Critical COVID-19 rarely associated with left ventricular systolic impairment

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Introduction: For some individuals infection with SARS-CoV-2 leading to COVID-19 can result in a life-threatening multi-system disease. Multiple potential pathophysiological mechanisms for the development of cardiovascular complications have been postulated [1]. Early reports suggested that more than a third of hospitalised patients undergoing TTE had evidence of LV impairment [2].

Purpose: To ascertain the incidence of ventricular impairment among critically ill adults with COVID-19 admitted to the intensive care unit (ICU).

Methods: Retrospective analysis of transthoracic echocardiograms (TTE) performed on patients admitted to ICU with COVID-19 between 10th March and 10th May 2020. Archived studies were reviewed by accredited professionals. Studies were performed according to a modified British Society of Echocardiography (BSE) Level 1 protocol [3], with the addition of right ventricle (RV) focused apical 4 chamber, as well as apical 2 and 3 chamber views, and without ECG synchronisation as per infection control protocols. In the majority of patients the left ventricular ejection fraction (LVEF) was estimated from biplane Simpson's method. The RV function was assessed using the TAPSE in most patients. In the remaining patients the LV or RV function was visually assessed.

Results: Of 179 patients admitted to ICU, 85 (47.5%) had at least one TTE of diagnostic quality. Studies were performed a median of 7 days after ICU admission (IQR 3–17 days). Baseline and clinical characteristics and key echocardiographic measurements are summarised in table 1. The majority of patients were undergoing mechanical ventilation at the time of the scan (94.1%). One hundred and fifty-nine patients (89%) had elevated Troponin T (≥14ng/L) on the day of the study. LV systolic impairment (LVSD) was present in 5 patients (6.0%). This was known to be chronic in 3 patients (1 with coronary artery disease, 1 with chemotherapy induced cardiomyopathy and 1 with dilated cardiomyopathy of unknown aetiology), whilst pre-morbid cardiac function was unknown in the other 2 patients. No patient had severe LVSD (LVEF ≤35%). RV systolic dysfunction (RVSD) was found in 25 patients (31.3%). Amongst patients receiving mechanical ventilation there was no significant difference in Positive End Expiratory Pressure (PEEP) between patients with and without RVSD (9.4cmH2O vs. 9.8cmH2O, p=0.64), however there was a non-significant trend towards lower PaO2/FiO2 (P/F ratio) amongst patients with RVSD (18.9kPa vs 25.7kPa, p=0.07).

Conclusions: In contrast to other studies which have reported high frequency of LV impairment amongst hospitalised patients with COVID-19 [3], de novo LVSD was rarely found in this study, occurring in just 2 patients (2.4%), and being severe in neither. RV dilatation and systolic impairment were commonly found. A trend towards lower P/F ratios in patients with RVSD suggests severity of lung injury may be a factor in developing RV impairment.

Table 1. Baseline characteristics and echocardiographic findings.

	Females	All patients	Males
Patient characteristics			
Age (years)		[n=85]	
Median (IQR)		57 (51-64)	
Sex, n (%)		[n=85]	
Female		32 (37.6)	
Male		53 (62.4)	
Ethnicity, n (%)		[n=84]	
White		25 (29.8)	
Mixed		0	
Asian		13 (15.5)	
Black		37 (44.0)	
Other		9 (10.7)	
BMI (kg/m²), n (%)		[n=78]	
<18.5		1 (1.3)	
18.5-24.9		20 (25.6)	
25.0-29.9		23 (29.5)	
30.0-34.9		19 (24.4)	
35.0-39.9		6 (7.7)	
≥40.0		9 (11.5)	
Co-morbidities, n (%)		[n=85]	
Hypertension		49 (57.6)	
Diabetes		33 (38.8)	
Coronary Artery Disease		7 (8.2)	
Chronic Kidney Disease		16 (18.8)	
Clinical characteristics at time of scan			
Mechanically ventilated, n (%)		80 (94.1)	
Mode of ventilation, n (%)		[n=66]	
Mandatory (PPV)		43 (65.2)	
Spontaneous (mixed)		19 (28.8)	
CPAP (NPV)		4 (6.1)	
Troponin T, median (IQR)		[n=63] 49 (23-142)	
Echocardiographic characteristics			
Quality of recorded images, n (%)			
Good		46 (54.1)	
Sub-optimal		14 (16.5)	
Poor		25 (29.4)	
Left Ventricular size			
LVIDd (mm), median (IQR)	[n=27] 43 (38-44)		[n=47] 46 (42-50)
LVEDV (ml) (A4Ch), median (IQR)	[n=22] 79 (64-96)		[n=33] 98 (87-128)
LVEDVI (ml/m²) (A4Ch), median (IQR)	[n=20] 36 (34-50)		[n=30] 47 (40-59)
LV dilated, n (%)		[n=75] 2 (2.7%)	
Left Ventricular function			
LV fractional shortening (%) (PLAX), median (IQR)		[n=72] 37 (31-42)	
LVEF (%) (A4Ch), median (IQR)		[n=55] 62 (58-67)	
LV Global Longitudinal Strain, median (IQR)		[n=40] -21.2 (-18.0 - -22.5)	
Visual assessment of LV systolic function, n (%)		[n=83]	
Hyperdynamic		17 (20.5)	
Normal		61 (73.5)	
Impaired		5 (6.0)	
Severely impaired		0	
Right Ventricular size			
RVOTd (mm) (PLAX), median (IQR)	[n=25] 30 (26-32)		[n=43] 34 (30-37)
RV basal diameter (mm) (A4Ch-RV), median (IQR)	[n=22] 37 (34-45)		[n=37] 44 (38-47)
RV mid diameter (mm) (A4Ch-RV), median (IQR)	[n=21] 32 (28-40)		[n=37] 36 (32-38)
RV length (mm) (A4Ch-RV), median (IQR)	[n=20] 71 (69-77)		[n=37] 80 (75-86)
RV dilated, n (%)		[n=59] 18 (30.5)	
Right ventricular function			
RV Fractional Area Change (%) (A4Ch-RV), median (IQR)	[n=17] 38 (27-45)		[n=33] 38 (32-42)
TAPSE (mm) (A4Ch-RV), median (IQR)		[n=67] 18 (16-20)	
RV Global Longitudinal Strain, median (IQR)		[n=35] 23.0 (-19.5 - -24.8)	
Visual assessment of RV systolic function, n (%)		[n=80]	
Normal		55 (68.8)	
Impaired		25 (31.3)	
Miscellaneous			
TR Vmax (m/s), median (IQR)		[n=39] 2.9 (2.5-3.3)	
Pericardial effusion, n (%)		[n=81]	
Trivial		2 (2.5)	
Small (<10mm)		1 (1.2)	