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Assessment of Subtle Left Ventricular Systolic Dysfunction in Hospitalized COVID-19 Patients by Echocardiography Speckle Tracking: A Cross-sectional Study

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ABSTRACT
Background: Heart problems, especially in hospitalized patients, have been linked to the COVID-19 pandemic. It is possible for them to be subtle deficits in left ventricular (LV) systolic function even when there are no significant cardiac symptoms. Clinical results may be greatly improved with early detection of such dysfunction.
Objectives: To evaluate subtle changes in LV systolic function in patients hospitalized with moderate to severe COVID-19.
Patients and methods: In this cross-sectional study, 65 individuals hospitalized with moderate to severe COVID-19 were included. Echocardiographic evaluations were performed within the first 10 days of pulmonary infection.
Results: Cough and cardiac chest discomfort were significantly higher in patients with severe COVID-19 compared to moderate cases. There was a significant difference in the two groups' respiratory rates and blood pressures. Patients suffering from severe COVID-19 had a much-decreased LV ejection fraction (LVEF) when contrasted with those suffering from moderate illness. Additionally, global longitudinal strain values were significantly reduced in severe cases versus moderate cases. Among the total patient cohort, 23% demonstrated abnormal global longitudinal strain values, while another 6% had borderline results. LV systolic dysfunction was significantly more prevalent in severe cases, affecting 80% of these patients compared to 20% of those with moderate disease.
Conclusions: Traditional measures of EF are less sensitive than global longitudinal strain when it comes to detecting early signs of heart deterioration. Speckle monitoring echocardiography might be a useful tool for improving clinical outcomes and allowing for earlier intervention in hospitalized COVID-19 patients, especially those with advanced illness.
Keywords: COVID-19, Echocardiography, Left Ventricular Systolic Dysfunction, Speckle Tracking, Global Longitudinal Strain.

INTRODUCTION

The heart is primarily recognized for its role as a pumping organ, with its contractile efficiency serving as a fundamental measure of cardiovascular health [(1)]**.** Extensive research has highlighted the significance of evaluating left ventricular (LV) systolic function, not only for diagnosing cardiac disorders but also for assessing prognosis and optimizing treatment approaches [(2)]**.** In order to determine which heart failure patients will benefit from implanted defibrillators and biventricular pacing, a precise evaluation of LV ejection fraction (LV-EF) is crucial [(3)]**.**

One of the most important non-invasive imaging modalities for assessing hemodynamic state and heart function is two-dimensional echocardiography (2DE) [(4)], because of its significance in COVID-19 patients experiencing hemodynamic instability and other multi-organ dysfunctions [(5)]. Unfortunately, 2DE is not always done because of worries about spreading infections, and there have not been many studies looking at its use in COVID-19 patients [(6)].

The SARS-CoV-2 virus, which is responsible for the COVID-19 pandemic, might impact the cardiovascular system [(7)]. Patients infected with COVID-19 are more likely to die or require admission to an intensive care unit if they have myocardial injury [(8)]. Myocardial damage can occur in up to 30% of COVID-19 patients admitted to hospitals, according to research [(9)], though the underlying mechanisms remain unclear [(10)]. One proposed explanation is that respiratory failure and hypoxemia increase myocardial workload, placing additional strain on the heart [(11)].

Myocardial damage, thromboembolic events, arrhythmias, and heart failure have been linked to SARS-CoV-2. Echocardiography is a widely available and frequently utilized imaging modality for assessing cardiac function. Previous echocardiographic studies on COVID-19 patients have not demonstrated a decline in LV function when evaluated using LV-EF [(12)]. 2DE provides a more comprehensive analysis of regional LV function by offering volumetric measurements [(13)].

In hospitalized COVID-19 patients, this speckle tracking echocardiography (STE) study will identify mild LV systolic failure.

PATIENTS AND METHODS

Participating in this study were 65 hospitalized patients with COVID-19, which was defined by the World Health Organization (WHO) as ranging from moderate to severe [(14)]. Within the initial 10 days of lung infection or infiltration, STE scan was performed to evaluate any possible mild LV systolic dysfunction (LVSD).

Study design and setting

This cross-sectional study was conducted over an eight-month period, from October 2022 to June 2023, and included 65 patients. The evaluations of the LV systolic function by assessment of the EF using M-MODE and

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global longitudinal strain (GLS) using two-dimensional STE were performed within the first 10 days of COVID-19 infection or pulmonary involvement to identify subtle LVSD. The research was carried out across multiple isolation hospitals, including Badr University Hospital, 15th of May Hospital, Ain Shams Hospital, Al Kasr Al Ainy Hospital, and Mansoura Chest Disease Hospital.

Inclusion criteria: Moderate to severe COVID-19 patients aged 20–60 were diagnosed and classified according to WHO guidelines. Moderate cases were identified based on clinical symptoms and radiological findings indicative of pneumonia, with oxygen saturation (SpO_2) of at least 90% while breathing room air. Severe cases were defined by the presence of pneumonia accompanied by a respiratory rate exceeding 30 breaths per minute or an SpO_2 level below 90% on room air [(15)].

Exclusion criteria: People who had a history of heart disease, diabetes (as determined by HbA1C levels), or chronic renal disease were not included in the research. The results of the research were unaffected by the COVID-19 therapies.

Methodology

Each patient underwent a comprehensive assessment. A comprehensive medical history was taken, including demographic details (gender, age), medical history (diabetes mellitus, chronic kidney disease, heart disease), and the present symptoms of COVID-19. A thorough physical examination included measuring the patient's pulse, respiration rate, temperature, oxygen saturation, and blood pressure (both systolic and diastolic). Haemoglobin, white blood cell and platelet counts, creatinine, troponin I (normal value <0.04 ng/mL), and HbA1C values were considered essential laboratory testing.

LV functions by transthoracic echocardiography (TTE) speckle tracking

While evaluating LV function with TTE with speckle tracking, the patient was positioned in the left lateral decubitus posture, with their left arm draped over their head, for enhanced image collection. Improving picture quality was a top priority to guarantee accurate assessment of cardiac deformation. The frame rate was adjusted to 50 to 70 frames per second, and we made an effort to include all relevant myocardial structures for comprehensive evaluation throughout the cardiac cycle. The two-dimensional transdense TTE with Vivid GE machines ***(Vivid S 5/Vivid S6, USA)*** were used to evaluate GLS. Three different views—the apical long-axis, the four-chamber, and the two-chamber—were utilized to collect ECG-gated pictures. Images were digitally recorded at a frame rate of 50-70 fps for analysis. The endocardial boundary of the myocardial tissue was automatically traced using the Vivid GE machine, with human correction applied as needed. The GLS was automatically computed and shown as a bull's-eye map of the segments' strain. It is still difficult to define abnormal values for GLS because it changes with age, sex, and LV loading circumstances. However, according to the JACC Cardiovascular Imaging, GLS values of <16% are considered abnormal, while values >18% are deemed normal, and those ranging from 16% to 18% are classified as borderline. In the present study, GLS values >18% were considered normal. Additionally, LVEF <50% was defined as abnormal, based on the criteria outlined by **Fuster *et al.*** [(16)].

Ethics approval

The Faculty of Medicine, Helwan University, Egypt, Ethical Review Committee accepted the study protocol on August 9, 2022. Consent was taken from the patients or their 1st degree relative in case of patients on ventilator. All procedures followed the institutional research committee ethical norms and the Declaration of Helsinki.

Sample size calculation

The sample size was determined using G*Power 3.1.9.2 from the University of Kiel in Germany. A 0.05 alpha error and 90% research power were utilized as the foundation. Previous research has shown that LV-GLS can decrease by 40.9% to 80% in moderate and severe COVID-19 instances, which was used to perform the computation [(17)]. To account for a potential dropout, three additional patients were included, resulting in a total enrollment of 65 patients.

Statistical analysis

The SPSS v26 software, created and maintained by IBM in Chicago, IL, USA, was used to perform the statistical investigation. Histograms and Shapiro-Wilk tests were used to ensure that the data were standardized. The mean ± SD parametric data were further compared using independent t-tests. Quantitative variables will be presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t-test. Qualitative variables were presented as frequency and percentage and analyzed using the Chi-square or Fisher's exact test when appropriate. P value < 0.05 was considered significant.

RESULTS

Among 65 COVID-19 patients (55 moderate, 10 severe), age and gender did not differ significantly between both groups. Cough and cardiac chest pain were more frequent in severe cases. These patients also had higher respiratory rates and lower SpO_2 levels. SBP and DBP were significantly lower in severe cases. Most lab parameters were comparable, except for elevated troponin and creatinine in severe cases (**Table 1).**

Table (1): Basic Characteristics of Study Patients

		Total (n=65)	Moderate COVID-19 (n=55)	Severe COVID-19 (n=10)	Test Statistical	P
Age (Years)		46.32±8.59	46.16±8.4	47.2±9.99	t=0.349	0.729
Sex	**Male**	38(58.46%)	34(61.8%)	4(40%)	χ^2=1.66	0.198
	Female	27(41.54%)	21(38.1%)	6(60%)		
Cough		25(38.46%)	15(27.3%)	10(100%)	χ^2=18.9	**<0.001***
Cardiac Chest Pain		19(29.23%)	11(20%)	8(80%)	χ^2=14.72	**<0.001***
HR (beats/min)		93.32±11.08	93.11±11.25	94.5±10.56	t=0.363	0.718
RR (breaths/min)		22.08±5.03	20.71±4.06	29.6±2.59	t=6.66	**<0.001***
Temperature (°C)		37.9±0.72	37.91±0.74	37.8±0.6	t=0.459	0.648
SpO_2 (%)		90.91±6.94	93.65 ± 1.81	75.8±4.8	t=21.04	**<0.001***
SBP (mmHg)		113.1±11.1	115.35±6.89	100.4±19.6	t=4.44	**<0.001***
DBP (mmHg)		76.69±8.37	78.11±5.55	68.9±15.26	t=3.46	**<0.001***
Glycated HA1c (%)		5.13±0.37	5.14 ± 0.38	5.12 ± 0.34	t=0.128	0.899
Platelets ($x10^9$/L)		275.7±19.3	277.6±17.9	265.6±12	t=0.317	0.752
Hb (g/dL)		12.02 ± 1.56	12.05±1.58	11.84±1.51	t=0.387	0.700
WBCs ($x10^9$)		12.7 (7.6 – 17.6)	12.7 (7.6 – 17.6)	14.3 (9.6 – 18.4)	U=246	0.598
Troponin (ng/mL)		0.02 (0.01 – 0.16)	0.02 (0.01 – 0.04)	0.22 (0.09 – 0.42)	U=125.5	**0.007***
Creatinine (mg/dL)		1.1 (0.9 – 1.3)	1.1 (0.9 – 1.3)	1.2 (0.9 – 1.6)	U=209.5	**0.028***

Data are presented as mean ± SD or frequency (%). *Significant P value <0.05, t: Independent t-test, χ^2: Chi-square test, U: Mann-Whitney. HR: heart rate, RR: respiratory rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: hemoglobin, WBCs: white blood cells, SpO_2: Peripheral Oxygen Saturation.

In addition, severe COVID-19 cases had significantly lower EF (49.4 ± 11.29 vs. 61.69 ± 9.25), with a higher prevalence of abnormal EF (60% vs. 10.91%). GLS was significantly reduced in severe cases (-15.33 ± 3.63 vs. -19.36 ± 2.9), with a higher proportion of abnormal GLS (70% vs. 14.55%). LVSD measured with borderline and abnormal GLS was more frequent in severe cases (80% vs. 20%) (**Table 2).**

Table (2): Left Ventricular Systolic Function Assessed by Echocardiography in Moderate vs. Severe COVID-19 Patients

		Total (n = 65)	Moderate COVID-19 (n = 55)	Severe COVID-19 (n = 10)	Test Statistical	P value
EF		59.8±10.49	61.69±9.25	49.4±11.29	t=3.74	**<0.001***
Normal		53(81.5%)	49(89.09%)	4(40%)	χ^2=13.54	**<0.001***
Abnormal		12(18.5%)	6(10.91%)	6(60%)		
GLS		-18.74±3.3	-19.36±2.9	-15.33±3.63	t=3.85	**<0.001***
Abnormal		15(23.1%)	8(14.55%)	7(70%)	χ^2= 15.86	**<0.001***
Borderline		4(6.2%)	3(5.45%)	1(10%)		
Normal		46(70.8%)	44(80%)	2(20%)		
LVSD	**Abnormal+ borderline LV-GLS**	19(29.2%)	11(20.0%)	8 (80.0%)	χ^2=14.72	**<0.001***
	Normal	46(70.8%)	44(80%)	2(20%)		

Data are presented as mean ± SD or frequency (%). *Significant P value <0.05, t: Independent t-test, χ^2: Chi-square test, GLS: global longitudinal strain, GLS: normal ≤ –18%, borderline: –16% to –18%, abnormal: > –16%. EF: Ejection Fraction, normal > 50% and abnormal ≤ 50%. LVSD: left ventricular systolic dysfunction.

Significant correlations with GLS (%) included age (r = 0.260), systolic blood pressure (r = 0.561), diastolic blood pressure (r = 0.4971), respiratory rate (r = -0.278), SpO_2 (r = -0.402), troponin (r = 0.666), creatinine (r = 0.266), and EF (r = -0.868). Other variables showed non-significant correlations (**Table 3 and Figure 1).**

Table (3): Correlation between Global Longitudinal Strain (GLS) and Clinical and Laboratory Parameters

	GLS (%)	
	Pearson's r	**P value**
Age (years)	0.260	**0.036***
SBP (mmHg)	0.561	**< 0.001***
DBP (mmHg)	0.497	**<0.001***
RR (breaths/min)	-0.278	**0.024***
SpO_2 (%)	-0.402	**0.001***
Glycated HA1c (%)	-0.052	0.678
Platelets ($x10^9$/L)	-0.118	0.348
Hb (g/dL)	0.078	0.539
WBCs ($x10^9$)	0.004	0.977
Troponin (ng/mL)	0.666	**< 0.001***
Creatinine (mg/dL)	0.266	**0.032***
EF by M-mode (%)	-0.868	**<0.001***

*Significant P value <0.05, RR: respiratory rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: hemoglobin, WBCs: white blood cells, SpO_2: Peripheral Oxygen Saturation, EF: Ejection fraction, GLS: global longitudinal strain.

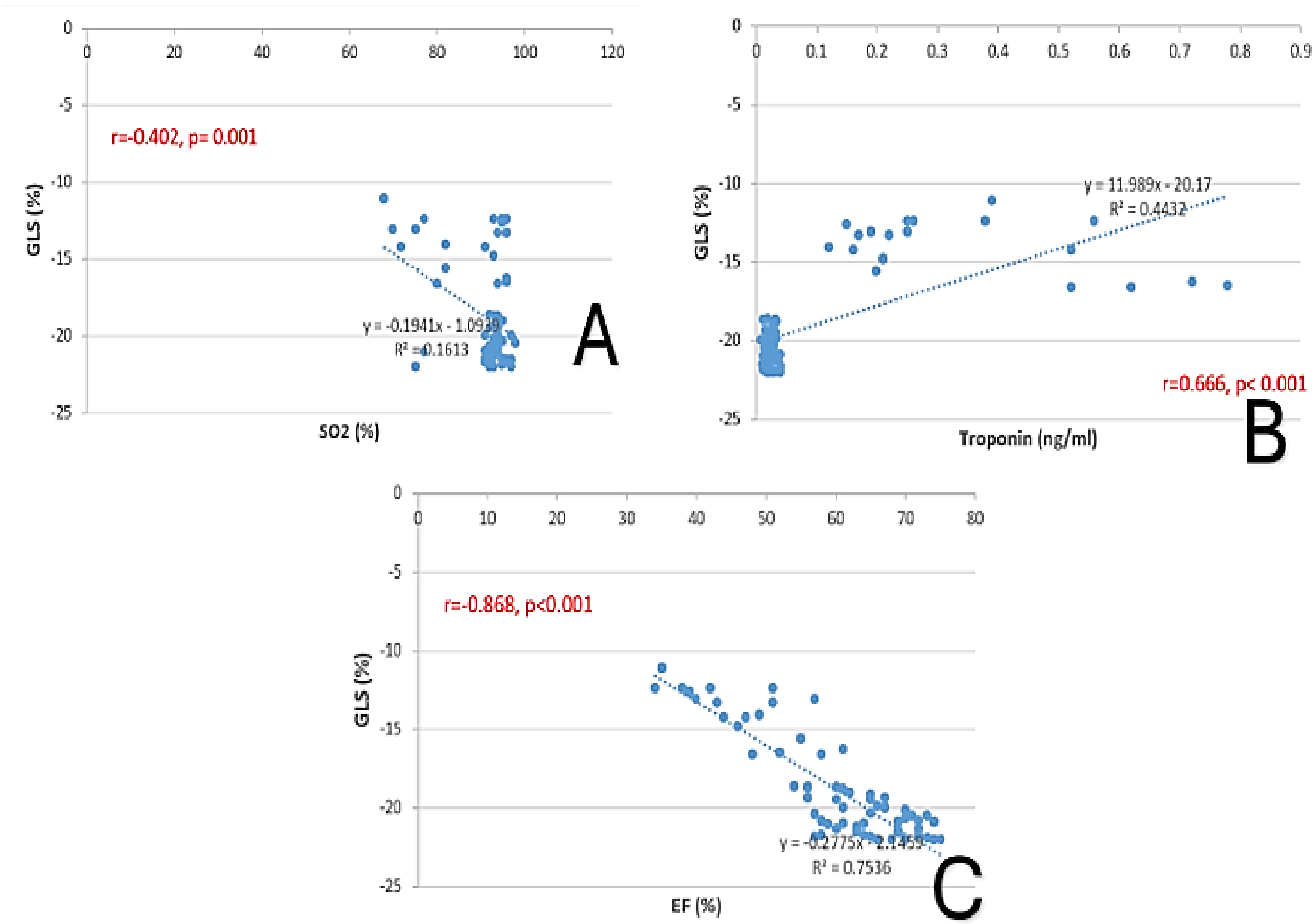

Figure 1: (A) Scatter diagram for negative correlation between GLS and SO_2 (%), (B) Scatter diagram for positive correlation between GLS and Troponin (ng/mL), (C) Scatter diagram for negative correlation between GLS and EF (%).

Both SpO_2 (OR = 16.62) and troponin (OR = 81.76) were shown to be independent significant predictors of abnormal GLS in the regression analysis. Notably, elevated troponin showed the strongest predictive value, indicating a link between myocardial injury and GLS impairment (**Table 4).**

Table (4): Logistic Regression Analysis of Predictors for Borderline and Abnormal Global Longitudinal Strain (GLS)

	Univariate regression analysis			Multivariate regression analysis		
	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
SpO_2 (%) <92.5	9.75	1.98- 47.9	**0.005***	16.62	1.05 - 263	**0.046***
SBP (mmHg) <114.5	9.33	2.2-37.9	**0.002***			
DBP (mmHg) <76.5	6.42	1.7-23.4	**0.005***			
Creatinine (mg/dL) >1.05	8.60	1.1-70.7	**0.046***			
Troponin (ng/mL) >0.08	47.70	8.6-265.1	**<0.001***	81.76	4.6 - 1449	**0.003***

*Significant as P value ≤0.05, CI: confidence interval. Variables excluded from multivariate model due to non-significance, SBP: systolic blood pressure, DBP: diastolic blood pressure, SpO_2: Peripheral Oxygen Saturation.

Troponin (ng/mL) demonstrated strong discriminating power between normal and abnormal GLS (AUC = 0.904, sensitivity = 86.7%, specificity = 95.7%, accuracy = 93.4%) and borderline GLS (AUC = 0.913, sensitivity = 75.0%, specificity = 73.9%, accuracy = 70.0%). In contrast, So_2 (%) showed weaker predictive ability for abnormal GLS (AUC = 0.663, accuracy = 50.8%) and borderline GLS (AUC = 0.652, accuracy = 60.0%), with lower specificity and positive predictive value (**Table 5**).

Table (5): ROC Analysis for Prediction of Borderline and Abnormal Global Longitudinal Strain (GLS)

	AUC	95% CI	Cut off point	Sensitivity	Specificity	PPV	NPV	Accuracy
Troponin (ng/mL)	0.904	0.79 - 0.98	>0.08	86.7%	95.7%	86.7	95.6	93.4%
SpO_2 (%)	0.886	0.78 - 0.98	<92.5	80.0%	70.0%	44.4	92.1	72.3%
SBP (mmHg)	0.845	0.71- 0.98	<114.5	73.3%	70.0%	42.3	89.7	70.7%
DBP (mmHg)	0.825	0.69 - 0.96	<76.5	73.3%	70.0%	42.3	89.7	70.7%
Creatinine (mg/dL) >1.05	0.623	0.45 - 0.79	>1.05	66.7%	40.0%	20.0	75.0	53.8%

AUC: Area under the curve, CI: Confidence interval, PPV: positive predictive value, NPV: negative predictive value., SBP: systolic blood pressure, DBP: diastolic blood pressure, SpO_2: Peripheral Oxygen Saturation.

DISCUSSION

Myocardial damage is one of the worst consequences that SARS-CoV-2 may cause to the cardiovascular system [18]. Systolic and diastolic RV and LV functions may be compromised, with right ventricular failure more prevalent in severely sick patients. Critically sick patients need TEE to quickly assess hemodynamic status and guide treatment [19].

Speckle-tracking echocardiography measures myocardial strain well, including GLS of the LV and strain parameters of the right ventricle (RV) [11].It plays a crucial role in diagnosing and predicting cardiac conditions. A 2DE is essential for detecting ongoing or new cardiac dysfunction in post-COVID patients with respiratory issues, as GLS is a highly sensitive marker that often declines before a measurable reduction in EF [20].

Our demographic study showed no significant age or gender differences amongst COVID-19 patients. While **Gherbesi *et al.*** [21] found no evidence of a strong relationship between age, gender, and COVID-19 strain distribution, **Bhatraju *et al.*** [22] observed significant correlations during hospitalization and up to three months later.

Regarding the vital signs, cough and cardiac chest pain were more prevalent in severe cases ($p < 0.001$), a result consistent with research by **Lombardi *et al.*** [23] that found that systemic inflammation and hypoxia often accompany respiratory symptoms and chest discomfort in patients with severe COVID-19 involving the heart. In addition, severe cases exhibited significantly higher respiratory rates and lower SpO_2 (both $p < 0.001$), reflecting more profound respiratory compromise.

Studies have linked hypoxia and tachypnea to increased myocardial stress, promoting cardiac injury and dysfunction [24]. In addition, our findings showed that severe cases had much lower systolic and diastolic blood pressures, which is in line with research suggesting that hemodynamic instability and hypotension are caused by COVID-19-induced vasodilation and systemic inflammation [25]. However, some studies, such as that by **Roshdy *et al.*** [26] found no significant blood pressure variations, suggesting potential variability in patient populations and treatment protocols.

While most test measures were unaffected, severe patients had increased troponin ($p = 0.015$) and creatinine ($P=0.028$). Excess troponin levels in COVID-19 patients with heart damage are related with cardiovascular disease, serious illness, mortality, and poor prognoses [27]. Increased creatinine is linked to severe illness, supporting previous evidence indicating renal failure in COVID-19 patients worsens cardiovascular consequences [28]. However, **Copur *et al.*** [29] discovered an elevated blood creatinine level, electrolyte problems, serum urea, and a substantially high incidence of acute kidney injury (AKI). Possible complicating factors, such as preexisting kidney disease, may explain why renal involvement is not the main cause of mortality or morbidity in COVID-19 patients.

Severe COVID-19 cases had considerably lower EF (49.4 ± 11.29 vs. 61.69 ± 9.25, $p < 0.001$) and greater prevalence of aberrant EF (60% vs. 10.91%, $p < 0.001$). **Çap *et al.*** [30] found that LVSD is prevalent in hospitalized COVID-19 patients and increases mortality rates. Severe cases had significantly lower GLS (-15.33 ± 3.63 vs. -19.36 ± 2.9, $p < 0.001$), along with a greater prevalence of abnormal and borderline GLS (70% vs. 14.55, $p = 0.004$). Our findings agree with **Shmueli *et al.*** [31]**, and De *et al.*** [32] who found decreased LVGLS in COVID-19-recovered patients.

Our findings support previous evidence that speckle-tracking echocardiography can detect early myocardial dysfunction before a significant reduction in EF occurs. Similarly, **Bhatia *et al.*** [33] found that COVID-19 patients exhibited subclinical cardiac dysfunction, as measured by decreased GLS, even if their EF was unaffected. Furthermore, a study by **Samy *et al.*** [18] found that almost all patients had symptoms of GLS impairment after contracting COVID-19, regardless of the intensity of their illness. A systematic echocardiographic study by **Szekely *et al.*** [34] reported significant GLS impairment in severe COVID-19 cases, reinforcing our findings.

Significant predictors of aberrant GLS were SpO_2 (OR = 16.62, $p = 0.046$) and troponin (OR = 81.76, $p = 0.003$), according to our regression analysis. The greatest predictive value for GLS impairment was elevated troponin (OR = 157.2, $p = 0.003$), suggesting that myocardial injury is the primary source of LV failure in severe COVID-19 patients. Supporting this, prior research has highlighted that increased troponin levels are a robust independent predictor of worse cardiac outcomes in COVID-19 [35].

Age ($r = 0.260$, $p = 0.036$), BP, respiratory rate, SpO_2, troponin, creatinine, and EF were all substantially linked with GLS. Progression of cardiac impairment is shown by the robust negative association between GLS and EF ($r = -0.868$, $p < 0.001$) in cases with severe COVID-19.

Troponin demonstrated strong predictive value for abnormal GLS (AUC = 0.904, sensitivity = 86.7%, specificity = 95.7%, accuracy = 93.4%), reinforcing its role as a key biomarker for myocardial injury in COVID-19. In contrast, SpO_2 showed weaker predictive ability (AUC = 0.663, accuracy = 50.8%), suggesting that while hypoxia contributes to cardiac dysfunction, it may not be a sole determinant of myocardial impairment.

CONCLUSION

This research demonstrates that limited LV dysfunction is prevalent in COVID-19 patients hospitalized, particularly those with advanced disease. The strong association between impaired GLS and troponin elevation underscores the importance of integrating STE echocardiographic assessment into routine clinical practice for COVID-19 management.

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