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Detection of Cardiac Involvement Using Speckle Tracking Echocardiography in COVID-19 Recovered Patients and Its Relation to Leucocyte Count.

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ORIGINAL STUDY

Detection of cardiac involvement using speckle tracking echocardiography in coronavirus disease 2019 recovered patients and its relation to leucocyte count

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Abstract

Background: **Globally, the coronavirus disease-2019 (COVID-19) pandemic remains a leading cause of morbidity and mortality. Our research aimed to assess the relationship between recovered COVID-19 patients' left ventricular (LV) systolic function and a range of inflammatory and hematological markers.**

Methods: **For cases; baseline clinical and biochemical data such as the complete blood count, serum ferritin, C-reactive protein (CRP), lactate dehydrogenase, and D-dimer were measured Echocardiographic parameters of LV functions, including global longitudinal strain (GLS), myocardial performance index, left atrial volume index, and ejection fraction, were done to all cases and controls.**

Results: **GLS was lower in post-COVID 19 in comparison to the control (−16.8 ± 7.9 vs. 20.6 ± 4.9, respectively, *P* 0.001), mean of GLS value changed among the three groups of COVID illness (mild: −16.6 ± 6.7 moderate: −14.5 ± 8.8 severe: −12.5 ± 5.7 %; *P* 0.001). GLS correlated positively with white blood cell 0.578, *P* 0.001.**

Conclusion: **Subclinical LV dysfunction was prevalent in 40 % of cases and more predominant in severe COVID illness. Impaired LV-GLS was associated with lower white blood cells.**

Keywords: Coronavirus disease 2019, Global longitudinal strain, Systolic function

1. Introduction

Globally, the coronavirus disease-2019 (COVID-19) pandemic remains a leading cause of morbidity and mortality [1]. Myocarditis, heart failure, cardiac arrhythmias, cardiogenic shock, coronary syndrome, and venous thromboembolism are among the cardiovascular symptoms associated with COVID-19 [2]. Furthermore, a limited number of researchers have found that COVID-19 patients had subclinical cardiac impairment [3,4]. Compared with conventional left ventricular (LV) systolic function, speckle tracking echocardiography (STE) derived global longitudinal strain (GLS) is a sensitive and accurate technique for the prediction of subclinical LV impairment [5]. In this work, we evaluated LVGLS in retrieved COVID-19. Our study aimed to assess the relationship between recovered COVID-19 patients' LV systolic function and a range of inflammatory and hematological markers.

2. Methods

Our study was conducted at Zagazig University Hospital and AL-Ahrar Teaching Hospital from March 2022 to November 2023. A total of 230 patients who had recently recovered from COVID-19 infection within 30−45 days were evaluated, this period was selected based on a previous study which promoted that period interval, actually, this period was expected that the acute clinical manifestations of

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active COVID illness were disappeared and almost patients who were hospitalized discharged and screened by reverse transcriptase PCR (RT-PCR) two negative PCR test results that are at least 24 h apart [6]. These patients were all previously diagnosed as COVID-19 positive utilizing the RT-PCR swab test. Patients were identified as recovered based on the criteria for discharge (more than three days of normal temperature, alleviated respiratory symptoms, and two negative PCR test results that are at least 24 h apart) and had at least 14 days of isolation. Patients who had impaired LV function (n = 20), poor image quality (n = 30), prior coronary artery disease (CAD) (n = 30), uncontrolled diabetes mellitus (DM) (n = 10), uncontrolled hypertension (HTN) (n = 20), and (n = 20) lost in follow-up were excluded from the study. Postexclusion, 100 cases were enrolled and screened by conventional and STE studies. For the control volunteers 40 participants were excluded due to poor image quality (n = 20), HTN (n = 10), and DM (n = 10) with the final 100 control participants enrolled in our study. (Fig. 1: design of flow chart).

For cases; At COVID-19 infection acute illness, baseline clinical and biochemical characteristics were obtained for each case. These included the complete blood count, lactate dehydrogenase (LDH), serum ferritin, C-reactive protein (CRP), and D-dimer.

A total of 100 cases and 100 control participants were assessed by (Commercial ultrasonic scanner VividE95, Horten, Norway; with a multifrequency phased array probe with a 1.5–3.6 MHz frequency range and phased-array transducers. The European Association of Echocardiography and the American Society of Echocardiography guidelines provided the basis for the standard assessment [7]. Two experienced and independent echocardiographers blinded to the patient's clinical data regarding the

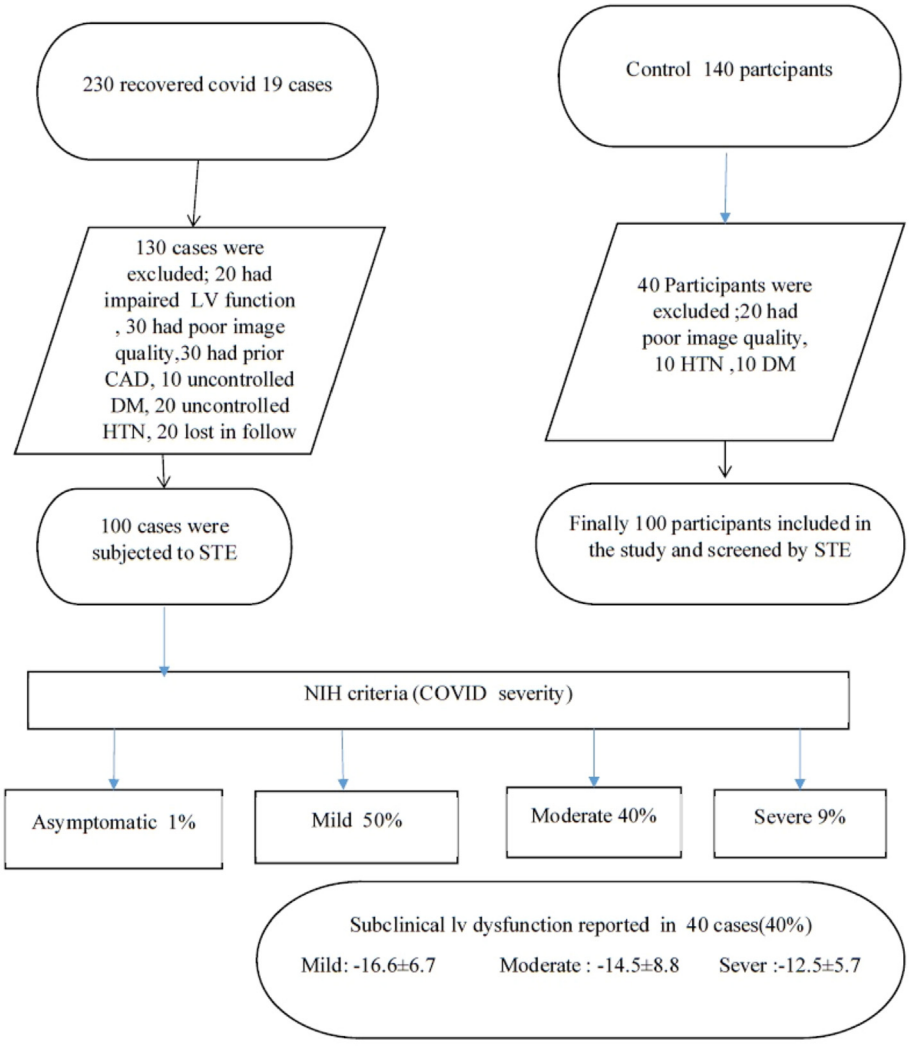

Fig. 1. Flow chart design.

start of disease duration symptoms and medical history performed left ventricular ejection fraction (LVEF) m-mode, left ventricle tissue doppler velocities (TDI) to assess systolic mitral annular velocity (s′) and LV myocardial performance index, modified biplane area–length method for the left atrial maximum volume index (LAVI); mL/m^2. Pulmonary artery systolic pressure was assessed based on a tricuspid regurgitant (TR) jet then the right atrial pressure values were added.

2.1. Assessment of the left ventricle using speckle tracking echocardiography (STE)

Two-dimensional STE images (same machine used in conventional assessment 9 with soft were included for STE) were acquired from the three, four, and two apical views of the left ventricle. Three consecutive beats' views were collected, and they were then saved in cine-loop format. The software system automatically created epicardial tracing for each view after manually specifying the endocardial border, creating GLS, the average value of 18 segments of the total three chamber views [7].

2.2. Ethical standard

The current study was approved by Faculty of Medicine Zagazig University with an IRB number 9265-22-3-2022.

Informed written consent was taken from all participants.

2.3. Statistical analysis

Version 21 of the SPSS program was used to analyze the data. In the case of continuous data, mean ± standard deviation (SD) was utilized, while percentages were shown for categorical data. For categorical variables, the χ^2 test or Fisher exact test was utilized. For mean comparisons of continuous data, on the other hand, the Student's *t*-test or Mann–Whitney *U* test was utilized. Based on the severity of COVID-19, the study used analysis of variance (ANOVA) with post-hoc analysis to compare the mean values of continuous variables among the three groups. Using the Spearman correlation coefficient test, the relationship between inflammatory indicators, laboratory indices, and LVGLS was analyzed.

3. Results

The mean age of post-COVID patients 46.33 ± 6.3 with 58 % of them were female, comorbidities such as HTN, DM, and smoking accounted for 20, 30, and 20 %, respectively. During COVID-19 infection 99 % of patients reported symptoms, the National Institute of Health (NIH) severity criteria [7] were utilized to categorize symptomatic patients into three groups: mild (50 %), moderate (40 %), and severe (9 %) disease. During active COVID infection myalgia/fatigue and loss of smell were the most predominant symptoms, reported in 50 % and 20 %, respectively. The most common symptoms during the post-COVID-19 recovery phase were palpitation, dyspnea, and fatigue 40, 30, and 20 %, respectively (Table 1).

A follow-up echocardiography was performed on average 38.4 ± 7.6 days after COVID-19 recovery. LV GLS was lower in post-COVID-19 in comparison to the control (−16.8 ± 7.9 vs. 20.6 ± 4.9, respectively), *P* 0.0001, otherwise, conventional echocardiographic parameters were not statistically significant (Table 2).

Table 1. Demographic characteristics of the studied groups.

–	Post-COVID 19 (n = 100)	Control (n = 100)	*P* value
Age (years)			
Mean ± SD	46.33 ± 6.3	44.12 ± 7.71	0.106
Variable	No (%)	No (%)	
Sex			0.618
Female	58 (58)	50 (50)	
BMI(kg/m^2)			
Mean ± SD	29.76 ± 3.6	28.54 ± 1.89	0.78
HTN	20 (20)	–	
DM	30 (30)	–	
Smoking	20 (20)		
Symptoms			
Asymptomatic	1 (1)	–	
Mild	50 (50)		
moderate	40 (40)		
Sever/critical	9 (9)		
Duration of symptoms/days	3.8 ± 1.5	–	
Duration of hospital stay	8.3 ± 7.4	–	
Symptoms on admission			
Fever	4 (4)		
Cough	6 (6)		
Dyspnea	4 (4)	–	
Chest pain	5 (5)		
Myalgia/Fatigue	6 (6)		
Loss of smell	50 (50)		
Loss of taste	20 (20)		
	5 (0.05)		
Post-COVID recovery symptoms			
Palpitation	40 (40)		
Dyspnea	30 (30)	–	
Fatigue	20 (20)		
Cough	6 (6)		
Syncope	3 (3)		
Pedal edema	1 (1)		

BMI, body mass index; DM, diabetes mellitus; HTN, hypertension.

Table 2. Echocardiographic data of the studied groups.

	Post-COVID 19 ($n = 100$)	Control ($n = 100$)	*P* value
EF m-mode %	60.6 ± 10.7	62.8 ± 12.8	0.98
LV EDD mm	61.6 ± 12.8	63.9 ± 14.7	0.67
LVESD mm	33.9 ± 9.7	30.7 ± 10.7	0.89
LV s wave cm/s	10.7 ± 7.9	11.7 ± 4.8	0.77
LV MPI	0.30 ± 0.02	0.31 ± 0.08	0.45
LA VI ml\m^2	24.7 ± 9.7	24.8 ± 3.9	0.56
PASP mmHg	27.6 ± 10.8	25.9 ± 9.9	0.76
Lv–GLS %	−16.8 ± 7.9	−20.6 ± 4.9	0.001

EF, ejection fraction; GLS, global longitudinal strain; LAVI, left atrial volume index; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular endsystolic diameter; MPI, myocardial performance index; PASP, pulmonary artery systolic pressure.

We subdivided post-COVID patients according to normal LV-GLS (20.6 ± 4.9) and reduced LV-GLS (−16.8 ± 7.9) to take a detailed overlook on demographic, laboratory, and echocardiographic data and to know whether they differ or not. G2 post-COVID with reduced strain had statistically significantly longer hospital stay in comparison to G1 post-COVID with normal strain (7.8 ± 1.8 vs 3.3 ± 2.4, respectively), G2 had statistically significant lower white blood cell (WBC) in comparison to G1 (5.8 ± 1.7 vs 7.4 ± 2.6, respectively). G2 had statistically significant higher CRP, LDH, S. Ferritien, D-dimer in comparison to G1. Conventional echocardiographic parameters were not significant (Table 3).

As impaired LVGLS (less than −20.6 %) was reported in (40 %) of post-COVID patients, there was a significant difference in the mean LV-GLS values among the three groups of symptomatic patients (mild: −16.6 ± 6.7 moderate: −14.5 ± 8.8 severe: −12.5 ± 5.7 %; *P* 0.001). (Table 4), and case demonstration Figs. 2–4.

LVGLS correlated positively with WBC 0.578, P.001 (Fig. 5).

LVGLS correlated negatively with other inflammatory mediators CRP, LDH, D-dimer, S. Ferritien (Table 5).

Table 3. Comparison of demographic, echocardiographic, and laboratory data between subjects with and without reduced left ventricular global longitudinal strain.

	G 1 Post COVID with normal LV-GLS ($n = 60$)	G 2 Post COVID with reduced LV-GLS ($n = 40$)	*P* value
Age (years)			
Mean ± SD	44.33 ± 6.3	45.12 ± 7.71	0.106
Variable	No (%)	No (%)	
Sex			0.118
Female	30 (50)	22 (55)	
BMI(kg/m^2)			
Mean ± SD	28.86 ± 4.6	29.53 ± 1.69	0.88
HTN	30 (50)	23 (57.5)	0.98
DM	20 (33.3)	12 (30)	0.78
Smoking	10 (16.7)	5 (12.5)	0.74
Asymptomatic	1 (1)	–	
Duration of hospital stay	3.3 ± 2.4	7.8 ± 1.8	0.03
Laboratory data			
WBC (10^3/μL)	7.4 ± 2.6	5.8 ± 1.7	0.02
Hb (gm/dl)	12.6 ± 4.9	11.9 ± 3.9	0.78
Neutrophils (10^3/μL)	4.8 ± 1.03	4.1 ± 1.3	0.56
Lymphocytes (10^3/μL)	1.3 ± 1.08	1.7 ± 1.3	0.03
PLTs (10^3/μL)	190 ± 12.9	200.6 ± 20.6	0.89
CRP (mg/L)	89.5 ± 22.9	120.8 ± 33.9	0.001
LDH (U/L)	379.9 ± 89.9	411.5 ± 79.9	0.001
D-Dimer (mg/L)	9.8 ± 5.9	14.7 ± 8.6	0.002
S. Ferritien (μg/L)	221.9 ± 76.9	338.9 ± 66.9	0.001
Echocardiographic data			
EF M-MODE	61.6 ± 12.8	60.5 ± 12.6	0.89
LV S wave	12.6 ± 6.1	13.8 ± 8.4	0.34
LAVI	26.8 ± 12.6	27.8 ± 11.5	0.65
LV MPI	30.9 ± 1.9	31.7 ± 1.02	0.67
PASP	28.9 ± 11.9	30.8 ± 12.9	0.87
LVGLS	−20.6 ± 4.9	−16.8 ± 7.9	0.001

BMI, body mass index; CRP, c −reactive protein; DM, diabetes mellitus; EF, ejection fraction; GLS, global longitudinal strain; Hb, hemoglobin; HTN, hypertension; LAVI, left atrial volume index; LDH, lactate dehydrogenase; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular endsystolic diameter; MPI, myocardial performance index; PASP, pulmonary artery systolic pressure; PLTS, platelets; WBC, white blood cell.

4. Discussion

The major finding of the current study was that (40 %) of the patients who had recovered from COVID-19 had subclinical LV systolic impairment. STE-derived LV-GLS is a reproducible and objective way for myocardial deformation assessment [8,9]. LV strain was impaired in post-COVID-19 recovery and was seen in 11 (22 %), 22 (55 %), and 7 (77.7 %) patients, respectively, in mild, moderate, and severe COVID-19 illness. It is worth mentioning that even mild COVID-19 patients suffered subclinical impairment. We could say that cardiac affection on COVID-19 is multi-factorial with the following mechanisms being postulated; direct myocardial compromise brought on by the cytopathic action of the virus; hypoxia-induced oxygen supply-demand mismatch; endothelial dysfunction and inflammation-induced micro or macrovascular thrombosis; and cytokine storm-induced systemic inflammatory response [10]. Furthermore, early myocardial fibrosis and peri-myocarditis were two signs of chronic cardiac inflammation, have been linked to LV impairment in COVID-19 recovered individuals [11,12]. Previous studies have demonstrated the value of LVGLS in cardiac involvement

Table 4. Distribution of abnormal left ventricular global longitudinal strain according to coronavirus disease severity.

	Mild $n = 50$ (50 %)	Moderate $n = 40$ (40 %)	Severe $n = 9$ (9 %)	P	Post-hoc
LV-GLS mean ± sd	−16.6 ± 6.7	−14.5 ± 8.8	−12.5 ± 5.7	0.001	P^*_1 0.002, P^*_2 0.001, P^*_3 0.04
Reduced strain n (%)	11 (22)	22 (55)	7 (77.7)	0.001	

P^*_1 mild versus moderate.
P^*_2 mild versus moderate.
P^*_3 moderate versus severe.
LV-GLS, left ventricular global longitudinal strain.
Sd, standard deviation.

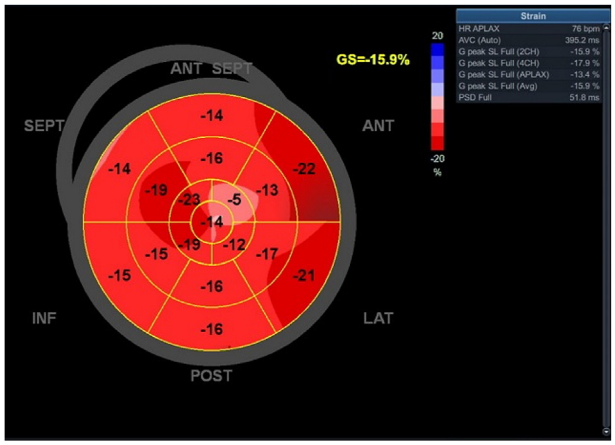

Fig. 2. Case demonstration of LV-STE at the postrecovery period of mild coronavirus disease 2019 illness, showing bull eye average GS = −15.9 %.

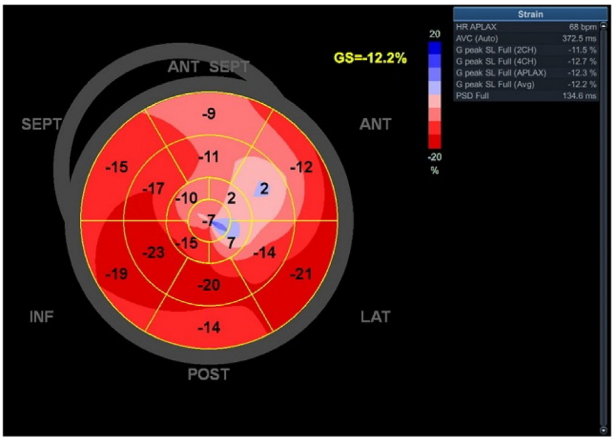

Fig. 4. Case demonstration of LV-STE at the postrecovery period of severe coronavirus disease 2019 illness, showing bull eye average GS = −12.2 %.

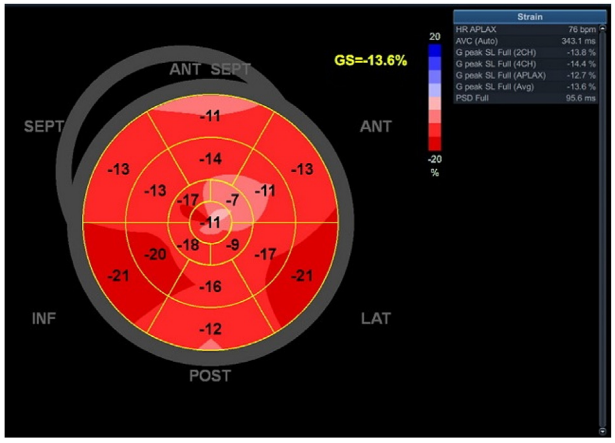

Fig. 3. Case demonstration of LV-STE at the post recovery period of moderate coronavirus disease 2019 illness, showing bulls eye average GS = −13.6 %.

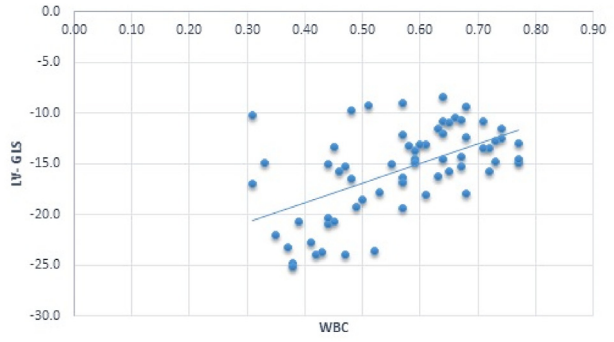

Fig. 5. Scatter dot graph showing positive correlation between left ventricular global longitudinal strain and white blood cell.

in patients who were actively infected with COVID-19 [4,13,14], but there is little data regarding the utility of GLS in patients with COVID-19 recovery. Özer *et al.* [15] noted that one month after being discharged from the hospital due to a COVID-19 infection, 28 (37.8 %) patients had impaired LVGLS.

Baruch *et al.* [16] reported residual LV systolic impairment as mean GLS values were impaired, in thirty percent of cases at a one-month follow-up.

Numerous investigations showed that patients with COVID-19 recovery frequently had cardiac MRI abnormalities, particularly late gadolinium enhancement (LGE) and aberrant native T1 and T2 values which show myocardial inflammation and edema [11,17–19].

Table 5. Pearson correlations between left ventricular global longitudinal strain and inflammatory parameters.

	LV- GLS
WBC	
r	0.578
P	0.001
CRP	
r	−0.425
P	0.008
LDH	
r	−0.685
P	0.002
Ferritin	
r	−0.451
P	0.004
D-dimer	
r	−0.544
P	0.009

CRP, c-reactive protein; LDH, lactate dehydrogenase; LVGLS, left ventricular global longitudinal strain; WBC, white blood cell.

Even though the 'gold standard' for detecting subclinical cardiac involvement is cardiac magnetic resonance (CMR) imaging, there are significant concerns about its cost and accessibility in low-income nations. In addition, it is nearly hard to follow up with CMR for every patient due to the massive burden of COVID-19 recovered cases. STE-imaging is a straightforward, affordable, and readily available method to identify subclinical cardiac dysfunction in these patients under such circumstances [20].

Our study found the mean LVGLS values (post recovery period) for the COVID-19 disease groups with mild, moderate, and severe cases were, respectively, -16.6 ± 6.7, -14.5 ± 8.8, and -12.5 ± 5.7; It reveals that, in comparison to patients with mild or moderate disease, those with severe COVID-19 infection had a greater risk of residual LV dysfunction, which could be explained by the significant positive correlation between LVGLS and inflammatory mediators (CRP, LDH, S. ferritin, D-dimer) so we could say that sever COVID illness at active COVID infection had the most impaired residual mean value of LVGLS at post-recovery period due to high level of inflammatory mediators. Özer *et al.* [15] found concordant findings; CRP levels correlate positively with LVGLS (postrecovery period). Derya *et al.* [21] reported that LVGLS correlates negatively with serum ferritin and LDH values.

The early study had investigated the relationship between inflammatory markers at active COVID time with residual LV GLS at the postrecovery period [21] but up to our knowledge, our study was the first one to find an association between WBCs at active infection and residual LV systolic impairment at postrecovery period. Our results revealed that WBCs were lower in COVID patients in comparison to control and were lower in post-COVID patients with reduced LVGLS in comparison to those with normal strain. Other research has shown that individuals with COVID-19 who had a high leucocyte count at hospital admission experienced more severe disease and mortality [22]. However, several additional investigations have indicated that reduced WBC counts [23] were similarly linked to a higher risk of severe morbidity and mortality in COVID-19.

Increased apoptosis and CD95 expression in T-cells, B-cells, and natural killer cells may be the cause of COVID-19 cases showing leukopenia signs, which leads to T-cell exhaustion and impairs the release of inflammatory cytokines [24,25]. Additionally, the overexpression of T cell immunoglobulin, mucin domain molecule 3 (TIM-3), and programmed death receptor 1 (PD-1) on $CD4^+$ and $CD8^+$ T-cells results in T cell exhaustion [24].

4.1. Conclusion

Subclinical LV dysfunction was prevalent in 40 % of cases and more predominant in severe COVID illness. Impaired LV-GLS was associated with lower WBCs.

4.2. Limitations

Relatively small number of cases. STE has its inherent limitations as image quality and being affected by other comorbidities. Almost of patients did not have echocardiography before COVID-19 illness so we could not exclude that this cardiac dysfunction did not present before COVID-19 but we depended on our exclusion criteria on cardiac history instead of absent old echocardiography before COVID-19.

4.3. Recommendations

Future larger sample studies with longer follow-up studies to confirm our results.

Ethics information

This work was carried out in accordance with the Declaration of Helsinki in which a The patients' written informed consent was obtained. The faculty of medicine, Zagazig university Hospitals Ethical Committee gave approval prior to the research begining. The aim and nature of the study as well as the risks were all explained to the patients or their relatives. The participants first guardians agreed that he/she would have the investigational nature of

the study, its inherent risks and benefits. Confidentiality of data was assured in which Participants' information was replaced with research identification codes (ID Codes), Patients can withdraw from the study at any time and still get the full medical service.

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Author contribution

Shaimaa Wageeh wrote the concept and the protocol design of the research, shared in the echocardiographic assessment of cases. Eman H. Seddik interpretated, assessed the statistcal results of research, wrote the manuscript and was the corresponding author Alaa Ramadan Youssuf shared in echocardiographic assessment of cases and control. All authors had read and reviewed the final manuscript.

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Conflicts of interest

There are no conflicts of interest.

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