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IMPACT OF PAST COVID-19 INFECTION ON THE SEVERITY OF CORONARY ARTERY DISEASE

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Introduction: The gold standard for the treatment of coronary artery disease (CAD) in the case of acute obstruction of the coronary arteries is myocardial revascularization through stent placement, balloon angioplasty, or coronary artery bypass grafting. Advances in myocardial revascularization, the active implementation of drug-eluting stents, and effective antithrombotic therapy have led to a significant reduction in the incidence of coronary restenosis in recent years.

The clinical manifestations of CAD are directly related to the degree of narrowing of the coronary artery lumen due to atherosclerotic plaques. One possible method for eliminating such narrowing is percutaneous coronary intervention (PCI) with stent implantation. However, in 2–3% of cases, restenosis, or the re-narrowing of the previously treated coronary segment, occurs within a relatively short period. This usually results from excessive growth of the inner layer of the arterial wall, known as neointima, which leads to the recurrence of angina symptoms. Notably, this restenosis can be clinically more significant than the original atherosclerotic plaque for which stenting was performed.

This invention relates to medicine, specifically cardiology, and may be applied to predict the risk of restenosis in coronary stents. A known method for predicting restenosis is based on clinical or anatomical indicators (e.g., Hirshfeld J.W. Jr. et al., 2021). Inflammation plays a key role in the pathogenesis of CAD by provoking endothelial dysfunction and initiating local immune-inflammatory responses.

Thus, one in every fourteen individuals has contracted a coronavirus infection, which in turn leads to the worsening of chronic diseases, particularly circulatory system disorders. Although not immediately, the pathogenesis of the new infection was eventually described. By the end of 2020, the negative impact of COVID-19 on the vascular endothelium had already been proven. This involved widespread vascular damage throughout the body, manifesting in the development of sequential processes—diffuse immune-inflammatory and thrombo-inflammatory syndromes [3].

It was soon discovered that during the COVID-19 pandemic, in-hospital mortality among patients with ST-segment elevation myocardial infarction increased. Moreover, it was found that the combination of heart failure and coronary artery disease had a significantly adverse effect on mortality risk in hospitalized COVID-19 patients [7,9]. A new study confirmed that hospitalized patients with COVID-19 in the United States had a high prevalence of myocardial injury, which was associated with lower survival rates and worse outcomes.

A 2022 study conducted by researchers in Washington was particularly revealing. It turned out that the risk of developing coronary artery disease after recovering from COVID-19 increased by 72%, and the incidence of heart attacks was 63% higher. Notably, the risk was elevated even in patients under the age of 65 who had no prior chronic conditions [12].

The results of these studies indicate that the risk of developing cardiovascular diseases within one year among patients with favorable outcomes after COVID-19 infection is significant [9].

In addition, potentially fatal complications of CAD, such as acute coronary syndrome following COVID-19, come to the forefront and, according to some data, may present as a clinical manifestation of severe COVID-19. Clearly, the analysis of the relationship between past COVID-19 infection and the exacerbation of cardiovascular diseases may confirm the role of inflammation in the development of atherosclerosis and its complications, as well as aid in developing new tools for risk prediction.

Materials and Methods: In this cross-sectional, retrospective, observational study, the objective was to investigate the impact of COVID-19 on the course of coronary artery disease (CAD) and the risk of developing acute myocardial infarction (AMI).

Research objectives:

- To review existing literature on the role of inflammation in the course of CAD and chronic heart failure (CHF);

- To analyze medical records of CAD patients and select cases according to inclusion criteria;
- To conduct a comparative analysis of data between the study group and the control group based on inclusion criteria;
- To identify the relationship between a history of COVID-19 infection and the progression of CAD.

Medical records of inpatients treated from March 2020 to December 2022 at the Department of X-ray Endovascular Surgery, ASMI, Andijan, were examined. The total sample size consisted of 28 patients. Two groups of patients with CAD were compared:

- Group I: 16 patients with a history of COVID-19 infection
- Group II: 12 patients without a history of COVID-19 infection

The average age in Group I was 56.3 ± 7.87 years; in Group II – 55.8 ± 9.36 years.

Inclusion criteria:

- Patients with exertional angina of functional class (FC) II–III;
- Age over 35 years;
- Presence of chronic heart failure (CHF) of FC II–III according to the New York Heart Association (NYHA);
- Underwent coronary angiography (CAG);
- Favorable treatment outcomes.

Exclusion criteria:

- Pregnant women;
- Children;
- Patients with organic heart valve lesions;
- Patients with other heart diseases in the absence of CAD;
- Patients with terminal CHF (FC IV per NYHA);
- Lethal outcomes.

After applying the inclusion and exclusion criteria, qualitative and quantitative research indicators were established:

- Degree of coronary artery occlusion based on CAG data: right coronary artery (RCA), circumflex artery (CA), and left anterior descending artery (LAD);
- Six-minute walk test (6MWT) results;
- Lipid profile – levels of cholesterol (Chol), triglycerides (TG), low-density lipoproteins (LDL), and high-density lipoproteins (HDL);
- Risk of developing acute myocardial infarction (AMI);
- Left ventricular ejection fraction (LVEF) based on echocardiography (EchoCG).

Based on these indicators, a database was created for further statistical processing. For statistical analysis:

- Qualitative variables were expressed in absolute numbers and percentages;
- Quantitative variables were expressed as mean values ± standard error of the mean.
- To determine the dependence between quantitative variables, Student's t-test was used.
- To determine the relationship between qualitative variables, Pearson’s chi-square test was used. Differences were considered statistically significant at $p < 0.05$.

Results: A total of 18 patients participated in the study — 8 in Group I and 10 in Group II. The results of the quantitative variable analysis are presented in Table 1, where:

- **N** – number of patients
- **M** – arithmetic mean
- **me** – median
- **m** – standard error of the mean
- **t** – Student's t-test value
- **p** – level of statistical significance

The quantitative indicators included:

- Left ventricular ejection fraction (LVEF)
- Six-minute walk test (6MWT) results
- Lipid profile parameters

- Degree of coronary artery stenosis based on coronary angiography (CAG) findings

Discussion: According to numerous studies conducted in recent years, the development of coronary artery disease (CAD) is significantly influenced not only by dyslipidemia but also by systemic inflammatory responses, which contribute to the formation of atherosclerotic plaques. At the same time, COVID-19, with its pathogenic effects in the form of systemic immune-inflammatory and thrombo-inflammatory responses, plays a major role in worsening the course of cardiovascular diseases by damaging the endothelium of blood vessels throughout the body.

Table 2.

	I Group				II Group					
Category	**N**	**M**	**me**	**M**	**N**	**M**	**me**	**m**	**t**	**p**
LVEF (%)	51	53,3	57,5	1,52	55	59,96	61	1,68	2,94	0,004
6MWT	47	280,2	270	9,16	54	311,4	300	11,45	2,13	0,035
LAD	27	75,93	80	3,64	37	60,54	70	3,4	3,09	0,003
CA	15	65,33	70	4,72	19	62,89	60	5,17	0,35	0,729
RCA	24	73,13	75	3,37	23	64,35	70	4,55	1,55	0,128

The hypothesis we relied on was the expected worsening of the course of coronary artery disease (CAD) in patients who had previously contracted COVID-19, even in mild to moderate forms. Among the patients in Group I, 8 individuals (45%) reported an increase in clinical symptoms after recovering from COVID-19, which partially supported our hypothesis prior to reviewing the full results. However, in order to confirm the accuracy of this assumption, it was necessary to examine the outcomes based on the parameters established before the study began.

The primary objective of the study was to determine how a history of COVID-19 affects the progression of CAD. Therefore, the first indicator we examined was the degree of coronary artery occlusion. We selected three key arteries for analysis: the left anterior descending artery (LAD), the circumflex artery (CA), and the right coronary artery (RCA).

The most statistically significant difference was observed in the degree of LAD stenosis based on coronary angiography (CAG) data. For patients in Group I, the average degree of stenosis was $75.93 \pm 3.64\%$, whereas in Group II, it was $60.94 \pm 3.4\%$ ($p = 0.003$). The degree of stenosis in the other arteries also showed a trend toward being more pronounced in Group I. For example, the average degree of CA stenosis was $65.33 \pm 4.72\%$ in Group I and $64.35 \pm 4.55\%$ in Group II ($p = 0.001$). The degree of RCA stenosis was $73.13 \pm 3.37\%$ in Group I versus $64.35 \pm 4.55\%$ in Group II ($p = 0.128$).

Based on these findings, COVID-19 appears to be one of the determining factors in the severity of ischemic myocardial damage.

Table 2.

Lipid levels in the blood serum of clinically healthy individuals and CAD patients

Group	**Total Cholesterol (mmol/L)**	**Triglycerides (mmol/L)**	**LDL (mmol/L)**	**HDL (mmol/L)**	**VLDL (mmol/L)**	**Atherogenic Index (units)**
Group I	4.6 ± 0.1	1.5 ± 0.1	2.6 ± 0.2	1.4 ± 0.1	0.4 ± 0.1	2.8 ± 0.3
Group II	6.8 ± 0.3	2.6 ± 0.1	5.2 ± 0.3	0.9 ± 0.4	0.7 ± 0.3	5.2 ± 0.2
p-value	p < 0.001	p < 0.05	p < 0.001	p < 0.05	p < 0.05	p < 0.01

The clinical lipid profile indicators presented in Figure 1 tended to show more favorable values in Group I patients, with the exception of LDL levels. For Group I, LDL was **3.01 ± 0.22 mmol/L,** while

for Group II, it was **2.90 ± 0.14 mmol/L.** The average HDL level in Group I was **1.1 ± 0.04 mmol/L,** compared to **1.03 ± 1.0 mmol/L** in Group II. Since HDL is considered an anti-atherogenic lipid fraction, its higher level in the COVID-19 group indicates a tendency toward a better lipid profile.

Total cholesterol (TC) in Group I was **4.48 ± 0.24 mmol/L**, and in Group II **4.56 ± 0.16 mmol/L.** Triglycerides (TG) also followed the overall trend of being lower in Group I. The average TG value in Group I was **1.62 ± 0.14 mmol/L**, compared to **1.89 ± 0.14 mmol/L** in Group II.

Based on these findings, it can be noted that atherosclerotic plaque formation is influenced not only by dyslipidemia but also by the condition of the vascular endothelium, which changes during systemic inflammatory responses. Since COVID-19 is associated with immune-inflammatory and thrombo-inflammatory syndromes, endothelial damage is an expected consequence—even with a favorable outcome—which increases the risk of more severe CAD progression.
In addition, the clinical course of CAD in Group I patients, as reflected in the 6-minute walk test (6MWT), differed significantly from that in Group II. The results presented in Figure 3 clearly illustrate the difference between the two groups:

- Group I: **208.2 ± 9.1 m**
- Group II: **311.4 ± 1.45 m** (p = 0.001)

Thus, the original hypothesis—that a history of COVID-19 is a factor in the more severe course of CAD—was confirmed in this study.
Considering that all patients had chronic heart failure (CHF) of functional class II–III according to NYHA, the left ventricular ejection fraction (LVEF) was also analyzed based on echocardiography (EchoCG) data. The graphical representation is shown in Figure 1.

- Group I: **LVEF = 53.3 ± 1.52%**
- Group II: **LVEF = 59.96 ± 1.68%** (p = 0.001)

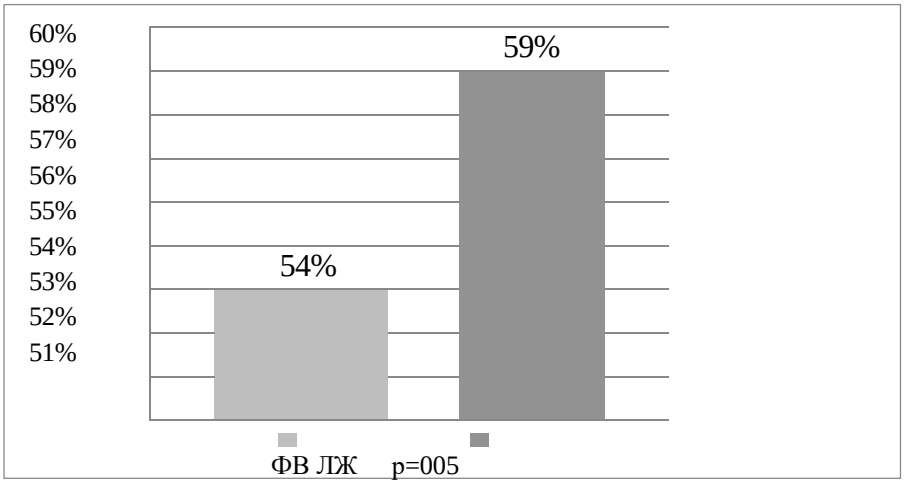

Figure 1 – Echocardiographic Data of Left Ventricular Ejection Fraction (LVEF) Based on Simpson's Method

Based on the data obtained, it can be concluded that a previous COVID-19 infection has a statistically significant effect on the contractile function of the myocardium, which worsens the general condition of patients and negatively affects their quality of life.
In addition, attention should be paid to the qualitative indicators presented in Figure 5. An important factor is that the primary incidence of coronary artery disease (CAD) in Group I was 31.3%, whereas in Group II, it was only 14.5%. CAD developed in 17.6% of patients in Group I compared to 10.9% in Group II. The odds ratio (OR) was 1.618, with a 95% confidence interval (CI) of 0.619–4.226, and the relative risk (RR) was 1.750, with a 95% CI of 0.575–5.322 (p = 0.321).
This suggests that a history of COVID-19 not only affects the progression of existing CAD but also increases the risk of developing the disease in patients who previously showed no coronary symptoms. There is also an increased risk of potentially fatal complications, including the acute myocardial infarction (AMI) considered in this study.

Based on the above, the original hypothesis proposed by the authors was confirmed by the results of this study. Unfortunately, since the study was conducted retrospectively at the Department of X-ray Endovascular Surgery, it was not possible to accurately assess the patients' condition over a follow-up period of two to four months after discharge.

In the future, this study could be expanded by involving outpatient specialists to monitor the dynamics of disease progression and patient condition after undergoing coronary angiography with stenting. **Conclusions:** The COVID-19 coronavirus infection affects the course of coronary artery disease (CAD) and chronic heart failure (CHF) through the development of a systemic inflammatory response, which leads to a worsening of the overall disease course. A hyperinflammatory, procoagulant state and endothelial dysfunction are key elements of the pathogenesis of COVID-19, increasing the risk of thrombosis. Undoubtedly, thrombotic and thromboembolic complications associated with COVID-19 are linked to more severe disease progression. Special attention should be given to the monitoring, prevention, and treatment of thrombotic and thromboembolic complications of COVID-19, regardless of its severity, and not only in hospitalized patients.

COVID-19 increases the tendency toward developing CAD (OR = 1.618, 95% CI). It also affects the **primary incidence** of CAD in patients who previously had no coronary symptoms. In Group I, primary CAD was diagnosed in 18 cases (31.3%), while in Group II only in 8 patients (14.5%).

COVID-19 infection significantly reduces myocardial contractile function. It also worsens CAD symptoms, as evidenced by the results of the six-minute walk test. Given the relatively better lipid profile in Group I patients, it can be concluded that **systemic inflammatory response plays a key role in the formation of atherosclerotic plaques.**

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