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Prognostic Value of Echocardiography in Recovered COVID-19 Patients: Review Article

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ABSTRACT

Global health is under danger due to COVID-19's overwhelming global spread. Despite the fact that the respiratory system is the primary organ affected by SARS-CoV-2, there is mounting evidence that it can also impact the cardiovascular system. A useful tool for assessing cardiovascular disease is echocardiography. It is affordable, broadly accessible, and offers data that can affect management. Leading international societies advice using only echocardiography when a potential clinical benefit exists, favoring focused assessments, and employing smaller portable equipment due to the danger of staff infection and equipment contamination during the procedure. Several investigations over the past few several different types of echocardiographic anomalies have been described for months in COVID-19 individuals. These findings are summarized in this review, which also discusses potential contributing mechanisms.

Keywords: COVID-19, Cardiovascular disease, Echocardiography, Review, Zagazig University.

INTRODUCTION

Impaired ventricular performance is linked to myocarditis, myocardial injury, arrhythmia, HF, venous thromboembolism, myocardial ischemia, and necrosis, all of which increase the risk of patient mortality. Numerous more direct or indirect cardiovascular problems are connected to COVID-19 infection [(1)].

Echocardiographic manifestations in COVID-19:

The 2DE is a crucial, noninvasive test that aids in determining hemodynamic and cardiac condition. Patients with COVID-19 who have multiple organ involvement, two-dimensional echocardiography (2DE) has become increasingly important, particularly in situations with Instability of hemodynamics Routine 2DE is not recommended for all patients due to the risk of infection, and there are presently no thorough 2DE studies in COVID-19 patients [(2)].

More COVID- 19 patients continue to report persistent symptoms, even weeks after the acute stage of a viral infection, such as lassitude, palpitations, impaired exercise tolerance, shortness of breath, chest pain, neurocognitive challenges, muscle pains and weakness, gloominess, anxiety, and other mental health problems [(3)]. In their investigation, ***Greenhalgh et al.*** classified this condition as post-acute. If symptoms persist for more than three months after the illness's beginning and extended if symptoms last for three to twelve weeks, COVID-19 [(4)]. Between 10% and 50% of instances are recorded in various studies, which is a significant variation in its reported occurrence [(5)]. Contrary to post-acute syndromes acquired after other serious illnesses, patients with the post-acute COVID-19 condition have been described with mild or severe manifestations of the disease, who did not require intensive care unit admission. Unknown are the causes of the post-acute COVID-19 syndrome. Some papers relate chronic viremia to relapse and/or reinfection with SARS-CoV-2, inadequate or nonexistent antibody responses, inflammatory and immunological reactions, deconditioning, and post-traumatic stress disorder. These conditions may increase the likelihood of myocardial damage and inflammation, which may lead to left ventricular dysfunction [(4)].

It has long been known that COVID-19 causes multiorgan involvement, and that cardiac dysfunction is linked to worse outcomes [(6)]. Due to the potential for disease transmission, although it is rarely used, 2DE is incredibly beneficial in this patient population [(7)]. It enables noninvasive assessment of the patient's hemodynamic and heart health, providing information on the severity of the illness [(6)]. Additionally, in these people, 2DE can be utilized to rule out obstructive diseases (such cardiac tamponade and pulmonary embolism) as well as hypovolemic shock (decreased cardiac output and collapsed IVC) [(8)].

Patients with COVID-19 have been proven to have worse prognoses when they have myocardial damage [(9)]. According to a recent report, the left heart of these patients may show myocardial involvement, segmental contraction abnormalities, or global hypokinesia as a result of hypoxia damage, respiratory strain, and inflammation [(10)] has demonstrated that a decrease in RVGLS (less than 23%) may have a predictive significance in COVID-19 patients, even though LVEF was not lowered. Then again, **Szekely *et al.*** [(11)] According to research, patients with myocardial damage and worse clinical circumstances had poorer RV function compared to those with normal troponin levels or moderate clinical symptoms. These patients' LV systolic performance did not differ considerably.

Another study revealed that patients with dilated and impaired RV function on echocardiography that was done had LV systolic function that was normal or hyperdynamic within 5 days of hospital admission [(12)].

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The severe patient group had lower values for LVEF, E wave, E/A ratios, and RV-FAC, but higher values for LVEDD, LVESD, and LA diameters. While TAPSE values were comparable in both groups, the severe group had larger RV, RA, and IVC diameters and greater sPAP.

Szekely *et al.* [11] found that valvular heart disease, LV diastolic dysfunction, and RV dilatation with or without dysfunction were the most common echocardiographic findings among individuals with COVID-19 infection. Three patients: 2 with mild aortic regurgitation and 1 with severe organic mitral regurgitation. The remaining 32 (or 32%) subjects had echocardiograms, and none of them had any abnormalities noted.

RV dilatation and dysfunction are common in COVID-19 pneumonia patients, and their presence is associated with a prothrombotic condition, inflammatory condition that is evident in higher D-dimer and CRP values. Many people with markedly reduced radial RV systolic function but relatively preserved longitudinal shortening have been identified by visual RV examination. This most likely explains why patients with impaired RV function have different objective assessments of FAC and tricuspid annular plane systolic excursion [12].

Patients with abnormal scans, as reported by ***Dweck et al.*** regarding echocardiographic results (n= 667) were older and more likely than individuals with normal scans (n= 549) to have valvular heart disease, heart failure, or a history of ischemic heart disease , but their rates of hypertension or diabetes mellitus were comparable. In both groups, the percentage of males was comparable. He also reported 479 (39%) patients with left ventricular anomalies, myocarditis in 35 (3%), takotsubo cardiomyopathy in 19, and new myocardial infarction in 36 (3%), according to echocardiographic data. 17, 12 and 9% of patients, respectively, were found to have mild, moderate, or severe left ventricular dysfunction. 397 individuals (33%) had right ventricular anomalies, with 19% having mild or moderate impairment and 6% having severe impairment. Less often reported conditions were right ventricular dilatation (15%), and elevated pulmonary artery pressures (8%) with a D shape left ventricle (4%). He also stated that 11 (1%) patients had cardiac tamponade, while 14 (1%) patients had endocarditis. One in seven patients (n= 182, 15%) had serious cardiac disease. This was described as significant cardiac tamponade or left or right ventricular dysfunction. Abnormalities on the echocardiogram were more likely to be found in patients who had chest pain with ST-segment elevation (71%), increased biomarkers (69%), left ventricular failure (60%), right ventricular failure (60%), or other clinical situations that requiring echocardiogram (72%). A sensitivity analysis limited to the 813 patients with confirmed COVID-19 infection revealed the following 50% of patients (409/813), or 1 in 7 patients (n= 119, 15%), had abnormal echocardiograms [13]. The According to **Sattarzadeh Badkoubeh *et al.*** [14] analysis of data from the mortality group, the mean sPAP was considerably greater in patients who died (mean sPAP, 32.78 mmHg versus 27.32 mmHg, P value 0.007). In addition, those with LV diastolic dysfunction were more likely to pass away than those with adequate LV diastolic function (P value 0.055). Significant valvular heart disease was associated with elevated sPAP concentrations (moderate or greater severity). According to the findings by **Khan *et al.*** [15] the majority of patients with abnormal transthoracic echocardiography (TTE) had valvular heart disease but no further pathology.

Tricuspid regurgitation (TR), the most common aberration, was seen in 26 (56.5%) individuals, followed by aortic regurgitation in 13 (28.3%) patients, and mitral regurgitation in 12 (26.1%) patients. Atrial dilatation was present in 14 (30.4%) individuals, and right ventricular (RV) systolic dysfunction was present in 12 (26.1%) patients. Most individuals had moderate. The average pulmonary artery systolic pressure was (PASP) being 47 (SD 12) mmHg. 11 of the 22 individuals out of 66 who had previously undergone a transthoracic echocardiography (TTE) exhibited worse alterations.

According to **Peng *et al.*** [16], abnormal echocardiogram findings were associated with the severity of the illness and its subsequent cardiovascular consequences.

Numerous studies have used troponin levels, ECG abnormalities, and echocardiography features to explain the COVID-19-related cardiac injury in detail [13-15]. There is no link between troponin and abnormal TTE, and the underlying causes of cardiac dysfunction as they relate to abnormal TTE are probably complicated. Despite the fact that direct virus invasion of the myocardium is one cause, Type II myocardial infarction (respiratory failure and reduced oxygen perfusion-related), cytokine storm-induced microangiopathy and stress cardiomyopathy are likely to be the most prevalent contributory causes [17,18,19].

According to **Tudoran *et al.*** [20] the outcomes of TTE evaluation varied greatly depending on how many weeks had passed since the acute sickness. Therefore, only 30 patients were assessed between 4 and 8 weeks following COVID-19, and 12 of them were diagnosed with PH, LVD, and type II or type III diastolic dysfunction. Eight individuals had a little pericardial effusion (between 3.5 and 5 mm), whereas seven others had a pericardium that was larger than 4 mm. Only type I or type II diastolic dysfunction was reported in seven patients. After 9 to 11 weeks of recovery, the remaining 120 patients were assessed, and only 2 of them had PH,

one of whom also had a diminished LV systolic function. Diastolic dysfunction was identified in 14 individuals, all of whom had high BMIs and concomitant LVH. In 9.33% of these individuals, an enlarged pericardium of greater than 2 mm and between 2 and 4 mm was detected. According to some authors, the majority of patients with post-acute COVID-19, particularly those with mild or moderate forms, recover completely over time, often within 4 to 6 weeks; but, according to others, symptoms can continue for 12 weeks or longer, earning the moniker "long COVID-19" [4, 21]. While **Turdan *et al*.** [20] discovered that the degree of cardiac abnormalities decreased in tandem with the passage of time after the diagnosis of COVID-19, the severity of cardiac alterations did not diminish. They also stated that the current assessment (like in PH, RVD, or decreased LV systolic function) demonstrated that other acute aspects, such as pericardial effusion, were discovered sooner within 6 weeks after the acute illness, while other abnormalities, such as diastolic dysfunction, and thickened blood vessels were found in the later stages of the illness. Regular ECG monitoring is critical for individuals with COVID-19, since early recognition of cardiac comorbidity can reduce the likelihood of adverse outcomes.

As in this instance, **Takotsubo cardiomyopathy** is usually accompanied by substantial QTc prolongation and the potential for life-threatening arrhythmias. 8 This risk may be exacerbated by the fact that many COVID-19 patients are treated with medications that prolong QT, such as chloroquine or haloperidol. **Takotsubo cardiomyopathy**, in addition to producing left ventricular failure, can also produce cardiogenic shock. When COVID-19 patients develop hemodynamic issues, echocardiography is indicated to rule out **Takotsubo cardiomyopathy**, myocarditis, or ischemia-induced heart failure [22].

Freaney *et al*. [23] established a link between LV diastolic dysfunction and Mortality and illness progression in COVID-19 patients; nevertheless, additional research with a bigger sample size is required to properly estimate this issue, given the significant concerns surrounding the higher prevalence of HFpEF in coronavirus-infected patients.

Pay close attention to RV systolic performance and the differential diagnosis of pulmonary hypertension in critically ill ICU patients (such as pulmonary thromboembolism). Many people with modest symptoms who are stable do not benefit from echocardiographic data [14].

Multiple mechanisms, including the increased afterload brought on by acute respiratory distress syndrome and pulmonary embolism, the cardiodepressant effects of cytokines, and direct viral injury to the right ventricle, were proposed to contribute to the right ventricular dysfunction of these patients in a review by **Park *et al*.** [24].

Inflammation, hypoxia, and hypercapnia result in alveolar injury, pulmonary capillary damage, and an increase in pulmonary vascular resistance in the lungs of COVID-19 patients. In consequence, RV afterloads increase. Inadequate MV settings may also contribute to increases in pulmonary arterial pressure and RV afterload [16]. The primary functions of the right side of the heart are circulation and oxygenation, which have direct implications for the clinical condition of COVID-19 patients. In ARDS patients, the development of RV dysfunction is a vicious cycle that worsens. In several patient populations, it has been demonstrated that RV dysfunction is a significant predictor of mortality. A considerable majority of persons with severe COVID-19 also required MV, because it is well recognized that MV has a hemodynamic influence on ventricular function. MV exacerbates right-heart dysfunction and pulmonary artery pressure increase. To avoid the adverse effects of MV on ventricular function, the standard should be to delay MV in each patient as long as possible [2].

According to ASE guidelines for COVID-19 patients, 2DE should only be performed if it would significantly impact the clinical course and should not be repeated if there have been no discernible changes in the patient's condition. When evaluating heart failure, arrhythmia, alterations in the ECG, and newly discovered cardiomegaly in COVID-19 patients, it is advised to use 2-DE. [16].

Use of personal protection equipment is advised, as is keeping contact times to a minimum. A comprehensive assessment of the patient's clinical condition and the results of all other tests should be used to decide whether to undertake 2DE. For patients who have been identified as having COVID-19 positivity, non-urgent echocardiography should be delayed [25].

CONCLUSION

Echocardiography is required in recovered COVID-19 patients as early detection of cardiac comorbidities may reduce unfavorable outcomes and prevent long-term cardiovascular sequelae. The COVID-19 infection has multiple direct or indirect cardiovascular effects.

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