



openheart Echocardiography in suspected coronavirus infection: indications, limitations and impact on clinical management

Megan M Sheehan,¹ Yoshihito Saijo ,² Zoran B Popovic,³ Michael D Faulx ³

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¹EC-10, Cleveland Clinic Lerner College of Medicine, Case Western Reserve University, Cleveland, Ohio, USA

²Cardiovascular Medicine, Tokushima University Hospital, Tokushima, Japan

³Cardiovascular Medicine, Cleveland Clinic Main Campus Hospital, Cleveland, Ohio, USA

Correspondence to
Dr Michael D Faulx; faulxm@ccf.org

ABSTRACT

Objectives To describe the use of echocardiography in patients hospitalised with suspected coronavirus infection and to assess its impact on clinical management.

Methods We studied 79 adults from a prospective registry of inpatients with suspected coronavirus infection at a single academic centre. Echocardiographic indications included abnormal biomarkers, shock, cardiac symptoms, arrhythmia, worsening hypoxaemia or clinical deterioration. Study type (limited or complete) was assessed for each patient. The primary outcome measure was echocardiography-related change in clinical management, defined as intensive care transfer, medication changes, altered ventilation parameters or subsequent cardiac procedures within 24 hours of echocardiography. Coronavirus-positive versus coronavirus-negative patient groups were compared. The relationship between echocardiographic findings and coronavirus mortality was assessed.

Results 56 patients were coronavirus-positive and 23 patients were coronavirus-negative with symptoms attributed to other diagnoses. Coronavirus-positive patients more often received limited echocardiograms (70% vs 26%, $p=0.001$). The echocardiographic indication for coronavirus-infected patients was frequently worsening hypoxaemia (43% vs 4%) versus chest pain, syncope or clinical heart failure (23% vs 44%). Echocardiography changed management less frequently in coronavirus-positive patients (18% vs 48%, $p=0.01$). Among coronavirus-positive patients, 14 of 56 (25.0%) died during hospitalisation. Those who died more often had echocardiography to evaluate clinical deterioration (71% vs 24%) and had elevated right ventricular systolic pressures (37 mm Hg vs 25 mm Hg), but other parameters were similar to survivors.

Conclusions Echocardiograms performed on hospitalised patients with coronavirus infection were often technically limited, and their findings altered patient management in a minority of patients.

INTRODUCTION

The COVID-19 pandemic has caused over 569 000 deaths in the USA as of 26 April 2021.¹ Pulmonary consequences of this disease, including acute respiratory distress

Key questions

What is already known about this subject?

► While it is known that echocardiographic markers of right ventricular (RV) impairment (RV enlargement and systolic dysfunction, increased estimated RV systolic pressure and abnormal right atrial and ventricular strain imaging) are predictive of increased mortality in patients hospitalised with COVID-19-related pulmonary disease, indications for echocardiography in patients with COVID-19 or how the findings of these echocardiograms, which are often limited, point-of-care studies, impact patient management in real time compared with those without COVID-19 is less well characterised.

What does this study add?

► This study adds evidence that echocardiograms performed in COVID-19-infected patients are often of limited technical quality, and their results alter patient care in a minority of cases, particularly when the indication for the study lacks cardiac-specific findings or complaints.

How might this impact on clinical practice?

► These findings might impact clinical practice by suggesting that echocardiography in hospitalised patients with COVID-19 infection be used more judiciously, with the use of predefined indications and employing more complete studies performed by trained technicians wearing appropriate personal protective equipment. Such an approach might conserve resources and protect echocardiographic providers by limiting the number of 'low-yield' studies.

syndrome (ARDS), are significant contributors to morbidity and mortality.^{2 3} Observational studies suggest that COVID-19 may also affect the heart, leading to myocarditis, arrhythmias and acute coronary syndrome.⁴⁻⁶ The role of diagnostic cardiac testing in the management of these patients is still unclear.

Echocardiography is readily accessible and may be used to assess for functional cardiac injury and to monitor complications of both disease and treatment in patients with

COVID-19. A study of 120 patients with COVID-19 by Li *et al* found that right ventricular (RV) longitudinal strain was a strong predictor of acute heart injury, ARDS and mortality.⁷ A large prospective international survey of 1216 COVID-19-positive patients from Dweck *et al* found that 55% had an abnormal echocardiogram.⁸ Echocardiographic findings changed management in only 33% of patients, most frequently in patients with pre-existing cardiac disease or elevated biomarkers. The American Society of Echocardiography recommends that echocardiography only be performed for patients infected with COVID-19 if expected to provide clinical benefit.⁹ Deciding which patients may benefit from echocardiography remains a challenge.

In this study, we characterise echocardiographic indications and parameters in a series of patients tested for COVID-19. We compare findings and management changes in patients with the disease to those who tested negative, with the goal to identify patients with COVID-19 who are more likely to benefit from echocardiography.

METHODS

Study population

This sample was taken from a prospective registry of all patients tested for COVID-19 at a large tertiary care centre. We included adult patients who were tested for COVID-19 from 11 March 2020 to 20 May 2020 on admission to the hospital and received an echocardiogram during their index COVID-19 hospitalisation. The patients were further grouped based on testing results. The patients were considered COVID-19-positive if confirmed by nasopharyngeal or oropharyngeal swabs, run with the use of the Centers for Disease Control and Prevention assay using Roche MagNA Pure extraction (Roche Life Science) and ABI 7500 DX PCR machines (Applied Biosystems/Thermo Fisher Scientific).

Clinical data

Demographics, comorbidities, presenting symptoms, medications, laboratory data within 24 hours of admission and outcomes were extracted from the electronic medical record. Laboratory testing measured on admission included blood urea nitrogen, creatinine, sodium, potassium, bicarbonate, glucose, white blood cell count, haemoglobin, haematocrit, platelet count, albumin, aspartate aminotransferase, alanine aminotransferase, C reactive protein, lactate, procalcitonin, interleukin-6, N-terminal pro b-type natriuretic peptide, troponin T, high-sensitivity troponin, creatine kinase, creatine kinase myocardial band, d-dimer, fibrinogen and ferritin. Outcomes of interest included need for intensive care, oxygen therapy, mechanical ventilation, shock, renal failure, use of vasopressors and mechanical circulatory support.

The index echocardiogram was identified for each patient as the first echocardiogram performed during hospitalisation. Echocardiogram type was classified

as complete, limited technician study or point of care. Echocardiogram indication was also extracted from the medical record and divided into six categories: abnormal biomarkers, shock, cardiac symptoms, arrhythmia, worsening hypoxaemia or clinical deterioration. Patients could have multiple indications. Cardiac symptoms included syncope, physical signs of heart failure and cardiac arrest. An echocardiogram was considered to have changed management if transfer to intensive care, medication changes, alterations in ventilation parameters or subsequent cardiac procedures occurred within 24 hours in response to results and was determined by chart review.

Echocardiographic assessment

All patients underwent comprehensive baseline echocardiograms using commercial instruments (Philips Medical Systems, Bothell, Washington, USA; General Electric, Milwaukee, Wisconsin, USA). The extracted conventional echocardiographic parameters were measured, according to the current American Society of Echocardiography guidelines.^{10 11} Echocardiographic analysis was completed using Syngo Dynamics (Siemens Healthcare, Erlangen, Germany). Left ventricular (LV) and left atrial (LA) volumes were measured using a modified biplane Simpson's method, and volume indexes were calculated as volume divided by body surface area. The peak early diastolic velocity and the peak early diastolic of mitral annular tissue motion velocities were measured. The ratio of mitral early inflow velocity to early mitral annular velocity (E/e') ratio was calculated. Tricuspid lateral annular systolic velocity was assessed using tissue Doppler imaging from the apical four-chamber view. Tricuspid annular plane systolic excursion was measured as the systolic displacement of the tricuspid lateral annulus, recorded on M-mode imaging. Pulmonary artery systolic pressure was estimated from the peak velocity of tricuspid regurgitation jet using the modified Bernoulli equation plus right atrial (RA) pressure evaluated from the inferior vena cava size and collapsibility.

Speckle tracking echocardiography (STE)

Two-dimensional STE was used to characterise global longitudinal strain (GLS). Images for LV-GLS were acquired from apical 4ch, 2ch and 3ch views, and images for LA-GLS, RV-GLS and RA-GLS were acquired from apical 4ch view.¹² All images were analysed in a blinded manner, offline using vendor-independent STE software (Velocity Vector Imaging V.2; Siemens, Mountain View, California, USA). For each of the individual apical views, tracking was visually inspected throughout the systole to ensure adequate border tracking, and the endocardial contours were adjusted manually when necessary. The endocardial wall was divided into 6 segments in each view (18 segments for LV and 6 segments for LA, RV and RA), and the case was excluded if it was not feasible to track 2 or more segments in each view. In the setting of LV and RV, negative peak strain value of the average of segmental

strain curves was considered LV-GLS and RV-GLS, respectively. In the setting of LA and RA, zero strain reference was set at the R wave in the ECG, and positive peak strain value of the average of segmental strain curves was considered LA-GLS and RA-GLS, respectively. When tracing the borders of the LA endocardium, the confluences of the pulmonary veins and the LA appendage were excluded. All measurements were made by an experienced investigator blinded to clinical information.

Statistical analysis

Baseline data are presented as median (IQR), mean (SD) and number (percentage) where appropriate. Categorical variables were compared using the χ^2 test or Fisher's exact test, and continuous variables were compared using Student's t-tests or Wilcoxon rank-sum test. Statistical testing was done using R V.4 (R Foundation for Statistical Computing, Vienna, Austria). A two-sided p value of <0.05 was considered to be statistically significant.

Patient and public involvement

Our study involved data taken from a large prospective registry of patients hospitalised at our institution for suspected COVID-19 infection. Given the observational nature of our study, we did not directly involve patients or the public in its overall design.

RESULTS

A total of 79 patients were included in the study, and 56 (71%) of these tested positive for COVID-19. Compared with those who tested negative, patients with COVID-19 were less likely to smoke (5.4% vs 34.8%, $p=0.001$) but were otherwise similar in age, sex, ethnicity and comorbidities (table 1). Of those who tested negative for COVID-19, five (22%) were diagnosed with bacterial pneumonia; five (22%) were diagnosed with another infectious process; one (4%) was diagnosed with viral pneumonia; four (17%) were diagnosed with non-infectious pulmonary processes; seven (30%) were diagnosed with heart failure exacerbation; and eight (35%) were diagnosed with another cardiac process such as tamponade.

Patients with COVID-19 had lower troponin T (0.03 ng/mL vs 0.13 ng/mL, $p=0.001$) and high-sensitivity troponin (33 ng/L vs 72 ng/L, $p=0.02$) at admission compared with those who tested negative (table 2). They also had lower lactate levels (1.6 mmol/L vs 2.8 mmol/L, $p=0.02$) and white blood cell counts ($6.1 \times 10^9/L$ vs $10.6 \times 10^9/L$, $p=0.002$). However, they were more likely to show pneumonia on a chest X-ray (70% vs 22%, $p<0.001$) and more often required oxygen therapy (89% vs 61%, $p=0.009$). Patients with COVID-19 had longer lengths of stay in both the hospital (17.7 days vs 6.1 days, $p=0.002$) and the intensive care unit (15.9 days vs 5.0 days, $p<0.001$).

Patients with COVID-19 more often received limited technician echocardiograms (70% vs 26%, $p=0.001$) later in their admission (median time from admission

Table 1 Demographic information and comorbidities stratified by COVID-19 status

	COVID-19-negative	COVID-19-positive	P value
Demographics			
N	23	56	
Age (mean \pm SD)	65.7 \pm 18.2	62.5 \pm 15.2	0.42
Gender (% male)	17 (73.9)	37 (66.1)	0.68
Ethnicity (%)			0.65
Black	9 (39.1)	25 (44.6)	
Caucasian	13 (56.5)	25 (44.6)	
Hispanic/Latino	1 (4.3)	4 (7.1)	
More than one	0 (0)	2 (3.6)	
Alcohol use			0.25
None	10 (43.5)	34 (60.7)	
Moderate (<14 drinks/week)	9 (39.1)	18 (32.1)	
Excessive (>14 drinks/week)	4 (17.4)	4 (7.1)	
Smoking			0.001
Never	5 (21.7)	29 (51.8)	
Former	10 (43.5)	24 (42.9)	
Active	8 (34.8)	3 (5.4)	
Drug use (%)	3 (13)	8 (14.3)	>0.99
Comorbidities (%)			
Cardiovascular disease	14 (60.9)	25 (44.6)	0.29
CAD	8 (34.8)	10 (17.9)	0.18
HFrEF	6 (26.1)	9 (16.1)	0.47
HFpEF	5 (21.7)	6 (10.7)	0.35
Aortic valve disease	4 (17.4)	3 (5.4)	0.20
Mitral valve disease	2 (8.7)	4 (7.1)	>0.99
Atrial arrhythmias	8 (34.8)	12 (21.4)	0.34
Ventricular arrhythmias	2 (8.7)	2 (3.6)	0.71
PAD	2 (8.7)	4 (7.1)	>0.99
Aortopathy	1 (4.3)	1 (1.8)	>0.99
Pulmonary disease	8 (34.8)	17 (30.4)	0.91
Stroke	4 (17.4)	4 (7.1)	0.33
Hypertension	14 (60.9)	37 (66.1)	0.86
Diabetes	5 (21.7)	19 (33.9)	0.42
Cancer history	5 (21.7)	9 (16.1)	0.78
CKD	5 (21.7)	15 (26.8)	0.85
Gastrointestinal disease	6 (26.1)	15 (26.8)	>0.99
Neurological disease	4 (17.4)	6 (10.7)	0.24
Psychiatric disease	10 (43.5)	11 (19.6)	0.06
Sleep apnoea	3 (13)	11 (19.6)	0.71

CAD, coronary artery disease; CKD, chronic kidney disease; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; PAD, peripheral arterial disease.

Table 2 Laboratory testing and outcomes stratified by COVID-19 status

	COVID-19-negative	COVID-19-positive	P value
Admission lab values, median (IQR)			
BUN (mg/dL)	25 (20–44)	24.5 (15–45)	0.65
Creatinine (mg/dL)	1.37 (0.92–2.02)	1.42 (0.91–1.94)	0.74
GFR (mg/min/1.73 m ²)	31.5 (23.5–48.0)	46 (30–60)	0.09
Sodium (mmol/L)	136 (134.5–139.0)	136.5 (134.8–139.0)	0.82
Potassium (mmol/L)	4.2 (3.85–4.65)	4.2 (3.9–4.5)	0.66
Bicarbonate (mmol/L)	21 (19–24)	23 (20–25)	0.46
Glucose (mg/dL)	136 (110.4–232.5)	131 (109.5–169.0)	0.27
WBC ($\times 10^9$ /L)	10.61 (8.25–14.52)	6.1 (4.6–8.4)	0.002
Haemoglobin (g/dL)	12.5 (10.95–14.25)	12.9 (10.9–14.2)	0.67
Haematocrit (%)	37.8 (33.6–43.35)	39.3 (35.0–42.5)	0.78
Platelets (k/uL)	206 (175.5–250)	186.5 (139.8–234.8)	0.19
Albumin (g/dL)	3.8 (3.4–4.0)	3.5 (3.1–3.8)	0.08
AST (U/L)	30 (24.5–64.0)	39.5 (31.9–74.5)	0.22
ALT (U/L)	20 (17.5–37.0)	26.5 (19–41)	0.60
CRP (mg/L)	6.1 (3.6–13.3)	10.4 (5.0–16.9)	0.41
Lactate (mmol/L)	2.8 (1.6–3.95)	1.6 (1.2–2.3)	0.02
Procalcitonin (ng/mL)	1.39 (0.75–2.92)	0.34 (0.18–0.66)	0.04
IL-6 (pg/mL)	115.6 (67.8–19 834.25)	62 (20.5–120.1)	0.39
NT Pro-BNP (pg/mL)	3712 (305–9787)	567 (181–2771)	0.11
Troponin T (ng/L)	0.13 (0.04–0.4)	0.03 (0.01–0.05)	0.001
Hs-troponin (ng/L)	72 (65.0–268.5)	33 (14–56)	0.02
Creatine kinase (U/L)	134.5 (81.25–238)	230 (98–461)	0.31
CK-MB (ng/mL)	3.3 (2.3–26.65)	3.8 (2.2–4.5)	0.56
D-dimer (ug/mL)	2840 (2150–8890)	1250 (610–3300)	0.08
Fibrinogen (mg/dL)	509 (343–679)	541 (434.5–667.5)	0.64
Ferritin (ug/L)	294.5 (167.62–506.35)	704 (459–1470)	0.13
Hospitalisation characteristics			
Hospitalisation required (%)	23 (100)	56 (100)	>0.99
Pneumonia on CXR (%)	5 (21.7)	39 (69.6)	<0.001
Oxygen therapy	14 (60.9)	50 (89.3)	0.009
ICU required	22 (95.7)	43 (76.8)	0.10
ICU length of stay (mean \pm SD)	5.0 \pm 4.1	15.9 \pm 11.7	<0.001
NIPPV (%)	2 (8.7)	7 (12.5)	0.92
Mechanical ventilation (%)	9 (39.1)	33 (58.9)	0.18
Ventilation length (days) (mean \pm SD)	6 \pm 5.9	13.9 \pm 11.8	0.06
Prone ventilation (%)	0 (0)	12 (21.4)	0.04
Dialysis	2 (8.7)	16 (28.6)	0.11
Shock (%)	13 (56.5)	31 (55.4)	>0.99
Vasoactive drips (%)	16 (69.6)	33 (58.9)	0.53
Mechanical circulatory support	3 (13)	4 (7.1)	0.69
Death (%)	8 (34.8)	14 (25)	0.55

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CK-MB, creatine kinase myocardial band; CRP, C reactive protein; CXR, chest X-ray; GFR, glomerular filtration rate; ICU, intensive care unit; IL-6, interleukin-6; NIPPV, non-invasive positive-pressure ventilation; NT Pro-BNP, N-terminal pro b-type natriuretic peptide; WBC, white blood cell.

Table 3 Echocardiographic indications and parameters stratified by COVID-19 status

	COVID-19-negative		COVID-19-positive		P value
N	Available results (out of 23)		Available results (out of 56)		
Echocardiogram type	23		56		0.001
Complete		14 (60.9)		12 (21.4)	
Limited technician		6 (26.1)		39 (69.6)	
Point of care		3 (13)		5 (8.9)	
Echocardiogram indication	23		56		
Time from admission to echocardiogram (days)	23	1 (1–1)	56	2 (1.0–4.3)	0.003
Cardiac-specific					
Abnormal biomarker		4 (17.4)		9 (16.1)	>0.99
Cardiogenic shock		3 (13)		3 (5.4)	0.48
Cardiac signs		10 (43.5)		13 (23.2)	0.13
Arrhythmia		2 (8.7)		5 (8.9)	>0.99
Not cardiac-specific					
Worsening hypoxaemia		1 (4.3)		24 (42.9)	0.002
Clinical deterioration		10 (43.5)		20 (35.7)	0.70
Change in management	23	11 (47.8)	56	10 (17.9)	0.01
LVEF % (median, IQR)	22	49 (31.5–60.0)	56	55 (47.3–60.0)	0.09
≥50		11 (47.8)		38 (67.9)	
40–49		4 (17.4)		11 (19.6)	
30–39		1 (4.3)		3 (5.4)	
<30		6 (26.1)		4 (7.1)	
Diastolic function	10		43		0.80
Grade 1		6 (26.1)		21 (37.5)	
Grade 2		3 (13)		7 (12.5)	
Grade 3/4		1 (4.3)		2 (3.6)	
RV size	19		52		0.18
Normal		17 (73.9)		42 (75)	
Mildly dilated		0		7 (12.5)	
Moderately dilated		2 (8.7)		3 (5.4)	
Severely dilated		0 (0)		1 (1.8)	
RV systolic function	18		51		0.25
Normal		11 (47.8)		39 (69.6)	
Mildly reduced		3 (13)		8 (14.3)	
Moderately reduced		3 (13)		4 (7.1)	
Severely reduced		1 (4.3)		1 (1.8)	
RVSP	13	36 (29–52)	33	28 (21–40)	0.35
Strain %					
LV strain	17	–11.8 (–15.9 to –6.3)	42	–14.5 (–16.3 to –11.6)	0.12
LA strain	12	15.9 (8.2–26.8)	31	31.4 (19.2–38.1)	0.07

Data displayed as median (IQR) or number (%).

LA, left atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; RV, right ventricular; RVSP, right ventricular systolic pressure.

to echocardiogram 2 days vs 1 day, $p=0.003$) (table 3). The imaging indication for those who tested positive was frequently worsening hypoxaemia (43% vs 4%), rather

than cardiac symptoms including chest pain, syncope or clinical signs of heart failure (23% vs 44%). Based on echocardiographic quality, we were able to analyse 58

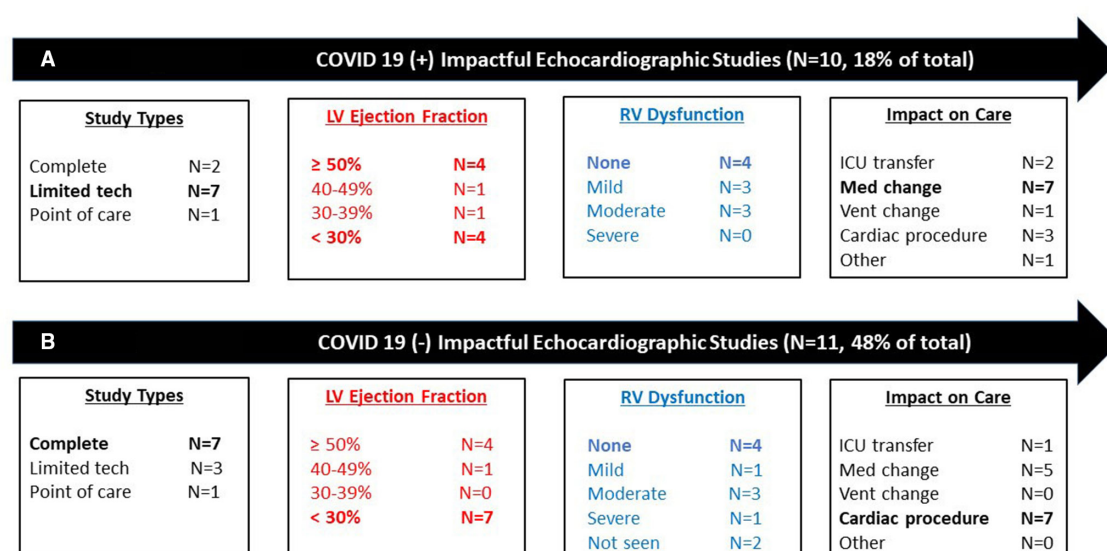


Figure 1 Echocardiographic information, findings and resultant changes in care for COVID-19-positive (A) and COVID-19-negative (B) patients. ICU, intensive care unit; LV, left ventricle; med, medication; RV, right ventricle; tech, technician; vent, ventilator.

patients (73%) in LV-GLS, 18 patients (23%) in RV-GLS, 43 patients (54%) in LA-GLS and 25 patients (32%) in RV-GLS. Echocardiographic parameters were closer to standard ranges for patients with COVID-19, including left ventricular ejection fraction (LVEF) (median of 55% vs 49%, $p=0.09$), LV-GLS (-14.5 vs -11.8 , $p=0.12$) and LA strain (31.4 vs 15.9 , $p=0.07$) (table 3). There were 38 (68%) patients with COVID-19 who had an ejection fraction $\geq 50\%$, compared with 11 (48%) of those without COVID-19. Other parameters were difficult to measure on several patients due to image quality (online supplemental table 1). Echocardiographic results changed management less frequently in COVID-19-positive patients (18% vs 48%, $p=0.01$; figure 1). Of the 10 COVID-19-positive patients who had alterations in management, 2 were transferred to intensive care; 7 had medication changes; 1 had a change in ventilation parameters; and 3 had a cardiac procedure performed.

COVID-19-positive outcomes

Of all COVID-19-positive patients, 14 out of 56 (25.0%) died during the initial hospitalisation. Of those who died, all required intensive care, 11 (79%) received mechanical ventilation, and 7 (50%) required prone ventilation. Those who died were older (mean age 65.8 vs 61.4 years) and more often had pre-existing cardiovascular disease compared with those who survived (57% vs 41%). Echocardiographic parameters for those who died compared with those who tested positive but survived their initial hospitalisation are presented in table 4. Those who died more often had echocardiography performed due to clinical deterioration (71% vs 24%) and had elevated right ventricular systolic pressures (RVSPs) (37 mm Hg vs 25 mm Hg), but other parameters were similar between groups, including LVEF (57.5% vs 55.0%).

DISCUSSION

In this observational study of 79 patients tested for COVID-19 who had echocardiographic imaging performed at a large tertiary care centre, we found that the vast majority of echocardiograms in COVID-19-positive patients were unremarkable. The majority of COVID-19-positive patients in our sample had LVEFs of $\geq 50\%$ and normal RV systolic function. This is different from reports from Dweck *et al*, who found that 55% of echocardiograms among COVID-19-positive patients were abnormal. This is surprising, as our patient sample has a higher burden of underlying cardiovascular disease and other comorbidities. However, patients in our sample often had cardiac imaging performed for non-cardiac-specific signs, rather than signs of heart failure or elevated biomarkers. This may explain the lower frequency of echocardiographic abnormalities in our study.

Compared with patients who were admitted to the hospital and ultimately tested negative for COVID-19, patients with COVID-19 received limited technician studies more frequently than a complete echocardiogram. There are several possible reasons for this finding. Imaging conditions may have been different among groups, as a larger number of COVID-19-positive patients underwent dialysis and mechanical ventilation during hospitalisation. These may have posed an additional challenge for complete examination at bedside for COVID-19-positive patients. It is also possible that clinicians used echocardiograms differently between the two groups and required less information from the COVID-19-positive group to make clinical decisions. During the study period, hospitals tried to minimise staff exposure to patients with COVID-19, which may also contribute to differences in imaging. Amid concerns of prolonged exposure for technicians and limited personal protective equipment, these

Table 4 Echocardiographic parameters for COVID-19-positive patients stratified by survival

	Survivors	Deaths	P value
N	42	14	
Echocardiogram type			0.59
Complete	10 (23.8)	2 (14.3)	
Limited technician	29 (69)	10 (71.4)	
Point of care	3 (7.1)	2 (14.3)	
Echocardiogram indication			
Cardiac-specific			
Abnormal biomarker	7 (16.7)	2 (14.3)	>0.99
Cardiogenic shock	2 (4.8)	1 (7.1)	>0.99
Cardiac signs	11 (26.2)	2 (14.3)	0.58
Arrhythmia	4 (9.5)	1 (7.1)	>0.99
Not cardiac-specific			
Worsening hypoxaemia	18 (42.9)	6 (42.9)	>0.99
Clinical deterioration	10 (23.8)	10 (71.4)	0.004
LVEF (%)	55 (48–60)	57.5 (46.0–63.8)	0.80
Diastolic function			0.57
Grade 1	17 (40.5)	4 (28.6)	
Grade 2	5 (11.9)	2 (14.3)	
Grade 3/4	1 (2.4)	1 (7.1)	
RV size			0.69
Normal	32 (76.2)	10 (71.4)	
Mildly dilated	6 (14.3)	1 (7.1)	
Moderately dilated	3 (7.1)	0 (0)	
Severely dilated	1 (2.4)	0 (0)	
RV systolic function			0.68
Normal	30 (71.4)	9 (64.3)	
Mildly reduced	6 (14.3)	2 (14.3)	
Moderately reduced	4 (9.5)	0 (0)	
Severely reduced	1 (2.4)	0 (0)	
RVSP	25 (20.3–38.9)	37 (30.5–65.0)	0.04
Strain %			
LV strain	–14.4 (–16.5 to –11.5)	–14.9 (–15.6 to –13.4)	0.46
LA strain	30.5 (19.9–37.8)	38.5 (23.2–45.0)	0.29

Data displayed as median (IQR) or number (%).

LA, left atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; RV, right ventricular; RVSP, right ventricular systolic pressure.

findings align with efforts to minimise risk for healthcare workers. However, as many of the echocardiographic parameters were within normal ranges for the COVID-19-positive cohort of patients, these studies may not be critical to management or clinical decision-making,

particularly among those without cardiac-specific signs of injury. Echocardiographic results changed management for relatively few patients with COVID-19 in this study, which is consistent with what others have found.¹³ Echocardiography should only be performed if there is an expected benefit in the management of the patient, and risk to healthcare workers and the patient should be carefully considered. In patients with suspected myocardial injury, further investigation may be warranted, as this is important for prognosis and may alter management for a select group of patients.¹⁴

We also found that patients who tested negative for COVID-19 and received echocardiograms were more ill than those who tested positive and received similar imaging. This is consistent with results from a similar study from Guo *et al*, in which clinical and echocardiographic parameters in a group of patients with COVID-19 were compared with retrospective analysis of echocardiograms from flu-positive patients. They found that flu-positive patients had a higher prevalence of LV failure and lower cardiac indices than patients with COVID-19.¹⁴ Similarly, patients in our study without COVID-19 often had greater lab abnormalities on initial presentation, such as elevated white blood cell counts and lactate levels, often required intensive care and had an in-hospital mortality rate of 35%. Their echocardiographic parameters were often worse. We studied patients with and without COVID-19 during the same time period, allowing us to compare changes in clinical practice and decision-making directly. Our findings suggest that the threshold for ordering an echocardiogram based on clinical presentation was lower for COVID-19-positive patients.

Finally, we found that elevated RVSP was frequently observed in patients with COVID-19 who died. RV failure is a known complication of severe ARDS, and resulting haemodynamic instability is an important cause of mortality in these patients.¹⁵ Our findings are consistent with previous reports in the COVID-19 population.^{16 17}

There are limitations to our study. There are likely differences between patients with COVID-19 for whom an echocardiogram was ordered and those who did not have one during their hospitalisation, which limits the generalisability of our study. There may be factors that we did not measure in our study that led to additional evaluation and imaging. There may also be undocumented changes in management as a result of the echocardiographic results which were not captured in our analysis. It is possible that results helped clinicians decide not to escalate care further. The quality of the images that were used for analysis also varied, which impacted the sample size for certain echocardiographic parameters. The low quality of images in the COVID-19-positive patients may have made it difficult for clinicians to make management decisions based on these results, which would lead us to underestimate changes in management. However, as the majority of images provided basic information regarding ventricular function, we believe that the reported normal

findings in these images are the reason for a lack of clinical action.

Our study is the first to our knowledge to compare echocardiographic indications and parameters in a cohort of patients tested for COVID-19 during the same time period, allowing us to compare directly for changes in clinical practice and diagnostics.

In summary, we found that the majority of echocardiograms performed in a cohort of COVID-19-positive patients during hospitalisation were unremarkable. These studies did not alter management in most cases, particularly among those without cardiac-specific signs of injury. Patients with COVID-19 who died were more likely to show RV dysfunction on echocardiography, consistent with the pathophysiology of SARS-CoV-2. Our findings suggest that hospital systems might consider limiting the use of echocardiography in patients with COVID-19 to those with unambiguous evidence of cardiovascular complication or injury where study findings are most likely to result in meaningful management changes that would better justify the risk of exposing echocardiographic technicians and physician sonographers to potential infection. For COVID-19-positive patients undergoing echocardiography, a point of emphasis should be made to obtain accurate estimates of RVSP and RV systolic function, given the negative prognostic implications of pulmonary hypertension and RV dysfunction in patients with COVID-19 infection.

Twitter Michael D Faulx @MichaelFaulx

Contributors MMS was responsible for study data acquisition, offline echocardiographic measurements, primary statistical analyses and manuscript draft preparation. YS was responsible for offline echocardiographic measurements, Methods section preparation and final manuscript editing. ZBP was responsible for study design input, supervision of offline echocardiographic measurement and final manuscript editing. MDF was responsible for primary study design, institutional review board submission, study database creation, study data acquisition, abstract authorship and final manuscript editing, and was the overall content guarantor.

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Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The study protocol was reviewed and approved by the Cleveland Clinic institutional review board (IRB 20-480) and deemed both ethical and low risk to study participants. Due to its low-risk nature, the study received consent exempt status. Our registry was subject to institutional review and was granted an exemption for informed consent owing to the use of deidentified patient data and minimal patient risk.

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Data availability statement Deidentified patient data are available in a password-protected RedCap database and as deidentified studies within a clinical imaging server, accessible by the authors. These data can be made available upon reasonable request.

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ORCID iDs

Yoshihito Saijo <http://orcid.org/0000-0002-1621-0200>

Michael D Faulx <http://orcid.org/0000-0002-6946-778X>

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