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Evaluation of echocardiographic parameters in military patients with post-Covid cardiac syndromes

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Abstract: The new coronavirus infection is dangerous not only due to its detrimental effect on the respiratory tract, but also due to the development of serious complications from the cardiovascular system based on the systemic inflammatory process. Today, it is relevant to study the impact of the complicated course of COVID-19 on the cardiovascular system—in particular, the study of indicators without initial changes in echocardiography.

Keywords: post-Covid cardiac syndrome (PCCS), chronic persistent myoendocarditis (CPME), chronic fibrinous pericarditis (CFP), chronic heart failure (CHF), ejection fraction (EF), shortening fraction (ShF), stroke volume (SV), end diastolic volume (EDV), end systolic volume (ESV).

Introduction. A comparison with the data of foreign authors on the development of pulmonary hypertension and remodeling of the heart chambers after COVID-19 should include the natural involvement of the right ventricle (predominantly biventricular nature of heart failure). The mechanism that causes the development of heart failure in post-Covid syndrome includes the direct effect of the virus on cells, a decrease in the level of active ACE2 receptors, inflammation and an immunological response, affecting the structural integrity of the myocardium, pericardium and cardiac conduction system, death of cardiomyocytes and fibrofatty replacement of desmosomal proteins, which play an important role in intercellular adhesion and contribute to the development of severe systolic and diastolic dysfunction of the LV [5,7]. Pulmonary hypertension can also develop, and as a result - dilatation of the right chambers of the heart, diffuse damage to the alveoli with subsequent organization and focal fibroproliferative diffuse damage to lung tissue. Foci of proliferation of myofibroblasts and fibrocystic degeneration of the lungs are often observed. This process may be triggered by proinflammatory cytokines and further predispose to bacterial colonization and subsequent secondary infection [6,8].

The aim. To assess the functional state of the heart in military patients with post-Covid cardiac syndromes using echocardiography.

Material and methods. Clinical examination of patients was carried out at the bases of the Military Medical Academy (Military Hospital). The study included 170 patients with cardiac post-Covid syndromes (average age 42±4.2 years), which developed 2-3 weeks after suffering from COVID-19 at various stages, pneumonia with lung damage. Lung involvement up to 70% was noted in 12 (7.0%), lung involvement up to 40% in 45 (26.5%) and lung involvement up to 15% in 58 (34.0%)

patients. And in 55 (32.3%) patients there was no lung damage. The diagnosis of COVID-19 was confirmed by PCR. Patients who had no previous pathology of the cardiovascular system.

The control group consisted of 35 practically healthy patients. Of these, n=164 (80%) men with an average age of 43±2.8 years and n=41 (20%) women with an average age of 40±2.0. Of the total number of patients examined, 35 developed chronic fibrinous pericarditis (CFP) (average age 40±2.8 years), 57 had arterial hypertension combined with dyslipidemia (average age 38±2.3 years), and 65 had chronic persistent myoendocarditis (CPME) (average age 28±3.4 years) and 18 with chronic heart failure (CHF) (average age 48±3.2 years).

Table №1

Echocardiography results in the study groups

Indicators	CFP (n=35)	AH+ dyslipidemia (n=57)	CHF (n=18)	CPME (n=65)	Control group (n=35)
Sinotubular connection of the aorta, mm	32,0±5,60	36,0±4,80*	40,0±6,0*	42,0±3,30**	28,0±2,80
LA volume index, ml	30,0±2,30	27,0±3,30	40,0±4,20**º	38,0±4,0*º	26,0±3,0
LV EDD, mm	42,0±4,30	45,0±4,50	58,0±3,80** º	54,0±4,70*º	40,0±3,20
LV ESD, mm	31,0±3,30	26,0±2,80	44,0±4,20*º	40,0±2,90*º	25,0±2,20
SWTh, mm	8,0±1,20	11,50±2,20*	8,0±1,0	7,0±1,30	8,0±1,50
PWTh, mm	8,0±1,30	12,0±1,50*º	8,0±1,70	7,0±1,30	8,0±0,80
Right ventricular wall, mm	4,20±0,60	4,0±0,80	5,50±1,10	4,0±0,70	3,50±0,50
Index RA volume, ml	28,0±5,30	32,0±5,80*	40,0±3,50** ºº	42,0±3,0**ººº	25,0±2,30
Sav, sm^2	3,20±0,20*	3,6±0,70*	4,2±0,50** ºº	4,5±0,20** ºº	2,40±0,40
Left ventricular myocardial mass, gr	165,±6,30	232±8,50***ºº	195±7,30**ºº	160±6,30	150±4,60
Index left ventricular myocardial mass, gr	69,0±5,30	122±4,30**ºº	115±3,50**ºº	62,0±4,80	54,0±3,30
LV EDV, ml	72,0±4,20	80,0±7,20	132,0±6,80***ººº	120±7,30**ºº	68,0±6,30

LV ESV, ml	25,0±2,50	28,0±4,20	69,0±4,50 ***ººº	55±3,70** *ººº	22,0±2,20
EDD basal part of the right ventricle, mm	38,0±4,20	40,0±3,80	48,0±5,30 ** º	44,0±5,0*	34,0±2,90
EDD middle part of the right ventricle, mm	36,0±3,30	38,0±4,20	46,0±3,60 **º	42,0±2,80* *	32,0±2,0
SV, ml	47,0±6,30	52,0±4,20	63,0±3,70 *º	65,0±4,30* º	46,0±3,0
EF,%	65,0±4,20	65,0±5,20	47,0±3,30 **ºº	54,0±4,50*	67,0±3,8
SHF,%	26,0±2,50	38,0±2,70ºº	24,0±3,30 **	25,0±3,0	37,0±3,30
RWTh, sm	0,38±0,03	0,52±0,05* *ºº	0,27±0,01	0,26±0,07	0,40±0,05
VCI, mm	18,0±2,30	19,0±2,0	23,0±2,80	22,0±3,0	16,0±1,50
MV peak E, m/s	53,0±6,30	55,0±5,80	74,0±7,20	68,0±4,8	60,0±5,30
MV peak A, m/s	69,0±6,30	74,0±8,20	55,0±5,30	53,0±3,8	42,0±3,0
E/A m/s	0,78±0,20	0,68±0,30	1,32±0,40	1,25±0,6	1,3±0,80
SMV, sm^2	4,30±1,20	5,20±2,20	6,20±2,40	5,0±2,0	4,5±1,80
TV peak E, m/s	45,0±3,50	53,0±5,50	39,0±4,20	52,0±6,3	57,0±5,80
TV peak A, m/s	53,0±4,50	48,0±3,80	52,0±6,30	40,0±3,2	42,0±3,70
TV E/A	0,80±0,30	1,10±0,50	0,7±0,10	1,1±0,80	1,2±0,6
Aorta, m/s	122,0±5,90	170±7,30* *ºº	185±8,30 **ºº	142,0±4,80	133,0±5,2
GP, mmHg	3,50±1,0	8,20±2,20* *ºº	9,30±3,30 **ºº	5,2±2,20	4,0±1,50
PA, m/s	96,0±3,30	102,0±5,60	169±7,20 **ºº	148±6,20* *ºº	98,0±2,50
GP, mmHg	2,20±1,0	2,60±0,80	4,50±2,0	4,0±1,80	2,4±1,0
Free fluid in the pericardial cavity, ml	290±8,60** *ººº	59,0±7,20	170±6,80 ***ººº	85,0±7,30* *	45,0±5,50
PASP mmHg	24,0±2,50	28,0±3,20	38,0±2,80 **º	35,0±3,30* º	25,0±2,20

*Note: *p<0.05, **p<0.01, ***p<0.001 – significant difference with the control group. ºp<0.05, ººp<0.01, ºººp<0.001 – significance of the difference between groups*

A comparative assessment of the echocardiographic characteristics of heart parameters in patients with post-Covid cardiac syndromes was carried out. The results of the work indicate that in patients with CPME, the expansion of the aorta

and sinotubular junction was noted in comparison with the group of patients with CPME by 23.80% (p <0.01). The expansion and thickening of the aortic walls is associated with the development of aortitis after a coronavirus infection. The virus enters endothelial cells through the angiotensin-converting enzyme II receptor and activates T-helper cells, which trigger cell-mediated and humoral immune responses [3]. These effects ultimately lead to the development of endothelialitis and leukocytoclastic vasculitis, in addition to a third type of hypersensitivity reaction leading to aortitis [10]. The following echocardiographic measurement of the chambers of the heart was the volume of the left atrium. The increase in the index volume of the left atrium in patients with CHF was greater in comparison with arterial hypertension combined with dyslipidemia by 32.50% (p <0.01), and the index volume of the right atrium in the group of patients with CPME compared with the group of patients with CAF more by 33.40% (p<0.01). When analyzing the indicators of the structural and functional state of the myocardium and central hemodynamics, assessed according to echocardiography, a significant decrease in the shortening fraction (SF%) in patients with CHF in comparison with the control group by 35.0% (p <0.01) was revealed, in patients with HPME by 32.43%. There was a trend towards a decrease in ejection fraction (EF%) in patients with CHF compared to the control group by 30% (p<0.01), and in patients with CPME it was lower by 19.40% (p<0.05). The index of left ventricular myocardial mass was significantly increased in patients with arterial hypertension combined with dyslipidemia in comparison with the control group by 64.65% (p<0.001), as well as in patients with CHF by 30.0% (p<0.001). 01). The volume of free fluid in the pericardial cavity in patients with CAF was significantly greater by 6.4 times (p <0.001) compared with the control group, and in patients with CHF there was also a significantly larger volume of fluid in the pericardium compared with the control group in 3. 7 times (p<0.001). The reasons for the increase in fluid volume arise due to autoimmune inflammatory processes in the pericardium [4]. The triggering factor is pathogens that trigger immune and inflammatory reactions and increase the release of cytokines and other bioactive substances. Since coronavirus is capable of causing a multisystem inflammatory process, with covid, heart structures are often involved in the pathological process. A large number of macrophages, neutrophils and other immune cells accumulate in the tissues of the cardiac sac [1,2,5].

The pulmonary artery systolic pressure (PASP) in patients with post-Covid cardiac syndromes turned out to be significantly higher in patients with CHF compared to the control group by 40.50% (p <0.01), and in the group of patients with CPME by 37.50 % (p<0.01).

Types of hypertrophy in patients with post-Covid cardiac syndromes

According to the results of the study, different types of myocardial hypertrophy were identified: concentric remodeling was noted in 28 (16.50%), concentric hypertrophy without dilatation in 15 (8.82%), concentric hypertrophy with dilatation in 6 (3.53%), eccentric hypertrophy without dilatation in 7 (4.20%).

The degree of dilatation of the left and right atrium in patients with post-Covid syndrome

The study revealed the development of dilatation of the heart chambers over a short period of time in patients who had suffered a coronavirus infection. In 30 (21.76%) there were signs of atrial dilatation of varying degrees: LA and RA dilatation grade 1 in 21 (19.0%), LA and RA dilatation grade 2 in 9 (14.0%).

Characteristics of right ventricular dilatation

According to our study, varying degrees of dilatation of the right ventricle were identified: degree 1 RV dilatation was noted in 24 (14.11%), degree 2 dilatation was detected in 14 (8.23%).

Left ventricular ejection fraction indicator

The study revealed that 102 (60%) had preserved ejection fraction (EF), 34 (20%) had a slight decrease in EF%, and 26 (13%) had a moderate decrease in EF%.

Characteristics of the degree of pulmonary hypertension in patients with post-Covid cardiac syndromes

n=56 (40%) had varying degrees of pulmonary hypertension (PH): PH 1st degree in n=34 (20%), PH 2nd degree in n=22 (13%).

Table№2

Characteristics of the state of the valve apparatus

Indicator	n/%
Aneurasmatic dilatation of the aortic walls	42/24,70
Aortic regurgitation 1st degree	29/17,0
Aortic regurgitation 2st degree	18/10,60
Mitral regurgitation 1st degree	53/31,20
Mitral regurgitation 2 st degree	26/15,30
Tricuspid regurgitation 1st degree	44/25,90
Tricuspid regurgitation 2 st degree	26/15,30
Pulmonary regurgitation 1 st degree	34/20
Pulmonary regurgitation 2 st degree	22/12,90

The mechanism of damage to the valve apparatus is due to the fact that ACE-2 is widely expressed in stromal fibroblasts of heart valves, especially aortic valves. The cytokine storm and subsequent effects of SARS-CoV-2 infection lead to valve damage, which may not be acutely evident due to the slowly progressive nature of valvular development [6,7].

Thus, from the data obtained it is clear that in patients with CHF and CPME there is a significant decrease in EF% and ShF% in comparison with the groups of

hypertension combined with dyslipidemia and with CFP. The increase in pulmonary artery systolic pressure was significantly higher in the listed groups, which contributes to the progression of dilatation of the RV, LA and RA.

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