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FEATURES OF THE DIURNAL PROFILE OF BLOOD PRESSURE AND REMODELING OF THE HEART AND VESSELS IN PEOPLE WITH COVID-19

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ABSTRACT

Introduction. The relevance of the study is due to the significant prevalence of COVID-19 and its cardiovascular complications worldwide.

Aim. The aim of the work is to assess the features of the processes of heart and vascular remodeling in different types of daily blood pressure profile after a coronavirus infection.

Materials and methods. 100 people were examined in outpatient settings 1 month after COVID-19 (62 men and 38 women), aged 63.14 ± 2.24 years. All examined participants underwent Doppler echocardiography, ultrasound examination of the brachiocephalic arteries, and daily blood pressure monitoring. Methods of primary statistical analysis were used to evaluate the results of the study.

Results. Left ventricular hypertrophy and IMC were diagnosed in 75.0 and 52.0% of patients; in 31.0% – non-dipper daily BP profile, in 14.0% – night-peaker, in 46.0% – dipper, in 9.0% – over-dipper. With LV hypertrophy and its normal geometry, the non-dipper daily BP profile was determined in 33.3 and 24.0%, an increase in IMC – in 45.2 and 6.4% of the examined. In 31.2% of people with eccentric hypertrophy, in 30.7% with concentric hypertrophy, in 29.4% with concentric remodeling, a non-dipper BP profile was detected. With LV hypertrophy and without it, the night-peaker daily BP profile was determined in 14.6 and 12.0% of cases. In 15.6% of those examined after COVID-19 with eccentric hypertrophy, in 11.5% with concentric hypertrophy, in 11.7% with concentric remodeling, a night-peaker BP profile was detected.

Conclusions. The results obtained indicate a significant impact of COVID-19 on the processes of cardiovascular remodeling, indicate the need to include daily BP monitoring, Doppler echocardiography, and vascular ultrasound in the algorithm for diagnosing and treating people with COVID-19.

Keywords: Arterial hypertension, Blood pressure, COVID-19, Arterial stiffness, Hypertension-mediated organ damage

INTRODUCTION

If we consider SARS-CoV-2 infection as a systemic lesion of the human body, it becomes obvious that the virus affects not only the respiratory, but also the cardiovascular, nervous, excretory and lymphatic systems, which is manifested in significant damage to the entire body [1, 2].

Cicco S. et al., 2023, showed that the SARS-CoV-2 virus uses the leading element of the renin-angiotensin-aldosterone system (RAAS) – angiotensin-converting enzyme (ACE) to penetrate cells [3]. ACE2 is expressed on endothelial cells of the vessels of the heart, kidneys and lungs. The main element of the RAAS, angiotensin II, affects the cardiovascular system by activating AT1 receptors, which in turn stimulates the processes of vasoconstriction, inflammation and fibrosis. Under

normal conditions, ACE2 plays a counter-regulatory role in the RAAS, forming anti-inflammatory, antioxidant and antifibrinolytic effects [4]. The SARS-CoV-2 virus reduces the expression of ACE2 on the cell surface, which significantly reduces the protective capabilities of the endothelium [5], leading to activation of the RAAS with endothelial damage [6, 7].

Mancia G. et al., in 2020 and 2025 [8, 9], showed that endothelial dysfunction and pathological remodeling of the heart and vessels are a common phenomenon in people with a previous coronavirus infection. Puntmann V. et al., 2022, indicated that symptoms of cardiovascular disease occur in 57% of patients with previous COVID-19, even if they were absent before infection. Researchers noted an increase in the right ventricular ejection fraction in patients [10].

Chlabicz M. et al., 2023, found that coronavirus infection led to changes in cardiac remodeling [11]. Podrug M. et al., 2023, indicated changes in remodeling and arterial stiffness [12]. The increase in vascular stiffness due to SARS-CoV-2 infection persists for a significant period of time, i.e., vascular aging processes are enhanced with loss of compliance and changes in the characteristics of the vascular walls [13].

Angeli F. et al., 2024, and Marozzi M. et al., 2025, demonstrated in their studies that in coronavirus disease, a persistent and significant increase in blood pressure is observed, with changes in its daily profile and the need for combined antihypertensive therapy [14, 15]. However, the mechanisms of the relationship between blood pressure and the processes of cardiovascular system remodeling after COVID-19 remain insufficiently studied to this day.

AIM

The aim of the study is to assess the features of heart and vascular remodeling processes in patients with different types of daily blood pressure profiles after coronavirus infection.

MATERIALS AND METHODS

100 people with a history of COVID-19 (62 men and 38 women), aged 63.14 ± 2.24 years, were examined in outpatient settings. The inclusion criteria in the study were: patients with arterial hypertension aged 45-65 years who had laboratory-confirmed COVID-19 and consulted a family doctor for dispensary supervision and agreed to participate in the study. Exclusion criteria: no confirmed COVID-19 disease, age >65 years, or disagreement with participation in the study. The study protocol was drawn up in accordance with the Declaration of Helsinki and agreed with the Ethics Committee. Examination and treatment of patients were carried out in accordance with the "Protocol for providing medical care for the treatment of coronavirus disease (COVID-19)" [16].

Structural and functional changes in the myocardium and vessels, as well as features of the diurnal blood pressure profile, were assessed using Doppler echocardiography, ultrasound examination of the brachiocephalic arteries, and daily blood pressure monitoring.

To evaluate the results of the study, primary statistical methods were used [17]. The author used ChatGPT (OpenAI, San Francisco, CA, USA) for language editing of the English text. The author reviewed and verified all AI-generated content to ensure accuracy and integrity.

RESULTS

Confirming the data of our previous work [18], left ventricular (LV) hypertrophy was found in 75

examined subjects, and among them, concentric hypertrophy and remodeling were found in 34,6 and 22,7%, eccentric hypertrophy in 42,7% of individuals (Fig. 1 a and 1 b).

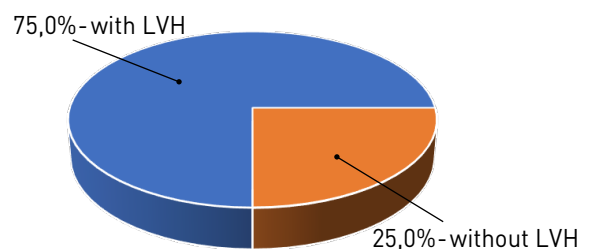


Fig. 1 a. Prevalence of hypertrophy left ventricle in individuals with Covid 19 survivor

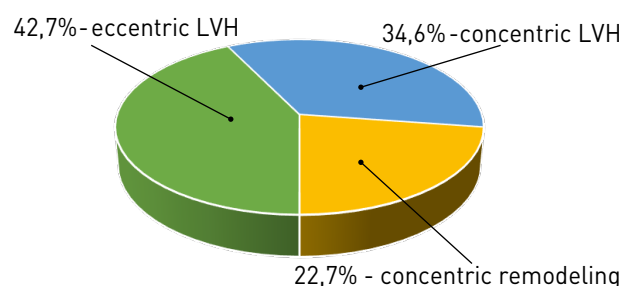


Fig. 1 b. Prevalence of different types left ventricular hypertrophy in individuals with Covid 19 survivor

Based on the normal circadian rhythm of blood pressure (BP), the types of daily BP curves were distinguished: dippers, non-dippers, night-peakers, over-dippers. In the examined individuals with and without LV hypertrophy, the non-dipper daily BP profile was found in 33,3 and 24,0%, night-peaker – in 14,6 and 12,0%, dipper – in 42,7 and 56,0%, over-dipper – in 9,4 and 8,0% of the examined individuals (Fig. 2).

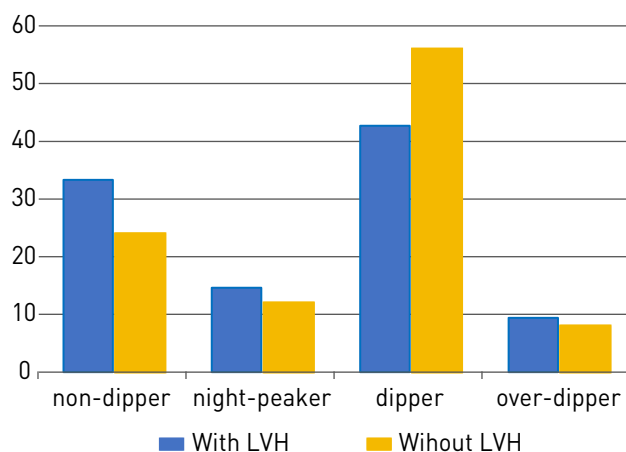


Fig. 2. Prevalence of different types of diurnal blood pressure profile

Diurnal blood pressure patterns were observed in patients with COVID-19 with different types of LV remodeling (Fig. 3).

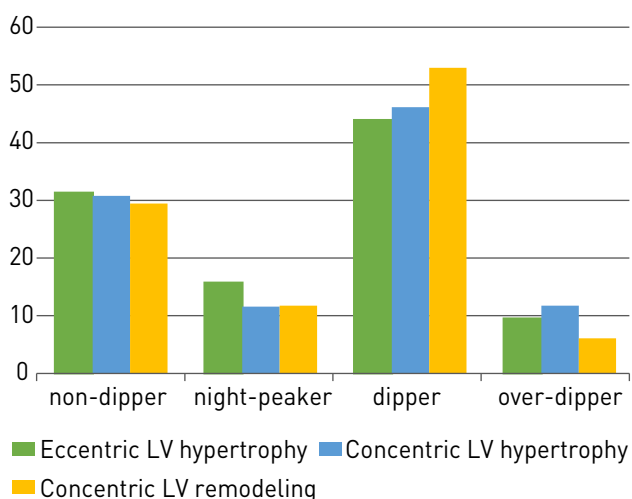


Fig. 3. Prevalence of blood pressure profiles in patients with COVID-19 and different types of LV remodelling

In individuals with concentric hypertrophy and remodeling, as well as with eccentric hypertrophy, the non-dipper diurnal BP profile occurred in 30.7%, 29.4%, and 31.2%; the night-peaker profile – in 11.5%, 11.7%, and 15.6%; the dipper profile – in 46.1%, 52.9%, and 43.8%; and the over-dipper profile – in 11.7%, 6.0%, and 9.4%, respectively.

Significant differences were observed when comparing daytime, night-time and diurnal blood pressure values in individuals with different blood pressure profiles (Table 1).

In individuals with a non-dipper BP profile, compared to the dipper subjects, daytime systolic blood pressure (SBP) significantly increased by 7.3% ($p < 0.05$) and diastolic blood pressure hypertension time index (DBP HI) by 109.6% ($p < 0.05$); nighttime systolic blood pressure (SBP HI) by 15.5% ($p < 0.001$), diastolic blood pressure (DBP) by 16.6% ($p < 0.001$), systolic blood pressure hypertension time index (SBP HI) by 180.5% ($p < 0.001$), DBP HI more than 2 times; daily minimum systolic blood pressure (minSBP) decreased by 13.9% ($p < 0.01$), mean systolic blood pressure (mean SBP) decreased by 8.4% ($p < 0.01$), maximum systolic blood pressure (max SBP) decreased by 10.3% ($p < 0.05$), minimum mean blood pressure (MinMBP) decreased by 18.2% ($p < 0.001$), mean blood pressure (MBP) decreased by 15.4% ($p < 0.01$), maximum mean blood pressure (MaxMBP) decreased by 6.2% ($p < 0.05$), systolic hypertension area index (SBP HI) decreased by 66.6% ($p < 0.001$) and diastolic hypertension area index (DBP HI) decreased by 50.9% ($p < 0.001$) (Table 1).

Table 1. Features of diurnal blood pressure profiles of individuals with Covid-19

Daily blood pressure monitoring indicators		Diurnal blood pressure profile		
		dipper	non-dipper	night-peaker
Day time	SBP	124,7±2,5	133,8±4,2*	134,8±4,7*
	SBP HI	12,8±3,6	24,8±6,7	40,4±10,5**
	DBP HI	13,6±3,7	28,5±6,7*	37,8±11,2***
Night time	SBP	108,4±1,7	125,2±3,8***	141,2±5,8***
	DBP	67,6±1,3	78,8±2,2***	85,9±3,5***
	SBP HI	11,8±3,4	33,1±7,5***	47,1±11,3***
	DBP HI	11,1±3,5	35,3±7,4***	50,2±12,1***
	VSBP	8,8±0,6	10,4±0,7	13,8±1,7**
	VDBP	7,3±0,6	8,4±0,6	10,1±1,5*
Daily time	min SBP	87,4±1,8	99,6±3,4**	97,5±3,8*
	mean SBP	121,0±1,9	131,2±3,5**	134,3±3,6**
	max SBP	156,4±3,5	172,5±5,7*	174,1±6,7*
	MinMBP	62,8±1,5	74,2±3,1***	75,6±1,8***
	MBP	88,5±1,7	102,1±3,2**	96,5±2,7*
	MaxMBP	117,4±2,6	124,7±3,6*	123,2±5,2
	MinPBP	20,6±0,7	22,7±1,5	26,0±2,1*
	mPBP	41,3±1,4	44,5±2,1	52,1±2,9***
	MaxPBP	67,4±3,7	78,2±4,5	86,1±7,7*
	SBP HI	1,2±0,1	2,0±0,4***	1,6±1,1***
	DBP HI	33,0±3,0	16,2±1,9***	18,9±3,4***

Note: * – $p < 0.05$, ** – $p < 0.01$, *** – $p < 0.001$

Compared with the dipper subjects, in individuals with the night-peaker BP profile, daytime SBP increased statistically significantly by 8.1% ($p<0.05$), hypertension time index SBP HI and DBP HI by 215.6% ($p<0.01$) and 177.9% ($p<0.001$); night-time SBP by 30.3% ($p<0.001$), DBP by 27.1% ($p<0.001$), SBP HI and DBP HI by both almost 3 times, variabilities of SBP (VSBP) and DBP (VDBP) by 56.8% ($p<0.01$) and 38.4% ($p<0.05$); daily minimum mean blood pressure (MinMBP) – by 11.6% ($p<0.01$), daily mean blood pressure (MBP) – by 10.9% ($p<0.01$), maximal mean blood pressure (MaxMBP) – by 20.4% ($p<0.001$), SBP – by 9.1% ($p<0.05$), max SBP – by 11.3% ($p<0.05$), minimum pulse BP (MinPBP) – by 2.6% ($p<0.05$), mean pulse BP (mPBP) – by 26.1% ($p<0.001$), maximum pulse BP (MaxPBP) – by 27.7% ($p<0.05$), SBP HI – by 33.3% ($p<0.001$) and DBP HI decreased – by 42.7% ($p<0.001$) (Table 1).

Significant differences were observed when comparing cardiac remodeling processes in individuals with COVID-19 and different BP profiles (Table 2).

In patients with a non-dipper BP profile, compared with individuals with a dipper, left atrial size increased by 6.3%, left atrial index – by 6.3%, left

atrial area – by 3.3%, left atrial area index – by 5.1%, LV end-diastolic size – by 4.3%, LV end-diastolic size index – by 4.2%, LV end-systolic size – by 14.3%, LV end-diastolic volume – by 8.4%, LV end-diastolic volume index – by 9.1%, LV end-systolic volume – by 13.5%, LV posterior wall myocardial thickness – by 11.1%, relative thickness of posterior wall of LV – by 25.0%, LV myocardial mass – by 10.1%, myocardial mass index – by 13.1%, right atrial area – by 5.6%, right atrial area index – by 1.6%, LV end-diastolic volume index – by 1.2%, and the LV ejection fraction decreased – by 2.6%, but all these features were not statistically significant (Table 2).

Compared with the dipper subjects, in subjects with the night-peaker BP profile, the left atrial area index significantly increased by 19.9% ($p<0.05$) and not statistically significantly: aortic root diameter – by 3.2%, left atrial size – by 3.1%, left atrial area – by 9.2%, left atrial index – by 6.3%, LV end-diastolic size – by 2.1%, LV end-diastolic size index – by 8.3%, LV end-systolic size – by 3.6%, LV end-diastolic volume – by 1.4%, LV end-diastolic volume index – by 10.1%, LV ejection fraction – by 2.9%, LV posterior wall myocardial thickness – by 22.2%, relative thickness of posterior wall of LV – by

Table 2. Features of the structural and functional state of the heart in people with Covid-19 depending on the daily blood pressure profile

Indicators	Diurnal blood pressure profile		
	dipper	non-dipper	night-peaker
Aortic root diameter	3,1±0,1	3,1±0,1	3,2±0,1
LA atrial size	3,2±0,1	3,4±0,1	3,3±0,1
LA atrial index	1,6±0,1	1,7±0,1	1,7±0,1
LA atrial area	15,3±0,8	15,8±0,6	16,7±1,3
LA atrial area index	7,8±0,3	8,2±0,2	9,3±0,9*
LV end-diastolic size	4,7±0,07	4,9±0,1	4,8±0,3
LV end-diastolic size index	2,4±0,1	2,5±0,1	2,6±0,1
LV end-systolic size	2,8±0,1	3,2±0,2	2,9±0,1
LV end-diastolic volume	99,7±3,6	108,1±4,6	101,1±7,6
LV end-diastolic volume index	50,6±1,8	55,2±1,8	55,7±4,7
LV end-systolic volume	33,3±1,7	37,8±2,5	32,8±3,2
LV ejection fraction	69,0±0,8	68,1±1,3	71,0±6,3
LV posterior wall myocardial thickness	0,9±0,1	1,0±0,2	1,1±0,1
Relative thickness of posterior wall of LV	0,4±0,1	0,5±0,1	0,5±0,1
Interventricular septal thickness	1,2±0,1	1,2±0,1	1,3±0,1
LV myocardial mass	213,5±12,6	235,0±14,1	259,7±28,3
Myocardial mass index	106,2±5,5	120,1±4,3	128,6±10,2
Right atrial area	12,5±0,7	13,2±0,6	12,7±0,5
Right atrial area index	6,6±0,3	7,0±0,2	7,2±0,2

Note. * – $p<0.05$

25.0%, interventricular septal thickness – by 8.3%, LV myocardial mass – by 21.6%, myocardial mass index – by 20.9%, right atrial area – by 1.6%, right atrial area index – by 9.1%, and LV end-systolic volume decreased by 1.5% (Table 2).

Features of diurnal blood pressure profiles in individuals who have had COVID-19 are associated with both different types of left ventricular remodeling and thickening of the intima-media complex (IMC) of extracranial vessels [18].

Intima-media complex thickening (>0.9 mm) was observed in 52.0% of individuals with COVID-19, while it was not present in 48.0%; among individuals with and without LV hypertrophy – in 57.3 and 36.0%; in those examined with a non-dipper diurnal blood pressure profile – 45.2 and 6.4%, night-peaker – 50.0 and 7.1%, dipper – 39.1 and 10.9%, over-dipper – 44.4 and 11.1%, respectively.

Depending on the daily blood pressure profile of the subjects, the structural and functional state of their extracranial vessels is presented (Table 3).

When comparing individuals with a non-dipper BP profile with individuals with a dipper BP profile, the maximum blood flow velocity of the common carotid artery (RCCA) increased by 0.5%, and the

diameters of the left common carotid artery (LCCA) increased by 16.6% and the left vertebral artery (D LHA) by 33.3%, and the maximum blood flow velocity of the left common carotid artery (LCCA) decreased by 2.5%, maximum blood flow velocities of the right internal carotid artery (RICA) by 3.8%, the right internal vertebral artery (RVA) by 8.8%, the left internal vertebral artery (RVA) by 7.7%, and the intima-media complex by 11.1%, but these changes were not statistically significant.

Compared with the dipper subjects, in individuals with the night-peaker BP profile, the diameters of the RCCA increased by 14.3%, the LCCA by 16.6%, the LVA by 33.3%, the intima-media complex by 11.1%, and the maximum blood flow velocities of the RCCA decreased by 7.4%, the LCCA by 11.1%, the RICA by 11.9%, the RVA by 10.8%, the LVA by 7.0%, and the LICA resistance index by 14.2%, but these changes were also not significant.

Significant changes were observed only with a decrease in the maximum blood flow velocity of the left internal carotid artery (LICA) – by 3.3% ($p<0.05$) in non-dippers and by 14.2% ($p<0.05$) in night-peakers, and an increase in the diameter of the RICA by 20.0% ($p<0.05$) in night-peakers.

Table 3. Features of the structural and functional state of extracranial vessels in people with Covid-19 depending on the daily blood pressure profile

Structural and functional state of extracranial vessels	Diurnal blood pressure profile		
	dipper	non-dipper	night-peaker
Diameter of the RCCA	0,7±0,01	0,7±0,2	0,8±0,1
Maximum blood flow velocity of the RCCA	86,1±2,6	86,5±2,7	79,7±4,5
RCCA resistance index	0,7±0,01	0,7±0,01	0,7±0,02
Diameter of the LCCA	0,6±0,01	0,7±0,03	0,7±0,01
Maximum blood flow velocity of the LCCA	87,5±2,3	85,3±3,2	77,8±3,4
LCCA resistance index	0,7±0,01	0,7±0,01	0,7±0,01
Diameter of the RICA	0,5±0,1	0,5±0,01	0,6±0,02*
Maximum blood flow velocity of the RICA	73,4±2,7	70,6±1,7	64,7 ±3,4
RICA resistance index	0,7±0,01	0,7±0,01	0,7±0,03
Diameter of the LICA	0,5±0,01	0,5±0,02	0,5±0,01
Maximum blood flow velocity of the LICA	75,2±2,22	72,7±1,8*	64,5±3,1*
LICA resistance index	0,7±0,01	0,7±0,02	0,6±0,03
Diameter of the RVA	0,3±0,01	0,3±0,01	0,3±0,01
Maximum blood flow velocity of the RVA	46,5±1,8	42,4±1,7	41,5±2,2
RVA resistance index	0,7±0,01	0,7±0,01	0,7±0,01
Diameter of the LVA	0,3±0,01	0,4±0,01	0,4±0,01
Maximum blood flow velocity of the LVA	45,3±1,4	41,8±2,0	42,1±3,2
LVA resistance index	0,7±0,01	0,7±0,01	0,7±0,01
Intimate media complex	0,9±0,01	0,8±0,01	0,10±0,01

Note. * – $p<0.05$

DISCUSSION

A significant increase in cardiovascular disease has been noted among COVID-19 survivors, including those who did not require hospitalization [19]. Huseynov A. et al., 2023, indicated that on average 30% of patients who have recovered from COVID-19 experience persistent cardiopulmonary symptoms (including shortness of breath, palpitations, decreased physical performance, and cardiac arrhythmias) that persist for weeks or even months after acute SARS-CoV-2 infection [20]. Bielecka E. et al., 2024 [21], showed that during coronavirus disease, there is activation of the sympathetic and inhibition of the parasympathetic nervous system, and as a result, blood pressure and heart rate increase [22]. However, the etiopathogenetic relationships of neurotropic effects with the autonomic and immune systems, or with the blood coagulation, anticoagulant, and fibrinolytic systems remain poorly understood [23]. Of particular note are the studies by Hultström M. et al., 2020, which indicate the presence of hyperreninemia, hypercholeolemia, and hypernatremia with cardiac volume overload in individuals who have had severe COVID-19 [24], and Saeed S. et al., 2021, which confirm the possibility of hypertension in those who did not previously have it, and blood pressure dysregulation in previously controlled hypertension [25].

In the work of Bielecka E. et al., 2024, [21] it was shown that in patients with DMAT, the average daily, daytime and nighttime BP, and DBP were significantly increased compared to the pre-pandemic group; the prevalence of persistent uncontrolled hypertension was also significantly higher.

According to the results of our studies, among patients with COVID-19, LV hypertrophy was detected in 75.0% of those examined, and among them, concentric hypertrophy and remodeling were found in 34.6 and 22.7%, respectively, eccentric hypertrophy – in 42.7%; among individuals with and without LV hypertrophy, the non-dipper daily BP profile was found in 33.3 and 24.0%, night-peaker – in 14.6 and 12.0%, dipper – in 42.7 and 56.0%, over-dipper – in 9.4 and 8.0% of the examined. In individuals with concentric hypertrophy and remodeling, as well as with eccentric hypertrophy, the non-dipper daily BP profile was found in 30.7, 29.4, and 31.2%; night-peaker – in 11.5, 11.7, and 15.6%; dipper – in 46.1, 52.9, and 43.8%; over-dipper – in 11.7, 6.0, and 9.4%, respectively.

Our results are confirmed by a number of previously conducted studies. In the work of Brown J. et al., 2024, it was shown that in patients with COVID-19, the LV ejection fraction at rest significantly changed

compared to healthy individuals [26]; Rohun J. et al., 2024, described that heart failure after coronavirus disease should be considered as a manifestation of impaired function of the left and right ventricles, as well as their diastolic dysfunction [27]; Kapusta J. et al., 2023, showed that cardiac arrhythmias (atrial fibrillation, ventricular and supraventricular extrasystoles) are independent predictors of myocardial dysfunction after coronavirus disease [28]. Ingul C. et al., 2022, indicated that three months after COVID-19, diastolic dysfunction occurs three times more often than without it, and premature ventricular contractions were observed in 20% of those examined [29]. Wang W. et al., 2022, noted a significantly higher risk of ischemic heart disease, myocarditis, and heart failure among patients with COVID-19 [30].

Diurnal features of blood pressure in patients with COVID-19 correlate with various factors of cardiac and vascular remodelling. In this study, an increase in the intima-media complex (>0.9 mm) was observed in more than half of the subjects; among subjects with and without LV hypertrophy, in 57.3 and 36.0%; in subjects with a non-dipper daily blood pressure profile, in 45.2 and 6.4%, night-peaker, in 50.0 and 7.1%, dipper, in 39.1 and 10.9%, and over-dipper, in 44.4 and 11.1%, respectively. Our results indicate the need for long-term extended monitoring of cardiovascular complications in patients with COVID-19.

CONCLUSIONS

1. In people with COVID-19, the features of the circadian rhythm of blood pressure were determined: the non-dipper daily blood pressure profile was found in 31.0%, night-peaker – in 14.0%, dipper – in 46.0%, over-dipper – in 9.0%; changes in cardiac and vascular remodelling – in 75.0 and 52.0% of the examined, respectively.

2. The non-dipper daily blood pressure profile was determined in 33.3 and 24.0%, thickening of the intima-media complex – in 45.2 and 6.4% of people with hypertrophy and normal geometry of the left ventricle. In eccentric hypertrophy – in 31.2%, in concentric hypertrophy – in 30.7%, in concentric remodelling – in 29.4% of people with COVID-19, the non-dipper BP profile was determined. In such patients, compared with the examined dipper, daytime SBP significantly increased by 7.3% and the DBP HI – by 109.6%; nighttime SBP – by 15.5%, DBP – by 16.6%, SBP HI – by 180.5%, DBP HI – by more than 2 times; daily min SBP – by 13.9%, ser SBP – by 8.4%, max SBP – by 10.3%, min MBP – by 18.2%, MBP – by 15.4%, max MBP – by 6.2%, SBP HI – by 66.6% and DBP HI decreased – by 50.9%. There were no

significant structural and functional changes in the heart in those examined with the non-dipper profile, and in the vessels – the maximum blood flow velocity of the LICA increased – by 3,3%.

3. The night-peaker BP profile was detected in 14,6 and 12,0% of individuals with and without left ventricular hypertrophy. With eccentric hypertrophy – in 15,6%, with concentric hypertrophy – in 11,5%, with concentric remodeling – in 11,7% of individuals with COVID-19, the night-peaker BP profile was determined. Compared with the dipper subjects examined, in individuals with the night-peaker BP profile, daytime SBP significantly increased by 8,1%, SBP HI and DBP HI – by 215,6% and 177,9%; nighttime SBP – by 30,3%, DBP – by 27,1%, SBP HI and DBP HI – both almost 3 times, VSBP and VDBP –

by 56,8% and 38,4%; daily min SBP – by 11,6%, mean SBP – by 10,9%, max SBP – by 11,3%, min MBP – by 20,4%, MBP – by 9,1%, Min PBP – by 2,6%, mPBP – by 26,1%, Max PBP – by 27,7%, SBP HI – by 33,3% and decreased DBP HI – by 42,7%. Compared with the dipper subjects, in individuals with a night-peaker BP profile, the LV atrial area index significantly increased by 19,9%, the diameter of the RICA by 20,0%, and the maximum blood flow velocity of the LICA decreased by 14,2%.

4. Our results indicate the need to introduce 24-hour BP monitoring, doppler echocardiography and ultrasound of the brachiocephalic arteries into the diagnostic algorithm for individuals with COVID-19, and to implement an assessment of their dynamics in the treatment of this pathology.

Article Declarations

Prospects for further research. Further research will be aimed at developing an algorithm for the diagnosis and treatment of coagulopathy in people who have had coronavirus disease. The results of the study were obtained by the authors during the research work of the Department of Family Medicine of Shupyk National University of Health of Ukraine on the topic «Development and Justification of Programs for the Prevention and Treatment of Patients With Comorbid Pathology of Organs and Systems» [state registration number 0122U-002416; term: 2022-2026].

Study limitations. The authors of the manuscript consciously acknowledge that the limitations of this review are due to both the previously outlined framework of topics, time periods and types of studies, and the availability of sources. The search strategy covered PubMed (<https://pubmed.ncbi.nlm.nih.gov/>).

Primary data and materials. The author of the manuscript consciously certifies that primary medical documentation (medical histories, outpatient cards, examination protocols, results of laboratory and instrumental examinations of specific patients) and statistical databases were not used in the work.

Research ethics. The researchers followed all protocols and procedures prescribed by the Biomedical Research Ethics Committee, as well as the requirements of Ukrainian health legislation and the provisions of the Declaration of Helsinki (2000). The results of the study were obtained by the authors during the research work of the Department of Family Medicine of Shupyk National University of Health of Ukraine on the topic «Development and Justification of Programs for the Prevention and Treatment of Patients With Comorbid Pathology of Organs and Systems» [state registration number 0122U-002416; term: 2022-2026].

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1. Mokhtari T, Hassani F, Ghaffari N, Ebrahimi B, Yarahmadi A, Hassanzadeh G. COVID-19 and multiorgan failure: A narrative review on potential mechanisms. *J Mol Histol.* 2020 Dec;51(6):613-628. <https://doi.org/10.1007/s10735-020-09915-3>.
2. Babicki M, Kapusta J, Kołat D, Kałuzińska-Kołat Ż, Mastalerz-Migas A, Jankowski P, Chudzik M. Cardiac symptoms in patients 3-6 months after contracting COVID-19- data from the polish STOP-COVID registry. *BMC Infect Dis.* 2025 Apr 9;25(1):489. <https://doi.org/10.1186/s12879-025-10774-0>.
3. Cicco S, Vacca A, Albanese F, Susca N, Desantis V, Magistro A, Cazzato G, Cicco G, Sablone S, Cariddi C, Marozzi MS, Catena C, Brosolo G, Marcante S, Ingravalle G, Dalfino L, Lauletta G, Pappagallo F, Solimando AG, Grasso S, Maiorano E, Introna F, Sechi LA, Ria R. Immune disturbance leads to pulmonary embolism in COVID-19 more than classical risk factors: a clinical and histological study. *Intern Emerg Med.* 2023 Oct;18(7):1981-1993. <https://doi.org/10.1007/s11739-023-03383-9>.
4. Xue Y, Sun S, Cai J, Zeng L, Wang S, Wang S, Li J, Sun L, Huo J. Effects of ACEI and ARB on COVID-19 patients: A meta-analysis. *J Renin Angiotensin Aldosterone Syst.* 2020 Oct-Dec;21(4):1470320320981321. <https://doi.org/10.1177/1470320320981321>.
5. Cicco S, Vacca A, Cittadini A, Marra AM. Long-Term Follow-Up May be Useful in Coronavirus Disease 2019 Survivors to Prevent Chronic Complications. *Infect Chemother.* 2020 Sep;52(3):407-409. <https://doi.org/10.3947/ic.2020.52.3.407>.
6. Mokhtari T, Hassani F, Ghaffari N, Ebrahimi B, Yarahmadi A, Hassanzadeh G. COVID-19 and multiorgan failure: A narrative review on potential mechanisms. *J Mol Histol.* 2020 Dec;51(6):613-628. <https://doi.org/10.1007/s10735-020-09915-3>.
7. Mancuso P, Gidaro A, Gregato G, Raveane A, Cremonesi P, Quarna J, Caccia S, Gusso L, Rusconi S, Giacomelli A, Cogliati C, Bertolini F. Circulating endothelial progenitors are increased in COVID-19 patients and correlate with SARS-CoV-2 RNA in severe cases. *J Thromb Haemost.* 2020 Oct;18(10):2744-2750. <https://doi.org/10.1111/jth.15044>.
8. Mancia G, Rea F, Ludergrani M, Apolone G, Corrao G. Renin-Angiotensin-Aldosterone System Blockers and the Risk of Covid-19. *N Engl J Med.* 2020 Jun 18;382(25):2431-2440. <https://doi.org/10.1056/NEJMoa2006923>.
9. Mancia G, Kreutz R, Brunström M, Burnier M, Grassi G, Januszewicz A, Muiesan ML, Tsioufis K, Agabiti-Rosei E, Algharably EAE, Azizi M, Benetos A, Borghi C, Hitij JB, Cifkova R, Coca A, Cornelissen V, Cruickshank JK, Cunha PG, Danser AHJ, Pinho RM, Delles C, Dominiczak AF, Dorobantu M, Doumas M, Fernández-Alfonso MS, Halimi JM, Járαι Z, Jelaković B, Jordan J, Kuznetsova T, Laurent S, Lovic D, Lurbe E, Mahfoud F, Manolis A, Miglinas M, Narkiewicz K, Niiranen T, Palatini P, Parati G, Pathak A, Persu A, Polonia J, Redon J, Sarafidis P, Schmieder R, Spronck B, Stabouli S, Stergiou G, Taddei S, Thomopoulos C, Tomaszewski M, Van de Borne P, Wanner C, Weber T, Williams B, Zhang ZY, Kjeldsen SE. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). *J Hypertens.* 2023 Dec 1;41(12):1874-2071. doi: 10.1097/HJH.0000000000003480. Epub 2023 Sep 26. Erratum in: *J Hypertens.* 2024 Jan 1;42(1):194. <https://doi.org/10.1097/HJH.0000000000003621>.
10. Puntmann VO, Martin S, Shchendrygina A, Hoffmann J, Ka MM, Giokoglu E, Vanchin B, Holm N, Karyou A, Laux GS, Arendt C, De Leuw P, Zacharowski K, Khodamoradi Y, Vehreschild MJGT, Rohde G, Zeiher AM, Vogl TJ, Schwenke C, Nagel E. Long-term cardiac pathology in individuals with mild initial COVID-19 illness. *Nat Med.* 2022 Oct;28(10):2117-2123. <https://doi.org/10.1038/s41591-022-02000-0>.
11. Chlabicz M, Dubatowka M, Jamiolkowski J et al. Enlarged dimensions of cardiac chambers and the root of the aorta as a significant cardiovascular impact of pandemic COVID-19: a population-based study. *Eur J Prev Cardiol.* 2023, 30(Supplement_1). <https://d-nb.info/1369271336/34>.
12. Podrug M, Koren P, Dražić Maras E, Podrug J, Čulić V, Perissiou M, Bruno RM, Mudnić I, Boban M, Jerončić A. Long-Term Adverse Effects of Mild COVID-19 Disease on Arterial Stiffness, and Systemic and Central Hemodynamics: A Pre-Post Study. *J Clin Med.* 2023 Mar 8;12(6):2123. <https://doi.org/10.3390/jcm12062123>.
13. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, Clement DL, Coca A, de Simone G, Dominiczak A, Kahan T, Mahfoud F, Redon J, Ruilope L, Zanchetti A, Kerins M, Kjeldsen SE, Kreutz R, Laurent S, Lip GYH, McManus R, Narkiewicz K, Ruschitzka F, Schmieder RE, Shlyakhto E, Tsioufis C, Aboyans V, Desormais I; ESC Scientific Document Group. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J.* 2018 Sep 1;39(33):3021-3104. doi: 10.1093/eurheartj/ehy339. Erratum in: *Eur Heart J.* 2019 Feb 1;40(5):475. <https://doi.org/10.1093/eurheartj/ehy686>.
14. Angeli F, Zappa M, Verdecchia P. Global burden of new-onset hypertension associated with severe acute respiratory syndrome coronavirus 2 infection. *Eur J Intern Med.* 2024 Jan;119:31-33. <https://doi.org/10.1016/j.ejim.2023.10.016>.

15. Marozzi MS, Fucile I, Panettieri I, Pagani M, Curcio R, Corvasce F, Falcone GS, Mancusi C, Izzo R, Vacca A, Cicco S. COVID-19 induces greater difficulty in blood pressure control due to increased arterial stiffness. *Intern Emerg Med*. 2025 Nov;20(8):2421-2432. <https://doi.org/10.1007/s11739-025-04138-4>.
16. Poryadok «Protokol nadannya medychnoyi dopomohy dlya likuvannya koronavirusnoyi khvoroby (COVID-19)» https://www.dec.gov.ua/wp-content/uploads/2022/02/2020_762_nakaz_covid19.pdf.
17. Babak V, Biletskiy A, Pristavka O, Pristavka P. Statystychna obrobka danix. 2001; 387.
18. Вознюк О. Особливості ремоделювання серця та судин у пацієнтів, які перенесли коронавірусну хворобу. USMYJ [Інтернет]. 22 грудня 2025 р. [цитовано 19 березня 2026 р.];158(4):51-4. Доступно за посиланням: <https://mmj.nmuofficial.com/index.php/journal/article/view/599>
19. Xie Y, Xu E, Bowe B, Al-Aly Z. Long-term cardiovascular outcomes of COVID-19. *Nat Med*. 2022 Mar;28(3):583-590. doi: 10.1038/s41591-022-01689-3.
20. Huseynov A, Akin I, Duerschmied D, Scharf RE. Cardiac Arrhythmias in Post-COVID Syndrome: Prevalence, Pathology, Diagnosis, and Treatment. *Viruses*. 2023 Jan 29;15(2):389. <https://doi.org/10.3390/v15020389>.
21. Bielecka E, Sielatycki P, Pietraszko P, Zapora-Kurel A, Zbroch E. Elevated Arterial Blood Pressure as a Delayed Complication Following COVID-19-A Narrative Review. *Int J Mol Sci*. 2024 Feb 2;25(3):1837. <https://doi.org/10.3390/ijms25031837>.
22. Marques KC, Silva CC, Trindade SDS, Santos MCS, Rocha RSB, Vasconcelos PFDC, Quaresma JAS, Falcão LFM. Reduction of Cardiac Autonomic Modulation and Increased Sympathetic Activity by Heart Rate Variability in Patients With Long COVID. *Front Cardiovasc Med*. 2022 Apr 29;9:862001. <https://doi.org/10.3389/fcvm.2022.862001>.
23. Dani M, Dirksen A, Taraborrelli P, Torocastro M, Panagopoulos D, Sutton R, Lim PB. Autonomic dysfunction in 'long COVID': rationale, physiology and management strategies. *Clin Med (Lond)*. 2021 Jan;21(1):e63-e67. <https://doi.org/10.7861/clinmed.2020-089>.
24. Hultström M, von Seth M, Frithiof R. Hyperreninemia and low total body water may contribute to acute kidney injury in COVID-19 patients in intensive care. *J Hypertens*. 2020 Aug;38(8):1613-1614. <https://doi.org/10.1097/HJH.0000000000002531>.
25. Saeed S, Tadic M, Larsen TH, Grassi G, Mancia G. Coronavirus disease 2019 and cardiovascular complications: focused clinical review. *J Hypertens*. 2021 Jul 1;39(7):1282-1292. <https://doi.org/10.1097/HJH.0000000000002819>.
26. Brown JT, Saigal A, Karia N, Patel RK, Razvi Y, Constantinou N, Steeden JA, Mandal S, Kotecha T, Fontana M, Goldring J, Muthurangu V, Knight DS. Ongoing Exercise Intolerance Following COVID-19: A Magnetic Resonance-Augmented Cardiopulmonary Exercise Test Study. *J Am Heart Assoc*. 2022 May 3;11(9):e024207. <https://doi.org/10.1161/JAHA.121.024207>.
27. Rohun J, Dorniak K, Faran A, Kocharńska A, Zacharek D, Daniłowicz-Szymanowicz L. Long COVID-19 Myocarditis and Various Heart Failure Presentations: A Case Series. *J Cardiovasc Dev Dis*. 2022 Nov 30;9(12):427. <https://doi.org/10.3390/jcdd9120427>.
28. Kapusta J, Babicki M, Pieniawska-Śmiech K, Kałuzińska-Kołat Ż, Kołat D, Jankowski P, Kasprzak JD, Wejner-Mik P, Białek-Bodzak A, Chudzik M. Clinical and electrocardiographic correlates of myocardial dysfunction after COVID-19 in nonhospitalised patients in long-term follow-up. Data from the polish long-covid cardiovascular study. *J Med Virol*. 2023 Dec;95(12):e29331. <https://doi.org/10.1002/jmv.29331>.
29. Ingul CB, Grimsmo J, Mecinaj A, Trebinjac D, Berger Nossen M, Andrup S, Grenne B, Dalen H, Einvik G, Stavem K, Follstad T, Josefsen T, Omland T, Jensen T. Cardiac Dysfunction and Arrhythmias 3 Months After Hospitalization for COVID-19. *J Am Heart Assoc*. 2022 Feb;11(3):e023473. <https://doi.org/10.1161/JAHA.121.023473>.
30. Wang W, Wang CY, Wang SI, Wei JC. Long-term cardiovascular outcomes in COVID-19 survivors among non-vaccinated population: A retrospective cohort study from the TriNetX US collaborative networks. *EClinicalMedicine*. 2022 Nov;53:101619. doi: 10.1016/j.eclinm.2022.101619. Epub 2022 Aug 11. Erratum in: *EClinicalMedicine*. 2023 May;59:101968. <https://doi.org/10.1016/j.eclinm.2023.101968>.

ОСОБЛИВОСТІ ДОБОВОГО ПРОФІЛЮ АРТЕРІАЛЬНОГО ТИСКУ ТА РЕМОДЕЛЮВАННЯ СЕРЦЯ ТА СУДИН У ЛЮДЕЙ З COVID-19

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РЕЗЮМЕ

Вступ. Актуальність дослідження обумовлена значною поширеністю COVID-19 та його серцево-судинних ускладнень в усьому світі.

Мета. Мета роботи – оцінити особливості процесів ремоделювання серця і судин при різних типах добового профілю артеріального тиску після перенесеної коронавірусної інфекції.

Матеріали та методи. В амбулаторних умовах обстежено 100 осіб через 1 місяць після перенесеного COVID-19 (62 чоловіки і 38 жінок), віком $63,14 \pm 2,24$ року. Усім обстеженим проводили доплерокардіографію, ультразвукове дослідження брахіоцефальних артерій, добове моніторування артеріального тиску. Для оцінювання результатів дослідження використовували методи первинного статистичного аналізу.

Результати. Гіпертрофії лівого шлуночка та КІМ діагностовано у 75,0 і 52,0% пацієнтів; у 31,0% – добовий профіль АТ non-dipper, у 14,0% – night-peaker, у 46,0% – dipper, у 9,0% – over-dipper. При гіпертрофії ЛШ та його нормальній геометрії добовий профіль АТ non-dipper визначався у 33,3 і 24,0%, збільшення КІМ – у 45,2 та 6,4% обстежених. У 31,2% осіб з ексцентричною гіпертрофією, у 30,7% – з концентричною гіпертрофією, у 29,4% – з концентричним ремоделюванням виявлено профіль АТ non-dipper. При гіпертрофії ЛШ та без неї добовий профіль АТ night-peaker визначався в 14,6 і 12,0% випадків. У 15,6% обстежених після перенесеного COVID-19 з ексцентричною гіпертрофією, у 11,5% – з концентричною гіпертрофією, у 11,7% – з концентричним ремоделюванням виявлено профіль АТ night-peaker.

Висновки. Отримані результати свідчать про наявність значного впливу COVID-19 на процеси серцево-судинного ремоделювання, вказують на необхідність включення добового моніторування АТ, доплерокардіографії та ультразвукового дослідження судин в алгоритм діагностики та лікування осіб з перенесеним COVID-19.

Ключові слова: Артеріальна гіпертензія, Кров'яний тиск, COVID-19, Артеріальна жорсткість, Ураження органів, спричинене гіпертензією

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