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FEATURES OF THE COURSE OF NON-ST ELEVATION MYOCARDIAL INFARCTION IN PATIENTS WITH A HISTORY OF COVID-19

<i>Aim</i>	To study the clinical course of non-ST segment elevation myocardial infarction (NSTEMI) in hospitalized patients after COVID-19 and to evaluate the effect of baseline characteristics of patients on the risk of complications.
<i>Material and methods</i>	The study included 209 patients with NSTEMI; 104 of them had had COVID-19. The course of myocardial infarction (MI) was analyzed at the hospital stage, including evaluation of the incidence rate of complications (fatal outcome, recurrent MI, life-threatening arrhythmias and conduction disorders, pulmonary edema, cardiogenic shock, ischemic stroke, gastrointestinal bleeding).
<i>Results</i>	Mean age of patients after COVID-19 was 61.8±12.2 years vs. 69.0±13.0 in the comparison group (p<0.0001). The groups were comparable by risk factors, clinical data, and severity of coronary damage. Among those who have had COVID-19, there were fewer patients of the GRACE high risk group (55.8% vs. 74.3%; p<0.05). Convalescent COVID-19 patients had higher levels of C-reactive protein and troponin I (p<0.05). The groups did not significantly differ in the incidence of unfavorable NSTEMI course (p>0.05). However, effects of individual factors (postinfarction atherosclerosis, atrial fibrillation, decreased SpO ₂ , red blood cell concentration, increased plasma glucose) on the risk of complications were significantly greater for patients after COVID-19 than for the control group (p<0.05).
<i>Conclusion</i>	Patients with NSTEMI, despite differences in clinical history and laboratory data, are characterized by a similar risk of death at the hospital stage, regardless of the past COVID-19. Despite the absence of statistically significant differences in the incidence of in-hospital complications, in general, post-COVID-19 patients showed a higher risk of complicated course of NSTEMI compared to patients who had not have COVID-19. In addition, for this category of patients, new factors were identified that previously did not exert a clinically significant effect on the incidence of complications: female gender, concentration of IgG to SARS-CoV-2 ≥200.0 U/l, concentration of C-reactive protein ≥40.0 mg/l, total protein <65 g/l. These results can be used for additional stratification of risk for cardiovascular complications in patients with MI and also for development of individual protocols for evaluation and management of NSTEMI patients with a history of COVID-19.
<i>Keywords</i>	COVID-19; post-COVID syndrome; myocardial infarction
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Local and systemic inflammation are known to play significant role in the development of atherosclerosis and the pathogenesis of acute coronary syndrome (ACS) [1]. While smoking, arterial hypertension (AH), obesity, dyslipidemia, and other well-studied factors cause endothelial damage, dysfunction, and local inflammation, some patients with ACS do not have such risk factors.

This provides an incentive to identify and study other predictors. Previous studies have confirmed the hypothesis of a viral etiology for myocardial infarction (MI) along with the role of systemic inflammation in the development and progression of cardiovascular diseases (CVDs) [2–4]. Systemic inflammation due to cytokine expression during the infectious process can trigger the activation

of macrophages and lymphocytes comprising atherosclerotic plaque, as well as its growth, cap rupture, and atherothrombosis [5, 6]. Several published studies have shown that patients with elevated plasma levels of inflammatory markers are at a high risk of adverse cardiovascular events [7, 8].

COVID-19 is an infectious disease caused by the novel Beta variant of SARS-CoV-2 coronavirus leading to severe respiratory syndrome. While the majority of patients have mild or asymptomatic COVID-19, its symptoms are more severe in about 20% of patients [9]. Local inflammation and cytolysis can result in the release of large amounts of pro-inflammatory cytokines (interleukin-6, interleukin-1, tumor necrosis factor) [10, 11], whose elevated levels of cytokines are correlated with the severity of COVID-19 [12]. It is also significant that levels of inflammation markers remain elevated in some patients in the post-COVID-19 period despite the resolution of symptoms [13]. Elevated levels of cardiospecific troponin may be indicative of viral damage of the myocardium [14].

In this connection, a special at-risk group consists of cardiovascular patients. The phenotypic resemblance between cardiovascular patients predisposed to more severe course of COVID-19 determines a considerable risk of adverse cardiovascular events in the acute and long-term periods. [15]. Persistent inflammation, oxidative stress, endothelial and microcirculatory damage, as well as procoagulant conditions, can contribute to the development of adverse cardiovascular events and aggravate MI in patients with a history of COVID-19 [5, 8, 16].

Objective

To study the clinical course of non-ST-elevation myocardial infarction (NSTEMI) during hospital stay and its outcomes in patients with a history of COVID-19 along with the effect of the baseline patient characteristics on the risk of complications.

Material and methods

We analyzed the data of patients treated for NSTEMI in V.P. Demikhov City Clinical Hospital from July 2020 to March 2021. The study included patients of 18 years and older with NSTEMI, hemodynamically significant coronary artery stenosis shown by coronary angiography (CAG) and without respiratory infections at the time of admission. Exclusion criteria were verified COVID-19 at the time of admission

or COVID-19 developed during hospital stay, COVID-19 vaccination, acute myocardial injury due to percutaneous coronary intervention (PCI) or surgical treatment.

All patients underwent PCR for COVID-19 and SARS-CoV-2 antibody tests at admission. Previous COVID-19 infection established using medical history and information collected from discharge summaries or other medical documents was verified by elevated titers of specific antibodies. Asymptomatic cases were identified by increased SARS-CoV-2 IgG titers without assessing previous symptoms. MI was diagnosed in accordance with the fourth Universal Definition of MI Expert Consensus Document (2018) of the European Society of Cardiology [17].

Patients underwent a generally accepted volume of examinations, including complete blood count, biochemical blood test, and coagulogram. The following examinations were performed: electrocardiography (ECG) at admission and later; echocardiography; CAG followed by PCI if indicated.

Anti-SARS-CoV-2 antibody titers were estimated semi-quantitatively using a CL 6000i analyzer (Shenzhen Mindray Bio-Medical Electronics Co.; China); reference values <2 U/mL for IgM and <10 U/mL for IgG were used.

The data were processed using Excel 2016 applications (Microsoft, USA), Statistica 10 (StatSoft Inc, USA) and SAS JMP 11 (SAS, USA). Normally distributed quantitative data were expressed as the mean and standard deviation (M (SD)), and non-normally distributed data were presented as the median and interquartile range (Me [25th percentile; 75th percentile]). The normality of data distribution was verified using the Lilliefors test (based on the Kolmogorov-Smirnov test). The Mann-Whitney U-test was used to evaluate the significance of differences between the groups. Qualitative variables were expressed as the absolute values and percentages (n (%)). Pearson's chi-squared test or Fisher's exact test for small samples were used to determine the statistical significance of differences between the qualitative variables. Relative risk (RR) and 95% confidence interval (CI) were calculated to analyze the dependence of the outcomes on the factors of interest. Statistical significance was defined as $p < 0.05$.

Results

The study included 209 patients divided into two groups. The study group included patients with

Table 1. General patient characteristics

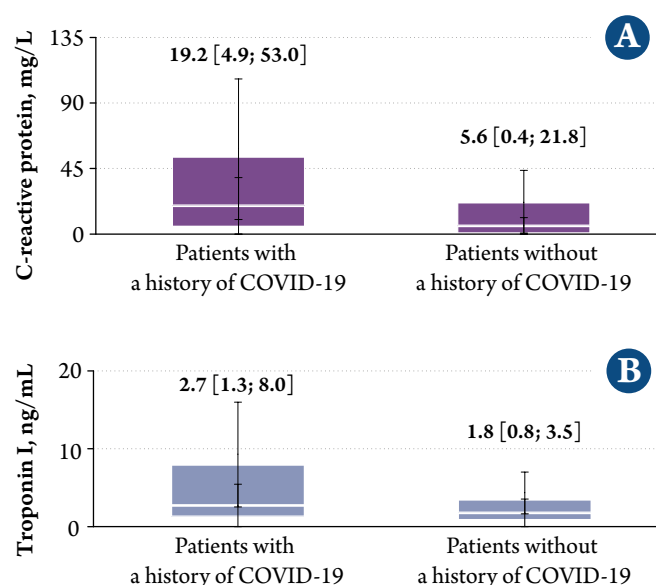
Parameter	Study group (n=104)	Control group (n=105)	p
Age, years	61.8±12.2	69.0±13.0	<0.0001
Male	51 (49.0)	64 (61.0)	0.0842
History of angina	80 (76.9)	93 (88.6)	0.0258
Postinfarction cardiosclerosis	35 (33.7)	39 (37.1)	0.5979
Burdened family history	19 (18.3)	19 (18.1)	0.9740
Diabetes mellitus	34 (32.7)	29 (27.6)	0.4242
Arterial hypertension	80 (76.9)	85 (81.0)	0.4750
Obesity	67 (64.4)	62 (59.0)	0.4252
Time after COVID-19, months	49.0 [34.0; 82.0]	-	-
IgM, U/mL	0.5 [0.3; 3.6]	-	-
IgG, U/mL	85.5 [39.5; 233.3]	-	-
Anatomical location of MI			
• Anterior	47 (45.2)	66 (62.9)	0.0112
• Inferior	37 (35.6)	19 (18.1)	
• Other	20 (19.2)	20 (19.0)	
AHF class (Killip)			
• I	77 (74.0)	74 (70.5)	0.5611
• II	13 (12.5)	20 (19.0)	
• III	12 (11.5)	10 (9.5)	
• IV	2 (1.9)	1 (1.0)	
High risk (GRACE)	58 (55.8)	78 (74.3)	0.0150
Multi-vessel coronary artery disease	27 (26.0)	32 (30.5)	0.4695

MI – myocardial infarction; AHF – acute heart failure.

a history of COVID-19 and positive serological test (n=104), while the control group included patients without a history of COVID-19 and with negative serological test (n=105). Patients has been previously compared by basic clinical and anamnestic characteristics, data of laboratory tests and examinations [18]. The comparison of the two groups is summarized in Table 1.

The patients of the study group were statistically significantly younger and had a history of exertional angina less frequently. The distributions of comorbidities, anamnestic data and risk factors were comparable between the groups (p>0.05).

A total of 28.2% of patients (n=59) took acetylsalicylic acid before the onset of NSTEMI, and 5.7% (n=12) took P2Y12 inhibitors. Oral anticoagulants were administered by 12% of patients (n=25), while angiotensin-converting enzyme inhibitors were received by 21.1% of patients

Figure 1. Levels of C-reactive protein (Figure 1A; p=0.0007) and troponin I (Figure 1B; p=0.0091) in the patient groups


(n=44). Beta-blockers and statins were taken by 21.5% (n=45) and 21.1% (n=44), respectively. The groups were comparable by the main classes of drugs administered before the onset of NSTEMI (p>0.05).

Patients' clinical status did not differ statistically significantly between the groups (p>0.05). The percentage of patients with high risk according to the GRACE score was statistically significantly lower than in the control group (p=0.015). The median GRACE score was 150.0 [130.0; 164.0] in the study group, which was statistically significantly lower than 159.0 [140.0; 186.0] in the control group (p=0.0169). Inferior MI was more common in the study group (p=0.0112). The number of patients with multi-vessel coronary artery disease is comparable between the groups (p=0.4695). Symptom-related coronary artery was stented in 98.1% of cases (n=102) in the study group and in 92.3% (n=97) in the control group (p=0.1010). PCI of other coronary arteries was performed equally frequently in both groups (p=0.3355). Complete revascularization was achieved in 53.9% of patients (n=56) in the study group and 58.1% (n=61) in the control group (p=0.5371).

Left ventricular ejection fraction (LVEF) was comparable in both groups (p=0.1687). Pulmonary artery systolic pressure (PASP) was statistically significantly higher in the study group (35.0 [30.0; 47.0] mm Hg vs. 30.0 [29.0; 35.0] mm Hg, respectively; p=0.0097). Echocardiographic signs of pericardial effusion were more common

Table 2. Clinical course of MI during hospital stay

Parameter	Study group (n=104)	Control group (n=105)	p
Time in hospital, days	8.0 [5.0; 9.2]	8.0 [6.0; 10.0]	0.3786
Death	7 (6.7)	5 (4.8)	0.5407
Complicated hospital period	20 (19.2)	19 (18.1)	0.8331

Table 3. Frequency of complications during hospital stay

Parameter	Study group (n=104)	Control group (n=105)	p
Recurrent myocardial infarction	1 (1,0)	0	0,3138
Ventricular fibrillation	1 (1,0)	1 (1,0)	0,9946
AV block grade 3	3 (2,9)	3 (2,9)	0,9905
CVA	2 (1,9)	0	0,1533
GI bleeding	0	3 (2,9)	0,0825
Pulmonary edema	12 (11,5)	10 (9,5)	0,6351
Cardiogenic shock	2 (1,9)	1 (1,0)	0,6214

AV – atrioventricular; GI – gastrointestinal; CVA – cerebrovascular accident.

among patients of the study group (7.7% vs. 1.0%, respectively; $p=0.0164$).

Figure 1 presents graphs comparing the levels of C-reactive protein (CRP) and troponin I in the patient groups. Both indicators were statistically significantly higher in the study group.

The duration of hospital stay was about 8 weeks in both groups. Table 2 contains data on the clinical course of MI during hospital stay. In both groups, the majority of patients had uncomplicated MI: 80.8% ($n=84$) in the study group and in 81.9% ($n=86$) in the control group. The mortality rate was 6.7% in the study group and 4.8% in the control group ($p=0.541$).

A complicated course was reported when one or more clinical conditions developed; these included: fatal outcome; MI recurrence; pulmonary edema; cardiogenic shock; ventricular fibrillation; complete atrioventricular block; acute cerebrovascular accident; gastrointestinal bleeding. The incidence of complications during hospital stay was comparable between the groups: $RR=1.06$ (95% CI 0.60–1.87; $p=0.8331$). There was no statistically significant difference in the distribution of these complications between the groups (Table 3). In the study group, recurrent MI was reported in one patient, while two patients had ischemic stroke. There were no such cases in the control group.

The ROC-analysis allowed establishing the cut-off points for quantitative indicators based on clinical and statistical significance, and the effects

Table 4. Analysis of the relative risks of complications in patients with NSTEMI during hospital stay

Factor	Study group		Control group	
	RR (95% CI)	p	RR (95% CI)	p
Female	3,85 (1,38–10,74)	0,0038	1,4 (0,62–3,16)	0,4114
PICS	7,89 (2,85–21,81)	<0,0001	2,9 (1,25–6,75)	0,0095
Bronchial asthma	3,49 (1,51–8,06)	0,0177	2,84 (1,20–6,76)	0,0318
Atrial fibrillation	5,93 (2,99–11,77)	<0,0001	2,45 (1,12–5,40)	0,0273
Multi-vessel coronary artery disease	3,88 (1,77–8,51)	0,0004	2,53 (1,12–5,73)	0,0224
SpO ₂ <89.0%	7,67 (4,05–14,50)	<0,0001	4,4 (2,11–9,16)	0,0015
LVEF<50 %	3,59 (1,49–8,66)	0,0021	3,56 (1,10–11,52)	0,0180
Erythrocytes<3.5×10 ¹² /L	6,27 (3,94–9,97)	<0,0001	3,91 (1,70–8,96)	0,0028
Hematocrit<35.0%	5,21 (2,64–10,30)	<0,0001	3,91 (1,70–8,96)	0,0028
Hemoglobin<110.0 g/L	4,05 (2,02–8,10)	0,0002	4,64 (2,10–10,25)	0,0003
Leukocytes≥9.0×10 ⁹ /L	2,6 (1,03–6,61)	0,0312	1,67 (0,66–4,22)	0,2733
Glucose≥8.0 mmol/L	7 (2,51–19,49)	<0,0001	3,24 (1,20–8,75)	0,0134
Total protein<65.0 g/L	4,24 (1,88–9,58)	0,0009	2,81 (0,97–8,15)	0,0738
CRP≥40.0 mg/L	3,67 (1,54–8,71)	0,0033	2,6 (0,64–10,56)	0,1816
IgG≥200.0 U/L	3,66 (1,61–8,35)	0,0010	-	-
GFR<47.0 mL/min/1.73 m ²	11,35 (2,77–46,42)	<0,0001	6,86 (0,95–49,69)	0,0518

NSTEMI – non-ST-elevation myocardial infarction; CI – confidence interval; RR – relative risk; PICS – postinfarction cardiosclerosis; LVEF – left ventricular ejection fraction; SpO₂ – blood oxygen saturation; CRP – C-reactive protein; GFR – glomerular filtration rate.

of the identified factors on the risk of complications were analyzed in both groups (Table 4).

Although most of the identified factors significantly increased the risk in both groups, RR was higher in most cases in the study group than in the control group. Several factors identified to have a statistically significant effect only in the study group included female sex, erythrocyte count $\geq 9.0 \cdot 10^9/L$, CRP ≥ 40.0 mg/L, total protein < 65 g/L, and glomerular filtration rate < 47.0 mL/min/1.73 m². Anti-SARS-CoV-2 IgG titer ≥ 200.0 U/L represents a new risk factor for complications during hospital stay.

Discussion

According to the findings of the analysis, MI patients with a history of COVID-19 are younger and include more women. Our findings are consistent with the data from the RECORD-3 and GRACE registers [19, 20]. Patients in our study cohort had more risk factors than patients in the Moscow ACS register [21]. In both groups, about 30% of patients smoked and had diabetes mellitus, which is a higher than that contained in the register. In addition to a higher incidence of MI, our patients more frequently noted clinical signs of exertional angina.

Echocardiography showed significantly higher PASP in patients with a history of COVID-19 and signs of pericardial effusion compared to COVID-19-naïve patients. According to previous studies, the incidence of pericardial effusion may reach 15% among convalescents [22, 23]. Pericardial effusion is a consequence of pericarditis associated with COVID-19 [24] and increased PASP as a consequence of post-COVID-19 fibrosis [25–27]. These conditions may have a considerable effect on the course of NSTEMI both during hospital stay and in the long term [28]. Twelve-month outcomes were assessed in a study including 705 patients with primary MI and increased PASP [29]. Patients with higher PASP had a higher risk of death than others (RR=3.8; 95% CI 1.76–8.39; $p=0.001$).

Although patients in the study group were significantly less likely to have a high risk (GRACE score), the median score was 140 in both groups despite lower estimated values.

The course of NSTEMI is significantly associated not only with myocardial damage, but also with comorbidities. The risk analysis did not reveal any significant effect of COVID-19 on the mortality and severity of MI. Despite the younger age of the convalescents, both groups were comparable by all complications. In patients with a history of COVID-19, RR was higher for almost all the selected factors than in the group of naïve patients; in some cases, new risk factors were identified. Elevated levels of CRP and higher prevalence of female sex were recognized as significant risk factors only in the group of patients with a history of COVID-19. These characteristics are noted to be typical risk factors for the development of post-COVID-19 syndrome [30]. Our findings can be used to further stratify the risk for NSTEMI patients. Although increased IgG titer may be considered as an independent risk factor for complicated MI during hospital stay in future studies, age- and sex-adjusted multivariate analysis will be required.

This study is limited by the single-center design and relatively small size of the sample.

Conclusion

The analysis showed that post-COVID-19 patients with myocardial infarction have a higher relative risk of complications during hospital stay as compared to individuals without a history of COVID-19 due to the effects of the considered factors. Moreover, female sex, C-reactive protein levels ≥ 40.0 mg/L, total protein < 65 g/L, and IgG ≥ 200.0 U/L may be considered as risk factors for an adverse outcome of myocardial infarction during hospital stay in post-COVID-19 patients. The findings show that patients with a history of COVID-19 have a high risk of adverse cardiovascular events. Following up patients with non-ST-elevation myocardial infarction and remote history of COVID-19 is of particular research interest.

No conflict of interest is reported.

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