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enabled through remote communication solutions can remove sonographer uncertainty in determining when enough images have been acquired to answer the clinical question, thereby increasing efficiency. Such technological solutions, along with other strategies to limit sonographer exposure, may be valuable tools for adhering to the American Society of Echocardiography’s recommendations for performing echocardiograms during the COVID-19 pandemic.1,3,4

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Reduced Cardiac Function by Echocardiography in a Minority of COVID-19 Patients 3 Months after Hospitalization

Studies have shown cardiac abnormalities in a majority of hospitalized patients with ongoing COVID-19 disease.1 There are, however, conflicting results regarding ventricular function in patients recovered from COVID-19. One recent echocardiographic study showed abnormalities in ventricular function in nearly one-third of patients after 3 months.2 Cardiovascular magnetic resonance studies have revealed a high frequency of cardiac involvement in patients recovered from COVID-19.3

All 92 patients were recruited prospectively at the time of hospitalization as a part of the NOR-Solidarity study evaluating the effect of repurposed antiviral drugs on hospitalized adult (≥18 years) COVID-19 patients.4 Fourteen Norwegian hospitals did echocardiographic examinations of the patients 3 months after hospitalization. All measurements were performed at the core laboratory at Oslo University Hospital Rikshospitalet, according to current guidelines.5 Intra- and interobserver reproducibility for left ventricular (LV) global longitudinal strain (GLS) in 10 random patients showed intraclass correlation coefficients of 0.90 (P = .001) and 0.94 (P < .001). Thirty-five healthy individuals matched for age and gender were used as controls.

The COVID-19 patients were 59 ± 13 years old (69% male). Twenty-five percent had hypertension, 16% had diabetes, and 16% had chronic heart disease prior to COVID-19.

Three months after hospitalization, all patients had normal LV ejection fraction (LVEF ≥ 53%). In the COVID-19 patients as a whole, LV GLS was reduced compared with the control group (–18.6% ± 2.2% vs –20.1% ± 2.0%, P = .001), but only 14 patients experienced LV GLS < –17%. Of these, eight patients had LV hypertrophy, including four with known hypertension. In the six remaining patients, reduced LV GLS could not be attributed to hypertrophic or any other known premorbid cardiac diseases.

During hospitalization, 18 (20%) patients went through the intensive care unit, but only three needed mechanical ventilation. There was no difference in frequency of impaired LV GLS in intensive care unit patients compared with non-intensive care unit patients (3/18 vs 11/74, P = .344). We could not find any significant differences in LV GLS in the antiviral treatment groups (remdesivir –18.6% ± 2.3%, hydroxychloroquine –18.6% ± 2.3%) compared with controls (–18.5% ± 2.2%, P = 1.00).

Right ventricular (RV) function was normal in all COVID-19 patients and was similar to the control group when assessed by fractional area change and free wall longitudinal strain but was slightly lower when assessed by tricuspid annular plane systolic excursion. The results at 3 months are detailed in Table 1.

Table 1 Echocardiographic data in 92 patients 3 months after recovery from COVID-19 compared with healthy individuals

| Study patients (n = 92) | Healthy individuals (n = 35) | P value |
|------------------------|-----------------------------|---------|
| LVEF, %                | 63 ± 6                      | 61 ± 6  | .241   |
| LV GLS, %              | –18.6 ± 2.2                 | –20.0 ± 2.2 | .001 |
| LVEDd, cm/m²           | 2.4 ± 0.3                   | 2.5 ± 0.3 | .247   |
| LVEDV, mL              | 120 ± 29                    | 130 ± 25 | .144   |
| LVESV, mL              | 47 ± 15                     | 50 ± 12  | .496   |
| LAVi, mL/m²            | 27.9 ± 7.8                  | 22.3 ± 6.4 | .245  |
| RV FAC, %              | 48 ± 7                      | 47 ± 6  | .519   |
| RV FwLS, %             | –28.4 ± 4.6                 | –28.3 ± 3.9 | .974 |
| TAPSE, cm              | 2.3 ± 0.3                   | 2.4 ± 0.2 | .022   |
| E/e                    | 8.4 ± 2.4                   | 7.4 ± 2.2 | .082   |
| Estimated SPAP, mm Hg  | 29.0 ± 7.5                  | 22.8 ± 5.5 | .002   |

Data are presented as mean ± SD.

| FAC: Fractional area change; FwLS, free wall longitudinal strain; LAVi, left atrial volume index; LVEDd, LV end-diastolic diameter (normalized by body surface area [cm/m²]); LVEDV, LV end-diastolic volume; LVESV, LV end-systolic volume; SPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion.

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After 3 months, 12 patients (13%) had elevated high-sensitivity cardiac troponin (>99% percentile), but only one of these had decreased LV GLS.

In summary, our prospective study showed normal LV function by LVEF in all patients 3 months after hospitalization for COVID-19. However, LV GLS was reduced in 15% of the patients. Furthermore, we could not find any relationships between reduced GLS and disease severity (treatment at intensive care unit) or elevated high-sensitivity cardiac troponin after 3 months. The majority of the patients with reduced GLS had arterial hypertension prior to COVID-19 infection; this might explain the impaired LV function, but we cannot exclude that this was caused by COVID-19. Cardiac function might potentially be influenced by the antiviral medications, but we could not find such effect. Our study was, however, not originally designed for this purpose.

The number of patients is small and increases the risk of both type I and type II statistical errors. Another limitation is lack of comparison with echocardiographic examination during hospitalization. Our study underscores the need for more studies on long-term cardiovascular consequences after COVID-19.

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