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Right heart condition in patients with COVID-19 pneumonia

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Aim. To assess right heart condition in patients with coronavirus disease 2019 (COVID-19) pneumonia.

Material and methods. One hundred and five patients with COVID-19 pneumonia were divided into 3 groups depending on the involvement of lung parenchyma: group I — 0-25%, II — 25-50%, III — 50-75%. The clinical status of patients was assessed using the NEWS2 and SHOCS-COVID scales. A complete blood count and biochemical blood tests were performed to determine the level of N-terminal pro-brain natriuretic peptide (NT-proBNP) and troponin I. Echocardiography was performed to assess the right heart structural, hemodynamic and functional parameters.

Results. In patients with COVID-19 pneumonia, with an increase in lung parenchyma involvement, the intensity of systemic inflammatory response increased: C-reactive protein, group I — (4 [1,9; 35] mg/l), in III — (70,5 [33; 144] mg/l) (p_{I-III}=0,012); myocardial stress marker level increased: NT-proBNP, group I — 77 [48; 150] ng/l, group III — 165 [100; 287] ng/l (p_{I-III}=0,047). The dependence of NT-proBNP on C-reactive protein level was revealed (r=0,335, p=0,03). Intergroup comparison did not reveal significant differences between the main right heart functional parameters: TAPSE, Tei index (PW and TDI), FAC of the right ventricle (RV) (p>0,05). However, differences in the tricuspid annular peaks were found as follows: group I — 0,14 [0,12; 0,14] m/s, group II — 0,14 [0,12; 0,15] m/s, group III — 0,16 [0,14; 0,17] m/s (p_{I-II}=0,012, p_{I-III}=0,014) and RV global longitudinal strain: group I — 19,63±7,72%, group III — 27,4±5,93% (p_{I-III}=0,014). The relationship between the RV global longitudinal strain and SHOCS-COVID score was confirmed (r=0,381; p=0,024).

Conclusion. Patients with COVID-19 pneumonia showed no signs of right heart dysfunction. The development of RV hyperfunction was noted. Most likely, this is a compensatory mechanism in response to acute RV afterload. NT-proBNP increase under conditions of an inflammatory response may indicate myocardial stress. The results obtained allow to expand our understanding of the right heart condition in patients with COVID-19 pneumonia.

Keywords: COVID-19, echocardiography, NT-proBNP, right heart, global longitudinal strain.

Relationships and Activities: none.

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A new coronavirus infection (COVID-19) caused by the SARS-CoV-2 virus is characterized by multisystem complications, the leading one being respiratory system damage [1]. Cardiovascular damage occurs in patients with COVID-19 in 20-30% of cases. Several pathophysiological mechanisms of cardiac damage are discussed: hypoxia, direct viral myocardial damage, systemic inflammatory response syndrome, hypercoagulation [2, 3].

Special attention should be paid to right heart dysfunction in patients with COVID-19, the frequency of which, according to Insgro G, et al., is 20-39% and often remains undiagnosed [3]. The main concept of right heart dysfunction formation is the formation of a vicious circle — increase of post-load (pulmonary vascular resistance) on the right ventricle (RV) and increase of end-systolic RV volume [4].

The risk group for developing RV dysfunction includes patients with extremely severe COVID-19 complicated by acute respiratory distress syndrome and needing artificial lung ventilation [5, 6].

In the literature available to us, there is insufficient data characterizing the state of right heart in patients with COVID-19 viral pneumonia who have a mild, moderate, severe course and do not require treatment in the intensive care unit.

The study goal is to evaluate the state of right heart in patients with COVID-19-associated pneumonia.

Material and methods

A single-center prospective study enrolled 105 patients with COVID-19 (polymerase chain reaction "+") and viral pneumonia confirmed by chest spiral computed tomography (SCT). The age of the subjects ranged from 27 to 83 years (Me 52 years, IQR [44;61]), of whom 61 (58%) were men. Respiratory support for patients with acute respiratory failure (n=83) was performed in the volume of low-flow oxygenation, the average rate of oxygen mixture delivery was 8,6±2,4 l/min.

Enrollment criteria:

1. Presence of positive result of the polymerase chain reaction on SARS-CoV-2.

2. Presence of viral pneumonia, confirmed by SCT data.

Exclusion criteria from the study:

1. Systolic dysfunction of left ventricle (LV) according to echocardiography (Echo).

2. Severe concomitant pulmonary and cardiovascular pathology: chronic obstructive pulmonary disease, bronchial asthma, acute cerebrovascular accident with marked neurological deficit, postinfarction cardiosclerosis, permanent atrial fibrillation, severe impaired renal function.

The NEWS2 and SHOCS-COVID scales were used to assess the patients' clinical status [7, 8]. SCT with determination of the volume of pulmonary parenchyma damage using MULTI-VOX software was performed (the volume of pulmonary parenchyma lesion did not exceed 75%). General clinical blood test, biochemical blood test with determination of troponin I and N-terminal promoted natriuretic peptide (NT-proBNP) concentration, Echo on day 10±2,5 from the onset of symptoms (equipment Siemens SC2000, Germany), with in-depth assessment of structural (RV diameters, RV wall thickness, volumes of right atrium (RAP)), hemodynamic (systolic pulmonary artery pressure (SPAP), mean pulmonary artery pressure (mPAP), maximal gradient in pulmonary artery) and functional (systolic and diastolic function of RV with calculation of Tei indices and global longitudinal deformation (GLS) of RV and RA) parameters were performed.

Patients received combination drug therapy for SARS-CoV-2-associated infection according to the Provisional Guidelines of the Russian Ministry of Health Version 9 [9].

The study was approved by the Local Ethics Committee of N.I. Pirogov Russian National Research Medical University, Protocol No. 203 dated January 21, 2021. Written informed consent was obtained from all participants prior the enrollment.

According to the study design, patients were divided into 3 groups according to the severity of viral pneumonia according to SCT data. The volume of pulmonary parenchyma damage in group I was 0-25%, in group II — 25-50%, in group III — 50-75% (Table 1).

Statistical processing of the obtained data was performed using the application package IBM SPSS 26 for Windows (USA). Quantitative measures were assessed for normality using the Shapiro-Wilk test (for <50 subjects) or the Kolmogorov-Smirnov test (for >50 subjects). Quantitative indices with normal distribution were described using arithmetic mean (M) and standard deviations (SD), 95% confidence interval limits. In the absence of normal distribution, quantitative data were described using the median (Me) and the lower and upper quartiles [Q1;Q3]. Comparison of three or more groups for a quantitative indicator with a normal distribution was performed using a one-factor analysis of variance, a posteriori comparisons were made using Tukey's criterion (provided that the variances are equal). Comparisons of three or more groups for quantitative index whose distribution differed from normal were made using the Kruskel-Wallis test, a posteriori comparisons were made using the Dunn test with Hill's correction. Comparisons of

Table 1

Characteristics of patients depending on the volume of lung parenchyma damage

	Group I (n=12)	Group II (n=61)	Group III (n=32)	p
Age, years	48,9±17	51,6±13,1	53,1±10,3	0,807
Men, %	7 (58,35)	33 (54)	21 (65)	0,564
NEWS2, score	1,6±0,9	2,3±1	3,2±1,5	0,045*
SHOCS-COVID, score	7,5±3,7	9±2,4	12,8±2,2	0,001*
SpO_2, %	97 [95;98]	93 [92;94]	90 [86;91]	<0,001*
Laboratory data				
Leukocytes, thousands	5,6±2,3	7,7±4,1	8,6±3,4	0,006* p_{I-III}=0,006*
Lymphocytes, thousands	1,2 [0,9;1,7]	1,0 [0,6;1,2]	0,9 [0,7;1,3]	0,124
CRP, mg/l	4 [1,9;35]	48,2 [22,2;91,8]	70,5 [33;144]	0,012* p_{I-III}=0,012*
LDH, U/l	242,92 [215;245]	332 [278;378]	367 [250;420]	0,018* p_{I-II}=0,048* p_{I-III}=0,031*
Fibrinogen, g/l	4,3 [3,6;4,9]	5,8 [4,1;7,3]	5,5 [3,2;7,8]	0,046* p_{I-II}=0,048*
NT-proBNP, ng/l	77 [48;150]	96 [49;212]	165 [100;287]	0,045* p_{I-III}=0,047*
Troponin I, ng/ml	<0,02	<0,02	<0,02	>0,05

Note: data are presented as M±SD or Me [Q1;Q3], depending on type of value distribution of the indicator under study. * — differences in the indicators are statistically significant (p<0,05).

Abbreviations: LDH — lactate dehydrogenase, SHOCS-COVID — rating scale of clinical state of patients with COVID-19, CRP — C-reactive protein, NEWS2 — The National Early Warning Score, NT-proBNP — N-terminal brain natriuretic peptide, SpO_2 — oxygen saturation.

percentages in the analysis of multifield contingency tables was performed using Pearson's chi-square test. Direction and closeness of correlation between two quantitative indicators were assessed using Pearson correlation coefficient (with normal distribution of the compared indicators) and Spearman rank correlation coefficient (with distribution other than normal). A predictive model characterizing the dependence of a quantitative variable on factors was developed using the linear regression method. The differences between the groups were considered significant at p<0,05.

Results

The patients in the groups were comparable in age (p=0,807) and gender (p=0,564). Significant differences were obtained between the groups according to NEWS2 scores: in group I — 1,6±0,9, in group II — 2,3±1, in III — 3,2±1,5 (p=0,045); SHOCS-COVID scores: in group I — 7,5±3,7, in II — 9±2,4, in III — 12,8±2,2 (p=0,001); oxygen saturation (SpO_2): in group I — 97 [95;98]%, in II — 93 [92;94]%, in III — 90 [86;91]% (p<0,001).

In addition, the study of systemic inflammation markers revealed significant differences between the level of leukocytes in patients of groups I (5,6±2,3 thousand) and III (8,6±3,4 thousand) (p_{I-III}=0,006). There were no significant differences in absolute number of lymphocytes (p=0,124), but there was a tendency to lymphopenia in patients with a more severe course of coronavirus infection. There were significant differences in C-reactive protein levels between group I (4 [1,9;35] mg/l) and group III (70,5 [33;144] mg/l) (p_{I-III}=0,012), fibrinogen between group I (4,3 [3,6;4,9] g/l) and group II (5,8 [4,1;7,3] g/l) (p_{I-II}=0,048). The level of total lactate dehydrogenase, which indirectly reflects the pathological process intensity in lung tissue, also differed significantly between groups I (242,92 [215;245] U/l) and II (332 [278;378] U/l) (p_{I-II}=0,048), I and III (367 [250;420] U/l) (p_{I-III}=0,031).

The NT-proBNP level in patients in groups I and II were within normal limits, in contrast to patients in group III, where they were moderately outside normal limits (165 [100;287] ng/l) and were significantly higher than in patients in group I (p_{I-III}=0,047). The troponin I level in the three groups were within normal limits (Table 1).

Thus, the most severe patients, in terms of clinical status and severity of systemic inflammatory reaction (SIR), were in group III.

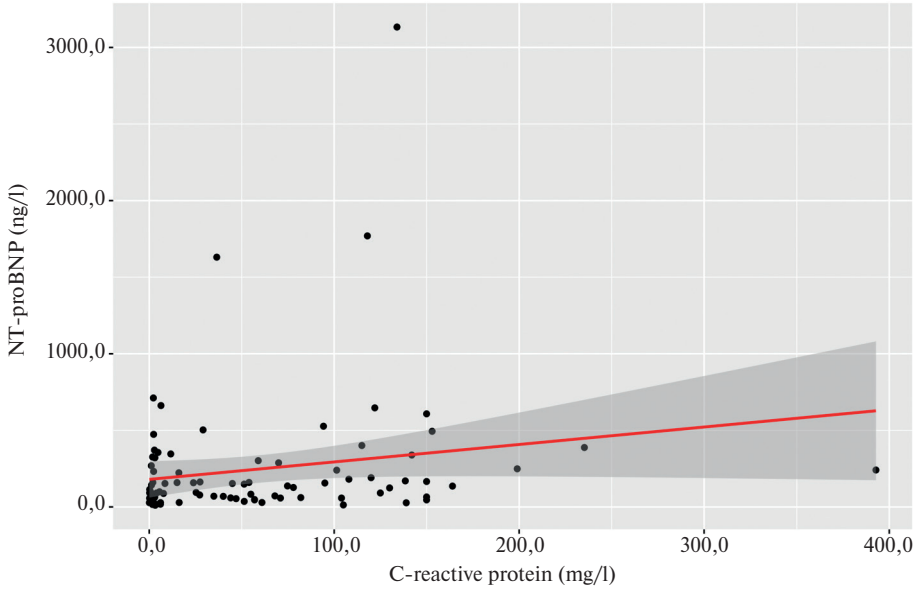

Figure 1. Dependence of myocardial stress marker (NT-proBNP) level on SIR's (CRP) marker in patients with COVID-19-associated pneumonia.
Abbreviation: NT-proBNP — N-terminal brain natriuretic peptide.

The relationship between level of the main marker of SIR — C-reactive protein (CRP) and the marker of myocardial stress — NT-proBNP was of interest. A statistically significant direct correlation of average strength between the above indicators was found (r=0,335, p=0,03). A prognostic model was built to describe the dependence of NT-proBNP level on the degree of SIR tension. The observed dependence is described by a pairwise linear regression equation: $Y_{NT\text{-}proBNP}=1,14\times X_{CRP}+178,702$. It was found that a 1-mg/l increase in CRP should be expected to increase NT-proBNP by 1,14 ng/l (Figure 1).

To assess the condition of the right parts of the heart, an Echo was performed, the results of which are presented in Table 2.

In all patients, LV ejection fraction (EF) was within normal limits (61±3,1%).

Comparison of the RV structural characteristics demonstrated that group III patients had a larger mean RV diameter than group I patients ($p_{I\text{-}III}=0,005$). Significant differences were revealed between all groups in terms of the minimum indexed volume of RA (p=0,038). Otherwise, there were no statistically significant differences in the structural characteristics of RV.

The study of hemodynamic parameters showed that SPAP reached the highest values in group III and was significantly higher than in patients of groups I and II ($p_{I\text{-}III}=0,001$, $p_{II\text{-}III}=0,001$). There was also an increase in mPAP with an increase in the volume of lung parenchyma damage. The values in group I significantly differed from those in groups II and III ($p_{I\text{-}II}=0,017$, $p_{I\text{-}III}=0,018$, respectively). The revealed differences are regular, taking into account the increase of hypoxemic vasoconstriction of the small circulatory circle vessels and dysregulation of vasoactive substances production in pulmonary vessels [10].

In the study of the right heart functional state, the peak s' rate — movement of the free wall of tricuspidal annulus tended to increase on tissue Doppler study. Significant differences in this parameter were found when comparing groups I and II, I and III ($p_{I\text{-}II}=0,012$, $p_{I\text{-}III}=0,014$, respectively). There were no differences in the parameters that most accurately determine the presence of right heart dysfunction: TAPSE, Tei index (PW and TDI), RV FAC (p>0,05).

When analyzing RV GLS as an index of RV systolic function, significant differences were obtained in comparison of groups I and III ($p_{I\text{-}III}=0,014$). Similar changes were revealed in the analysis of RA GLS values — the highest value was obtained in group III patients, and it was significantly higher than in group II patients ($p_{II\text{-}III}=0,002$).

Thus, there was no right heart dysfunction in patients with COVID-19-associated pneumonia. At the same time, higher RV and RA GLS values in group III patients may indicate right heart hyperfunction against the background of pronounced SIR. This observation is indirectly confirmed by the found direct correlation of average strength between the SHOCS-COVID score and RV GLS (r=0,381; p=0,024). The higher rate of peak s' of tricuspidal annulus in group III can be taken into account when discussing the formation of RV hypercontractility.

Table 2

Echo parameters of RV in patients with COVID-19-associated pneumonia

RV indicators	Group I (n=12)	Group II (n=61)	Group III (n=32)	p
RV/LV	0,8 [0,79;0,86]	0,8 [0,73;0,9]	0,8 [0,74;0,85]	0,898
RV, parasternal access, cm	2,7 [2,48;2,82]	2,8 [2,6;3,0]	2,9 [2,68;3,02]	0,267
RV basal diameter, apical access, cm	3,5 [3,4;3,9]	3,8 [3,4;4,1]	3,9 [3,58;4,12]	0,279
RV middle segment, apical access, cm	2,75 [2,6;3,02]	3,1 [2,8;3,5]	3,25 [3,08;3,6]	0,007* p_{I-III}=0,005*
RV length, apical access, cm	6,6 [6,3;7,45]	6,9 [6,2;7,5]	6,7 [5,78;7,62]	0,809
RV wall thickness, cm	0,6 [0,58;0,65]	0,5 [0,49;0,6]	0,6 [0,5;0,64]	0,081
Maximum volume of RA ind., mm^3	28,4 [15,55;28,5]	25,1 [20,82;27]	24,9 [22,45;28,75]	0,743
Minimum volume of RA ind., mm^3	8,9 [7,1;10,9]	11,5 [8,78;14,97]	9,2 [6,75;12,3]	0,046*
Maximum gradient per PA, m/s	2,15 [1,98;2,45]	2,2 [1,8;2,8]	2,6 [1,95;2,9]	0,585
Diameter of PA trunk, cm	2,0 [1,9;2,1]	2,2 [2,0;2,3]	2,2 [2,1;2,3]	0,06
SPAP, mm Hg	27,5 [24,75;30,25]	30,0 [26,0;32]	34 [31,0;36,25]	<0,001* p_{I-III}=0,001* p_{II-III}=0,001*
mPAP, mm Hg	10,7 [10,0;14,07]	16,7 [13,8;23,10]	19 [12,7;23,1]	0,017* p_{I-II}=0,017* p_{I-III}=0,018*
TAPSE, cm	2,25 [2,18;2,38]	2,3 [2,1;2,6]	2,35 [2,1;2,5]	0,919
RV ESA ind., mm^2	5,3 [4,3;6,45]	6,0 [4,9;6,8]	5,5 [4,57;7,95]	0,654
RV EDA ind., mm^2	10,0 [8,85;11,65]	10,9 [9,7;12,5]	10,5 [9,0;13,05]	0,475
RV FAC, %	45 [36,0;47,4]	44 [39,1;51,5]	46,5 [39;51,95]	0,686
RV e', m/s	0,12 [0,1;0,15]	0,14 [0,11;0,17]	0,12 [0,11;0,14]	0,081
RV a', m/s	0,15 [0,14;0,18]	0,15 [0,12;0,18]	0,16 [0,14;0,2]	0,255
RV s', m/s	0,14 [0,12;0,14]	0,14 [0,12;0,15]	0,16 [0,14;0,17]	0,004* p_{I-III}=0,014* p_{II-III}=0,012*
RV E, m/s	0,45 [0,43;0,54]	0,55 [0,49;0,64]	0,55 [0,46;0,6]	0,041* p_{I-II}=0,036*
RV A, m/s	0,43 [0,38;0,47]	0,45 [0,4;0,51]	0,47 [0,4;0,5]	0,461
RV E/A	1,1 [0,97;1,2]	1,2 [0,92;1,5]	1,1 [1,0;1,3]	0,294
RV E/e'	4,05 [3,42;4,4]	4,0 [3,27;4,7]	4,12 [3,75;4,83]	0,657
RV DT, m/s	194 [182;220]	200 [162;227]	202 [170;224]	1,000
Tei index (PW)	0,21 [0,14;0,4]	0,2 [0,15;0,27]	0,21 [0,12;0,29]	0,803
Tei index (TDI)	0,3 [0,25;0,46]	0,33 [0,24;0,45]	0,38 [0,29;0,52]	0,308
RV e'/a'	0,8 [0,6;0,95]	0,8 [0,7;1,0]	0,7 [0,6;0,8]	0,029* p_{II-III}=0,025*
RV GLS, %	19,63±7,72	22,64±5,44	27,4±5,97	0,015* p_{I-III}=0,014*
RA GLS, %	30,07±8,98	26,72±9,47	35,13±8,37	0,003* p_{II-III}=0,002*
RA EF, %	61 [49,5;70]	50 [44,75;62]	59 [49,5;73,0]	0,028*

Note: data are presented as M±SD or Me [Q1;Q3], depending on type of value distribution of the indicator under study. * — differences in the indicators are statistically significant (p<0,05).

Abbreviations: ind. — indexed rate, PA — pulmonary artery, RV — right ventricle, RV/LV — ratio of basal diameter of right ventricle to basal diameter of left ventricle, RV EDA — right ventricle end-diastolic area, RV ESA — right ventricle end-systolic area, the RV FAC — fraction shortening of the right ventricle, RA — right atrium, SPAP — systolic pulmonary artery pressure, mPAP — mean pulmonary artery pressure, RA EF — right atrium ejection fraction, RV GLS — right ventricle global longitudinal strain, RA GLS — right atrium global longitudinal strain, TAPSE — tricuspid annular plane systolic excursion.

Discussion

A small number of publications devoted to the study of the right heart in patients with COVID-19-associated pneumonia have been found in the literature available to us.

In the study of Szekeley Y, et al. (2020) [11] conducted a comprehensive assessment of the cardiovascular system in patients hospitalized with COVID-19. 100 patients were divided into 3 groups depending on the conducted respiratory support. Group 1 included patients without respiratory insufficiency, group 2 included patients with moderate respiratory insufficiency who received non-invasive respiratory support, group 3 included patients on mechanical ventilation. The study of Echo parameters of RV revealed no differences in the end-diastolic area of RV (p=0,85), end-systolic area of RV (p=0,45), RV shortening fraction (p=0,08), systolic excursion of tricuspidal annulus (p=0,63), peak s' of tricuspidal annulus (p=0,55), Tei index (p=0,73). Thus, in patients with different severity of the disease there were no significant differences in structural and functional RV parameters, which coincides with the results we obtained, except for the rate of peak s' of tricuspidal annulus. Taking into account that in our study there were no patients with an extremely severe course of COVID-19, nevertheless there were higher values of this parameter.

The study by Bursi F, et al. (2020) [12] included 49 patients with COVID-19. The authors retrospectively analyzed Echo parameters of the right heart in surviving (n=33) and deceased patients (n=16). SPAP in the group of deceased patients corresponded to moderate pulmonary hypertension (39±11 mm Hg), but this index did not differ significantly from the values in the group of deceased patients (30±7 mm Hg, p=0,06). In addition, the deceased group showed RV dysfunction compared with the group of deceased patients, these are low TAPSE values of 18±3 mm (vs 21±5 mm, p=0,033) and RV GLS of 12±4% (vs 17±5%, p=0,008). In the present study, SPAP in patients with severe COVID-19-associated pneumonia (group III) was moderately elevated. It is interesting to note that in comparison with the study of Bursi F, et al. (2020), despite the severe course of COVID-19-associated pneumonia, there was no decrease in TAPSE in patients, and the GLS index increased, which can be interpreted as part of the development of RV hypercontractility.

Golukhova EZ, et al. (2020) [4] conducted an Echo study in 109 patients with COVID-19 in order to assess the right heart dysfunction in different variants of the course of COVID-19-associated pneumonia. 2 groups were identified — with stable (n=86) and progressive (n=23) COVID-19. In the group with progressive disease were observed moderate right heart dilatation: increase basal RV diameter — 44,3±6,6 mm (vs of 40,3±4,9 mm, p=0,002), the average RV diameter — 37,7 mm (vs 34,2±6,1 mm, p=0,032), indexed RA volume — 32,1 [26,3;42,2] ml/m^2 (vs 24,7 [19,4;33,7] ml/m^2, p=0,009). A number of functional indices of RV did not differ and remained within normal values: RV shortening fraction (p=0,937), TAPSE (p=0,167), Tei index (PW) (p=0,672), Tei index (TDI) (p=0,755). The only parameter that showed significant differences in the intergroup comparison was RV GLS (21,7% in group 1, 16,9% in group 2, p=0,001). The authors concluded that a decrease in this index may correspond to early systolic RV dysfunction.

The study of Li Y, et al. (2020) [13], in which RV GLS was studied as a possible predictor of death in patients with COVID-19, showed high prognostic value (cut-off point 23%, Se 94,4%, Sp 64,7%).

Myocardial GLS determination is a sensitive method for determining early systolic dysfunction of the right and left heart [14, 15]. At the same time, most methods of determining the RV functional state may not provide the necessary information on its systolic or diastolic function. Determination of RV GLS in combination with parameters of pulmonary hemodynamics [16] is the most valuable method for determination of RV function.

The main difference between the above studies and the present one is the examination of patients with an extremely severe and progressive course of COVID-19. Our study enrolled patients with a lung parenchyma damage volume of up to 75%. As a result, it was found not a decrease, but an increase in GLS of RV and RA in patients in the group with severe COVID-19-associated pneumonia. Most probably, this phenomenon is caused by compensatory RV hyperfunction in response to acutely increased postload. This was indirectly confirmed by an increase in NT-proBNP. It is known that NT-proBNP level increases as a result of atrial and/or ventricular distension, or increased myocardial postload, even when LV EF is normal [17].

Conclusion

This study demonstrates that patients with stable but severe COVID-19-associated pneumonia did not show Echo signs of right heart dysfunction. This is most likely due to the short duration of the disease at the time of investigation, absence of cardiomyocyte damage, which is confirmed by normal troponin I level and preserved LV EF in all examined patients. It was found that increase of RV GLS index (hypercontractility) with increasing severity of COVID-19-associated pneumonia and

its direct correlation with SHOCS-COVID scale may indicate the presence of compensatory RV hyperfunction in patients in response to acute post-loading. The NT-proBNP dynamics and its relationship with C-reactive protein (the main indicator of SVR severity) may indicate the presence of myocardial stress in systemic inflammatory response in COVID-19. These results can potentially be used in clinical practice in the comprehensive assessment of the cardiovascular system in patients with COVID-19-associated pneumonia outside the intensive care unit.

Relationships and Activities: none.

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