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ORIGINAL ARTICLE

Right heart echocardiography findings in hypoxic pneumonia patients during the COVID-19 pandemic in a South African population

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Abstract

Aims The right ventricle is affected by Coronavirus disease 19 (COVID-19) via multiple mechanisms, which can result in right ventricular dysfunction (RVD). This study aimed to provide an assessment of right heart function using conventional echocardiography and advanced strain imaging, in patients with hypoxic pneumonia during the COVID-19 pandemic.

Methods and results This study was an observational, prospective, single-centre study, including adults with hypoxic pneumonia, in two groups: COVID-19 pneumonia; and non-COVID-19 pneumonia. Bedside echocardiography was performed according to a pre-specified protocol and all right heart measurements were done as per standard guidelines. Right ventricular free wall strain (RVFWS) was measured using Philips® QLAB 11.0 speckle tracking software. Descriptive and comparative statistics were used to analyse data. Spearman Rank Order Correlations were used to determine the correlation between right ventricular (RV) parameters and clinical parameters. Univariate and multivariate logistic regression analyses were performed to characterize the predictors of in-hospital mortality. We enrolled 48 patients with COVID-19 pneumonia and 24 with non-COVID-19 pneumonia. COVID-19 patients were significantly older with a higher frequency of hypertension and diabetes and a trend towards a lower severity of illness score. Mean RVFWS yielded the highest estimates for the prevalence of RVD (81%), with no difference between the two pneumonia groups. Median Tricuspid Annular Plane Systolic Excursion (TAPSE) and right ventricular systolic excursion velocity (RVS') were not significantly different between COVID-19 (TAPSE 17.2 and RVS' 12), and non-COVID-19 pneumonia (TAPSE 17.8 and RVS' 12.1) with *P* values of 0.29 and 0.86, respectively. Non-COVID-19 pneumonia patients with moderate to severe hypoxaemia (PF < 150) were at greater risk of an elevated RV Systolic Pressure >30 mmHg respiratory rate = 3.25 (CI 1.35–7.82) on admission. Troponin levels discriminated between COVID-19 survivors (6 ng/L) and non-survivors (13 ng/L), $P = 0.04$. The mortality rate for COVID-19 was high (27%) compared to non-COVID-19 pneumonia (12%).

Conclusion Patients with COVID-19 pneumonia had a similar admission prevalence of RVD when compared to patients with non-COVID-19 pneumonia. Despite preserved traditional parameters of RV systolic function, RVFWS was diminished in both groups, and we propose that RVFWS serves as an important marker of the subclinical disease of RV.

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Study population, definitions, inclusion, and exclusion criteria

We screened all consecutive adult patients during working hours on weekdays, who were persons under investigation for SARS-CoV-2 virus, and were admitted between 20 October 2020 and 11 March 2021. Patients were included if they had hypoxic physician-diagnosed or chest x-ray (CXR)-diagnosed pneumonia and met the criteria for severe disease or critical illness. Severe disease was defined as oxygen saturation ≤92% with a respiratory rate ≥30 and therefore requiring supplemental oxygen support without the need for invasive or non-invasive ventilation.[18] Critical illness was defined as hypoxaemia and the need for additional ventilatory support, in the form of non-invasive ventilation.[18] The study investigation was performed within 48 h from admission and before the initiation of invasive mechanical ventilation. There was no serial imaging, outcome was determined as either survival (discharge from hospital) or death and was assessed retrospectively, with no intervening imaging or clinical follow-up during the admission pertinent to the study. We excluded patients if they had *mycobacterium tuberculosis* and *Pneumocystis jiroveci pneumonia*, were pregnant, had a known chronic lung disease, chronic cardiac disease, specifically ischaemic heart disease, cardiomyopathy, had organic valvular heart disease, either known or incidental, or had a history of pulmonary or cardiac surgery, and if the imaging was poor quality. This study focussed on right heart echocardiographic findings and excluded all patients with a left ventricular ejection fraction (LVEF) < 50%, in an attempt to exclude right heart abnormalities due to left heart systolic dysfunction.

Patient demographic, clinical, laboratory, and hospital survival data were extracted from clinical notes. The following scores were calculated from admission data: simplified acute physiology score 2 (SAPS2),[19] and Sequential Organ Failure Assessment (SOFA) scores,[20] We further classified patients using the Horowitz Index for Lung Function: ratio of arterial oxygen partial pressure (PaO_2 in mmHg) to fractional inspired oxygen (FiO_2) expressed as a fraction (P/F) ratio of below 150 or ≥ 150.

Two- dimensional echocardiography

Transthoracic echocardiography was performed on all patients in the left lateral or supine position using an S5-1 transducer on a Philips® portable CX 50 system (Amsterdam, the Netherlands). The images were obtained according to a standardized protocol. The data were transferred and analysed offline using the Philips® Xcelera workstation. In addition to LVEF, the following RV systolic functional parameters were measured: A pulse-wave tissue doppler imaging (TDI) was performed in the apical four-chamber view using a sample volume size (6 mm) adequate to cover the basal RV longitudinal excursion, and we measured time from beginning of isovolumetric contraction to the peak of the S wave in TDI (time to peak) to non-invasively estimate the mean pulmonary pressure as a surrogate for pulmonary hypertension as compared to right heart catheterization.[21] The cut-off time used to define pulmonary hypertension was 127 ms.[21] RV pulse-wave Doppler was performed to assess the RV E/A ratio and E/E' ratio, as a surrogate of RV diastolic function. All measurements relating to the RV were performed by an experienced Cardiologist based on the ASE guidelines and using the guidelines on the RV for cut-off of RVD[22] and the values described in the specific population.[23] RVFWS cut-off of −23 was used and has been feasible and reproducible in our unit with intra-observer and inter-observer variability coefficients of 7% and 7.6%, respectively.[24]

Right ventricle strain analysis

RVFWS was derived from a modified A4C RV view. Once three points, namely the RV apex, medial, and lateral tricuspid annulus, were defined, the software automatically traced the endocardial and epicardial border. Philips® QLAB version 11.0 software allowed offline semi-automated analysis of speckle-based strain. This results in the division of the RV into six standard segments in the A4C view. The region of interest, once created, can be manually adjusted as needed to allow for adequate speckle tracking. RVFWS is less impacted by cardiac motion, the left ventricle, and the volume status,[25,26] and gives both regional and average assessments. RVFWS has previously been shown to be more sensitive at diagnosing RVD in some conditions, than TAPSE and Doppler tissue imaging.[27] The RVFWS was obtained by averaging three lateral segments (the basal, mid, and apical RV wall). A cut-off of −23% was used, with more negative values indicating better myocardial mechanics.[28] The interventricular septum was excluded from the analysis. The longitudinal ε curves for each segment and a mean curve of all segments were generated by the software. These curves were used to derive peak negative RVFWS.

Outcome measures

The primary outcome was to describe the prevalence of RV dysfunction. The secondary outcomes included a description of RV dimensions, RV function, the relationship between RV function, and troponin, and D-Dimer, to describe the relationship between RVFWS and oxygenation, and to explore the predictors of mortality.

Data collection

Study data were collected and managed using REDCap® electronic data capture tools hosted at the University of Witwatersrand (Research Electronic Data Capture).[29,30]

Statistical analysis and sample size

Statistical analyses were performed using Statistica® version 13.3 (TIBCO Software Inc., USA). Continuous variables were expressed as median [interquartile range (IQR)], and proportions/percentages were used for categorical variables. Continuous data were compared using the Mann–Whitney *U* test while proportions were compared using the chi-square test. A *P*-value < 0.05 was considered statistically significant. Spearman Rank Order Correlations were used to determine the correlation between RV parameters and clinical parameters. Univariate and multivariate analyses using logistic regression analysis were performed to characterize the predictors of in-hospital mortality using biochemical and echocardiographic factors. A convenience sample was employed.

Ethics considerations

Approval was received from the University Human Research Ethics Committee (Medical), M200728, and is registered on the National Health Research Database, South Africa, GP_202008_140. Written informed consent from the patient or surrogate was obtained as per local ethics committee guidelines.

Results

Patient characteristics

We enrolled 72 hypoxic pneumonia patients between 20 October 2020 and 11 March 2021. We enrolled 48 patients with COVID-19 pneumonia (COVID-19) and 24 with non-COVID-19 pneumonia. *Figure 1* describes the participant selection process. The clinical characteristics of the cohort are shown in *Table 1*. Baseline arterial blood gas and biomarkers are provided in *Table 2*. Compared to the COVID-19 cohort, the non-COVID-19 cohort had significantly higher d-dimers. There was a weak correlation between troponin and RV diastolic dysfunction [RV E/A 0.26 ($P < 0.05$) and RV E/E' 0.27 ($P < 0.05$)].

Primary outcome

The highest overall percentage prevalence of RVD was obtained using RVFWS, 81% [confidence interval (CI) 76–85%, $n = 67$]. *Figure 2* demonstrates the percentage prevalences of RVD using different echocardiographic methods among COVID-19 and non-COVID-19 pneumonia participants. *Figure 3* demonstrates the measurement of RVFWS in two patients from the cohort.

Secondary outcomes

Right ventricular wall thickness and inferior vena cava (IVC) diameter were both significantly higher in the non-COVID-19 pneumonia group, shown in as *Table 3*. Despite a statistically greater E/A ratio in the non-COVID-19 compared to the COVID-19 pneumonia group, both

Table 2 Admission blood gas variables, biomarkers, and total lung ultrasound score

Variable	All		Non-COVID-19 pneumonia		COVID-19 pneumonia		*P*-value
	n	Mean (SD)/median [IQR]	*n*	Mean (SD)/median [IQR]	*n*	Mean (SD)/median [IQR]	
PaO_2 mmHg	72	45 [31–62]	24	43 [29–57]	48	46 [32–63]	0.47
FiO_2	72	0.33 [0.21–0.6]	24	0.21 [0.21–0.5]	48	0.4 [0.21–0.60]	0.14
P/F ratio	72	132 [93–192]	24	143 [96–218]	48	126 [81–183]	0.62
pH	72	7.43 [7.40–7.46]	24	7.42 [7.38–7.45]	48	7.44 [7.40–7.46]	0.38
$PaCO_2$ mmHg	60	37 (7.4)	24	36 (9.4)	45	37 (8.5)	0.83
BE meq/L	70	0.3 [−3.0–3.9]	24	−2.7 [−4.1–3.9]	46	1.3 [−1.5–4.0]	0.13
CRP mg/L	72	128 [60.5–211]	24	106 [63–225]	48	132.5 [56–192]	0.92
d-dimer mg/L	66	1.15 [0.38–3.56]	21	2.08 [1.08–3.84]	45	0.82 [0.35–3.18]	0.02[a]
Troponin ng/L	62	8 [5–20]	16	9 [5–19]	46	7 [5–21]	0.65
Total LUS	72	8 [4–12]	24	7.5 [4.4–12.5]	48	8 [4–11.5]	0.56

[IQR], interquartile range with median; (SD), standard deviation with mean; CRP, C reactive protein; PaO_2, partial pressure of oxygen; FiO_2, fraction of inspired oxygen; P/F, Horowitz index for Lung Function (P/F ratio); $PaCO_2$, partial pressure of carbon dioxide; BE, base excess.
[a]Statistically significant.

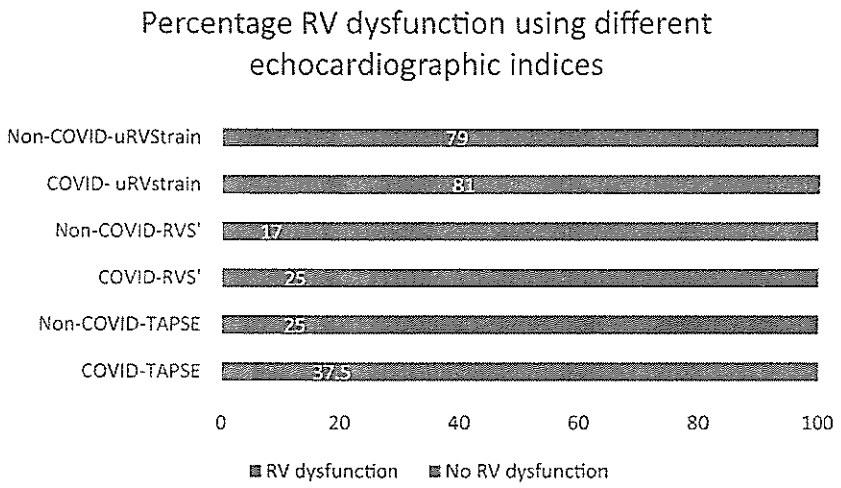

Figure 2 Prevalence (%) of RV dysfunction according to traditional echocardiographic parameters (RVS' and TAPSE) and RVFWS amongst COVID-19 and non-COVID-19 patients at admission.

different right heart echocardiography findings between the COVID-19 survivors and non-survivors. See *Table 4* for factors associated with mortality.

We generated a logistic regression model using six variables including an oxygenation marker (PaO_2), a marker of ventilation ($PaCO_2$), a metabolic indicator (bicarbonate), severity of illness score (SAPS II), COVID-19 status, and a marker of RV strain (average strain) to determine independent predictors of mortality. Four parameters with a *P*-value <0.5 on univariate analysis were included in the final model. A higher SAPS II score ($P = 0.04$) and COVID-19 positivity (0.05) were independent predictors of mortality.

Discussion

Our main finding was a high prevalence (around 80% vs. 35%) of RVD amongst patients presenting with hypoxic pneumonia. RVFWS was superior to traditional RV parameters (TAPSE, RVS', and PASP) for assessing early RV systolic dysfunction in both patient cohorts, detecting more cases of RV systolic dysfunction in both COVID-19 and non-COVID-19 pneumonia groups.

It is difficult to ascertain the true degree of RVD that occurs specifically due to COVID-19 hypoxic pneumonia owing to the confounding effects of disease severity, varying P/F ratios, clinical settings (hospitalized vs. intensive care), and variability in the timing of echocardiography with regard disease progression, however, the prevalence of RVFWS is reported to vary between 12.6%[31,32] and 86%.[26]

Sun *et al.*[31] demonstrated similar prevalences of RVD among COVID-19 pneumonia and bacterial pneumonia. Their prevalences were lower than ours and may be explained by the use of traditional parameters of TAPSE and RVS'. Other differences included an older population, and a very low human immunodeficiency virus (HIV) rate. In addition, neither severity of illness scores nor P/F ratios were described. *Bleakley et al.* also found a higher proportion of RVD in critically

Table 4 Factors associated with mortality

Variables	All survivors {*n* 56}	All non-survivors {*n* 16}	*P* value	COVID-19 survivors {35}	COVID-19 non-survivors {13}	*P* value
Time from isovolumetric contraction to the peak of the S wave (TTP) (ms)	93 [83–117] {55}	101 [90–114.5] {16}	0.85	96.5 [88–112] {34}	99.0 [90.0–112.0] {13}	0.99
TAPSE (mm)	17.6 [15.6–21.9] {56}	16.6 [14.15–21.15] {16}	0.23	17.4 [15.3–22] {35}	15.6 [14.0–21.0] {13}	0.14
RVSP (mmHg)	10 [7–23] {42}	10 [10–24] {11}	0.42	10 [10–20.3] {23}	10.0 [10.0–14.0] {11}	0.77
RVS' (cm/s)	12.0 [10.2–13.4] {56}	12.3 [9.15–17.05] {16}	0.55	12.0 [9.6–13.8] {35}	12.7 [10.4–16.9] {13}	0.37
E (m/s)	53.92 (±16.78) {56}	55.05 (±15.67) {16}	0.81	52.3 (±15.9) {35}	56.0 (±16.2) {13}	0.55
A (m/s)	58.87 (±17.73) {55}	68.69 (±21.04) {16}	0.35	61.1 (±17.2) {35}	71.8 (±18.1) {13}	0.53
E/A	0.85 [0.75–1.16] {55}	0.82 [0.61–1.09] {16}	0.23	0.84 [0.72–1.07] {35}	0.80 [0.6–0.88] {13}	0.37
E'	10.53 (±3.00) {55}	10.02 (±2.89) {16}	0.93	9.8 (±2.3) {34}	9.5 (±2.5) {12}	0.53
A'	13.8 [11.3–15.8] {56}	15.1 [12.6–199] {15}	0.21	13.4 [11.3–15.7] {35}	15.1 [12.7–18.2] {12}	0.29
E/E'	5.30 [3.83–6.58] {55}	5.47 [4.06–7.91] {16}	0.46	5.4 [4.0–6.6] {34}	5.7 [5.0–8.1] {12}	0.33
RVFWS (%)	−13.48 (±7.08) {52}	−16.31 (±5.88) {16}	0.43	−13.5 (±7.5) {31}	−15.6 (±6.2) {13}	0.25
RV wall thickness (cm)	0.60 [0.57–0.75] {53}	0.6 [0.51–0.68] {16}	0.36	0.6 [0.54–0.69] {32}	0.6 [0.55–0.63] {13}	0.78
RV base (mm)	31.4 [27.4–36.8] {53}	30.2 [26.96–35.9] {16}	0.77	31.6 [27.4–39.3] {31}	28.2 [26.2–35.6] {13}	0.39
RV length (mm)	59.5 [51.7–66.6] {54}	66.5 [57.5–74.35] {16}	0.04[a]	58.0 [41.1–64.7] {31}	61.0 [56.8–68.5] {13}	0.14
Tricuspid annular size (mm)	29.52 (±4.90) {54}	27.11 (±5.16) {16}	0.74	29.4 (±4.6) {34}	26.2 (±5.0) {13}	0.18
IVC diameter (mm)	11.4 [9.00–13.80] {53}	10.2 [8.5–13.85] {16}	0.76	10.3 [8.8–12.1] {32}	10.2 [8.0–13.1] {13}	0.78
RA length (mm)	42.01 (±7.54) {54}	25.54 (±5.36) {16}	0.16	43.0 (±7.5) {33}	40.4 (±5.9) {13}	0.19
RA width (mm)	35.73 (±7.22) {53}	40.96 (±5.41) {16}	0.92	35.2 (±6.6) {33}	32.4 (±6.3) {13}	0.19
CRP mg/L	122 [58–193.5] {56}	137.5 [79.5–244] {16}	0.52	130 [51–180] {35}	135 [80–264] {13}	0.65
d-dimer mg/L	1.15 [0.38–2.76] {56}	1.58 [0.58–5.19] {16}	0.34	0.49 [0.35–1.73] {35}	1.01 [0.39–4.65] {13}	0.20
troponin ng/L	7 [5–17] {56}	13 [7–29] {16}	0.04[a]	6 [5–17] {35}	13 [8–25] {13}	0.04[a]

[a]Statistically significant, [IQR] interquartile range with median, (SD) standard deviation with mean, interquartile range, {} number, ms, milliseconds; mm, millimetres; mmHg, millimetre mercury; *n*, number; TAPSE, Tricuspid Annular Plane Systolic Excursion; RVSP, right ventricular systolic pressure; RVS', right ventricular systolic excursion velocity; cm/s, centimetre per second; RVFWS, right ventricular free wall strain; RA, right atrial; E/E', wave, A A' wave, RV, right ventricular, IVC, inferior vena cava, LVEF, left ventricular ejection fraction, CRP, C reactive protein.

revised it, and approved the submitted version, and agrees both to be personally accountable for the author's contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature. B.F.J.: conception, design of the work, substantively revised it, and approved the submitted version, and agrees both to be personally accountable for the author's contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature. S.O.: conception, design of the work, analysis, and interpretation of data, substantively revised it, and approved the submitted version, and agrees both to be personally accountable for the author's contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature. R.M.: design of the work, data collection, and sample collection, interpretation of data, drafted the work, substantively revised it, and approved the submitted version, and agrees both to be personally accountable for the author's contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

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