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Predictors of Reduced Left Ventricle Global Longitudinal Strain One Year After COVID-19 Pneumonia

Aim	To identify predictors of decreased left ventricular global longitudinal strain (LV GLS) using the method of speckle-tracking in gray scale one year after COVID-19-associated pneumonia in patients without ischemic heart disease (IHD), previous pulmonary embolism (PE), peripheral thrombosis, and atrial fibrillation (AF).
Material and methods	The study included 156 patients from the Prospective Registry of People After COVID-19-Associated Pneumonia, with optimal visualization quality according to echocardiography (EchoCG), without IHD, AF, history of pulmonary embolism (PE), and peripheral thrombosis. The patients underwent clinical examination in the hospital during the acute period and at 3 and 12 months after discharge from the hospital. To identify earlier predictors of LV GLS impairment, clinical, laboratory, and instrumental data obtained in the hospital and at 3 months of discharge were compared based on the presence of LV GLS impairment one year after discharge (43 patients with reduced LV GLS and 113 patients with normal LV GLS). An LV GLS value ≥18% was considered reduced.
Results	At 3 months after discharge from the hospital, LV GLS impairment was detected in 34 (21.8%) of 156 patients, and 12 months later, in 43 (27.6%; p=0.211) of 156 patients. In contrast to the group with normal LV GLS, the majority of the group with reduced LV GLS were men (74.4% vs. 37.2%; p=0.001). In this group, body mass index (BMI) was significantly higher (29.9±4.3 kg/m^2 vs. 28.1±4.5 kg/m^2; p=0.011), and biological (11.6% vs. 2.7%; p=0.024) and hormonal therapy was administered more frequently (38.1% vs. 22.3%; p=0.049). The final predictive model for LV GLS impairment included male gender (odds ratio (OR), 5.65; 95% confidence interval (CI), 1.22–14.37; p <0.001), BMI (OR, 1.11; 95% CI, 1.01–1.23; p=0.040), left ventricular end-systolic volume index (LVESVI) (OR, 1.10; 95% CI, 1.01–1.22; p=0.046) and right ventricular outflow tract (RVOT) acceleration time (OR, 0.98; 95% CI, 0.95–0.99; p=0.027).
Conclusion	One year after COVID-19-associated pneumonia, a decrease in LV GLS was observed in 27.6% of patients without IHD, AF, history of PE, and peripheral thrombosis and was associated with male gender, increased BMI and LVESVI, and shortened RVOT acceleration time as measured 3 months after discharge from the hospital. The decrease in LV GLS one year after discharge was not associated with the severity of the disease, length of stay in the hospital, or biological and hormonal therapy.

Keywords	COVID-19-associated pneumonia; echocardiography; speckle-tracking in gray scale; left ventricular global longitudinal strain
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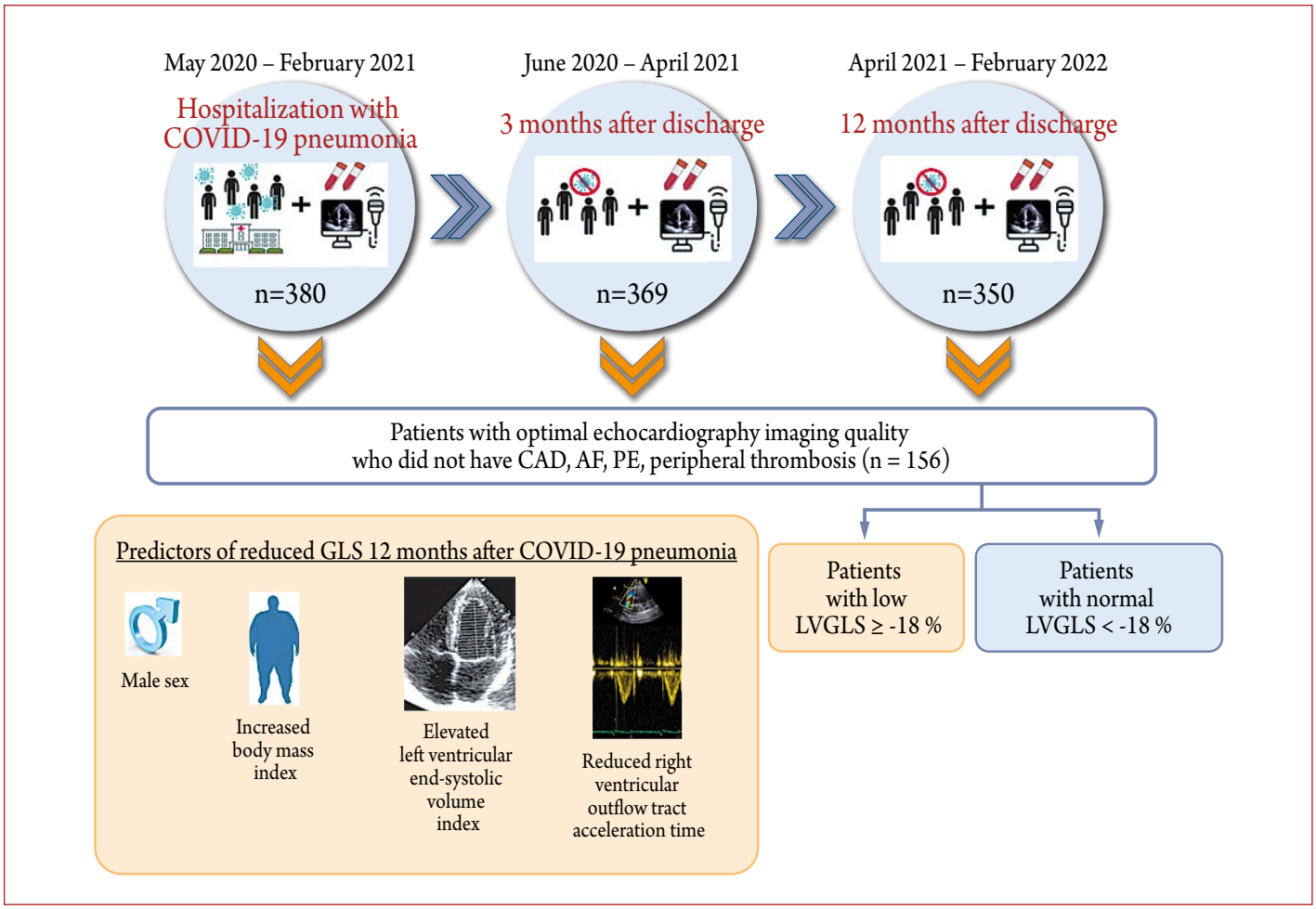
Introduction

The findings of prospective observations in patients with a history of COVID-19 show both an increased risk of cardiovascular complications and subclinical involvement of the respiratory, cardiovascular, and coagulation systems and kidneys, even after mild to moderate COVID-19 [1]. Given the tremendous number of those who had COVID-19, the timely detection of systolic and diastolic myocardial dysfunction is one of the most important tasks of modern cardiology. The left ventricular global longitudinal strain (LVGLS) is a highly sensitive marker of such dysfunction. We also suggest that there are modifiable predictors of abnormal LVGLS in the long term after discharge, which can be targeted to prevent the development of subclinical systolic dysfunction and thus enhance the prognosis for patients.

Central illustration. Predictors of Reduced Left Ventricle Global Longitudinal Strain One Year After COVID-19 Pneumonia



LVGLS, left ventricular global longitudinal strain.

Objective

Identify predictors of a decrease in LVGLS 12 months after documented COVID-19 pneumonia using speckle tracking echocardiography (STE) in patients without pulmonary embolism (PE), peripheral thrombosis, coronary artery disease (CAD), atrial fibrillation (AF).

Material and methods

The observational study was conducted on data from the Prospective Register of Patients with a History of COVID-19 associated Pneumonia (certificate of state registration No. 2021622535). The observation complies with the Declaration of Helsinki, is approved by the local ethics committee (minutes No. 159 dated July 23, 2020) and registered in the international register of clinical trials (ClinicalTrials.gov identifier: NCT04501822). The hospital data of patients were studied by discharge summaries. All patients underwent clinical examinations and laboratory tests, including computed tomography (CT) of the lungs (Toshiba Aquilion-64), determination of pulse wave velocity and the ankle brachial index (VaSera VS-1000 Series sphygmomanometer), and echocardiography 3 and 12 months after discharge.

Heart failure with preserved ejection fraction (HFpEF) was diagnosed according to the HFA-PEFF algorithm [2]. Echocardiography was performed using a Vivid S70 system. DICOM echocardiograms were processed in TomTec. Echocardiography measurements and inclusion criteria have been previously described in detail [3]. According to the current guidelines, the lower normal limit was LVGLS ≤ 18 % [4, 5]. LVGLS was measured manually by one expert to exclude inter-researcher variability [4]. Data of patients with a history of PE and peripheral thrombosis were not included since the effect of hypercoagulation on echocardiography parameters cannot be excluded in these cases. Data of patients with CAD were not included to level the effect of ischemic factor on LVGLS. Data of patients with AF were also not included, since LVGLS can be studied only in sinus rhythm. The following laboratory parameters were estimated: creatinine, C-reactive protein, D-dimer during the hospital stay; standard deviation of the red blood cell distribution width, liver function tests, creatinine, ferritin, C-reactive protein (including high sensitivity

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CRP), interleukin-6, tumor necrosis factor, brain natriuretic peptide 3 months after discharge.

Of the 380 subjects, 26 patients were excluded due to: newly diagnosed malignancy (n=2), pregnancy (n=3), change of residence (n=3), refusal for various reasons (n=10), failure to undergo the examinations on time (n=7), and inadequate echocardiogram quality (n=1).

At Visit 1 and Visit 2, 271 and 244 patients, respectively, had optimal echocardiograms. Changes in LVGLS were traced in 206 patients. CAD, PE, AF, and peripheral thrombosis were diagnosed in 50 patients. The final analysis included data of 156 patients with the median age of 52 [44; 56] years, including 52.6 % of female patients.

According to the CT scan of the lungs, mild, moderate, severe, and critical lesions were observed in 15.7 %, 36.6 %, 34.0 %, and 13.7 % of patients, respectively, during hospitalization. 10.5 % of patients were treated in intensive care units.

The examined patients were divided into groups depending on LVGLS values 12 months after discharge: normal LVGLS (n=113) and reduced LVGLS (n=43). The examination results were compared at hospitalization and 3 months after discharge to detect earlier signs of abnormal LVGLS in the long term after COVID-19 pneumonia.

Statistical analysis of the data obtained was performed in SPSS 21.0 (SPSS Inc., Chicago, IL, USA) and Statistica 12.0. The distribution of variables was estimated using the Kolmogorov–Smirnov test. Normally distributed quantitative data were presented as the means and standard deviations (M±SD) and non-normally distributed quantitative variables were expressed as the medians and interquartile ranges (Me [Q1–Q3]). The intergroup comparisons were carried out depending on the type of distribution of quantitative data using the Student's t-test for independent groups or the Mann–Whitney test. The chi-squared test and Fisher's exact

Table 1. Clinical parameters of patients 3 months after COVID-19 pneumonia depending on LVGLS 12 months after discharge

Parameter		Normal LVGLS (n=113)	Abnormal LVGLS (n=43)	p
Male		42 (37.2)	32 (74.4)	0.001
Age, years		50.2±10.9	48.2±12.2	0.446
Data obtained during hospital treatment				
Length of hospital stay, days		13.2±4.7	14.1±6.1	0.585
Severity of pneumonia according to CT, degree	1	19 (17.1)	5 (11.9)	0.584
	2	42 (37.8)	14 (33.3)	
	3	37 (33.3)	15 (35.7)	
	4	13 (11.7)	8 (19.0)	
Treatment in ICU		10 (8.9)	4 (9.3)	0.942
Biological therapy (tocilizumab, sarilumab)		3 (2.7)	5 (11.6)	0.024
Hormonal therapy (prednisolone)		25 (22.3)	16 (38.1)	0.049
Follow-up data 3 months after discharge				
Body mass index, kg/m2		28.1±4.5	29.9±4.3	0.011
Residual lung damage		26 (23.0)	15 (34.9)	0.128
Systolic blood pressure, mm Hg		126.7±16.5	128.3±14.1	0.482
Diastolic blood pressure, mm Hg		83.3±11.8	85.6±11.9	0.374
Ankle brachial index, mean		1.08±0.09	1.09±0.09	0.671
Pulse wave velocity, mean, m/s		13.2±2.4	13.8±2.8	0.363
Arterial hypertension		71 (63.4)	30 (69.8)	0.456
HFA-PEFF score, %	0–1	72 (63.7)	26 (60.5)	0.129
	2–4	39 (34.5)	14 (32.6)	0.818
	5–6	2 (1.8)	3 (7.0)	0.715
Diabetes mellitus type 2		10 (8.8)	5 (11.6)	0.559
Heart rhythm disorders		34 (30.1)	9 (20.9)	0.253
Adherence to treatment	do not adhere	4 (3.7)	1 (2.5)	0.126
	adhere fully	47 (43.5)	16 (40.0)	
	adhere poorly	22 (20.4)	9 (22.5)	
	no prescriptions	35 (32.4)	14 (35.0)	

The data expressed as the means and standard deviations (M±SD), the absolute numbers and percentages of patients (n (%));
LVGLS , left ventricular global longitudinal strain; CT, computed tomography; ICU, intensive care unit;
HFA-PEFF, the Heart Failure Association diagnostic algorithm for heart failure with preserved ejection fraction.

test were used to compare the categorical variables. The differences were statistically significant with a two-tailed level of significance $p<0.05$. Logistic regression analysis with stepwise selection of variables was conducted to identify factors associated with abnormal LVGLS. If factors had a similar mechanism of the effect (for example, the degree and risk arterial hypertension (AH), systolic and diastolic blood pressure), the analysis included the most statistically significant one. The intra-study reproducibility of LVGLS measurements was assessed by blind measurements made twice using a Bland–Altman method. An intraclass correlation coefficient (ICC) was also calculated.

Table 2. Echocardiography parameters of patients 3 months after COVID-19 pneumonia depending on LVGLS 12 months after discharge

Parameter		References		Normal LVGLS (n=113)	Abnormal LVGLS (n=43)	p
		Male	Female			
LV end-diastolic volume	mL	106±22	76±15	85.6±22.0	97.9±23.7	0.004
	mL/m²	54±10	45±8	44.5 [38.3; 51.8]	46.3 [40.2; 57.3]	0.120
LV end-systolic volume	mL	41±10	28±7	26.5±8.3	33.7±14.1	0.001
	mL/m²	21±5	16±4	13.5 [11.2; 16.1]	15.5 [12.6; 18.8]	0.014
LV mass (M-mode)	g	88–224	67–162	161.3±32.3	179.2±39.1	0.003
	g/m²	49–115	43–95	82.5 [74.6; 94.1]	89.8 [77.9; 99.2]	0.086
LV systolic volume, mL				65.4±18.6	70.4±28.3	0.384
LVEF (Simpson 2D)	%	62±5	64±5	69.2±3.9	67.7±4.11	0.076
LV outflow tract deceleration time, ms				214.6±32.5	198.4±26.6	0.004
LV isovolumic relaxation time (IVRT)	ms	74±7**		99.8±20.6	98.2±24.8	0.434
LV early diastolic filling deceleration time (DT)	ms	181±19**		212.2±54.4	218.6±68.1	0.868
Early LV diastolic filling velocity, E, cm/s				73.5 [61.0; 86.0]	67.0 [60.0; 78.0]	0.197
Late LV diastolic filling velocity, E, cm/s				67.5 [58.5; 80.0]	69.0 [56.0; 81.0]	0.845
E/A		1.28±0.25**		1.06 [0.80; 1.34]	0.97 [0.80; 1.35]	0.371
Early diastolic lateral mitral annular velocity, e' later	cm/s	≥10***		11.9±3.8	10.7±3.9	0.122
Early diastolic septal mitral annular velocity, e' sept	cm/s	≥7***		9.2±2.9	8.2±3.2	0.034
E/e'		<9***		6.8 [6.2; 8.0]	7.9 [6.3; 9.4]	0.069
Left atrial maximum volume	mL	18–58*	22–52*	45.7±12.1	47.3±15.4	0.838
	mL/m²	16–34		22.8 [19.9; 27.5]	22.3 [19.1; 25.8]	0.342
Left atrial ejection fraction, %				61.3±8.2	61.2±9.0	0.803
Epicardial fat thickness, mm				7.19±1.6	7.23±1.8	0.741
Right atrial maximum volume	mL	–	–	30.6±10.7	33.4±12.1	0.134
	mL/m²	25±7	21±6	15.7 [12.4; 18.7]	15.9 [12.3; 18.9]	0.524
RV anteroposterior dimension	mm	21–35		25.4±2.3	26.2±1.4	0.035
	mm/m²			13.5±1.4	13.1±1.3	0.136
RV diastolic area	cm²	10–24	8–20	15.1±4.1	17.2±3.8	0.001
	cm²/m²	5.0–12.6	4.5–11.5	7.6 [6.5; 8.8]	8.3 [7.3; 9.8]	0.022
RV fractional area change (RVFAC)	%	≥35		52.7±8.9	50.3±9.3	0.203
Tricuspid annular plane systolic excursion (TAPSE)	mm	≥17		22.9±2.5	23.0±2.5	0.763
RV sphericity index, basal, mm				0.45±0.09	0.46±0.07	0.364
RV sphericity index, middle, mm				0.38±0.07	0.39±0.08	0.684
Number of patients with clear CW spectrum of tricuspid regurgitation, n (%)				85 (75.2)	32 (74.4)	0.918
Peak tricuspid regurgitation velocity	cm/s	≤2.8***		2.16±0.32	2.16±0.39	0.961
Tricuspid annular S' velocity	cm/s	≥9.5		10.2±2.7	9.7±2.4	0.358
RV outflow tract acceleration time, ms				117.2±25.8	104.2±17.3	0.002

The data are expressed as the medians and interquartile ranges (Me [Q1–Q3]), the means and standard deviations (M±SD), and numbers and percentages of patients (n (%)). Reference values are provided source [4]; * – from source [6]; ** from source [7] (for 41–60-year-old age group); *** – from source [2]. LVGLS, left ventricular global longitudinal strain.

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Results

Abnormal LVGLS was found in 34 (21.8 %) and 43 (27.6 %) of 156 patients examined 3 months and 12 months after discharge, respectively (p=0.211). The main clinical characteristics are presented per groups in Table 1.

In contrast to the normal LVGLS group, the majority of the reduced LVGLS group was male patients, the mean body mass index (BMI) was statistically significantly higher in this group and corresponded to overweight. There were statistically significant intergroup differences in the frequency of using biologically active and hormonal therapy during the hospital stay: e.g., tocilizumab, sarilumab, and prednisolone were more often prescribed in the reduced LVGLS group. The groups did not differ in other clinical characteristics (frequency and structure of complaints 3 months after discharge, frequency and degree of AH and diabetes mellitus type 2, frequency of residual lung abnormalities in CT, mean ankle-brachial index, and pulse wave velocity, etc.).

As for the laboratory findings, the reduced LVGLS group had higher serum levels of creatinine both during the hospital stay (90.7±24.2 μmol/L vs. 78.7±16.9 μmol/L; p=0.010) and 3 months after discharge (76.8±13.1 μmol/L vs. 71.4±11.9 μmol/L; p=0.009). Three months after discharge, the relative red blood cell distribution width was smaller in this group (47.0±3.7 % vs. 48.2±3.4 %; p=0.023) with trends to higher levels of ferritin (92.6 [39.6; 165.8] mg/mL vs. 49.8 [21.8; 130.0] mg/mL; p=0.056) and alanine aminotransferase (50.3±9.3 U/L vs. 20.7±6.7 U/L; p=0.059). There were no differences between the groups in other laboratory tests.

The mean values of the echocardiography parameters were within the normal range (Table 2). No subjects had reduced left ventricular ejection fraction (LVEF), but it tended to be lower in the reduced LVGLS group. In this group, patients had statistically significantly higher LV end-systolic volume (LVESV) and LV mass, shorter deceleration time in the LV outflow tract, lower systolic septal mitral annular velocity, and a trend to a higher E/e' ratio. The reduced LVGLS group was characterized by a larger right ventricular (RV) diastolic area and a higher acceleration time in the RV outflow tract.

In addition to the factors, which differed between the groups, potentially significant factors were included in the further analysis: age, length of hospital stay», and severity of pneumonia, the volume of lung damage during the hospital stay (maximum) and 3 months after discharge; the presence and structure of complaints, the presence and severity of AH, signs of chronic heart failure (CHF), LVEF, parameters of LV diastolic function, and adherence to treatment as assessed 3 months after discharge. Of all the many factors potentially affecting LVGLS, the four most significant ones were selected by logistic regression. They were used construct the prediction model for LVGLS abnormalities in the long term after COVID-19 pneumonia: male sex (odds ratio (OR) 5.65; 95 % confidence interval (CI) 2.22–14.37; p<0.001), BMI (OR 1.11; 95 % CI 1.01–1.23; p=0.040), left ventricular end-systolic volume index (LVESVI; OR 1.10; 95 % CI 1.01–1.22; p=0.046), and acceleration time in the RV outflow tract (OR 0.98; 95 % CI 0.95–0.99; p=0.027) 3 months after discharge.

Good comparability was demonstrated for the LVGLS measurement results: the intraoperator reproducibility (the Bland–Altman method) was 2.9 % with a coefficient of variation of 1.05 for 84 repeated blind measurements; ICC=0.88±0.04.

Discussion

LVGLS is a highly sensitive marker of subclinical LV dysfunction and is of great significance for the diagnosis of HFpEF. The HFA-PEFF algorithm detected HFpEF in only 5 patients (2 patients with normal LVGLS and 3 patients with abnormal LVGLS). The majority (63.7 % of patients with normal LVGLS and 60.5 % of patients with decreased LVGLS) did not have CHF, and about 30 % of patients in both groups potentially had initial stage of HFpEF (HFA-PEFF 2–4). Diastolic stress test should be performed for the latter to exclude HFpEF.

Based on the results of logistic regression analysis, 4 parameters were selected from the baseline set of variables describing the clinical and laboratory status of patients, systolic and diastolic functions of the ventricles of the heart. Those parameters had an independent association with abnormal LVGLS in the long term after the disease—a demographic parameter (male sex), anthropometric parameter (BMI), and two echocardiographic parameters evaluated 3 months after discharge: LV systolic function and pulmonary vascular resistance.

The male sex, according to our findings, was the strongest predictor of abnormal LVGLS. It increased 5.65-fold the risk of LVGLS abnormalities in the long term after COVID-19 pneumonia. This is consistent with the results of population-based studies conducted before the COVID-19 pandemic. The Korean population study showed male patients had worse values of peak systolic strain than female patients [8], similar results were obtained in other ethnic groups [9]. As for the elderly population, the majority, as well as our patients, have AH; the ARIC study also showed lower LVGLS in male patients [10]. The same association was observed in patients with untreated AH [11].

In the Russian population study of persons over 55 years old, the association of LVGLS and AH was shown, which significantly depends on body weight [12]. In

our study, LVGLS abnormalities had no independent association with AH, which was probably due to the younger age of patients.

BMI was the other strongest predictor of abnormal LVGLS. An extra 1 kg/m2 increased the risk of reduced LVGLS by 11 % in the long term after COVID-19 pneumonia. Tudoran et al. [13] showed that, 1.5–2.5 months after COVID-19, LVGLS abnormalities were associated with age, BMI, and creatine kinase MB levels during the hospital stay. According to our data, none of the laboratory parameters showed any association with LVGLS abnormalities during the hospital stay or 3 months after discharge. This is probably due to the longer follow-up period in our study. We deliberately did not include the results of clinical examinations and laboratory tests conducted 12 months after discharge to identify earlier predictors of abnormal LVGLS. STE indicators were not included in the analysis 3 months after discharge since indicators commonly used in clinical practice as predictors of LVGLS abnormalities should be preferred. In our study, such echocardiography parameters included a parameter characterizing systolic function—LVESVI (an extra 1 mL/m2 increased the risk of detecting reduced LVGLS by 10 %) and a marker of increased pulmonary vascular resistance—acceleration time in the RV outflow tract (a 1 ms decrease increased the risk by 2 %).

As well as Croft et al. [14], Tryfou et al. [15], and Baycan et al. [16], we found no correlations between LVGLS and severity of COVID-19 and lung involvement during the hospital stay. This confirms that, in the long term after recovery, the risk of subclinical myocardial dysfunction does not depend on the severity of symptoms during the acute disease. It is also noteworthy that there are no independent associations between hormonal and biological therapy conducted in the acute period and LVGLS abnormalities in the long term after COVID-19 pneumonia.

Thus, according to our findings, sex, BMI, and generally accepted echocardiography indicators of early recovery period are sufficient to predict LVGLS abnormalities in the long term after COVID-19 pneumonia. According to our data, BMI is the modifiable predictor of reduced LVGLS. Therefore, targeted weight loss can serve as a tool to prevent subclinical LV systolic dysfunction and therefore be considered as an effective measure to reduce the risk of CHF after COVID-19.

Limitations

Our sample is limited to patients with optimal echocardiography imaging, i.e., it does not reflect the condition of all patients with the history of COVID-19 pneumonia. The study is also limited by the lack of data on myocardial strain before COVID-19 and in the acute period of the infection. Thus, the observed myocardial strain abnormalities could be attributed to the direct action of the virus or its indirect influence through the development/aggravation of cardiovascular pathology, but they could also be present before COVID-19.

Conclusion

Twelve months after COVID-19 pneumonia, a decrease in left ventricular global longitudinal strain is observed in 27.6 % of patients without coronary artery disease, atrial fibrillation, pulmonary embolism, and peripheral thrombosis and is associated with male sex, increased body mass index, elevated left ventricular end-systolic volume index, and reduced acceleration time in the right ventricular outflow tract estimated 3 months after discharge from the hospital. The decrease in left ventricular global longitudinal strain is not associated with the severity of pneumonia, the length of hospital stay, biological and hormonal therapy.

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