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ORIGINAL INVESTIGATION

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A pilot study on right ventricular longitudinal strain as a predictor of outcome in COVID-19 patients with evidence of cardiac involvement

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Abstract

Aims: The aim of this investigation was to evaluate echocardiographic parameters of cardiac function and in particular right ventricular (RV) function as a predictor of mortality in patients with coronavirus disease-2019 (COVID-19) pneumonia.

Methods and Results: This prospective observational study included 35 patients admitted to a UK district general hospital with COVID-19 and evidence of cardiac involvement, that is, raised Troponin I levels or clinical evidence of heart failure during the first wave of the COVID-19 pandemic (March–May 2020). All patients underwent echocardiography including speckle tracking for right ventricular longitudinal strain (RVLS) providing image quality was sufficient (30 out of 35 patients). Upon comparison of patients who survived COVID-19 with non-survivors, survivors had significantly smaller RVs (basal RV diameter 38.2 vs 43.5 mm $P = .0295$) with significantly better RV function (Tricuspid annular plane systolic excursion (TAPSE): 17.5 vs 15.3 mm $P = .049$; average RVLS: 24.3% vs 15.6%; $P = .0018$). Tricuspid regurgitation (TR) maximal velocity was higher in survivors (2.75 m/s vs 2.11 m/s; $P = .0045$) indicating that pressure overload was not the predominant driver of this effect and there was no significant difference in left ventricular (LV) ejection fraction. Kaplan–Meier and log-rank analysis of patients split into groups according to average RVLS above or below 20% revealed significantly increased 30-day mortality in patients with average RVLS under 20% (HR: 3.189; 95% CI: 1.297–12.91; $P = .0195$).

Conclusion: This study confirms that RVLS is a potent and independent predictor of outcome in COVID-19 patients with evidence of cardiac involvement.

KEYWORDS

coronavirus, COVID-19, right ventricular function, right ventricular strain

1 | INTRODUCTION

Coronavirus disease 2019 (COVID-19), the disease caused by SARS-Coronavirus-2 (SARS-CoV-2), has posed unprecedented challenges to healthcare systems around the world. The disease has spread rapidly to most countries around the world and has demonstrated significant morbidity and mortality.

While typically presenting as a respiratory illness, COVID-19 commonly affects other organs including the cardiovascular system.[1,2] Involvement of the heart is usually detected by elevated troponin levels with or without ECG changes and can be associated with symptoms of heart failure and chest pain. Multiple studies have demonstrated that cardiac involvement is linked to a worse outcome.[3–5]

Echocardiography is recommended as initial cardiac imaging in COVID-19 patients with evidence of cardiac involvement[6] and appears to significantly alter clinical management in a third of patients.[7]

In its severe form, COVID-19 viral interstitial pneumonia manifests similarly to acute respiratory distress syndrome (ARDS) resulting in respiratory failure and the need for artificial ventilatory support.[8,9] Applying the relatively broad Berlin definition of ARDS,[10] the incidence of ARDS in hospitalized COVID-19 patients is up to 70%. While COVID-19 demonstrates unique disease features, there appear to be significant similarities in pulmonary hemodynamics and their effect on cardiac function between COVID-19 pneumonia and non-COVID-19-related ARDS.[11]

In non-COVID-19-related ARDS, impaired right ventricular (RV) function on echocardiography has been identified as a predictor of patient deterioration and poor overall outcome.[12]

Recent echocardiographic data supports that a dilated right heart is associated with poor outcome in patients with COVID-19 pneumonia.[13] Further, reduced RV function evaluated on speckle tracking analysis correlated with poor outcome in a Chinese patient cohort.[14]

Here, we sought to investigate RV function and particularly right ventricular longitudinal strain (RVLS) and its correlation with patient outcome in COVID-19 patients. This study was performed in a UK district general hospital-based patient cohort during the first wave of the COVID-19 pandemic.

2 | MATERIALS AND METHODS

2.1 | Patients

This is a prospective observational study on 35 consecutive patients admitted to Stoke Mandeville or Wycombe Hospitals (Buckinghamshire NHS Foundation Trust) between March and May 2020 with COVID-19 pneumonia, who also underwent transthoracic echocardiography (TTE). Inclusion criteria were as follows: (a) Symptoms and radiologic findings consistent with COVID-19 pneumonia, (b) detection of Cov-SARS-2 RNA on RT-PCR on throat and nose swab samples, (c) evidence of myocardial damage defined as: (a) elevated high sensitivity Troponin I levels (>200 ng/mL), or (b) a dynamic Troponin rise from >50 to >100 ng/mL on two measurement at least three hours apart or (c) clinical evidence of heart failure upon cardiology review. Patients with known underlying heart disease due to severe pulmonary hypertension, severe valvular disease, left-sided heart failure with a left ventricular ejection fraction of <35%, or suboptimal echocardiographic images (5 patients) were excluded from the study. The patients were followed up for 40 days after admission, or until death. No deaths occurred later than 30 days so for clarity, the Kaplan–Meier curves were chosen to end at 30 days. Of the 35 patients, 11 were mechanically ventilated at the time of echocardiogram.

2.2 | Clinical data

Demographics and clinical data were obtained via review of the patients' paper and electronic records.

2.3 | Echocardiograms

Echocardiography was performed using portable echocardiography machines (VIVID S70N) by British Society of Echocardiography (BSE) accredited echocardiographers. In accordance with the BSE guidelines on performing echocardiograms on patients with proven or suspected COVID-19, level 1 scans were obtained with limited views (parasternal long axis, apical four-chamber view, subcostal view). ECGs traces were not routinely used, and tissue doppler was not routinely acquired.

All measurements were performed remotely by two independent investigators blinded to the clinical data. Data sets with insufficient image quality for RV strain analysis were excluded from RV strain analysis, but were used for the measurement of the remaining echo parameters if the image quality was satisfactory (N = 4). This indicates a feasibility of RVLS in 86% of all cases, that is, 30 out of 35 had sufficient image quality for RVLS analysis.

2.4 | Echocardiographic analysis

Echocardiographic measurements were performed remotely in post-processing analysis. Right atrial (RA) and RV size and volumes were measured from the apical four-chamber view. Basal and mid RV diameters were measured in end-diastole. RV area was measured in both end-diastole and end-systole, and RV fractional area change was calculated as a percentage (RVD – RVS)/RVD*100. RA volume was measured in end-systole. Tricuspid annular plane systolic excursion (TAPSE) was measured from the apical four-chamber view as systolic apical displacement of the tricuspid annulus in post-processing or directly obtained MMode acquisitions. IVC dimensions were obtained from subcostal views and maximal diameters were measured.

RV strain analysis (Figure 1) was performed using the vendor non-specific GE 2D ECHOPAC software in the apical four-chamber

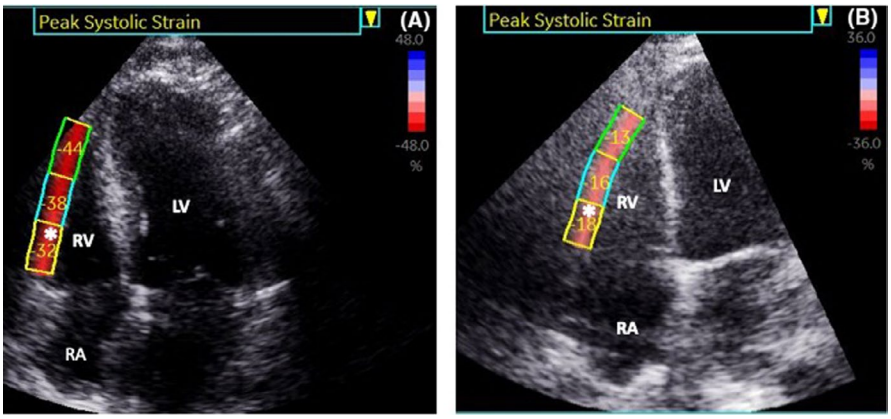

FIGURE 1 Examples of echocardiographic images with defined regions of interest for RV strain analysis in the basal, mid-ventricular and apical segments of the RV free wall. A, showing preserved RVLS and B, showing a patient with reduced RVLS

view. The RV free wall was manually defined as region of interest followed by automatic speckle tracking analysis of RVLS. The RV free wall was automatically divided into a basal, mid, and apical wall sections and RVLS values are presented individually and as overall average, with more negative values corresponding with better myocardial deformation. To avoid confusion, absolute strain values were used. Therefore, more positive values would imply better myocardial mechanics. If image quality was insufficient for speckle tracking in ≥1 segment(s), the case was excluded from strain analysis.

2.5 | Statistical analysis

Statistical analysis was performed using Microsoft Excel and Graphpad Prism software. Continuous data are expressed as mean ± standard error of the mean (SEM). All continuous variables were tested for normality using histograms, and Q–Q. Comparison of means was performed using the two-way Student's *t* test, and a *P*-value of $< .05$ was considered statistically significant. Categorical data are given as percentages indicating frequency and were compared using Fischer's exact test.

Survival curves were created using Kaplan–Meier analysis. Individual survival curves were compared using log-rank tests. Comparison in clinical and echocardiographic parameters was conducted in groups divided into: (a) survivors and non-survivors (ie, patients who passed away during their hospital admission), as well as (b) cases with an average RVLS of ≤20 and ≥20. To our knowledge, there is no generally accepted normal value of RVLS. Therefore, the cutoff of 20 used here was chosen as it was closest to the mean of RVLS values of our cohort and was also in accordance with previously reported cutoff values in RV strain studies.[15]

TABLE 1 Delineating clinical parameters in all included patients and according to mortality with statistical comparison of survivors vs non-survivors

	All patients (n = 34)	Survived (n = 19)	Deceased (n = 15)	Significance (survived vs deceased)
Avg. Age	72 (±2.6)	70 (±3.3)	75 (±4.0)	*P* = .345
Gender (%male)	79%	84%	73%	*P* = .672
Hypertension	53%	63%	40%	*P* = .2998
Diabetes	35%	53%	13%	**P* = .029
CKD	32%	37%	27%	*P* = .715
IHD	9%	5%	13%	*P* = .574
Current Smoker	6%	5%	7%	*P* = .888
Cer. Vasc. Dis.	9%	11%	7%	*P* = 1.000
Airway disease	9%	16%	0%	*P* = .2380
Peak hs-Troponin I	971 (±363)	1125 (±526)	775.5 (±500)	*P* = .633
D-dimer	9379 (±2124)	10 496 (±3083)	8616 (±2705)	*P* = .651
LDH	447 (±44.2)	459.2 (±62.8)	429.9 (±62.7)	*P* = .744
Ferritin	1271 (±285.2)	1071 (±191.4)	1573 (±661.4)	*P* = .479
Hba1C	57.0 (±5.1)	65.6 (±3.2)	38.4 (±6.2)	***P* = .001

Abbreviations: Cer. Vasc. Dis. = cerebro vascular disease; CKD = chronic kidney disease; IHD = ischemic heart disease.

"Airway disease": indicating a history of either asthma or chronic obstructive pulmonary disease.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

2.6 | Ethical approval

This study was approved by a local ethics commission and by the Health Research Authority and Health and Care Research Wales. IRAS project ID: 284 335; REC reference: 20/HRA2536.

3 | RESULTS AND DISCUSSION

3.1 | Clinical parameters

The clinical parameters of all patients included in the study are shown in Table 1, alongside a breakdown of the data according to mortality. The mean age of all included patients was 72 years of age and 79% of patients were male. Approximately half of all patients were known to be hypertensive (53%) and approximately one third had diabetes (35%) and chronic kidney disease (32%). Nine percent (9%) of patients had a history of chronic airways disease or ischemic heart disease. Apart from diabetes, there were no other significant differences in age or sex distribution or medical background between survivors and non-survivors of COVID-19. Peak hs-Troponin I, D-dimer, Ferritin, and LDH levels were significantly elevated in all patients with no significant differences between survivors and non-survivors. Accompanying the lower number of patients with diabetes in non-survivors, there was also a significant reduction in average Hba1C levels.

3.2 | Echocardiographic parameters associated with mortality

The echocardiographic parameters of all patients included in the study are shown in Table 2, alongside a breakdown of the data according to mortality. Compared to survivors of COVID-19 pneumonia, non-survivors had significantly increased basal (38.2 vs 43.5 mm $P = .0295$) and mid-ventricular (26.0 vs 30.4 $P = .0390$) RV diameters and significantly reduced TAPSEs (17.5 vs 15.3 mm $P = .049$). There was a trend toward increased diastolic and systolic RV area as well as RA volume in non-survivors which did not reach statistical significance and there was no significant difference in RV fractional area change or IVC diameter.

Total right ventricular longitudinal strain (RVLS) was significantly reduced in non-survivors compared to survivors. (24.3% vs 15.6%, $P = .0011$). This was also the case when RVLS was separated into basal (23.4% vs 17.5%, $P = .0288$), mid-ventricular (24.6% vs 15.5%, $P = .0011$), and apical segments (26.3% vs 13.7%, $P = .0001$). TR

TABLE 2 Delineating echocardiographic parameters in all included patients according to mortality with statistical comparison of survivors vs deceased patients

	All patients (n = 34)	Survived (n = 19)	Deceased (n = 15)	Significance (survived vs deceased)
TAPSE	16.5 (±0.56)	17.5 (±0.71)	15.3 (±0.81)	*$P = .049$
RVD 1	40.5 (±1.4)	38.2 (±1.8)	43.5 (±1.9)	*$P = .0295$
RVD 2	27.9 (±1.2)	26.0 (±1.5)	30.4 (±1.9)	*$P = .0390$
RVD 3	69.9 (±1.5)	69.6 (±2.3)	70.3 (±1.7)	$P = .8108$
RV D area	19.7 (±1.3)	17.9 (±1.7)	22.0 (±1.8)	$P = .1164$
RV S area	12.5 (±1.0)	11.6 (±1.5)	13.6 (±1.4)	$P = .3196$
RV fract. area. change	38% (±2%)	38.1% (±2.7%)	38.4% (±3.2%)	$P = .9426$
RA volume	39.0 (±3.4)	32.2 (±3.6)	40.6 (±4.0)	$P = .1318$
TR Vmax	2.74 (±0.12)	2.75 (±0.16)	2.11 (±0.12)	**$P = .0045$
IVC diam.	20.1 (±0.9)	19.5 (±1.3)	21.1 (±1.2)	$P = .3648$
LV EF	61.0% (±2.3%)	62.5 (±2.6)	59.1 (±4.0)	$P = .4875$
Avg. RVLS (n = 30)	20.4% (±1.62)	24.3% (±1.92)	15.6% (±1.69)	**$P = .0018$
Bas. RVLS (n = 30)	20.7% (±1.38)	23.4% (±1.86)	17.5% (±1.79)	*$P = .0288$
Mid RVLS (n = 30)	20.5% (±1.50)	24.6% (±1.86)	15.5% (±1.72)	**$P = .0011$
Apical RVLS (n = 30)	19.8% (±1.90)	26.3% (±2.07)	13.7% (±1.96)	***$P = .0001$

Abbreviations: Avg. RVLS = average right ventricular longitudinal strain; Bas. RVLS = basal segment right ventricular longitudinal strain; IVC diam. = diameter of the inferior vena cava; LV EF = left ventricular ejection fraction; Mid RVLS = mid-ventricular segment right ventricular longitudinal strain; RV D area = RV diastolic area; RV S area = RV systolic area; RVD1 = basal RV diameter; RVD2 = mid-ventricular RV diameter; RVD3 = RV length from base to apex; TR Vmax = maximal velocity of the tricuspid regurgitation jet.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

maximal velocity was higher in survivors compared to non-survivors (2.75 m/s vs 2.11 m/s; $P = .0045$) and there was no significant difference in LV ejection fraction (62% vs 59%; $P = .4875$).

3.3 | Mortality associated with RVLS under 20%

The absolute average RVLS in all included patients was 20.4%. Accordingly, patients were split into two groups with an avg. RVLS above or below 20%. Kaplan–Meier curves (Figure 2A) and log-rank analysis of these groups revealed significantly increased 30-day mortality in patients with mean RVLS under 20% (HR: 3.189; 95% CI: 1.297–12.91; $P = .0195$) indicating that it predicts increased mortality in patients with COVID-19 pneumonia.

Separating the patients into two groups according to TAPSE (>16 mm vs <16 mm), Kaplan–Meier and log-rank analyses revealed a similar trend of RV longitudinal function, indicating increased mortality in the reduced TAPSE group; however, this did not reach statistical significance (HR: 2.353; 95% CI: 0.8520–7.057; $P = .1049$).

3.4 | Clinical and echocardiographic parameters associated with RVLS

Comparing the two groups of patients with an absolute mean RVLS > 20% and mean RVLS < 20% (Table 3) revealed no significant differences with regards to clinical parameters including age, gender, hypertension, diabetes, ischemic heart disease, cerebrovascular disease, smoking or airway disease, that is, asthma or COPD. Blood tests showed a non-significant trend toward higher peak troponins and higher D-dimer and Ferritin levels in patients with average RVLS < 20%. Interestingly, Hba1C levels were significantly lower in patients with lower average RVLS although there was no significant difference in the prevalence of diabetes. Overall, 11 of the 30 patients were intubated and mechanically ventilated at the time of echocardiogram. 7 out of 17 (41%) patients with RVLS > 20% and 4 out of the 13 (31%) patients with RVLS < 20%, indicating that mechanical ventilation did not have a significant effect on RVLS measurements ($P = .7084$).

With regards to other echocardiographic parameters, the group of patients with mean RVLS < 20% had significantly more dilated

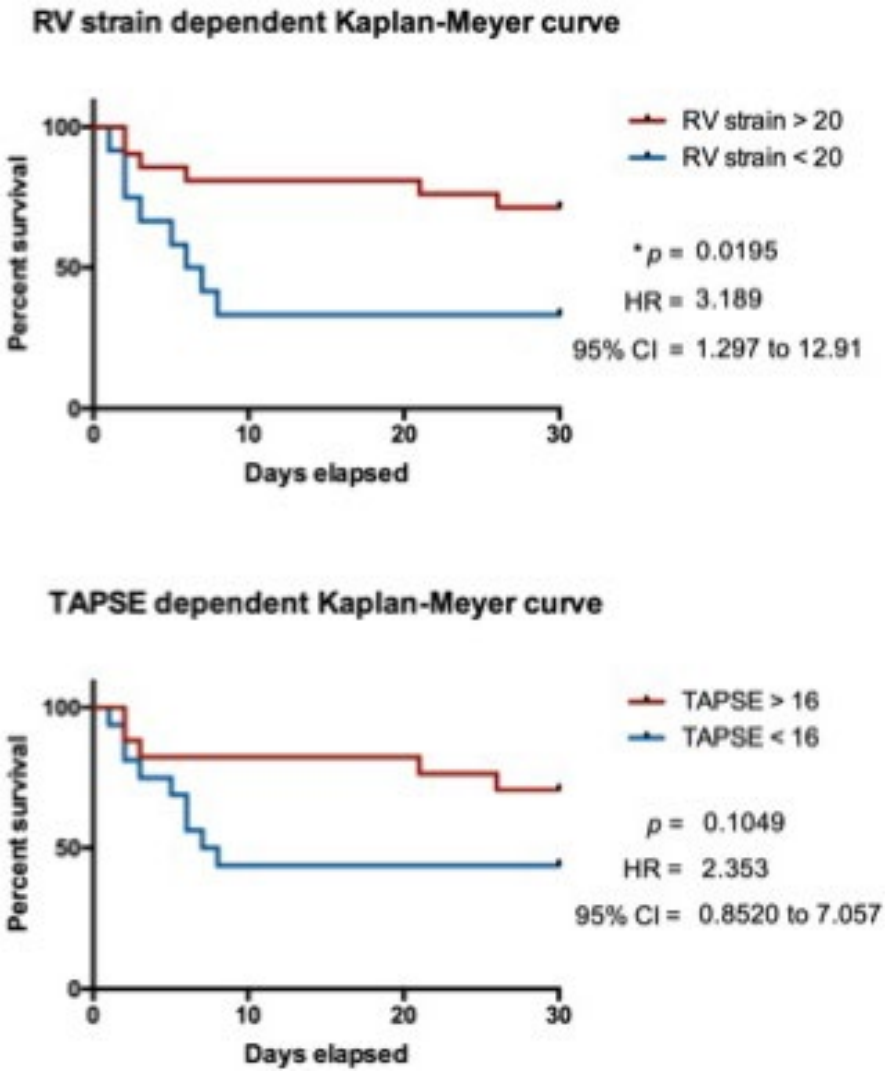

FIGURE 2 Kaplan–Meier survival curves and log-rank analysis of the patients separated into two groups according to RVLS (>20 and <20) and TAPSE (>16 mm vs <16 mm), revealed significantly increased 30-day mortality in patients with mean RVLS under 20% (HR: 3.189; 95% CI: 1.297–12.91; $P = .0195$) and a trend of increased mortality in the reduced TAPSE group; however, this did not reach statistical significance (HR: 2.353; 95% CI: 0.8520–7.057; $P = .1049$)

TABLE 3 Delineating clinical and echocardiographic parameters in patients with RVLS > 20 and < 20%

	All patients (n = 30)	RVLS > 20% (n = 17)	RVLS < 20% (n = 13)	Significance
Age	71 (±2.6)	70 (±3.9)	73 (±4.2)	*P* = .6338
Gender (% males)	80%	82%	77%	*P* = 1.0000
Hypertension	53%	41%	69%	*P* = .1590
Diabetes	33%	41%	23%	*P* = .4404
CKD	27%	18%	38%	*P* = .2420
IHD	3%	0%	8%	*P* = .4333
Current smoker	10%	12%	8%	*P* = 1.0000
Cer. vasc. dis.	7%	6%	8%	*P* = 1.0000
Mech. Ventilated	37%	41%	31%	*P* = .7084
peak hs. Trop I	767 (±287)	479 (±208)	1144 (±602)	*P* = .2583
D-dimer	10 369 (±2325)	9212 (±2721)	12 540 (±4480)	*P* = .5082
LDH	474 (±47)	482 (±71)	464 (±62)	*P* = .8565
Ferritin	1394 (±308)	1101 (±221)	1761 (±636)	*P* = .2956
Hba1C	53.9 (±4.9)	63.6 (±7.3)	42 (±3.5)	**P* = .0234
LV EF	61.1% (±2.5%)	63.7 (±2.7%)	57.7 (±4.5)	*P* = .2408
TR Vmax	2.72 (±0.12)	2.80 (±0.17)	2.57 (±0.14)	*P* = .4224
IVC D	2.01 (±0.1)	2.01 (±0.13)	2.01 (±0.13)	*P* = .9862
RA vol.	39.8 (±3.8)	33.3 (±3.6)	48.4 (±6.7)	**P* = .0442
RVD1	4.1 (±0.1)	3.8 (±0.2)	4.4 (±0.2)	**P* = .0429
RVD2	2.8 (±0.1)	2.6 (±0.2)	3.1 (±0.2)	*P* = .0607
RVD3	7.0 (±0.2)	6.8 (±0.2)	7.2 (±0.3)	*P* = .2020
TAPSE	16.7 (±0.6)	17.2 (±0.7)	16.1 (±1.1)	*P* = .3676
RV D area	20.3 (±1.4)	18.0 (±1.5)	23.4 (±2.4)	**P* = .0490
RV S area	12.8 (±1.1)	10.7 (±1.1)	15.4 (±2.0)	**P* = .0331
RV area change	38.8% (±2.1%)	41.7 (±2.2%)	34.9 (±3.7%)	*P* = .1131

Abbreviations: Cer. vasc. dis. = cerebrovascular disease; CKD = chronic kidney disease; IHD = ischemic heart disease; IVC diam. = diameter of the inferior vena cava; LV EF = left ventricular ejection fraction; Mech. Ventilated = mechanically ventilated; RV D area = RV diastolic area; RV S area = RV systolic area; RVD1 = basal RV diameter; RVD2 = mid-ventricular RV diameter; RVD3 = RV length from base to apex; TR Vmax = maximal velocity of the tricuspid regurgitation jet.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

right ventricles (RVD1: 4.4 vs 3.8 cm, *P* = .0442; RVd area: 23.4 vs 18 cm^2 *P* = *.0490*; RVs area: 15.4 vs 10.7 cm^2, *P* = *.0331*) and larger RA (RA vol.: 48.4 vs 33.3 mL, *P* = .0442). There was a non-significant trend toward lower TAPSE and lower RV fractional area change in patients with mean RVLS of < 20%.

There was no significant difference in maximal TR velocity, IVC diameter or left ventricular EF between groups, indicating that pressure or volume overload of the right ventricle or biventricular failure is not the primary driver of differences in RVLS.

4 | DISCUSSION

COVID-19 affects the heart in as much as half of patients [1,7,16] and cardiac damage does appear to persist for some time even after clinical recovery.[17] The pathologic processes affecting the heart are not entirely understood[18,19] but likely range from direct viral infection of the myocardium to indirect reactive inflammation as well as microvascular or macrovascular thrombosis.[20,21] Pressure overload of the right ventricle in the context of severe lung disease is a further potential contributor in particular to RV damage. RV function measured on standard echocardiography and as strain on speckle tracking analysis is impaired in patients with non-COVID ARDS and this impairment correlates with poor outcome.[22]

In the present study, we show that reduced RV function measured as an absolute RVLS of < 20% correlates significantly with increased mortality. The patient population in this study was significantly older than the population in similar studies,[13,14,23] predominantly male and had significant comorbidities. The applied inclusion criteria, that is, elevated troponins or clinical signs of heart failure

selected specifically for patients with evidence of myocardial involvement resulting in a high-risk cohort. In these patients with evidence of cardiac damage, RVLS was the best predictor of mortality, whereas LV systolic function did not differ between survivors and non-survivors. Interestingly, maximal velocity of TR was higher in survivors and trended to be higher in patients with RVLS > 20% indicating that pressure overload was not present at the time of echocardiogram and making intrinsic right heart failure the more likely cause. An increased RVLS in COVID-19 survivors in the presence of an increased TR Vmax, and pulmonary artery systolic pressure, may actually reflect good RV longitudinal contractile reserve partly responsible for the better clinical outcome seen in this group. We therefore suggest that RVLS may be an independent clinical predictor in COVID-19 and could be helpful in risk stratification of cases. These data confirm similar recently published results.[14]

To our knowledge, this is the first observational study on RV function in COVID-19 patients with predetermined evidence of myocardial injury. This pre-specified inclusion criterion makes this study representative of clinical practice as echocardiography is usually performed in patients with clinical evidence of myocardial involvement rather than in unselected COVID-19 inpatients. Our data show that RVLS analysis was both feasible in a UK district general hospital adult population and an independent predictor of patient outcome.

RV strain parameters can be obtained remotely and with relative ease from 2D echocardiographic acquisitions, thereby reducing the exposure of the operator to patients with COVID-19. We therefore suggest measuring RVLS in patients with evidence of myocardial damage, that is, elevated troponin levels or clinical suspicion of heart failure, can be an effective tool in predicting clinical outcomes and possibly the need for high level intervention.

4.1 | Limitations

The main limitation of this study is the limited number of patients which may explain the unexpected finding of a low number of patients suffering from diabetes in the deceased group.

Further there is potential bias as pulmonary embolism was not excluded in all patients. Performing CT pulmonary angiography (CTPA) in all study participants was not deemed ethically justified. However, 10 patients underwent CTPA according to clinical indications demonstrating pulmonary embolism in a total of 4 patients. Three of these patients survived, indicating that there is no clear predominance of PE in the group of non-survivors however, with the majority of patients not having undergone CTPA this cannot be excluded.

5 | CONCLUSION

The data we present in this observational echocardiographic study on patients with COVID-19 pneumonia confirm that RVLS is a potent and independent indicator of mortality and suggests that this is not simply related to RV pressure overload but likely secondary to intrinsic RV failure. We suggest that performing RV strain analysis in COVID-19 patients with evidence of cardiac involvement can assist in predicting outcomes.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Raw data were generated at Buckinghamshire NHS Foundation Trust. Derived data supporting the findings of this study are available within the article and from the corresponding author AS on request.

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