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Original article

Parameters of myocardial electrical instability in patients after myocardial infarction comorbid with a novel coronavirus infection (COVID-19)

Anastasia A. Tonkoglaz [1], Elena V. Averyanova [1], Yulia A. Barmenkova [1], Maryam A. Yangurazova [2], Marina V. Lukyanova [1], Valentin E. Oleynikov [1]

[1] Penza State University, Penza, Russia
[2] N.N. Burdenko Penza Regional Clinical Hospital, Penza, Russia

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Abstract: *Objective* — This article aims to assess parameters of myocardial electrical instability and arrhythmic events in patients after myocardial infarction (MI), with and without ST-segment elevation, comorbid/noncomorbid with a novel coronavirus infection (COVID-19) using a long-term electrocardiographic (ECG) monitoring.
Methods — The study included 64 subjects: 25 (39%) patients with MI comorbid with COVID-19 (MI+C group) and 39 (61%) patients with MI noncomorbid with a novel coronavirus infection (MI group). The mean age of patients was 54.3±6.8 years. A long-term ECG monitoring for 97.4 (95% CI 77.9-115.2) hours was performed with Astrocard®-Telemetry system (Meditek JSC, Russia), starting from the 4th day of MI. Rhythm and conduction disorders, along with ischemic episodes were recorded; an analysis of ventricular late potentials, heart rate turbulence, and QT dispersion was carried out.
Results — There were no differences in the frequency of delayed afterdepolarizations in MI and MI+C groups: 15-28% and 18-33% of patients, respectively. An analysis of turbulence parameters did not reveal statistically significant differences between the groups. Such arrhythmic events as frequent supraventricular extrasystole and life-threatening arrhythmias (ventricular extrasystole of grade 4A and higher sensu B. Lown and M. Wolf) were recorded significantly more often in the MI+C group than in the MI group: 48% vs. 20.5% (p=0.021) and 24% vs. 5.1% (p=0.026), respectively.
Conclusion — The novel coronavirus infection (COVID-19) exacerbates myocardial electrophysiological heterogeneity in the acute cardiovascular event and is associated with an increase in clinically significant arrhythmic events.

Keywords: myocardial infarction, long-term ECG monitoring, life-threatening arrhythmias, novel coronavirus infection (COVID-19).

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Correspondence to Valentin E. Oleynikov. Address: Department of Internal Medicine, Medical Institute, Penza State University, 40 Krasnaya St., Penza 440026, Russia. Phone: +79022033140. E-mail: v.oleynikof@gmail.com.

Introduction

The 2019 novel coronavirus disease (COVID-19) pandemic has become a major public health concern worldwide. A typical symptom of a new variant of COVID-19 is bilateral pneumonia with the development of acute respiratory distress syndrome. Despite the fact that most patients with COVID-19 have the symptoms of fever, dry cough and shortness of breath, approximately 10% of the patients have complications in the form of acute myocardial injury. It is characterized by an elevated troponin, a reduction in left ventricular systolic function, the development of cardiogenic shock, along with myocarditis and arrhythmia even in patients without cardiovascular disease [1].

Some hospitalized patients develop an acute COVID-19 cardiovascular syndrome with various clinical manifestations, such as an acute cardiac injury with cardiomyopathy, ventricular arrhythmias and hemodynamic instability in the absence of atherosclerotic lesion of coronary arteries. The cause of the latter is debated: presumably, it is related to myocarditis, microvascular damage, and systemic endothelial injury [2]. A combination of viral load with a severe coronary event significantly increases the risk of fatal arrhythmias leading to sudden cardiac death [3].

Myocardial injury associated with COVID-19 can occur as both type I and type II myocardial infarction (MI), since it is caused by microvascular ischemic damage to cardiomyocytes even in the absence of coronary atherothrombosis. MI comorbid with COVID-19 is characterized by the progression of heart failure symptoms and the emergence of life-threatening arrhythmias [4-7].

The objective of our study was to evaluate arrhythmic events and parameters of myocardial electrical instability in patients with MI comorbid with a new coronavirus infection (COVID-19) using long-term ECG monitoring.

Material and Methods

Research methods

The study included 64 patients aged 54.3±6.8 years with MI, with and without ST-segment elevation, hospitalized in N.N.

Burdenko Penza Regional Clinical Hospital (Penza, Russia). The study was conducted in compliance with 1964 Declaration of Helsinki and Good Clinical Practice guidelines. The study protocol was approved by the Ethics Committee at Penza State University (Penza, Russia). All patients included in the study signed a voluntary informed consent.

The study included patients who the following criteria: age of 35-70 years old; presence of MI; with or without ST-segment elevation, confirmed by a 12-lead resting electrocardiogram (ECG); an increase in high-sensitivity troponin I, and availability of echocardiographic data. The main exclusion criteria were as follows: recurrent myocardial infarction; non-sinus rhythm; NYHA classes III-IV of chronic heart failure; decompensated chronic diseases.

Long-term (48-120 hours) Nehb 3 lead ECG monitoring (LM ECG_{48-120}) was performed using the Astrocard®-Telemetry system (Meditek JSC, Russia), starting from the 4th day of MI. The mean duration of ECG monitoring was 97.4 (95% CI 77.9-115.2) hours. Based on the obtained records, we analyzed the ischemic profile, cardiac arrhythmias, heart conduction disorders, heart rate turbulence (HRT), ventricular late potentials (VLPs), and QT dispersion at 24, 48, 72, 96, and 120 hours.

The ischemic dynamics of the ST-segment was recorded during episodes of its displacement (≥10 mV) (elevation and depression) from the isoline at a distance of 80 ms from the J point with a duration of at least 1 min. When assessing cardiac arrhythmias and heart conduction disorders, frequent ventricular extrasystoles (VE) >30 per hour, ventricular arrhythmias of grade 4A and higher sensu B. Lown and M. Wolf, supraventricular extrasystoles (SVE) >20 per hour, along with tachyarrhythmias and heart conduction disorders, were taken into account.

HRT was assessed only in the presence of premature ventricular contractions (PVC) by the following parameters: turbulence onset (TO) and turbulence slope (TS). TO values less than 0% and TS values greater than 2.5 ms/RR were considered normal [8-9].

Analysis of VLPs was performed in automatic mode with the search for the reference QRS complex. Measurements included the duration of the filtered QRS interval (QRSf), the duration of high-frequency low-amplitude (HFLA) signals at the end of the complex, and the root-mean-square (RMS) amplitude of the last 40 ms of the QRS. VLPs were recorded when at least two of the following parameters deviated: QRSf>114 ms, HFLA>38 ms, RMS<20 μV [8].

The QT interval, i.e., the duration from the onset of the Q wave to the apex (QTa) and the end (QTe) of the T wave, was measured automatically. In addition, an analysis was made of the dispersion of the QT interval duration (dispQTa, dispQTe) and its standard deviation (sdQTa, sdQTe) [8].

All patients underwent sampling of biological material for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) ribonucleic acid (RNA) with a nasopharyngeal swab using the polymerase chain reaction (PCR) method. If necessary, an additional determination of immunoglobulins to SARS-CoV-2 was performed. SARS-CoV-2 RNA was detected in 25 (39%) patients with MI. To assess the features of electrophysiological changes in the myocardium and arrhythmic events in MI comorbid with COVID-19, two groups were formed: 25 patients with MI and COVID-19 (39%) were included in the MI+C group, and 39 patients with MI without COVID-19 (61%) were included in the MI group.

Comparative characteristics of examined patient groups and medical treatment

Main demographic and anamnestic parameters were compared between the groups (*Table* 1). Both groups were comparable in terms of most characteristics; however, the patients in the MI+C group were statistically significantly older (p=0.005). As for laboratory parameters, the glucose level was significantly higher in the MI+C group (p=0.001), which was most likely due to pronounced carbohydrate metabolism disorders against the background of viral load and glucocorticoid treatment. It should be noted that the level of troponin I was significantly higher in the MI group (p=0.017).

Treatment of patients in both groups was performed in accordance with the clinical guidelines [10, 11]. Significant differences were observed only in diuretic therapy, since diuretic medicines were more often prescribed to the patients in the MI+C group, compared with the M group, because of more pronounced congestion: 10 (40%) vs. 5 (12.8%), respectively (p=0.028).

Statistical data processing

Licensed Statistica 13.0 software by StatSoft, Inc. (USA) was used for statistical data processing.

Table 1. Comparative characteristics of the MI and MI+C patient groups

Index	*MI group (n=39)*	*MI+C group (n=25)*	*p*
Age, years	53.4 (39-60.1)	63 (54-69)	0.005
Male/female, n (%)	35 (90%)/4 (10%)	21 (88%)/4 (12%)	0.772
History of IHD, n (%)	10 (26%)	6 (20%)	0.828
AH, n (%)	30 (77%)	22 (88%)	0.883
Burden of hereditary diseases, n (%)	15 (38%)	4 (16%)	0.102
Smoking, n (%)	25 (64%)	15 (60%)	0.795
PCI, n (%)	26 (67%)	12 (48%)	0.222
Pain-PCI time frame, hours	8.1 (3.5-15.5)	8.7 (5.2-16.8)	0.411
Left ventricular anterior/posterior wall MI, n (%)	28 (72%)/11 (28%)	17 (68%)/8 (32%)	0.966
TCF, mmol/L	4.44 (4.01-4.8)	4.95 (4.07-5.8)	0.51
LDL, mmol/L	3.42 (2.8-4.04)	2.71 (1.8-3.6)	0.11
HDL, mmol/L	0.97 (0.8-1.14)	0.99 (0.9-1.1)	0.63
Blood glucose, mmol/L	5.79 (5.4-6.2)	7.17 (6.46-7.88)	0.001
High-sensitivity troponin I, pg/mL	46.714 (28.188-65.240)	19.347 (13.315-37.362)	0.017

IHD, ischemic heart disease; AH, arterial hypertension; PCI, percutaneous coronary intervention; MI, myocardial infarction; TCF, total cholesterol fraction; LDL, low-density lipoprotein; HDL, high-density lipoprotein. The table presents means and 95% CI.

Table 2. Dynamics of TO and TS values in MI and MI+C patient groups at different LM ECG$_{48-120}$ time intervals

Parameter	*Group*	*24th hour*	*48th hour*	*72nd hour*	*96th hour*	*120th hour*	*Mean*
TO, %	MI	-1.65 (-2.49; -0.81)	-1.83 (-2.86; -0.79)	-1.61 (-2.21; -1.01)	-1.98 (-2.77; -1.18)	-1.89 (-2.96; -1.83)	-1.06 (-1.51; -0.62)
	MI+C	-2.16 (-3.1; -0.4)	-0.82 (-2.49; 0.85)	-1.26 (-2.12; -0.39)	-2.08 (-4.63; 0.59)	-0.84 (-1.57; 0.89)	-0.47 (-0.77; -0.17)
	p_{24-120}	0.791	0.176	0.368	0.310	0.160	0.161
TS, ms/RR	MI	12.68 (9.64; 15.7)	12.46 (8.85; 16.08)	12.79 (9.76; 15.82)	12.62 (9.22; 16.01)	15.87 (10.52; 21.2)	7.36 (4.99; 9.71)
	MI+C	11.4 (4.97; 17.83)	11.22 (4.98; 17.45)	9.49 (7.38; 11.59)	8.57 (4.58; 11.55)	11.49 (4.62; 27.6)	5.21 (3.56; 7.87)
	p_{24-120}	0.597	0.599	0.085	0.233	0.473	0.413

TO, the onset of turbulence; TS, the turbulence slope. *Mean* refers to the overall average for 120 hours of monitoring. The table presents means and 95% CI. p_{24-120} – statistically significant differences between groups.

Table 3. Frequencies of ischemic and arrhythmic events in MI and MI+C patient groups

Parameter	*MI group (n=39)*	*MI+C group (n=25)*	*p*
Frequent single SVE, n (%)	8 (20.5%)	12 (48%)	0.021
SVT, n (%)	4 (10.25%)	5 (20%)	0.274
Frequent single VE, n (%)	16 (41%)	8 (32%)	0.462
Life-threatening arrhythmias (coupled VE, VT, VF, R/T-type VE), n (%)	2 (5.1%)	6 (24%)	0.026
Ischemic episodes, n (%)	8 (20.5%)	9 (36%)	0.172
SA and AV blocks, n (%)	8 (20.5%)	3 (12%)	0.379

SVE, supraventricular extrasystole; SVT, supraventricular tachycardia; VE, ventricular extrasystole; VT, ventricular tachycardia; VF, ventricular fibrillation; SA, sinoatrial block; AV, atrioventricular block.

Table 4. Dynamics of dispQTe and sdQTe values in MI and MI+C patient groups at different LM ECG$_{48-120}$ time intervals

Parameter, ms	*Group*	*24th hour*	*48th hour*	*72nd hour*	*96th hour*	*120th hour*	*Mean*
dispQTe	MI	40.8 (28.2-53.4)	40.7 (29.9-51.5)	37.7 (25.5-49.9)	42.8 (11.1-64)	48 (11.4-75)	40.88 (28.4-52.5)
	MI+C	23.5 (18.5-32.5)	24.2 (16.9-31.6)	26.4 (18.8-34)	37.8 (17.65-58)	34.1 (11.9-56.2)	26.3 (18.7-33.9)
	p_{24-120}	0.01*	0.004*	0.037*	0.306	0.611	0.014*
sdQTe	MI	11.9 (8.4-15.4)	11.4 (7.8-15.1)	12.4 (8.6-16.3)	18 (7.9-28.1)	16.2 (5.2-27.2)	12.4 (8.6-16.2)
	MI+C	19.4 (13-25.7)	19.2 (13.9-24.5)	16.8 (11.3-22.3)	20.7 (6.1-35.2)	23.3 (10.9-47.4)	19.1 (12.9-25.3)
	p_{24-120}	0.012*	0.005*	0.048*	0.294	0.617	0.021*

dispQTe, QT interval duration dispersion to the end of the T-wave; sdQTe, standard deviation of QT interval duration dispersion to the end of the T-wave. *Mean* refers to the overall average for 120 hours of monitoring. p_{24-120} – statistically significant differences between groups. The table presents means and 95% CI.

All quantitative data were presented with confidence intervals (CI) of the mean and Student's t-distribution. For qualitative comparisons, we employed Pearson's chi-squared test ($\chi 2$) for independent samples. McNemar's test was used for pairwise comparisons. Independent samples with normally distributed characteristics were compared using Student's t-test. Non-normally distributed traits were compared between the groups using Mann-Whitney U test for independent samples. Statistical significance was assumed at $p<0.05$ [12].

Results

We did not find any differences between the groups in the number of patients with recorded afterdepolarization. Depending on the ECG monitoring duration, VLPs were recorded in 15-28% patients in the MI group, and in 18-33% patients in the MI+C group. According to the 72-hour LM ECG$_{48-120}$ data, higher QRSf values were observed in the latter group: 103 (97; 105) ms vs. 95 (94; 98) ms (p=0.009) (*Figure* 1).

The dynamics of the examined HRT parameters indicates that pathological HRT was less frequently detected in the MI group due to a disorder in the rapid response to VE, viz., the TO parameter, LM ECG$_{48}$, in 2 (5.1%) vs. 4 (16%) patients (p=0.027) by the end of the monitoring. There were no significant differences in the absolute values of TO and TS at different times of LM ECG$_{48-120}$ (*Table* 2).

The above differences in the state of myocardium electrophysiological processes in the MI and MI+C groups were revealed during the registration of arrhythmic events (*Table* 3). Frequent SVEs were more commonly recorded (p=0.021) in the MI+C group of patients; however, there were no differences in the registration of frequent VEs in both groups. Life-threatening ventricular arrhythmias of the grade 4A and higher sensu B. Lown and M. Wolf were significantly more frequently recorded in the MI+C group: 24% vs. 5.1% (p=0.026). At the same time, there were no significant differences between the groups in the registration of ischemic events and conduction disorders in the form of episodes of sinoatrial (SA) and atrioventricular (AV) blocks (p=0.172 and p=0.379, respectively).

When analyzing the QT interval, we obtained significant differences between the groups in terms of dispQTe and sdQTe parameters (*Table* 4).

On the second day of monitoring, as well as when comparing the mean values for the entire period, dispQTe parameter was significantly higher in the MI+C group (*Figure* 2), and its means were 40.88 (95% CI 13.1-33) ms vs. 26.3 (95% CI 17.9-29.2) ms in the MI group (p=0.014).

Lower values of sdQTe parameter were detected in MI group at similar time intervals, as well as when comparing the mean values for 120-hour monitoring (p_{24}=0.012, p_{48}=0.005, p_{72}=0.048, p_{mean}=0.021, respectively). Higher values of dispQTe and sdQTe parameters in the MI+C group implied the pronounced destabilization processes of myocardial electrical activity in patients.

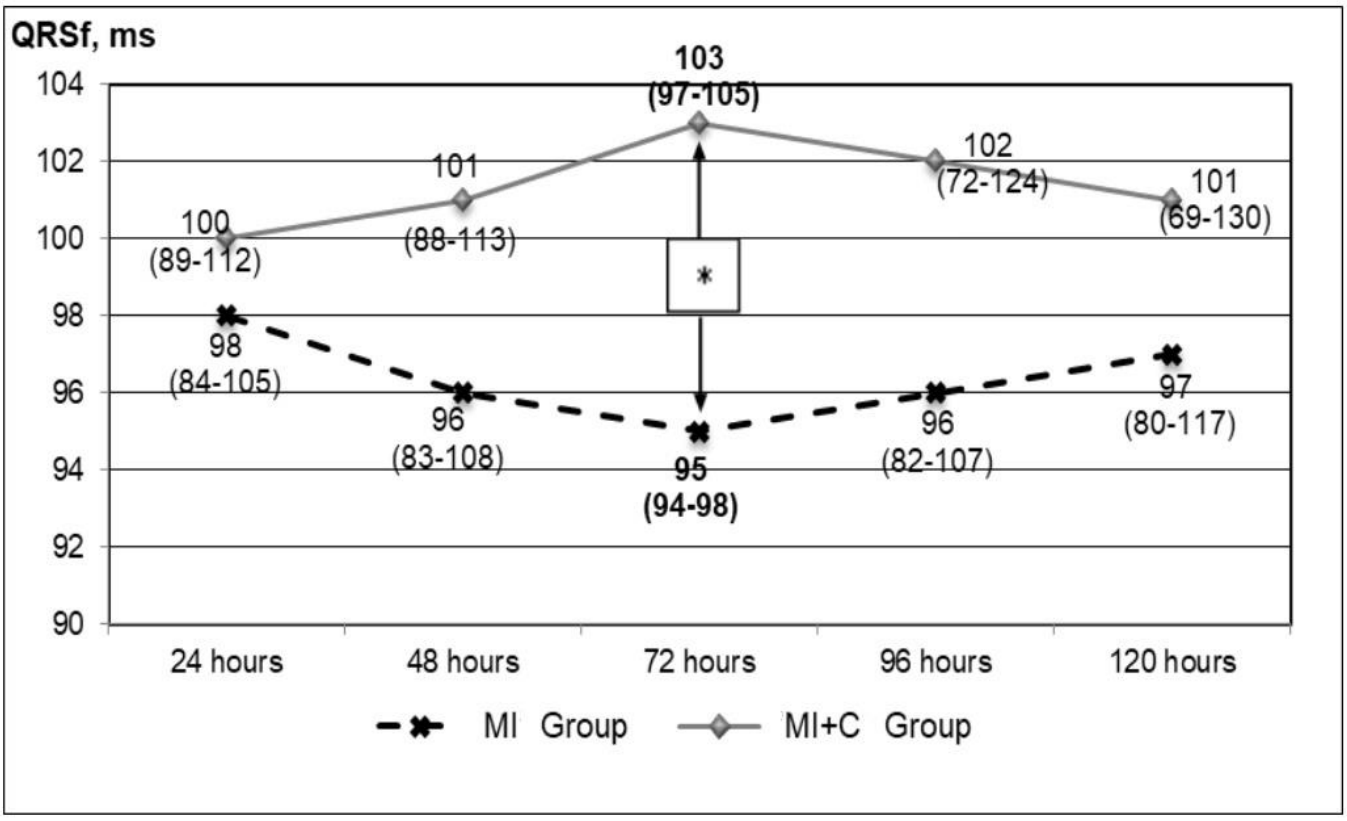

Figure 1. Dynamics of QRSf parameter values in MI and MI+C patient groups at different LM ECG$_{48\text{-}120}$ time intervals. QRSf is filtered QRS interval duration.

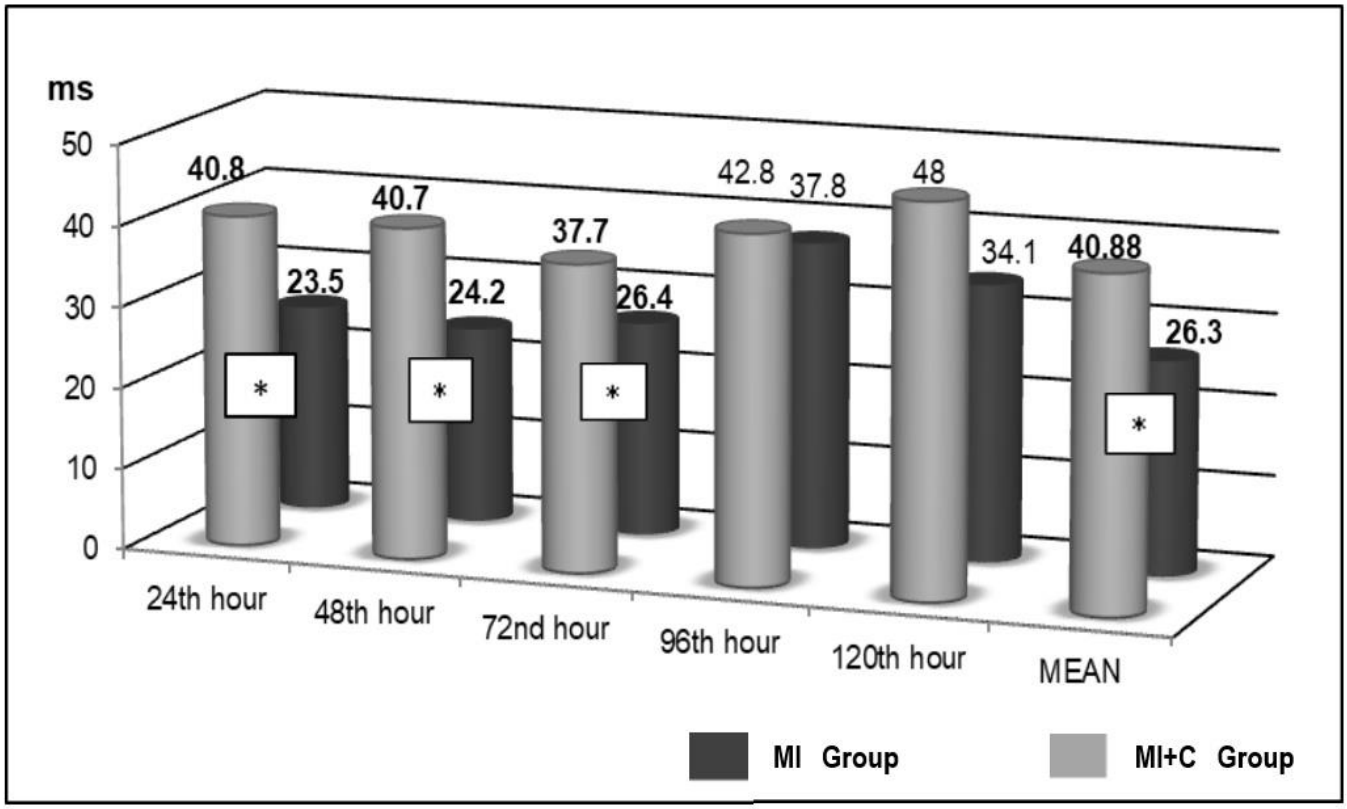

Figure 2. Dynamics of dispQTe parameter values in MI and MI+C patient groups at different LM ECG$_{48\text{-}120}$ time intervals. dispQTe is QT interval duration dispersion to the end of the T-wave; MEAN refers to the overall average for 120 hours of monitoring; * p<0.05 are statistically significant differences between groups.

Discussion

A number of authors note a severe course and a high death rate in patients with COVID-19 comorbid to cardiovascular pathology [13-16]. According to Seecheran R. et al., exacerbation of chronic cardiovascular diseases is associated with an increase in myocardial metabolism against the background of a reduction in coronary blood flow in conditions of a viral infection. This imbalance, combined with direct myocardial injury and inflammatory response, significantly increases the risk of acute coronary syndrome and arrhythmias [17]. The publications present data mainly on changes in routine resting ECG and arrhythmic events recorded in the acute stage of COVID-19 disease. However, we did not find information on the assessment of the severity of electrophysiological changes in the myocardium in acute coronary accident in combination with a new coronavirus infection. In this study, we compared the nature of arrhythmias and the parameters of myocardial electrical instability in patients with MI alone vs. MI exacerbated by a novel coronavirus infection (COVID-19), based on long-term ECG monitoring data.

Many articles have been published on the diversity of arrhythmic events in COVID-19. According to Angeli F. et al., ECG changes, such as atrial fibrillation, tachycardia-bradycardia syndrome, ST-segment and T wave changes, and right bundle branch block were recorded in 26% of patients with COVID-19 within 30 days from the onset of the disease [18]. Wang D. et al. found that such arrhythmic cardiac events as sinus tachycardia, atrial fibrillation and flutter, supraventricular tachycardia, sinus bradycardia, AV block, idioventricular rhythm, and sick sinus syndrome were recorded in 17-44% of patients with COVID-19 [19].

The development of arrhythmic episodes in patients directly depends on the severity of COVID-19. According to some authors, pathological supraventricular activity, life-threatening arrhythmias (ventricular extrasystole of grade 4A and higher sensu B. Lown and M. Wolf), SA and AV conduction disorders are most often detected in severe electrical disorders of the heart after MI, with and without ST-segment elevation, comorbid or noncomorbid with COVID-19 [5, 14, 15]. According to our data, frequent single SVE and life-threatening high-grade ventricular arrhythmias were more often established in patients with MI in combination with COVID-19 than in patients without coronavirus infection, 48% vs. 20.5% (p=0.021) and 24% vs. 5.1%, respectively (p=0.026). Apparently, the combination of acute damage to cardiomyocytes against the background of MI and viral activity leads to more pronounced disorders of electrophysiological processes in the myocardium, resulting in frequently detected clinically significant arrhythmias.

Kochi A.N. et al. suggested that arrhythmias in COVID-19 may occur primarily due to various factors: hypoxia caused by direct viral damage to the lung tissue and myocardium; abnormal immune response or ischemia; increased myocardial afterload caused by pulmonary hypertension; and electrolyte disorders [20]. As stated by Siripanthong B. et al., cell-mediated cytotoxicity is among potential arrhythmogenic mechanisms in COVID-19, when CD8+ T lymphocytes migrate to cardiomyocytes, causing inflammatory changes in the myocardium. Proinflammatory cytokines promote activation of T lymphocytes via cytokine storm, which leads to the release of more cytokines causing myocardial damage [21]. Tse G. et al. concluded that both structural and electrophysiological remodeling in viral myocarditis led to an increase in the duration of repolarization and the occurrence of abnormal conduction. Increased repolarization could cause triggered activity leading to impulse circulation by the re-entry mechanism in combination with inhomogeneous conduction [22]. Long B. et al. thought that arrhythmic complications in patients with COVID-19 were provoked by a cytokine storm, progressive myocardial hypoxia, electrolyte imbalance, coronary artery spasm, and microthrombosis. In his opinion, they were most often manifested by sinus tachycardia and bradycardia, atrial fibrillation, persistent supraventricular tachycardia, along with ventricular fibrillation and tachycardia [23].

The results of the comparison between the groups established a more pronounced electrophysiological instability in the combination of MI with COVID-19, due to the cumulative effect of coronary artery atherothrombosis, direct exposure to the virus, and systemic inflammatory response. Pathological HRT revealed in patients with viral load exceeded that in patients without COVID-19 by 10.9% (p=0.027). High values of the dispQTe and sdQTe parameters in the group of patients with COVID-19 demonstrated the severity of the electrical heterogeneity of the myocardium, due to the inhomogeneity of the repolarization phase in different areas of the myocardium. Viral activity of SARS-CoV-2 triggers an entire cascade of pathological processes: from endothelial dysfunction of small coronary vessels to severe damage to cardiomyocytes by immune complexes, followed by the formation of zones of myocardial fibrosis, which leads to even more significant changes in parameters associated with arrhythmic myocardial readiness [24, 25]. Bhatla A. et al. showed an increased risk of arrhythmic events when cardiovascular diseases were associated with COVID-19. In the course of the monitoring of 700 patients, we observed 9 cardiac arrests, 25 cases of primary paroxysms of atrial fibrillation, 9 clinically significant bradyarrhythmias, and 10 episodes of supraventricular tachycardias [26].

The results of comparing the groups of patients with MI vs. MI+C yielded a more pronounced destabilization of electrophysiological characteristics of the myocardium, since there were higher values of QRSf and QT dispersion, and pronounced disorders in the autonomic regulation of the heart rhythm (frequent registration of pathological HRT and severe arrhythmias).

To date, among available publications, we did not find any of them devoted to examining arrhythmic events during long-term ECG monitoring in patients with MI in combination with a novel coronavirus infection. The presented results correlated with ECG_{24} data on the characteristics of arrhythmias obtained by other researchers. Our data reflect the contribution of COVID-19 to the cardiotoxic effect, which exacerbates the severity of heterogeneous electrophysiological processes in the myocardium during infarction. Undoubtedly, MI is the main cause of disorders in the electrical balance stability of cardiomyocytes. COVID-19 aggravates the imbalance of myocardial electrical activity in combination with electrolyte shifts, formation of a large number of circulating immune complexes, a cytokine storm, and damage to the vascular endothelium. These changes lead to arrhythmic events, the prognostic value of which has yet to be explored.

Our study limitations were as follows: non-enrollment of patients with severe acute respiratory distress syndrome associated with COVID-19 (because of failure to undergo LM ECG_{48-120} in the intensive care unit) and patients with comorbidities at the decompensation stage against the background of COVID-19

progression; unwillingness of patients (especially those living in remote regions) to participate due to prolonged (over four days) wearing of ECG recording device on their bodies. It should be noted that dynamic monitoring of all patients who underwent LM ECG_{48-120} was performed at the Research Center of the Department of Internal Medicine, Medical Institute, Penza State University and at N.N. Burdenko Penza Regional Clinical Hospital (Penza, Russia). A repeat LM ECG48-120 is expected to be performed 12 months after MI.

Conclusion

The 2019 novel coronavirus infection (COVID-19) aggravates electrophysiological heterogeneity in patients in the acute period of myocardial infarction and is associated with an increase in clinically significant arrhythmic events.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with 1964 Declaration of Helsinki and its later amendments, or comparable ethical standards. This article does not contain any studies, involving animals, performed by any of the authors.

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Authors:
Anastasia A. Tonkoglaz – PhD student, Department of Internal Medicine, Medical Institute, Penza State University, Penza, Russia. https://orcid.org/0000-0002-5647-9837.
Elena V. Averyanova – PhD, Associate Professor, Department of Internal Medicine, Medical Institute, Penza State University, Penza, Russia. https://orcid.org/0000-0001-9925-2096.
Yulia A. Barmenkova – PhD, Instructor, Department of Internal Medicine, Medical Institute, Penza State University, Penza, Russia. https://orcid.org/0000-0001-5111-6247.
Maryam A. Yangurazova – Cardiologist, Department of Emergency Cardiology, N.N. Burdenko Penza Regional Clinical Hospital, Penza, Russia. https://orcid.org/0000-0002-2764-4071.
Marina V. Lukyanova – PhD, Associate Professor, Department of Internal Medicine, Medical Institute, Penza State University, Penza, Russia. https://orcid.org/0000-0002-2080-2639.
Valentin E. Oleynikov – DSc, Professor, Chair of the Department of Internal Medicine, Medical Institute, Penza State University, Penza, Russia. https://orcid.org/0000-0002-7463-9259.