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Post Covid-19 Evaluation of Patients With Cardiac Complaints By Using Echocardiography

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ABSTRACT

The aim of the study was to evaluate the presenting complaint, cardiovascular sequelae prevalence and type and demographic characteristics of patients who had no previous disease and had cardiac complaints after COVID-19, and to show the presence of cardiac sequelae objectively by evaluating every patient without chronic diseases by echocardiography and electrocardiography.

In this study, we evaluated symptomatic patients by using echocardiography.

In this study, there were included patients who recovered from COVID-19 disease and presented to the cardiology outpatient clinics. These patients were divided into two groups: those presenting within the first 30 days after recovery and those presenting between 30 and 60 days after recovery. Thirty patients who had not had COVID-19 before, who applied to the cardiology outpatient clinics, were selected as the control group.

The rate of dizziness, palpitations, chest pain and blood pressure dysregulation did not differ significantly between group I(patients presenting within 30 days after recovery), group II(patients presenting between 30 and 60 days after recovery), and group III(control group) ($p > 0.05$). Dyspnea rate in group I was significantly higher than group II and group III. There was a difference between groups in left ventricular ejection fraction. In group III, sPAP ≥ 35 mmHg ratio was significantly lower than group I and group II ($p < 0.05$). sPAP ≥ 35 mmHg ratio did not differ significantly ($p > 0.05$) between group I and group II.

While dyspnea was an important complaint in the first periods, it decreased over time. In addition, it was observed that right ventricular functions and left ventricular diastolic functions improved over time. It is noteworthy that there are more patients with pericardial effusion compared to the control group, which shows us that the inflammation process is continuing.

Keywords: Covid-19, Echocardiography, Cardiac

Introduction

Coronavirus 2019 disease (COVID-19) is an infection of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (1). COVID-19 disease has been continuing to cause serious mortality and morbidity worldwide, since WHO declared the COVID -19 outbreak as a pandemic (2). Cardiovascular involvement is one of the most important causes of mortality and morbidity (3). It was shown that covid-19 increases morbidity and mortality in patients with chronic heart disease (3). Studies indicated that COVID-19 indirectly or directly causes arrhythmia, thromboembolism, myocarditis(3).

In echocardiographic studies about patients being infected with active sars-cov-2, especially left ventricle systolic functions are preserved, while diastolic dysfunction occurs in the left ventricle (4,5). Right ventricular dysfunction was indicated to occur because of increased vascular resistance as a result of lung involvement (5). Particularly, increased right atrium volume and IVC diameter were seen in elevated systemic pulmonary artery pressure (5,6). Pericardial effusion has been detected in some patients (5,6). On the other hand, it was suggested that the severity of right ventricular involvement is associated with mortality (6).

Although the effect of Covid-19 disease on the cardiovascular system was shown in studies, studies about the patients recovered from COVID-19 are very limited (7,8). Studies suggest that serial measurements of respiration function should be measured with thorax CT when echocardiography is required (9,10). Our aim is to evaluate the complaints in postcovid patients who applied to the cardiology outpatient clinic together with the prevalence, the type of cardiovascular sequelae and demographic characteristics. We evaluated each patient without chronic disease by using echocardiography and ECG in order to show the presence of cardiac sequelae objectively. Also, we used other methods if indicated.

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Material and Method

Our randomized single-blind study was selected from 646 patients who admitted to our center's cardiology outpatient clinics between December 2020 and February 2021, and selected from 118 patients who had recovered from COVID-19 disease 30-60 days ago, 30 patients (0-30 days) and 30 patients (30-60 days) without comorbid disease and 30 control groups selected from 528 patients who applied to the outpatient clinic.

Heart failure, diabetes, hypertension, chronic obstructive pulmonary disease, interstitial lung disease, asthma, pulmonary hypertension, pulmonary embolism, coronary artery disease, valvular heart disease, cardiomyopathy, chronic kidney disease, obstructive sleep apnea syndrome, under 18 and over 65 years of age, patients who did not give the consent, pregnant patients and oncological patients were excluded from the study.

Van Yüzüncü Yıl University Ethics Committee approved the ethics committee approval.

Echocardiographic examination after at least 15 minutes of rest, in the left lateral position (2-dimensional, M-mode, Doppler echocardiography) using the Vivid E9 device and X5-1 transthoracic probe (Vivid 9 Pro, General Electric Medical Systems, Milwaukee, Wisconsin) through parasternal and apical windows. Echocardiography was performed on each participant following the standard images and techniques (parasternal long axis, parasternal short axis, apical four chambers and apical two chambers) included in the European Echocardiography Society's (EEC) guidelines (17). The person performing the echocardiogram is blind to when patients have had the COVID-19 infection.

In echocardiography, measurements were taken for the left and right ventricle. For this purpose, using the parasternal long axis, left ventricular end-diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD), interventricular septum (IVS) and posterior wall thickness (PWD), aortic root diameter (AoD) and end-systolic left atrial diameter (LA) measured in centimeters. Left ventricular ejection fraction (LVEF) was measured in apical 2 chambers and apical 4 chambers using Simpson method. The mitral peak early filling rate (E wave), late diastolic filling rate (A wave), E / A ratio, deceleration time (Dec. T) and early diastolic mitral lateral annular velocity (e ') were measured in the apical 4 spaces. Pulmonary artery diameter (PA) was measured in the parasternal short axis.

In apical four chamber view; right ventricle diameters (D1, D2 and D3), right atrium area and volume, tricuspid annular plane systolic excursion (TAPSE) with M Mode, systolic tricuspid lateral annular velocity (TDI S '), fractional area change (RV-FAC), systolic pulmonary artery pressure (sPAP) was measured using the tricuspid regurgitation peak velocity. It was decided whether pericardial effusion was present or not by examining all standard images.

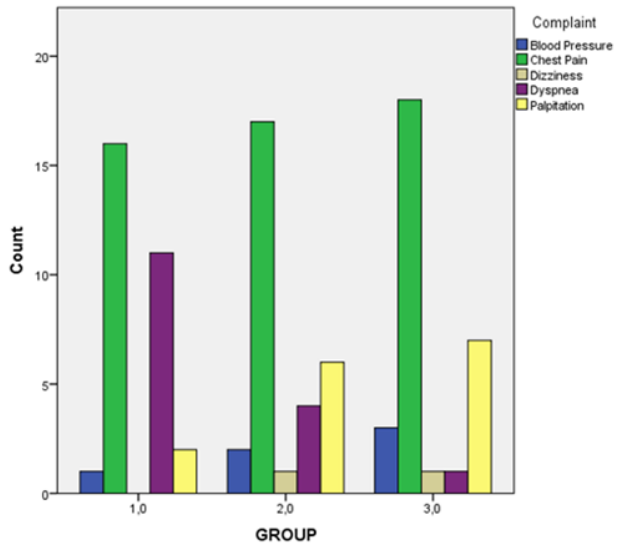
The pericardial space contains 10-50 ml of fluid. Pericardial effusion consists of exudate caused by inflammation or transudate caused by decreased absorption of pericardial fluid. Pericardial effusion on echocardiography was classified as mild (<10 mm), medium (10-20 mm) and wide (> 20 mm) in measurements at diastole.

After 30 minutes of rest, 12-lead (Nihon-Kohden brand) electrocardiography (ECG) recording of the participants was obtained. Measurements were made at room temperature (20 °C-24 °C), with at least 2 hours of fasting, no smoking for the last 24 hours, no alcohol consumption in the last day, and no heavy physical activity.

In our study, 12-lead ECG was recorded at a paper speed of 25 mm / s in the supine position. All ECGs were scanned in the scanner and the data was transferred to a personal computer to reduce possible errors and used for 400% magnification with Adobe Photoshop software. An average of three values were calculated for each derivation. PR distance was calculated as the time (msec) from the onset of the P wave to the R wave. The QRS complex was measured from the beginning of the Q wave to the end of the S wave. The QT interval was measured from the beginning of the QRS complex to the end of the T wave, and Bazett formula was used for the corrected QT for the heart rate. ($QTc = QT\sqrt{}$ (R-R interval).

Palpitations are defined as being aware of the person's heartbeat. The duration and time of the complaints were questioned. Blood tests were taken from the patients, rhythm holter was requested after secondary causes were excluded. VES, AVNRT, SVE and sinus tachycardia were detected in patients with rhythm holter. EPS was applied to patients who were found to have AVNRT and ST as a result of rhythm holter.

The blood pressure dysregulation was determined according to the blood pressure measurement and the patient's statement during the examination, and according to the blood pressure holter report. Holter results were evaluated according to the 2017 guidelines of the American Heart



Graph 1: patients' complaints
Group l: patients presenting within 30 days after recovery
Group ll: patients presenting between 30 and 60 days after recovery
Group lll: people who were not previously infected with SARS-CoV-2

Association. Systolic <120 mmHg and diastolic <80 mmHg normal blood pressure, systolic 120-129 and diastolic <80 mmHg elevated blood pressure, systolic 130-139 mmHg or diastolic 80-89 mmHg Stage 1 hypertension, systolic ≥ 140 mmHg or diastolic ≥ 90 mmHg Stage 2 was evaluated as hypertension and treatment was initiated.

We made the angina classification according to the 2013 ESC stable angina clinical classification.

Angina has 3 features:

1) Substernal pressure or feeling of heaviness that spreads to the chin, arms, back, which lasts for 2 - 15 minutes

2) Occurrence with exercise, emotional stress or sympathetic discharge that will increase myocardial oxygen demand

3) Relieving with rest or sublingual nitroglycerin

In typical angina, the effort test was performed first, if the effort test was positive, catheter angiography was recommended, if the effort test was negative, CT angiography was performed. If the CT angiography result was positive, catheter angiography was performed. In atypical angina, the same pattern was followed.

Treatments in non-angina were arranged according to echo findings.

Exertional dyspnea is the shortness of breath. A number of factors can cause exertional dyspnea, but they are usually related to insufficient tissue oxygenation by the blood. Patients with dyspnea were evaluated by echocardiography and were referred to respiratory clinic for evaluation with lung function test.

Statistical Analyses: Mean, standard deviation, median, lowest, highest, frequency and ratio values were used in the descriptive statistics of the data. The distribution of variables was measured with the Kolmogorov Simirnov test. ANOVA (Tukey test), independent sample t test, Kruskal-Wallis, Mann-Whitney U test were used in the analysis of quantitative independent data. Chi-square test was used in the analysis of qualitative independent data, and Fischer test was used when Chi-square test conditions were not met. SPSS 27.0 program was used in the analyzes.

Results

The age of the patients did not differ significantly between group I, group II, and group III (p> 0.05).

The gender distribution between group I, group II and group III did not show difference significantly (p> 0.05). There is no significant difference in terms of blood pressure ratio between group I, group II and group III (p> 0.05). (Table 1)

Smoking rate in group III was significantly higher than in group I and group II (p <0.05). The rate of smoking did not differ significantly between group I and group II (p> 0.05). (Table 1) The rate of dizziness, palpitations, chest pain and blood pressure dysregulation did not differ significantly between group I, group II, and group III (p> 0.05). Dyspnea rate in group I was significantly higher than group II and group III. Dyspnea rate did not differ significantly between group II and group III (p> 0.05). (Table 1) There is no significant difference in the types of angina between group I, group II, and group III (p> 0.05). (Table 1) Pericardial effusion rate did not differ significantly between group I and group II (p> 0.05). (Table 1) The rate of hospitalization did not differ significantly (p> 0.05) between group I, group II and group III. (T able 1)

In Graph 1, there are complaints at the admission to the cardiology polyclinic according to the groups.

There is no significant difference in AR, LA, LVEDD, LVESD, IVS, E, A, Dec.T, e' lateral, E/A, E/e', RV d2, RA, TDI S', sPAP, HR, PR, QRS, QT, QTc values between group I, group II, and group III (p> 0.05). (Table 2)

Table 1: The Demographic Characteristics of The Consultations

		Group I	Group II	Group III	p	
		Mean±s.d./n-%	Mean±s.d./n-%	Mean±s.d./n-%		
Age		41,4 ± 13,9	46,1 ± 10,6	43,2 ± 10,2	0,306	A
Sex	female	13 43,3%	14 46,7%	15 50,0%	0,875	X^2
	Male	17 56,7%	16 53,3%	15 50,0%		
Height (cm)		170,4 ± 9,7	167,8 ± 9,2	167,9 ± 9,1	0,606	A
Weight (kg)		76,7 ± 16,6	77,4 ± 14,9	74,6 ± 13,0	0,788	A
BMI (kg/m2)		26,2 ± 4,5	27,3 ± 5,3	26,2 ± 2,5	0,584	A
Blood Pressure	≥140 mmHg	0 0,0%	1 3,3%	2 6,7%	p>0.05	X^2
Smoking		2 6,7%	2 6,7%	10 33,3%	0,004	X^2
Complaints						
Dizziness		0 0,0%	1 3,3%	1 3,3%	p>0.05	X^2
Palpitation		2 6,7%	6 20,0%	7 23,3%	0,166	X^2
Dyspnea		11 36,7%	4 13,3%	1 3,3%	0,003	X^2
Chest Pain		16 53,3%	17 56,7%	18 60,0%	0,873	X^2
Blood Pressure disregulation		1 3,3%	2 6,7%	3 10,0%	p>0.05	X^2
Angina	Type I	2 12,5%	1 6,3%	6 33,3%	0,055	X^2
	Type II	0 0,0%	6 37,5%	3 16,7%		
	Type III	14 87,5%	9 56,3%	9 50,0%		
pericardial effusion	(-)	23 76,7%	26 86,7%		0,317	X^2
	(+)	7 23,3%	4 13,3%			
Hospitalization	(-)	27 90,0%	25 83,3%		0,448	X^2
	(+)	3 10,0%	5 16,7%			
A ANOVA (Tuke test) / X^2 Chi square test						

BMI: body mass index t Independent sample
Type l: Typical angina, Type ll: Atypical angina, Type lll: Non-anginal chest pain
Group l: patients presenting within 30 days after recovery
Group ll: patients presenting between 30 and 60 days after recovery
Group lll: people who were not previously infected with SARS-CoV-2

In group III, RV d1 value and RV d2 value were significantly lower than group I and group II ($p < 0.05$). The RV d1 value and RV d2 value did not differ significantly between group I and group II ($p > 0.05$). (Table 2)

The RV-FAC value in group I was significantly lower than group III ($p < 0.05$). RV-FAC values in group II did not differ significantly ($p > 0.05$) from group I and group III. (Table 2)

TAPSE value in group I was significantly lower than group II ($p < 0.05$). TAPSE value in group III did not differ significantly ($p > 0.05$) from group I and group II. (Table 2)

The % EF value in group III was significantly higher than in group I and group II ($p < 0.05$). % EF value in group II was significantly higher than group I ($p < 0.05$). (Table 2)

In group III, IVC; INSP/EXP> %50 ratio was significantly higher than group I and group II ($p < 0.05$). The ratio of IVC; INSP/EXP> %50 ratio did not differ significantly ($p > 0.05$) between group I and group II. (Table 2)

In group III, sPAP ≥ 35 mmHg ratio was significantly lower than group I and group II ($p < 0.05$). sPAP ≥ 35 mmHg ratio did not differ significantly ($p > 0.05$) between group I and group II. (Table 2)

HR, PR, QRS, QT, QTc values did not differ significantly ($p > 0.05$) between group I, group II, and group III. (Table 2)

The age and gender distribution of the patients in the group with and without dyspnea did not differ significantly ($p > 0.05$). Height and BMI values did not differ significantly ($p > 0.05$) in the groups with and without dyspnea. The weight in the

Table 2: The Ratio of The Number of İnpatients Per Period To The Number of Patients Consulted

		Group I			Group II			Group III			p	
		Mean±s.d./n-%			Mean±s.d./n-%			Mean±s.d./n-%				
Aortic root (cm)		2,45	±	0,32	2,54	±	0,35	2,36	±	0,26	0,100	K
LA (cm)		2,98	±	0,43	3,08	±	0,40	2,96	±	0,28	0,457	A
LVEDD (cm)		4,62	±	0,43	4,51	±	0,70	4,55	±	0,41	0,834	K
LVESD (cm)		3,26	±	0,39	3,18	±	0,59	3,18	±	0,22	0,454	K
IVC (cm)		0,90	±	0,16	0,99	±	0,19	0,99	±	0,12	0,087	K
PW (cm)		0,75	±	0,10	0,92	±	0,47	0,82	±	0,10	0,022	K
PA (cm)		2,00	±	0,27	2,01	±	0,20	1,98	±	0,16	0,944	K
E (cm/s)		0,75	±	0,19	0,72	±	0,13	0,79	±	0,08	0,150	A
A (cm/s)		0,70	±	0,24	0,68	±	0,18	0,66	±	0,09	0,873	K
Dec. T (msec)		232,7	±	64,7	230,8	±	54,3	223,9	±	26,5	0,765	K
e' lateral (cm/s)		0,11	±	0,03	0,11	±	0,02	0,12	±	0,02	0,051	K
E/A ratio		1,13	±	0,34	1,12	±	0,34	1,20	±	0,15	0,538	A
E/e' ratio		7,31	±	2,15	6,34	±	1,64	6,59	±	1,26	0,206	K
RV d1 (cm)		3,79	±	0,52	3,72	±	0,51	3,39	±	0,35	0,000	K
RV d2 (cm)		3,17	±	0,58	3,19	±	0,54	3,03	±	0,31	0,359	K
RV d3 (cm)		5,46	±	0,58	5,50	±	0,53	5,11	±	0,24	0,000	K
RV-FAC (%)		47,4	±	8,4	49,3	±	9,1	50,9	±	3,7	0,045	K
TAPSE (cm)		2,19	±	0,34	2,44	±	0,31	2,31	±	0,26	0,015	K
RA (ml/m2)		37,8	±	12,2	37,9	±	11,8	33,5	±	8,1	0,207	A
TDI S' (cm/s)		14,4	±	2,2	14,3	±	2,1	14,0	±	1,2	0,866	K
sPAP (mmHg)		33,0	±	6,2	31,6	±	5,4	27,5	±	2,1	0,431	K
EF (%)		64,8	±	2,7	63,5	±	2,5	66,2	±	1,9	0,000	K
IVC; INSP/EXP> %50*	(-)	10		33,3%	8		26,7%	2		6,7%	0,035	X^2
	(+)	20		66,7%	22		73,3%	28		93,3%		
sPAP ≥ 35 mmHg	(-)	22		73,3%	25		83,3%	30		100,0%	0,012	X^2
	(+)	8		26,7%	5		16,7%	0		0,0%		
HR, bpm		78,2	±	11,2	80,1	±	14,0	80,7	±	13,1	0,816	K
PR, msec		151,3	±	18,9	152,0	±	22,0	150,0	±	21,4	0,864	K
QRS, msec		88,8	±	13,5	87,8	±	8,7	89,8	±	13,5	0,739	K
QT, msec		356,0	±	28,2	355,2	±	25,4	352,7	±	30,6	0,920	K
QTc, msec		402,6	±	21,4	407,3	±	17,7	404,7	±	19,2	0,769	K

A ANOVA (Tuke test)/K Kruskal-wallis (Mann-whitney u test) / X^2 Chi-square test

LA left atrial, LVEDD left ventricular end diastolic diameter, LVESV left ventricular end systolic diameter, IVS interventricular septum, PW posterior wall diameter, PA pulmonary artery, Dec. T deceleration time, RV d1-d2-d3 right ventricle diameters, RV-FAC right ventricular fractional area change, TAPSE tricuspid annular plane systolic excursion, RA right atrial, TDI S' tissue Doppler imaging systolic wave S' velocity, sPAP systolic pulmonary artery pressure, IVC inferior vena cava, INSP/EXP inspiration / expiration, HR heart rate *IVC; INSP/EXP> %50, collapse of IVC during inspiration or not

group with dyspnea was significantly higher ($p < 0.05$) than the group without dyspnea. The ratio of TA in the group with and without dyspnea did not differ significantly ($p > 0.05$). The rate of smoking did not differ significantly ($p > 0.05$) in the group with and without dyspnea. P effusion rate was significantly lower in the group with dyspnea than in the group without dyspnea ($p < 0.05$). The rate of hospitalization in the group with and without dyspnea did not differ significantly ($p > 0.05$). (Table 3)

In the group with and without dyspnea, AR, LA, LVEDD, LVESD, IVS, E, PW, A, Dec. T, e'lateral, E/A, E/e ', RV d1, RV d2, RV d3, RA,

Table 3: There Are Departments İn Both Periods That Are Consulted

		Dyspnea (-)			Dyspnea (+)			p	
		Mean ±s.d./n-%			Mean ±s.d./n-%				
Age		43,4	±	12,4	44,9	±	13,1	0,675	t
Sex	female	22		48,9%	5		33,3%	0,294	X^2
	Male	23		51,1%	10		66,7%		
Height (cm)		167,1	±	9,4	172,6	±	8,7	0,078	t
Weight (kg)		73,1	±	16,3	84,0	±	11,9	0,034	t
BMI (kg/m2)		25,9	±	5,2	28,2	±	3,8	0,162	t
Blood Pressure ≥140 mmHg		1		2,2%	0		0,0%	1,000	X^2
Smoking		3		6,7%	1		6,7%	1,000	X^2
pericardial effusion	(-)	34		75,6%	15		100,0%	0,034	X^2
	(+)	11		24,4%	0		0,0%		
Hospitalization	(-)	39		86,7%	13		86,7%	1,000	X^2
	(+)	6		13,3%	2		13,3%		

t Independent Samples T Test / X^2 Chi-square test

TDI S', sPAP, TAPSE, RV-FAC, HR, PR, QRS, QT, QTc, % EF values did not differ significantly (p> 0.05). (Table 4)

The ratio of IVC; INSP/EXP> %50 in the group with dyspnea was significantly lower than the group without dyspnea (p <0.05). The rate of sPAP ≥ 35 mmHg in the group with dyspnea was significantly lower than the group without dyspnea (p <0.05). (Table 4)

HR, PR, QRS, QT, QTc values did not differ significantly (p> 0.05) in the group with and without dyspnea. (Table 4)

The age and gender distribution of the patients in the group with and without pericardial effusion did not differ significantly (p> 0.05). Height, weight, and BMI values did not differ significantly (p> 0.05) in the group with and without pericardial effusion. The ratio of TA in the group with and without pericardial effusion did not differ significantly (p> 0.05). The rate of smoking did not differ significantly (p> 0.05) in the group with and without dyspnea. The rate of hospitalization did not differ significantly (p> 0.05) in the group with and without pericardial effusion. (Table 5)

The rate of dizziness, palpitations, Chest Pain and Blood Pressure disregulation did not differ significantly (p> 0.05) in the group with and without pericardial effusion. The rate of dyspnea was significantly lower in the group with pericardial effusion than in the group without pericardial effusion (p <0.05). (Table 5)

In the group with and without pericardial effusion, AR, LA, LVEDD, LVESD, IVS, E, PW, A, Dec. T, E/A, E/e ', RV d1, RV d2, RV d3, RA , TDI S', sPAP, TAPSE , RV-FAC, HR, PR, QRS, QT, QTc,% EF values did not differ significantly (p> 0.05). (Table 6)

e' lateral value in the group with pericardial effusion was significantly lower than the group without pericardial effusion (p <0.05). In the group with pericardial effusion, RA was significantly higher than the group without pericardial effusion (p <0.05). (Table 6)

The ratio of IVC; INSP/EXP> %50 in the group with and without pericardial effusion did not differ significantly (p> 0.05). In the group with pericardial effusion, the sPAP ≥ 35 mmHg ratio was significantly lower than the group without pericardial effusion (p <0.05). (Table 6)

HR, PR, QRS, QT, QTc values did not differ significantly (p> 0.05) in the group with and without pericardial effusion. (Table 6)

In Figure 1, it is shown that pericardial effusion decreased 1 month after the medical treatment of the patients with pericardial effusion.

Discussion

We indicated that patients without a chronic disease who survived the COVID-19 infection applied especially to the cardiology outpatient clinic with exertional dyspnea and type 3 angina in the first 30 days. On echocardiography, we found deterioration in the parameters of the right ventricle and of the left ventricular diastolic function, and pericardial effusion. We found that the echocardiographic findings and complaints of

Table 4: There are Reasons For Consultation In Both Periods

		Dyspnea (-)			Dyspnea (+)			p	
		Mean ±s.d./n-%			Mean ±s.d./n-%				
Aortic root (cm)		2,48	±	0,35	2,53	±	0,28	0,661	t
LA (cm)		2,99	±	0,40	3,15	±	0,45	0,183	t
LVEDD (cm)		4,51	±	0,61	4,73	±	0,47	0,345	m
LVESD (cm)		3,15	±	0,51	3,43	±	0,37	0,062	m
IVC (cm)		0,94	±	0,18	0,96	±	0,18	0,480	m
PW (cm)		0,85	±	0,40	0,79	±	0,10	0,831	m
PA (cm)		1,98	±	0,23	2,08	±	0,27	0,232	m
E (cm/s)		0,72	±	0,16	0,77	±	0,16	0,342	t
A (cm/s)		0,68	±	0,18	0,73	±	0,30	0,952	m
Dec. T (msec)		229,7	±	58,4	238,0	±	63,2	0,641	t
e' lateral (cm/s)		0,11	±	0,02	0,11	±	0,03	0,816	m
E/A ratio		1,11	±	0,32	1,17	±	0,38	0,765	m
E/e' ratio		6,57	±	1,78	7,59	±	2,32	0,209	m
RV d1 (cm)		3,70	±	0,44	3,92	±	0,66	0,150	t
RV d2 (cm)		3,12	±	0,58	3,37	±	0,44	0,133	t
RV d3 (cm)		5,47	±	0,53	5,51	±	0,63	0,905	m
RV-FAC (%)		48,8	±	9,6	47,0	±	5,6	0,719	m
TAPSE (cm)		2,31	±	0,35	2,33	±	0,35	0,899	t
RA (ml/m2)		36,5	±	12,4	41,9	±	9,4	0,129	t
TDI S' (cm/s)		14,4	±	2,1	14,4	±	2,2	0,849	m
sPAP (mmHg)		31,4	±	5,9	34,2	±	5,3	0,182	m
EF (%)		64,3	±	2,4	63,7	±	3,5	0,736	m
IVC; INSP/EXP> %50*	(-)	10		22,2%	8		53,3%	0,023	X^2
	(+)	35		77,8%	7		46,7%		
sPAP ≥ 35 mmHg	(-)	39		86,7%	8		53,3%	0,007	X^2
	(+)	6		13,3%	7		46,7%		
HR, bpm		80,3	±	12,1	75,7	±	14,1	0,232	t
PR, msec		152,8	±	22,6	148,3	±	11,3	0,328	m
QRS, msec		87,6	±	9,6	90,3	±	15,4	0,932	m
QT, msec		353,2	±	25,2	362,7	±	30,3	0,448	m
QTc, msec		405,5	±	20,3	403,4	±	18,0	0,727	t

LA left atrial, LVEDD left ventricular end diastolic diameter, LVESV left ventricular end systolic diameter, IVS interventricular septum, PW posterior wall diameter, PA pulmonary artery, Dec. T deceleration time, RV d1-d2-d3 right ventricle diameters, RV-FAC right ventricular fractional area change, TAPSE tricuspid annular plane systolic excursion, RA right atrial, TDI S' tissue Doppler imaging systolic wave S' velocity, sPAP systolic pulmonary artery pressure, IVC inferior vena cava, INSP/EXP inspiration / expiration, HR heart rate *IVC; INSP/EXP> %50, collapse of IVC during inspiration or not

the patients who applied between 30 and 60 days after COVID-19 infection are parallel with the control group. Ejection fraction being indicator of left ventricular systolic function was found to be less than the other groups among those presenting at 30 and 60 days.

Studies have found that symptoms persist for a while in patients who survived COVID-19 (9,11). Some academic studies have reported that it takes up to 3-6 months (9). But we do not have enough data on whether these symptoms are permanent or not. For this reason, new definitions have been introduced as Long Covid and Acute Post COVID-19 Syndrome (11,12). Pulmonary recovery can take a long time due to major involvement of the respiratory tract in SARS-COV-2 infection (13,14). Depending on this effect, dyspnea is at the forefront in people who

Table 5: Presents The Clinical and Demographic Parameters of The Patients Consulted İn Covıd Services

		pericardial effusion (-)			pericardial effusion (+)			p	
		Mean ±s.d./n-%			Mean ±s.d./n-%				
Age		42,4	±	12,0	49,9	±	13,3	0,070	t
Sex	female	21		42,9%	6		54,5%	0,481	X^2
	Male	28		57,1%	5		45,5%		
Height (cm)		169,8	±	9,5	164,4	±	8,0	0,236	t
Weight (kg)		78,3	±	16,1	68,0	±	7,9	0,170	t
BMI (kg/m2)		27,0	±	5,1	25,0	±	0,9	0,405	t
Blood Pressure ≥140 mmHg		1		2,0%	0		0,0%	1,000	X^2
Smoking		2		4,1%	2		18,2%	0,150	X^2
Complaints	Dizziness	1		2,0%	0		0,0%	1,000	X^2
	Palpitation	7		14,3%	1		9,1%	0,973	X^2
	Dyspnea	15		30,6%	0		0,0%	0,000	X^2
	Chest Pain	23		46,9%	10		90,9%	0,207	X^2
	Blood Pressure disregulation	3		6,1%	0		0,0%	0,939	X^2
Angina	Type I	2		8,7%	1		11,1%	0,367	X^2
	Type II	6		26,1%	1		11,1%		
	Type III	15		65,2%	8		78,8%		
Hospitalization	(-)	43		87,8%	9		81,8%	0,631	X^2
	(+)	6		12,2%	2		18,2%		
t Independent sample t test / X^2 Chi-square test									

BMI: body mass index t Independent sample
Type l: Typical angina, Type ll: Atypical angina, Type lll: Non-anginal chest pain

survive COVID-19 (9,15,16). In our study, significant portion of the patients who applied to the cardiology outpatient clinic in the first 30 days after the infection had exertional dyspnea. Even 6 months after discharge in SARS and MERS, severe deterioration was observed in respiratory function tests (17). Therefore, patients with persistent exertional dyspnea should be closely monitored. In our study, we found that the number of applicants with exertional dyspnea between 30 and 60 days after COVID-19 infection decreased.

Myocarditis and pericarditis may occur after viral illnesses (18). SARS-COV2 is a cytopathic mRNA virus (13). Therefore, inflammatory events can be triggered due to the reaction against the virus (13,18). Thereby, myopericarditis or pericardial effusion is possible after SARS-COV-2. In studies were conducted in patients recovering from COVID-19, different rates of myopericarditis or pericardial effusion were detected with CMR(Cardiovascular magnetic resonance) (7,8,19). In our study, more patients with pericardial effusion were found in the first 30 days of Post COVID-19. The number of patients with pericardial effusion decreased between the 30-60 days of Post COVID-19, but it was never seen in the control group. These patients generally presented with chest pain and pericarditis symptoms was at the forefront.

Although most of the complaints of chest pain were in the foreground in all 3 groups, most common application was with type 3 angina in the first 30 days of Post COVID-19 and most of them were accompanied by pericardial effusion or pericarditis. It was observed that the symptoms of type 3 angina regressed with pericardial effusion or pericarditis on the Post COVID-19 30-60 days.

There are studies showing the QTc prolongation due to direct or indirect myocardial involvement in active COVID-19 infection (20,21). No pathology was detected in ECG in 3 groups of patients. In addition, it is remarkable that in the group of patients who survived COVID-19, smokers were less than the control group. As known, smoking is related to the cardiovascular diseases (22). In our study, no relationship was

Table 6: Echocardiography Results

		pericardial effusion (-)			pericardial effusion (+)			p	
		Mean ±s.d./n-%			Mean ±s.d./n-%				
Aortic root (cm)		2,52	±	0,34	2,38	±	0,29	0,225	t
LA (cm)		3,03	±	0,45	3,03	±	0,20	0,993	t
LVEDD (cm)		4,56	±	0,63	4,60	±	0,28	0,885	m
LVESD (cm)		3,21	±	0,53	3,25	±	0,29	0,954	m
IVC (cm)		0,94	±	0,19	0,97	±	0,06	0,495	m
PW (cm)		0,84	±	0,38	0,81	±	0,09	0,612	m
PA (cm)		2,00	±	0,25	2,03	±	0,18	0,521	m
E (cm/s)		0,75	±	0,14	0,68	±	0,22	0,183	t
A (cm/s)		0,69	±	0,22	0,70	±	0,18	0,586	m
Dec. T (msec)		228,4	±	60,4	246,8	±	53,7	0,355	t
e' lateral (cm/s)		0,11	±	0,02	0,09	±	0,02	0,019	m
E/A ratio		1,16	±	0,34	0,97	±	0,28	0,117	m
E/e' ratio		6,78	±	1,97	7,02	±	1,98	0,633	m
RV d1 (cm)		3,73	±	0,54	3,85	±	0,34	0,479	t
RV d2 (cm)		3,14	±	0,52	3,34	±	0,70	0,305	t
RV d3 (cm)		5,46	±	0,55	5,56	±	0,59	0,788	m
RV-FAC (%)		48,2	±	7,8	48,7	±	12,6	0,914	m
TAPSE (cm)		2,32	±	0,32	2,31	±	0,47	0,937	t
RA (ml/m2)		36,0	±	11,8	46,4	±	8,6	0,008	t
TDI S' (cm/s)		14,2	±	1,9	15,3	±	2,7	0,187	m
sPAP (mmHg)		31,3	±	5,2	35,6	±	6,6	0,106	m
EF (%)		64,1	±	2,7	64,5	±	2,7	0,992	m
IVC; INSP/EXP> %50*	(-)	13		26,5%	5		45,5%	0,216	X^2
	(+)	36		73,5%	6		54,5%		
sPAP ≥ 35 mmHg	(-)	41		83,7%	6		54,5%	0,034	X^2
	(+)	8		16,3%	5		45,5%		
HR, bpm		78,4	±	12,9	82,5	±	11,4	0,326	t
PR, msec		152,1	±	19,9	149,5	±	23,1	0,542	m
QRS, msec		88,4	±	11,3	87,9	±	11,6	0,780	m
QT, msec		356,2	±	27,7	352,7	±	22,0	0,936	m
QTc, msec		403,5	±	19,4	411,4	±	20,3	0,233	t

t Independent Samples T Test / m Mann-whitney u test / X^2 Chi-square test

LA left atrial, LVEDD left ventricular end diastolic diameter, LVESV left ventricular end systolic diameter, IVS interventricular septum, PW posterior wall diameter, PA pulmonary artery, Dec. T deceleration time, RV d1-d2-d3 right ventricle diameters, RV-FAC right ventricular fractional area change, TAPSE tricuspid annular plane systolic excursion, RA right atrial, TDI S' tissue Doppler imaging systolic wave S' velocity, sPAP systolic pulmonary artery pressure, IVC inferior vena cava, INSP/EXP inspiration / expiration, HR heart rate *IVC; INSP/EXP> %50, collapse of IVC during inspiration or not

found between smoking and dyspnea. However, those with dyspnea complaints had a high BMI.

There are studies using echocardiography to evaluate cardiac function in patients with active COVID -19 (4-6,23). Left ventricular diastolic dysfunction is discussed, especially parameters of right ventricule deteriorated (4,5). Involvement of right ventricule is associated with mortality (6). The main pathophysiological mechanism is the increase in systolic pulmonary artery pressure as a result of hypoxia caused by the attachment of SARS-COV-2 virus to the respiratory tract

epithelium (9,13). In our study, we found that right ventricular parameters were slightly impaired in patients who survived COVID-19 within the first 30 days. Especially right ventricule diameters were higher than the other 2 groups. Although the right ventricular fractional area change (RV-FAC) was within the normal range, it was found lower than the other two groups. The number of patients with systolic pulmonary artery pressure > 35 mmHg showing pulmonary artery resistance was higher. Increased pressure in the right heart reflects on the inferior vena cava, so the number of patients with an IVC (Inferior vena cava) collapse less than 50% in the first 30 days was more. The ratio of E / e' , which is one of the indicators of diastolic function and left ventricular filling pressure, was found to be higher than the other two groups, and E / e ' was found greater than 8 in 8 patients.

It was found that the number of the patients with systolic pulmonary artery pressure higher than 35 mmHg who survived COVID-19 and presented with dyspnea. In addition, the number of patients in which the inferior vena cava collapses < 50% in the inspiration is less.

In patients who survived COVID-19 and admitted between 30 and 60 days, we found that echocardiographically right ventricular functions improved and left ventricular diastolic parameters were similar to the control group. However, left ventricular systolic functions were found to be more impaired than the other two groups. Especially 2 patients with left ventricule systolic ejection fraction <60 of all patients were in this group.

We found mild pericardial effusion not exceeding> 10 mm in diastole in echocardiography more often in patients presenting in the first 30 days. Although there is no study about post COVID-19 with echocardiography, different rates of pericardial effusion were detected in studies with CMR (7,8). In the study conducted by Puntmann et al., 8 of 57 patients had pericardial effusion > 10 mm [7]. However, this study differs from our study in terms of sample space since patients with chronic diseases are not excluded. Mild pericardial effusion up to 8% was detected in CMR imaging was performed on healthy athletes (8). The number of patients with pericardial effusion has decreased in patients admitted on the 30 and 60 days of surviving COVID -19. Pericardial effusion increases the left ventricular filling pressure gradient and diastolic dysfunction develops as the left ventricle cannot be filled sufficiently in diastole (24). Among the parameters showing left ventricular diastolic function, and left ventricular filling pressure, it was found that E / A ratio and e' decreased and E/e' increased in patients recovering from COVID-19 and with pericardial effusion. Pericardial effusion increases the left ventricular filling pressure gradient, and diastolic dysfunction develops because the left ventricle cannot be filled sufficiently in diastole. Despite the preservation of right ventricular dimensions, the number of patients with sPAP > 35 mmHg was found more and volume increased in the right atrium.

Limitations: First of all, it is a single center study. The patients under the age of 18 were excluded, as consent was not obtained. The number of patients was limited, as we used patients without chronic disease. Therefore, larger cohort studies are needed to validate our results. Since blood samples were taken according to the complaints of each patient, the laboratory results of the patients were not compared. Our patients did not survive the COVID-19 disease severely. Most of the patients especially those who suffer from dyspnea could not be followed up.

Patients have an ongoing myocardial inflammation. Myocardial damage is associated with cardiac dysfunction. ECG changes due to myopericardial involvement seen in active COVID-19 patients were not seen in post COVID-19 patients. The symptoms of the patients changes over time. Although the sequelae could be followed and detected by echocardiography in the early period of post covid, it was observed that the patients recovered echocardiographically over time. Systolic dysfunction was detected in 2 patients. This raises the question of whether it causes myocardial damage due to the longer duration of inflammation. In the future, it supports the need for a multidisciplinary approach for therapeutic intervention in post COVID-19 patients. We state that echocardiography, which is a non-invasive, cheap and safe test to evaluate cardiac symptoms, is a follow-up tool for patients with post COVID-19 symptoms.

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Compliance With Ethical Standards: Conflict of interest

The authors declare that they have no conflict of interest.

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