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Cardiometabolic and echocardiographic characteristics of the cardiovascular phenotype of post COVID-19 condition

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ABSTRACT

Aim. To study the cardiometabolic and echocardiographic characteristics of COVID-19 convalescents, including patients with the cardiovascular phenotype of)post COVID-19 condition (PCC).

Materials and methods. The sample included 270 COVID-19 convalescents (62 without PCC and 208 with PCC). In the subgroup with PCC, 16 convalescents had a cardiovascular phenotype. The study took into account the data of anamnesis, anthropometry, several clinical and biochemical blood parameters, and instrumental diagnostic data (electrocardiography and echocardiography).

Results. In COVID-19 convalescents with PCC (n = 208), fasting plasma glucose levels were 1.10 times higher ($p < 0.001$), abdominal obesity (AO) was 5.52 times more common ($p < 0.001$), arterial hypertension (AH) was 4.96 times more common ($p < 0.001$), diastolic dysfunction grade I was 5.55 times more common ($p = 0.002$), and left ventricular hypertrophy was 7 times more common ($p = 0.005$). The indices of maximum blood flow velocity and pressure gradient in the pulmonary artery in convalescents with PCC were 1.08-fold ($p = 0.020$) and 1.14-fold ($p = 0.043$) lower, respectively. In COVID-19 convalescents with PCC (n = 16) and a cardiovascular phenotype, total cholesterol (TC) was 1.11 times higher ($p = 0.039$), low-density lipoprotein cholesterol (LDL-C) was 1.21 times higher ($p = 0.004$), high-density lipoprotein cholesterol (HDL-C) was 1.22 times lower ($p = 0.040$), non-high-density lipoprotein cholesterol (non-HDL-C) was 1.24 times higher ($p = 0.005$) compared with patients without a cardiovascular phenotype. An increase in TC, LDL-C, and non-HDL-C and a decrease in HDL-C are associated with the cardiovascular phenotype of PCC regardless of gender, age, body mass index, and lipid-lowering therapy.

Conclusion. According to the study, echocardiographic changes and cardiometabolic risk factors, such as AO, AH, and carbohydrate metabolism disorders, were more common in patients with PCC. The cardiovascular phenotype of PCC is associated with an increase in TC, LDL-C, non-HDL-C, and a decrease in HDL-C.

Keywords: COVID-19 convalescents, post COVID-19 condition, cardiovascular phenotype

Conflict of interests. The authors declare the absence of obvious or potential conflicts of interest related to the publication of this article.

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Compliance with the principles of ethics. The study was approved by the Ethics Committee of the Research Institute of Internal and Preventive Medicine, branch of ICG SB RAS, Novosibirsk (Minutes No. 71 dated November 10, 2020).

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Кардиометаболические и эхокардиографические характеристики сердечно-сосудистого фенотипа постковидного синдрома

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РЕЗЮМЕ

Цель. Изучить кардиометаболические и эхокардиографические характеристики реконвалесцентов COVID-19, в том числе пациентов с сердечно-сосудистым фенотипом постковидного синдрома (ПКС).

Материалы и методы. Выборка 270 реконвалесцентов COVID-19: 62 без ПКС и 208 с ПКС. В подгруппе с ПКС 16 реконвалесцентов имели сердечно-сосудистый фенотип. В ходе исследования учитывались данные анамнеза, антропометрии, ряда клинических, биохимических показателей крови, данных инструментальной диагностики (электрокардиографии и эхокардиографии).

Результаты. У реконвалесцентов COVID-19 с ПКС (n = 208) уровень глюкозы плазмы крови натощак был выше в 1,10 раза (p < 0,001), чаще встречались: абдоминальное ожирение (АО) в 5,52 раза (p < 0,001), артериальная гипертензия (АГ) в 4,96 раза (p < 0,001), диастолическая дисфункция I степени в 5,55 раза (p = 0,002) и гипертрофия левого желудочка в 7 раз (p = 0,005), показатели максимальной скорости кровотока и градиента давления в легочной артерии у реконвалесцентов с ПКС были ниже в 1,08 (p = 0,020) и 1,14 раза (p = 0,043) соответственно. У реконвалесцентов COVID-19 с ПКС (n = 16), имеющих сердечно-сосудистый фенотип, общий холестерин (ОХС) выше в 1,11 раза (p = 0,039), холестерин липопротеинов низкой плотности (ХС-ЛНП) выше в 1,21 раза (p = 0,004), холестерин липопротеинов высокой плотности (ХС-ЛВП) ниже в 1,22 раза (p = 0,040), холестерин липопротеинов невысокой плотности (ХС-неЛВП) выше в 1,24 раза (p = 0,005) по сравнению с пациентами без сердечно-сосудистого фенотипа. Увеличение ОХС, ХС-ЛНП, ХС-неЛВП и уменьшение ХС-ЛВП ассоциированы с сердечно-сосудистым фенотипом ПКС независимо от пола, возраста, индекса массы тела и гиполипидемической терапии.

Заключение. По данным исследования у пациентов с ПКС чаще встречались эхокардиографические изменения и кардиометаболические факторы риска, такие как АО, АГ и нарушения углеводного обмена. Сердечно-сосудистый фенотип ПКС ассоциирован с увеличением ОХС, ХС-ЛНП, ХС-неЛВП и уменьшением ХС-ЛВП.

Ключевые слова: реконвалесценты COVID-19, постковидный синдром, сердечно-сосудистый фенотип

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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Соответствие принципам этики. Все участники подписали информированное согласие на участие в исследовании и обработку персональных данных. Исследование одобрено этическим комитетом НИИТПМ – филиал ИЦиГ СО РАН (протокол № 71 от 10.11.2020).

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INTRODUCTION

According to the World Health Organization (WHO), 10–20% of new coronavirus infection (COVID-19) convalescents have consequences that manifest as new diseases, as well as decompensation of existing chronic non-communicable diseases, known as post COVID-19 condition (PCC).

In the acute course of COVID-19, the respiratory system is mainly affected through target cells having angiotensin-converting enzyme 2 (ACE2) receptors. However, ACE2 receptors are found in the cytoplasmic membrane of not only alveolar cells but also enterocytes of the small intestine, smooth muscle cells of the arteries, endothelial cells of the arteries and veins, and cells of tissues of the brain, esophagus, adrenal glands, bladder, etc. [1, 2].

Clinical cardiovascular manifestations are an important aspect of PCC [3]. Their structuring will greatly contribute to the search for a comprehensive, targeted approach to COVID-19 convalescents in order to diagnose and prevent complications early.

Aim. To study the cardiometabolic and echocardiographic characteristics of COVID-19 convalescents, including patients with the cardiovascular phenotype of post COVID-19 condition (PCC).

MATERIALS AND METHODS

A one-stage observational study of COVID-19 convalescents was performed at the Research Institute of Internal and Preventive Medicine, branch of the Institute of Cytology and Genetics, Siberian Branch of the Russian Academy of Sciences.

The study included 270 COVID-19 convalescents whose average age was 53.2 ±13.2 years. All the subjects were divided into subgroups depending on the presence of PCC: 62 without PCC (58.1% were men) and 208 with PCC (45.2% were men). The group of COVID-19 convalescents with PCC was formed taking into account the WHO criteria [4]. In the group of people with PCC, a cardiovascular phenotype was identified (16 people, 56.3% were men), represented by new onsets of cardiovascular diseases (CVD), as well as decompensation of pre-existing diseases of the cardiovascular system before COVID-19 infection. The structure of the cardiovascular phenotype is shown in Figure 1.

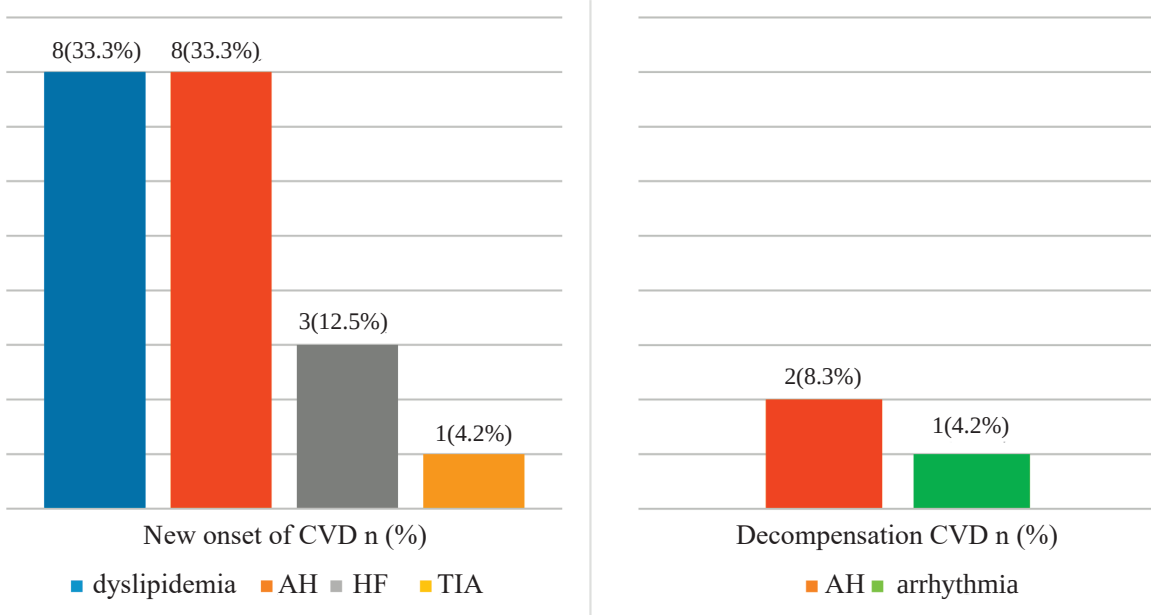

Fig. 1. The structure of the cardiovascular phenotype

Arterial hypertension (AH) was recorded at systolic blood pressure (SBP) ≥ 140 mm Hg and/or diastolic blood pressure (DBP) ≥ 90 mm Hg. Body mass index (BMI) was calculated using the formula: BMI (kg/m^2) = body weight (kg)/height (m^2) [5]. Abdominal obesity (AO) was recorded according to measurements of waist circumference > 94 cm (men) and > 80 cm (women).

Echocardiography (Echo) was performed in all patients using a Toshiba Aplio 500 color ultrasound scanner (Japan). The left ventricular myocardial mass (LVMM) was determined using the Penn Convention

formula [6]: LVMM (g) = 1.04 × ([LVEDD + IVSD + PWD]^3–[LVEDD]^3) – 13.6. Body surface area (BSA) according to the Du Bois and Du Bois formula: BSA (m^2) = 0.007184 × weight (kg)$^{0.425}$ × height (cm)$^{0.725}$ [7]. The calculation of the relative wall thickness (RWT) of the left ventricle (LV) was carried out according to the formula: RWT (units) = PWD × 2 / LVEDD [8]. The LV myocardial mass index (LVMI) was calculated using the formula: LVMI (g/m^2) = LVMM / BSA. The criteria for left ventricular hypertrophy (LVH) were the following parameters: (LVH (LVMM, g/height, m), ASE formula for overweight and obese patients: for men > 50 g/m$^{2.7}$, for women > 47 g/m$^{2.7}$, and for patients with normal body weight, indexing was carried out using BSA > 115 g/m^2 (men) and > 95 g/m^2 (women) [9].

Left ventricular diastolic dysfunction (LVDD) was assessed using Echo criteria: grade 1 LVDD was established if the ratio of LV filling pressure in the early diastole and atrial systole was (E/A) < 0.8, and the LV filling pressure in the early diastole (E) was < 50 cm/s; grade 2 LVDD was established if two of the following three criteria were positive: 1) the ratio of the early LV diastolic filling pressure and the left ventricular posterior wall in the early diastole (E/e' > 14); 2) left atrial volume indexed for body surface area (> 34 ml/m^2); 3) maximum tricuspid regurgitation rate > 2.8 m/s. Grade 3 LVDD was established when the E/A ratio was > 2 [10].

The erythrocyte sedimentation rate (ESR) was determined by the indirect Panchenkov's method (a space-dependent neutron kinetics model utilizing an integral representation of the Boltzmann equation). Biochemical parameters of blood (aspartate aminotransferase (AST), alanine aminotransferase (ALT), uric acid, fibrinogen, prothrombin index (PTI), activated partial thromboplastin time (APTT), C-reactive protein (CRP), fasting blood glucose, creatinine, total cholesterol (TC), high lipoprotein cholesterol densities (HDL-C), triglycerides (TG) were determined using Thermo Fisher Scientific kits (Finland) on a Konelab Prime 30i biochemical analyzer (Thermo Fisher Scientific, Finland). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula: LDL-C (in mmol/l) = TC – HDL-C – TG/2.2. Non-high-density lipoprotein cholesterol (non-HDL-C) was calculated using the formula: TC – HDL-C [11].

Statistical processing of the obtained results was performed using the SPSS software package (version 13.0). The results are presented as the median of the lower and upper quartiles *Me* [25;75]. We used the Mann–Whitney test to compare groups and univariate logistic regression analysis to evaluate the odds ratio. Spearman's rank correlation coefficient was used to assess correlations. The groups were compared regarding frequency using conjugation tables and the Pearson's chi-squared test. When testing statistical hypotheses, the critical level of significance was at $p < 0.05$.

RESULTS

According to demographic data, the age of convalescents with PCC was 1.18 times higher ($p = 0.003$) than that of convalescents without PCC (Fig. 2). No statistically significant differences were revealed regarding gender ($p = 0.075$).

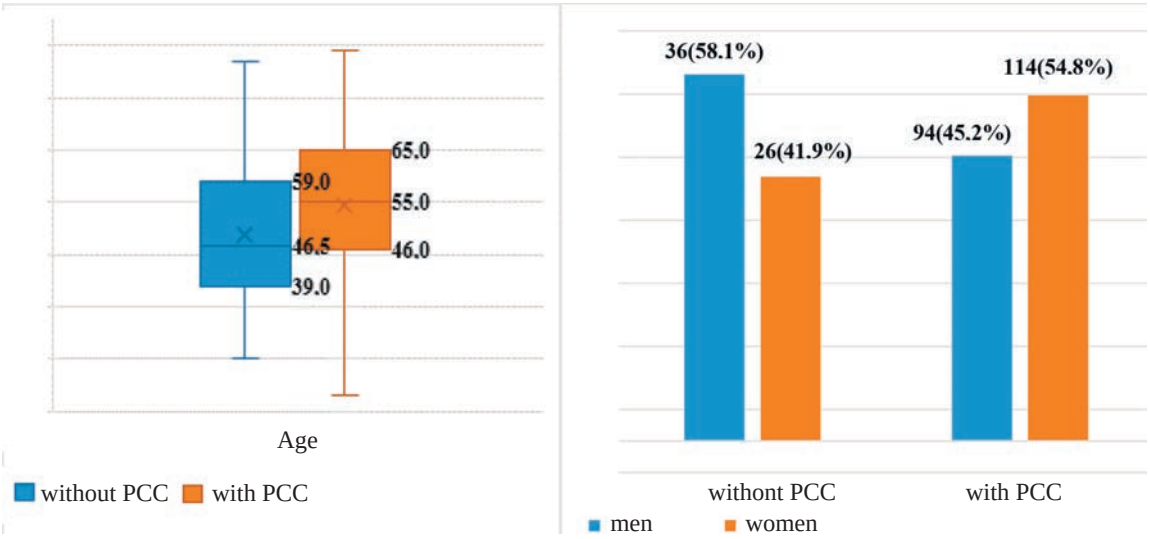

Fig. 2. Demographic data of COVID-19 convalescents with and without PCC

The analysis of anamnestic data revealed that at the outpatient and inpatient stages of treatment for the acute period of COVID-19, patients received various groups of medications. The analysis included records of only those patients for whom it was possible to clarify the data on medication intake. Thus, anticoagulant therapy was received by 6 (9.7% of 19) convalescents without PCC and 33 (15.9% of 72) convalescents with PCC (p = 0.264), oxygen therapy — by 2 (3.2% of 29) convalescents without PCC and 14 (6.7% of 84) with PCC (p = 0.193), glucocorticoid therapy — by 7 (11.3% of 25) convalescents without PCC and 30 (14.4% of 75) with PCC (p = 0.282), antibiotics – by 17 (27.4% of 26) convalescents without PCC and 62 (29.8% of 83) with PCC (p = 0.353), antiviral therapy — by 13 (21.0% of 25) convalescents without PCC and 35 (16.8% of 78) with PCC (p = 0.534).

Statistical processing of laboratory data revealed that in the group of people with PCC, fasting blood glucose and fibrinogen levels were 1.10 times higher (p < 0.001) and 1.13 times higher (p = 0.007), respectively (Fig. 3).

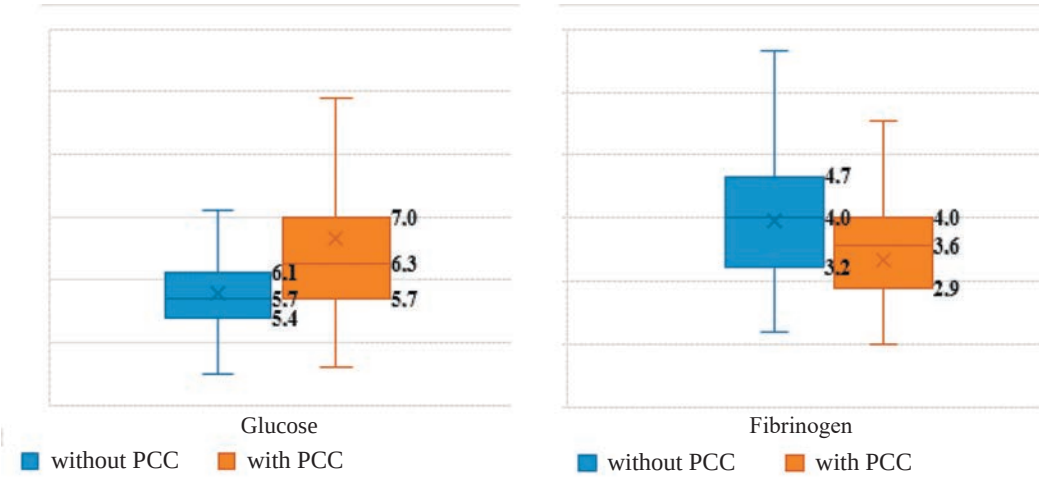

Fig. 3. Fasting blood glucose and fibrinogen levels in COVID-19 convalescents with and without PCC

According to Echo data, LVRWT was 1.07 times larger in patients with PCC, grade 1 LVDD was 5.55 times more common, LVH was 7 times more common compared with people without PCC. In individuals with PCC, AO and AH were also more common — by 5.52 and 4.96 times, respectively, than in individuals without PCC. When comparing the maximum blood flow velocity and the pressure gradient in the pulmonary artery in individuals with PCC, the parameters were 1.08 and 1.14 times lower, respectively (Table 1).

Table 1

Characterization of Morphofunctional Parameters of Individuals with and without PCC

Parameters	COVID-19 convalescents without PCC, n = 62	Convalescents of COVID-19 with PCC, n = 208	p
QT, s	0.36 [0.33;0.37]	0.35 [0.34;0.37]	0.565
QRS, s	0.08 [0.08;0.09]	0.08 [0.08;0.09]	0.975
Ao, mm	32.65 [30.00;35.00]	32.00[29.73;35.00]	0.469
LA diameter, mm	37.10 [34.00;40.00]	38.00 [35.00;42.00]	0.074
LA length, mm	49.00 [45.00;52.00]	50.00 [46.00;55.00]	0.069
RV, mm	27.50 [21.25;32.75]	26.00 [21.00;31.00]	0.537
IVSD, mm	10.15 [8.90;11.00]	10.50 [9.50;12.00]	0.075
LV, mm	50.10 [48.00;52.88]	51.00 [47.00;54.00]	0.347
LVESD, mm	31.00 [29.00;33.00]	31.90 [29.00;34.00]	0.429
PWd, mm	8.80 [8.00;9.48]	9.00 [8.30;10.00]	0.060
RA diameter, mm	35.00 [32.00;37.75]	35.00 [32.00;38.00]	0.679
RA length, mm	46.00 [43.00;52.00]	48.00 [44.00;51.00]	0.146
Mitral valve, m/s, Vmax	0.69 [0.52;0.82]	0.67 [0.55;0.75]	0.463
Mitral valve, gradient, mm Hg	1.90 [1.10;2.70]	1.80 [1.20;2.20]	0.434
Aortic valve, m/s, Vmax	1.23 [1.15;1.43]	1.30 [1.19;1.45]	0.264
Aortic valve, gradient, mm Hg	6.05 [5.35;8.20]	6.80 [5.70;8.40]	0.284
Pulmonary artery, m/s, Vmax	0.90 [0.80;1.04]	0.83 [0.73;0.96]	0.020
Pulmonary artery, gradient, mm Hg	3.20 [2.43;4.35]	2.80 [2.10;3.63]	0.043

End of table 1

Parameters	COVID-19 convalescents without PCC, *n* = 62	Convalescents of COVID-19 with PCC, *n* = 208	*p*
EF by the Simpson method, %	67.00 [63.25;69.00]	67.00 [63.00;70.00]	0.564
Mean pulmonary artery pressure, mm Hg	19.50 [14.00;22.75]	20.00 [15.00;24.00]	0.408
LVDD (grade 1), abs. (%)	22 (35.50)	122 (58.70)	0.002
LVMM, g	176.00 [143.50;194.75]	182.50 [153.00;224.00]	0.100
LVMI, g/m^2	88.23 [79.34;99.67]	92.15 [82.00;110.12]	0.052
LVRWT, U	1.51 [1.28;1.89]	1.62 [1.38;1.93]	0.049
LVH, abs. (%)	11 (17.70)	77 (37.00)	0.005

Note. AO – the diameter of the aortic root, LA – left atrium, RV – size of the right ventricle in a four–chamber section, IVSD – interventricular septum thickness during diastole, LVEDD – left ventricular end–diastolic diameter, LVESD – left ventricular end–systolic diameter, PWd – left ventricular posterior wall thickness during diastole, RA – right atrium, LVDD – left ventricular diastolic dysfunction, LVMM – left ventricular myocardial mass, LVMI – left ventricular myocardial mass index, LVRWT – relative wall thickness of the left ventricle, LVH – left ventricular hypertrophy

When analyzing demographic and anamnestic parameters in the group of COVID-19 convalescents with a cardiovascular phenotype, it was revealed that those with this phenotype were younger than other convalescents with PCC. Before the debut of COVID-19, blood pressure figures reached target values in people with decompensated cardiovascular phenotype, and optimal therapy was selected. The comparative characteristics of drug treatment are given in Table 2.

When analyzing cardiometabolic risk factors in the group with the cardiovascular phenotype, TC was 1.11 times higher, LDL-C was 1.21 times higher, HDL-C was 1.22 times lower, and non-HDL-C was 1.24 times higher than in other convalescents (Table 3).

Table 2

Comparative Characteristics of Drug Therapy for COVID-19 Convalescents with a Cardiovascular Phenotype and Other COVID-19 Convalescents, abs. (%)

Parameter	COVID-19 convalescents with a cardiovascular phenotype, *n* = 16	Other COVID-19 convalescents with PCC, *n* = 192	*p*
Angiotensin-converting enzyme inhibitors	1 (6.3%)	51 (26.6%)	0.070
Angiotensin II receptor blockers	0 (0.0%)	49 (25.5%)	0.020
Beta-blockers	2 (12.5%)	59 (30.7%)	0.121
Calcium channel blockers	0 (0.0%)	34 (17.7%)	0.065
Diuretics	0 (0.0%)	54 (28.1%)	0.013
Centrally acting antihypertensive agents	1 (6.3%)	8 (4.2%)	0.698

Table 3

Cardiometabolic Risk Factors in COVID-19 Convalescents with a Cardiovascular Phenotype and Other COVID-19 Convalescents, *Me* [25;75]

Parameters	COVID-19 convalescents with a cardiovascular phenotype, *n* = 16	Other COVID-19 convalescents with PCC, *n* = 192	*p*
Age, years	46.00 [41.25;55.00]	56.00 [46.00;65.00]	0.016
Men, abs (%)	9 (56.30)	85 (44.30)	0.355
BMI	28.03 [24.73;32.78]	29.03 [25.07;33.47]	0.605
WC, cm	97.00 [88.50;106.00]	100.00 [88.00;110.00]	0.390
Smoking, years, abs. (%)	6 (37.50)	66 (34.40)	0.801
PA < 3 h/week, abs. (%)	12 (75.00)	134 (69.80)	0.621
SBP, mm Hg	129.75 [118.13;140.00]	126.75 [115.63;135.00]	0.692
DBP, mm Hg	84.75 [73.13;90.00]	80.00 [75.00;87.38]	0.231
Heart rate, bpm	67.50 [57.25;71.50]	67.00 [62.00;75.00]	0.295
FBG, mmol/l	5.95 [5.50;6.45]	6.30 [5.70;7.00]	0.108
TC, mmol/l	5.86 [5.29;6.72]	5.27 [4.51;6.10]	0.039
LDL-C, mmol/l	4.05 [3.62;4.71]	3.29 [2.50;3.99]	0.004
HDL-C, mmol/l	1.06 [0.82;1.40]	1.29 [1.05;1.60]	0.040
Triglycerides, mmol/l	1.40 [1.09;2.42]	1.40 [0.96;2.09]	0.515
non-HDL-C, mmol/l	4.90 [4.20;5.70]	3.91 [3.12;4.75]	0.005
Fibrinogen, g/l	4.00 [2.88;4.61]	3.55 [2.88;4.00]	0.198

Note. Here and in Table 4: BMI – body mass index, WC – waist circumference, PA < 3 – physical activity less than 3 hours per week, SBP – systolic blood pressure, DBP – diastolic blood pressure, HR – heart rate, TC – total cholesterol, LDL-C –low-density lipoprotein cholesterol, HDL-C – high-density lipoprotein cholesterol, non-HDL-C – high-density non-lipoprotein cholesterol.

According to instrumental research methods, no differences were revealed between the cardiovascular phenotype and other individuals with PCC.

Logistic regression analysis identified the correlations of the cardiovascular phenotype with the level of TCH, LDL-C, HDL-C, and non-HDL-C (Table 4). The odds of having a cardiovascular phenotype increased twofold along with an increase in atherogenic lipid fractions and by 12.5 times along with a decrease in HDL-C.

Table 4

Logistic Regression Analysis of Parameters Associated with the Cardiovascular Phenotype of Post COVID-19 Condition

Parameter	Model 1 $Exp(B)_1$	*p*	Model 2 $Exp(B)_2$	*p*	Model 3 $Exp(B)_3$	*p*	Model 4 $Exp(B)_4$	*p*
Age, in year 1	0.951 (0.905–0.999)	0.045	0.947 (0.901–0.996)	0.034	0.961 (0.920–1.003)	0.071	0.944 (0.897–0.993)	0.026
Sex, M/W	0.677 (0.228–2.004)	0.481	0.728 (0.242–2.196)	0.574	1.008 (0.322–3.156)	0.989	0.803 (0.263–2.453)	0.700
BMI, per 1 kg/m^2	1.008 (0.918–1.106)	0.872	1.000 (0.911–1.098)	0.994	0.945 (0.849–1.052)	0.300	0.992 (0.901–1.092)	0.869
Hypolipidemic therapy, yes/no	0.329 (0.051–2.128)	0.243	0.346 (0.057–2.119)	0.251	0.247 (0.035–1.735)	0.160	0.316 (0.050–1.987)	0.219
TC, per 1 mmol/l	1.594 (1.023–2.483)	0.039	-	-	-	-	-	-
LDL-C, per 1 mmol/l	-	-	2.033 (1.213–3.407)	0.007	-	-	-	-
HDL-C, per 1 mmol/l	-	-	-	-	0.080 (0.012–0.524)	0.008	-	-
non-HDL-C, per 1 mmol/l	-	-	-	-	-	-	1.917 (1.218–3.017)	0.005

DISCUSSION

A number of studies reflect the cytotoxic effect of SARS-CoV-2 on cardiomyocytes, which is confirmed by increased markers of cardiovascular damage [12–14]. However, it remains controversial whether the long-term cardiovascular manifestations of COVID-19 are caused by the direct action of the virus on the heart tissue or are secondary due to the formation of systemic inflammation and hypoxia [15].

In our study, individuals with PCC were statistically more likely to have abdominal obesity. AO is known to affect immune function and endocrine metabolism. For instance, L. Shang et al. discussed obesity as a risk factor for the development of PCC [16]. H.W. Kim et al. described how, through the secretion of chemokines, perivascular adipose tissue leads to endothelial dysfunction, vasoconstriction, and proliferation of smooth muscle cells, which potentially contributes to the development of cardiovascular diseases [17].

Concomitant chronic diseases are well known to be risk factors in the development of severe forms of COVID-19. A number of studies confirm the association of arterial hypertension in patients with severe and fatal COVID-19 [18, 19]. However, high blood pressure is associated with old age, as well as other cardiovascular risk factors that affect the overall prognosis [3]

M.R. Dweck et al. studied a sample consisting of patients from 69 countries. As a result, changes in Echo parameters were detected in 55% of patients with acute COVID-19 [20]. Long-term studies of the heart were reflected in the work of I. Yaroslavskaya et al., which revealed a decrease in systolic and diastolic function of the left ventricle due to the presence of chronic heart failure and AH [21]. S.G. Kanorskiy et al. found a relationship between PCC and DD of the right ventricle, as well as a significant increase in the maximum and average pressure gradients on the aortic valve and the average pressure gradient on the mitral valve [22]. In our study, convalescents with PCC had grade 1 diastolic dysfunction of the left ventricle and Echo signs of left ventricular hypertrophy, which may be associated with heart damage according to the modern concepts of the mechanisms of damage to the cardiovascular system in COVID-19 [23].

When comparing the groups of convalescents with the cardiovascular phenotype and other phenotypes, we revealed a difference in the lipid profile. HDL-C has an antioxidant and immunomodulatory function. It also binds and neutralizes pathogenic lipids. However, during the inflammatory process, HDL-C is modified, which is accompanied by oxidative processes and the accumulation of oxidized forms of lipids. As a result,

accumulated LDL-C and TG lead to endothelial dysfunction and the development of cardiovascular complications [24].

Several studies have demonstrated an increase in blood lipids, in particular TG and LDL-C, in the acute and post-COVID period, regardless of the COVID-19 severity [25, 26]. In our study, high levels of TC, non-HDL-C, and LDL-C were observed in convalescents with a cardiovascular phenotype and a decrease in HDL-C.

CONCLUSION

Patients with PCC had changes in echocardiography more often, as well as cardiometabolic risk factors such as AO, AH, and impaired carbohydrate metabolism compared with convalescents without PCC. The cardiovascular phenotype of PCC is more associated with changes in the lipid profile, namely, an increase in total cholesterol, LDL-C, non-HDL-C, and a decrease in HDL-C.

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