T^o). The ECG was registered daily on the "Aksion EC3TC-3/6-04" (Russian Federation). Transthoracic echocardiography (EchoCG) was taken for each patient at least twice using the portable ultrasonic examination device GE HealthCare LOGIQ E, manufactured by General Electric (USA). Scores were calculated using the SOFA (Sepsis-related Organ Failure) and SAPS II (Simplified Acute Physiology Score) scales. Computed tomography (CT) of the chest organs was performed on the CT scanner GE Revolution EVO (Russian Federation). Laboratory tests were performed on the hematological (Mindray BC-6800, China) and biochemical (Roche Cobas c 311, Switzerland) analyzers, the cytokine tests (interleukins (IL): 1b, 2, 4, 6, 10, interferon γ (IFN- γ), tumor necrosis factor α (TNF- α) were tested using diagnostic kits (R and D Diagnostics Inc., USA) with the sensitivity of 1 pg/mL.

Statistic processing of data was performed in the SPSS 25.0 suite (IBM Corporation, Armonk, New York, USA, License No.5725-A54). Normality of distribution was assessed using the Shapiro-Wilk test. Descriptive statistics are presented as median and quartiles: Me (Q1; Q3). The Mann-Whitney U test was used for group comparisons. Multivariate logistic regression modeling was performed. Receiver operating characteristic (ROC) curve analysis was conducted using the probability of MI development predicted by the multivariate model. Results were considered statistically significant at $p < 0.05$.

Predictors	Group I, UA (n=35) Me (Q1; Q3)	Group II, MI (n=69) Me (Q1; Q3)	p-value
IL-1b, pg/mL	8.51 (8.25; 8.92)	9.17 (8.58; 9.74)	<0.001
IL-2, pg/mL	0.05 (0.04; 0.08)	0.08 (0.05; 0.11)	0.019
IL-4, pg/mL	10.91 (10.63; 11.23)	10.31 (9.79; 10.77)	<0.001
IL-10, pg/mL	86.60 (84.22; 89.68)	84.44 (81.72; 87.16)	0.003
IFN- γ , pg/mL	9.75 (9.36; 11.41)	8.88 (8.32; 9.56)	<0.001
TNF- α , pg/mL	22.21 (21.46; 23.92)	23.69 (21.56; 25.53)	0.026
IL-6, pg/mL	14.90 (13.90; 15.50)	15.84 (15.05; 16.53)	<0.001

Table 2. Characteristics of potential predictors (cytokine profile) of MI in ACS patients with severe COVID-19

Таблица 2. Характеристика потенциальных предикторов (цитокиновый профиль) ИМ у пациентов с ОКС на фоне тяжелой формы COVID-19

RESULTS

The average age of patients with the severe form of COVID-19 and ACS was 63.00 (54.25; 72.75) years, there were 56.7% women. The patients were admitted in the end of the first or beginning of the second week of the disease (days of admission in the comparison groups: 9.00 (7.00–11.00) and 9.00 (7.00–12.00), respectively, $p=0.369$). The groups were comparable in sex and age, duration of the disease and structure of concomitant pathology. The level of blood saturation with oxygen on admission was from 78.00% to 99.00%, the median values between the groups did not differ statistically (95.00% in patients of Group I and 94.00%, in Group II, $p=0.178$). More than 70 laboratory and instrumental indicators were analyzed in patients with ACS against the background of severe COVID-19; the most significant of them follow in **Tables 1** and **2**.

Using the obtained data, the risk of MI was evaluated using logistic regression method. On the first stage, univariate models were constructed, when each equation forcibly included only one risk factor (predictor). Using the results of these models, exponential coefficients of regression were derived interpreted as odds ratio (OR) and their 95% confidence intervals (95% CI) (**Table 3**). As expected, the indicators that had no differences in paired comparisons, turned out to be statistically insignificant

Indicator	OR [95% CI]	p-value
Age	1.02 (0.99–1.05)	0.260
CT lung involvement, %	1.07 (1.02–1.11)	0.002
SAPS II score	3.62 (2.01–6.49)	<0.001
SOFA score	1.06 (0.88–1.28)	0.559
Thickness of the pericardium cavity, mm	0.99 (0.84–1.17)	0.929
Systolic pressure in the PA (mm Hg)	1.03 (0.99–1.06)	0.155
Pulmonary acceleration time, ms	0.97 (0.96–0.99)	0.005
Banded neutrophils, %	1.80 (1.45–2.24)	<0.001
Segmentonuclear neutrophils, %	1.11 (1.06–1.17)	<0.001
Lymphocytes, %	0.88 (0.83–0.93)	<0.001
Platelets, 10 ⁹ /L	0.99 (0.98–0.99)	<0.001
Total protein, g/L	0.88 (0.82–0.93)	<0.001
C-reactive protein, mg/L	1.02 (1.01–1.03)	<0.001
CPK, U/L	1.00 (1.00–1.01)	<0.001
IL-1b, pg/mL	3.71 (1.79–7.67)	<0.001
IL-2, pg/mL	5.31 (1.42–19.89)	0.013
IL-4, pg/mL	0.33 (0.16–0.67)	0.002
IL-10, pg/mL	0.83 (0.73–0.94)	0.002
IFN-γ, pg/mL	0.36 (0.22–0.57)	<0.001
TNF-α, pg/mL	1.33 (1.06–1.68)	0.014
IL-6, pg/mL	1.95 (1.33–2.85)	0.001

Table 3. Assessment of MI risk in ACS patients with severe COVID-19 by univariate logistic regression: combination of models

Таблица 3. Оценка риска ИМ у больных с ОКС на фоне тяжелой формы COVID-19 методом одномерной логистической регрессии: совокупность моделей

predictors in the equations. The majority of studied laboratory and instrumental indicators manifested as risk factors with OR above one (1). Elevated values of the following indicators are associated with poor prognosis: lung involvement as seen on CT, SAPS II score, percentage of banded and segmentonuclear neutrophil leukocytes, concentration of the C-reactive protein, IL-1b, IL-2, TNF-α, IL-6 and activity of CPK in the blood serum. Indeed, all of these signs are markers of severity of COVID-19 progression. Conversely, the lower pulmonary acceleration time and lower concentrations of total protein, IL-4, IL-10, IFN-γ are associated with more favorable prognosis: the OR of these indicators is below one (1).

Then, different variants of multivariate models were constructed by the logistic regression method with step-by-step inclusion of predictors using the Wald algorithm. The models differed not only in their construction approach but also in their initial sets of potential predictors. The issue is that many of these predictors are closely interrelated and

therefore cannot be simultaneously selected by a stepwise algorithm for inclusion in the regression equation. Thus, the SAPS II severity scale is statistically significantly interrelated with all of the studied cytokines (correlation coefficients from 0.3 to 0.6 in absolute values). Thus, the inclusion of SAPS II score in the number of potential predictors ‘displaced’ the other risk factors from the equation, that were included in the prognostic model in the univariate or multivariate version without this severity scale. Some of the prognostic indicators were included in all constructed mathematical models, and some varied from one variant to another. In this paper, we make an example of the model with the best analytical characteristics (**Table 4**). Thus, the area under the ROC curve (AUC) was 0.99 ± 0.01, and Youden’s index at a threshold probability of 0.22 was 0.84. These results indicate that the constructed model has excellent discriminatory performance.

According to the constructed multivariate logistic regression, the probability of MI development in patients with COVID-19 with ACS can be calculated using the following equation:

$$p = 1 / (1 + e^{-(1,71X1 + 0,16X2 - 1,10X3 + 0,77X4 - 45,99)}),$$

where e – base of natural logarithms (rounded to 2.72); X1 – SAPS II score; X2 – CT lung involvement percentage; X3 – concentration of gamma-interferon in the blood serum in pg/mL; X4 – concentration of the tumor necrosis factor alpha in the blood serum in pg/mL.

The SAPS II scale turned out to be the most powerful prognostic factor of MI with the odds ratio (OR)=5.52 (95% CI: 1.86–16.42) (p=0.002). The second most influential predictor was the CT lung involvement degree with OR=1.17 (95% CI: 1.00–1.36) (p=0.045). TNF-α and IFN-γ, while keeping their roles of the risk factor and protective factor, turned out statistically insignificant (p=0.083 and p=0.056). Nevertheless, such levels of significance (more than 0.05, less than 0.10) in the exploratory mathematical models are viable, in the opinion of some authors. The decrease of their prognostic capacity is accounted for by the interrelation with two more powerful predictors, which turn out to be sufficient for the predicted variant of the ACS, viz. the myocardial infarction. The model had high predictive accuracy. The sensitivity, with the threshold probability of 0.22, was 98.6%, and the specificity was 85.7%.

At the next stage, this model was validated on a test dataset. The test set comprised 30 patients with COVID-19 who presented with ACS, which subsequently resolved as

Predictors in the model	Regression coefficient b	SE b	Wald’s statistics	OR (95% CI)	p-value
SAPS II score	1.71	0.56	9.45	5.52 (1.86–16.42)	0.002
CT lung involvement, %	0.16	0.08	4.00	1.17 (1.00–1.36)	0.045
TNF-α, pg/mL	-1.10	0.58	3.66	0.33 (0.11–1.03)	0.056
IFN-γ, pg/mL	0.77	0.44	3.01	2.16 (0.91–5.14)	0.083
Constant	-45.99	19.12	5.79	–	0.016

Table 4. Assessment of MI risk in ACS patients with severe COVID-19 by multivariate logistic regression using cytokine profile, lung CT and SAPS II score

Таблица 4. Оценка риска ИМ у больных с ОКС на фоне тяжелой формы COVID-19 методом многомерной логистической регрессии по цитокиновому профилю, КТ легких и шкале SAPS II

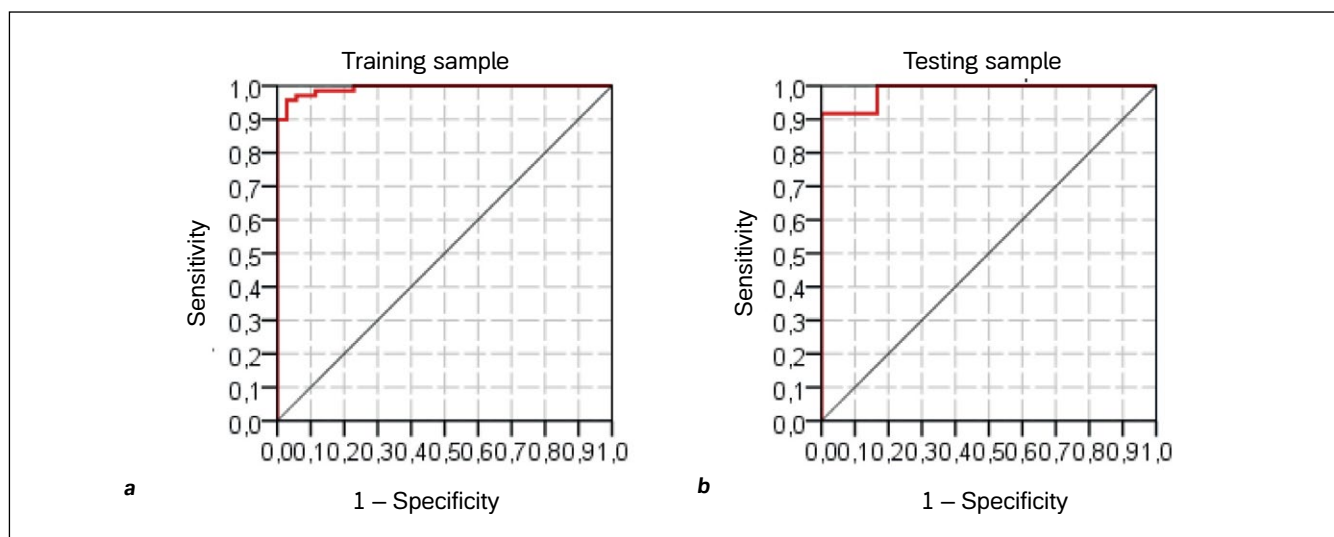


Figure 1. ROC curves of MI risk prediction in ACS patients with severe COVID-19 by multivariate logistic regression models: a – training sample, b – testing sample.

Рисунок 1. ROC-кривые прогнозирования риска ИМ у пациентов с ОКС на фоне COVID-19 по многомерным моделям логистической регрессии: а – по обучающей выборке, б – по тестовой выборке.

UA in 6 individuals and MI in 24. These patients were not included in the construction of the mathematical model described above. For all of them, the probability of MI risk was calculated using the regression coefficients obtained from the training dataset.

At the next stage, for patients in the main (training) and testing samples the ROC-curves were plotted (**Fig. 1**). The area under the ROC-curve for the testing sample was 0.98 ± 0.02 , and the Youden's index was 0.79.

The areas under the curve of these models are shown in **Table 5**.

DISCUSSION

As of today, the available literature describes individual models predicting ACS in COVID-19, despite the significant contribution of this complication in the overall mortality under the new coronavirus infection. Thus, M. Rashid et al. (2021) used the analysis of 517 cases of COVID-19 with ACS to report high in-hospital mortality (24.2%) in this group of patients and note its increase to 41.9% within 30 days after recovery from COVID-19 [9]. According to I.I. Serebrennikov et al. (2023), the cumulative mortality (60 days) in the COVID-19 with the ACS cohort was 48.3% [8]. There are several foreign and Russian studies

Tested model	AUC	SE AUC	p	95% CI
MI model: testing sample	0.99	0.01	<0.001	0.98–1.00
Testing of the MI model on the testing sample	0.98	0.02	<0.001	0.95–1.00

Notes: AUC – area under curve, SE AUC – standard error of AUC, p – statistic significance of difference from a useless classifier.

Примечания: AUC – area under curve – площадь под графиком, SE AUC – standard error of AUC – стандартная ошибка AUC, p – статистическая значимость отличия от бесполезного классификатора.

Table 5. Area under curve of the prediction model of MI risk assessment in ACS patients with severe COVID-19

Таблица 5. Площадь под ROC-кривой прогностической модели оценки риска ИМ у пациентов с ОКС на фоне COVID-19

focusing on predictors of adverse outcomes of ACS under COVID-19: level of ceramides (in the analysis of the metabolome profile), complex of 8 parameters (age, atrial fibrillation status, severe and extremely grave progression of the SARS-CoV-2 infection, acute kidney injury, chronic kidney failure of stage 2 and above, levels of ferritin, albumen, glucose), that predict the risk of a lethal outcome in the ACS but not its variant [10, 11]. In multicenter cohort studies performed in China and Iran, in the acute period of COVID-19 with the development of ACS, a correlation was identified between the calcification of coronary arteries, blood calcium level, and adverse outcome (in-hospital death) [12, 13]. The retrospective study of N.R. Smilowitz et al. (2020) demonstrated a close correlation between the degree and the duration of increase of cardiac troponin in COVID-19 patients with subsequent critical progression of ACS and lethal outcome [2]. There are individual Russian studies focusing on the outcome of ACS in the post-COVID period. There is a multivariate regression model of adverse outcomes of ACS in patients in the post-COVID period with the following predictors: chronic heart failure, presence of soluble fms-like tyrosine kinase-1, hypokinesia zones on EchoCG, carrier status of the TT/AA genotype of the rs2285666 genetic marker of the ACE2 gene. The sensitivity of the model is 93.5%, specificity, 21.8%, accuracy, 76.6% [14, 15]. We could not find in the available literature a model that would have predicted the variant of ACS development with high accuracy and specificity using predictors available in practical healthcare at the peak of the COVID-19 disease. Undoubtedly, such a model is needed to prevent development of negative scenarios of the acute coronary syndrome.

CONCLUSION

The proposed mathematical model allows for prediction of myocardial infarction in patients with a severe form of COVID-19 with a sensitivity of 98.6% and specificity of 85.7%. This provides not only practical but also scientific value, since the group of independent predictors includes not only the known factors determining the severity of the main disease, but also the two cytokines characterizing the immune response to the infection. ■

ADDITIONAL INFORMATION	ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ
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Study funding. The study was the authors' initiative without external funding.	Источник финансирования. Работа выполнена по инициативе авторов без привлечения финансирования.
Conflict of interest. The authors declare that there are no obvious or potential conflicts of interest associated with the content of this article.	Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с содержанием настоящей статьи.
Contribution of individual authors. A.V. Lunina: scientific data collection, systematization and analysis, writing of the manuscript. D.Yu. Konstantinov, L.L. Popova: study concept, detailed manuscript editing and revision. All authors gave their final approval of the manuscript for submission, and agreed to be accountable for all aspects of the work, implying proper study and resolution of issues related to the accuracy or integrity of any part of the work.	Участие авторов. А.В. Лунина – сбор и обработка научного материала, написание текста; Д.Ю. Константинов, Л.Л. Попова – разработка концепции исследования, редактирование текста. Все авторы одобрили финальную версию статьи перед публикацией, выразили согласие нести ответственность за все аспекты работы, подразумевающую надлежащее изучение и решение вопросов, связанных с точностью или добросовестностью любой части работы.
Statement of originality. No previously published material (text, images, or data) was used in this work.	Оригинальность. При создании настоящей работы авторы не использовали ранее опубликованные сведения (текст, иллюстрации, данные).
Data availability statement. The editorial policy regarding data sharing does not apply to this work.	Доступ к данным. Редакционная политика в отношении совместного использования данных к настоящей работе не применима.
Generative AI. No generative artificial intelligence technologies were used to prepare this article.	Генеративный искусственный интеллект. При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.
Provenance and peer review. This paper was submitted unsolicited and reviewed following the standard procedure. The peer review process involved 2 external reviewers.	Рассмотрение и рецензирование. Настоящая работа подана в журнал в инициативном порядке и рассмотрена по обычной процедуре. В рецензировании участвовали 2 внешних рецензента.

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