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Echocardiographic assessment of right ventricular dysfunction and outcome in patients with severe Covid-19 Pneumonia

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ABSTRACT

Background: Fatal cardiovascular complications and acute respiratory distress syndrome (ARDS) account for the majority of SARS-CoV-2-associated deaths. The objective of this research was to find transthoracic echocardiography (TTE) of right ventricular (RV) dysfunction parameters that can be utilized to predict outcomes in individuals with severe COVID-19 pneumonia;

Methods: This observational research included 90 cases with severe COVID-19 pneumonia subjected to TTE on the day of admission and 3rd day to determine the relationship between severity, mortality in severe COVID-19 pneumonia and RV function parameters;

Results: TAPSE, SPAP, RVD, RV-WT, and RV-FAC had significant differences among the two groups. PaO_2/FiO_2 and average MAP were significantly correlated with all RV parameters. Adjusted multivariate regression analysis on day 1 showed that TAPSE and SPAP followed by RVFAC were significantly related to mortality. While on day 3, it was revealed that RVFAC then SPAP were significantly related to mortality. SPAP, with a cutoff point >46 mmHg, was the most sensitive parameter, while the most specific to predict mortality was TAPSE, with a cutoff point ≤15 mm.

Conclusions: In cases with severe COVID-19 pneumonia, prediction of mortality can be performed by measuring RV parameters by TTE with high sensitivity and specificity.

Highlights

- Relation between RV and COVID-19
- RV TE parameters could be used to predict mortality and TTE has high sensitivity.

What are the main findings?

- TTE parameters of the RV had high sensitivity and specificity
- Prediction of death in patients with severe COVID-19 pneumonia.

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1. Introduction

The WHO in March 2020 reported a pandemic known as Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus (SARS CoV-2) [1]. It has a high degree of transmissibility and higher infectivity than influenza. It has a mortality rate ranging from <0.5% to >7% [2,3]. Fatal cardiovascular consequences plus acute respiratory distress syndrome (ARDS) account for the majority of SARS-CoV -2-related deaths. A much higher death rate was found in COVID-19 cases with myocardial injury than those without (59.6% vs. 8.9%) [4].

A range of SARS-CoV-2 cardiovascular manifestations involves cardiogenic shock, decompensated heart failure, myocardial infarction, and arrhythmias. It is unclear whether these signs are caused by SARS-CoV-2 directly or indirectly via myocardial dysfunction induced by cytokine [5,6]. In both medical and intensive care settings, echocardiography has become a valuable clinical approach, as it can provide information about heart–lung exchanges, current hemodynamic status also concomitant clinical problems. RV examination by echocardiography in COVID-19 patients is essential for evaluating the disease and tracking its progression. This examination should rely on practicable plus reproducible measurements, as it must be completed in a reasonably short duration to limit the contact hazard and because of the uncomfortable protective apparatus [7,8].

Directly and indirectly, the RV is included in COVID-19 development. Directly because of the COVID-19 infection lung tropism, resulting in hypoxic pulmonary vasoconstriction, interstitial pneumonia leading to an increase in afterload of RV. The function plus dimension of the RV affected by ventilatory therapy (non-invasive, invasive) influences via affecting heart–lung exchanges. The entire CVS (including the RV) is affected by COVID-19 activation of a systemic inflammation primarily by generating an elevation in sympathetic tone and troponin levels, volemic status

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alteration, and cytokine-negative inotropic effects [8–11]

The objective of this research was to find transthoracic echocardiography (TTE) of RV dysfunction parameters that can be utilized to predict outcomes in individuals with severe COVID-19 pneumonia.

2. Materials and methods

This research was performed on 90 adult cases with severe COVID-19 pneumonia (who meet any of the following: a. respiratory distress, respiratory rate (RR) ≥30 times/min, b. spO2 ˂93% at rest, c. PaO2/FiO2 ≤ 300 mmHg, * patients showing a rapid progression (>50%) on CT imaging within 24–48 hours should be managed as severe. d. Respiratory failure, need mechanical assistance) according to sample size calculation and were admitted Alexandria main university hospital's critical care department from 10/2021. By nasopharyngeal swabs, RT-PCR assays were utilized for confirmation of severe COVID-19 pneumonia in all cases [12]. Cases who were less than 18 years, pregnant females with systolic heart failure with valvular lesions, poor echocardiographic windows (obesity – COPD – pneumothorax), an underlying primary pulmonary parenchymal condition that may influence the right heart as (COPD, interstitial lung diseases), and significant arrhythmias were excluded.

All cases meeting the inclusion criteria were involved in our research. Informed consent was obtained before performing the research from the next of kin. Our institution ethics committee approved the research.

According to 30-day mortality, patients participating in the research were classified as survivors and non-survivors. Demographic characteristics, routine chest radiography, clinical characteristics and vital signs (temperature, non-invasive mean arterial pressure, heart rate, RR), Standard hematology (CBC) and biochemistry indices, biomarkers including C reactive protein, Interleukin-6 and D-dimer levels on admission and when indicated, baseline arterial blood gases (pH – $PaCO_2$ – PaO_2 : FiO_2 ratio – serum HCO_3) on admission and on need, mechanical ventilation (MV) days, intensive care unit (ICU) stay length, vasopressors (norepinephrine) or inotropes (dopamine or dobutamine) need and their doses, were analyzed between both groups. In addition, to evaluate the RV function parameters systolic pulmonary arterial pressure (SPAP), tricuspid annular plane systolic excursion (TAPSE), RV wall thickness, RV diameter, and RV-fractional area change (FAC), TTE was performed on admission and day 3. Using the 2010 guidelines of American society of echocardiography for adult's right heart evaluation, the following were assessed and recorded:

RV dimensions: RV internal diameters (base, mid cavity and apex to base) measuring in the 4-chamber apical view on TTE. **RV wall thickness**: It is measured in diastole from an apical or subcostal 4 view by M-mode or 2D imaging. **RV fractional area change (RVFAC)**: is a measure of RV systolic function and is defined as (end-diastolic area – end-systolic area)/end-diastolic area × 100). **Systolic pulmonary artery pressure (SPAP)**: by assessing the peak regurgitant jet velocity (v) with modified Bernoulli equation (RV systolic pressure = 4v2 + right atrial pressure), continuous-wave Doppler echocardiography and in the presence of tricuspid regurgitation, can determine the RV right atrial pressure gradient [13]. **Tricuspid annular plane systolic excursion (TAPSE)**: the distance of RV annular segment systolic excursion along its longitudinal plane from a conventional 4-chamber apical window measuring. TAPSE reflects RV longitudinal function [14].

IBM SPSS 20 software suite was used to analyze the data. The Student's t-test was used to examine the differences in age, PaCO2, PaO2/FiO2 ratio, pH, serum bicarbonate (HCO3), and RV parameters between the studied groups. Non-parametric data were analyzed using the Mann–Whitney test. In all groups, to compare the relationship between sex and comorbidities, the Chi-square test was employed. Using Pearson coefficients, the correlation between RV function parameters and (PaO2/FiO2 ratio, MV days, and average MAP) was determined. Utilizing the multivariate logistic regression model, the effect of variables on mortality was determined. To predict death, agreement (sensitivity and specificity) echocardiographic RV parameters were examined. Our institution's Community Medicine Department calculated the sample size using the Epi 7 program for sample size computation. A minimum sample size of 90 cases was important to get 95% confidence limits and 80% study power.

3. Results

According to 30-day mortality, cases were classified into 2 groups, survivors (group 1) (57 patients, 63.3%) and non-survivors (group 2) (33 patients, 36.7%). Demographic data (age, sex) were insignificantly different between both groups. The survivors had significantly higher pH, HCO_3 and PaO_2/FiO_2 ratio compared to non-survivors ($p = 0.041$, 0.046 and 0.006, respectively) and $PaCO_2$ was insignificantly different between both groups. The survivors had significantly lower HR, respiratory rate and temperature and significantly higher MAP on days 1 and 3 than non-survivors ($p < 0.05$). There was a significant difference between both groups regarding the Vasopressor (Norepinephrine) requirements ($p < 0.001$). The survivor group has significantly a shorter duration of vasopressor use than the non-survivor group ($p = 0.020$). The survivor group has significantly lower CRP, IL-6 and D. dimer

compared to the non-survivors. There was a significant difference between both groups regarding the inotrope's requirements (*p* value 0.004). Regarding need for MV, it was significantly higher among non-survivors' group ($p < 0.001$). Survivors had more days of MV plus higher length of ICU stay than non-survivors ($p < 0.05$). Survivors had a significantly higher RV-FAC, TAPSE plus RV wall thickness and significantly lower SPAP and RV diameter on days 1 and 3 than the non-survivors' group ($p < 0.001$) (Table 1).

On days 1 and 3, RV function parameters were significantly correlated with PaO2/FiO2 ratio and average MAP, while they were insignificantly correlated with MV days (Table 2).

In multivariate regression, on day 1, TAPSE & SPAP, then RVFAC ($p < 0.05$) were significantly related to

Table 1. Comparison between survivors and non-survivors of ARDS regarding baseline characteristics and right ventricular function parameters.

Characteristics	Survivors (*n* = 57)	Non-survivors (*n* = 33)	P value
Age[years] (mean ± SD)	46.60 ± 14.57	45.27 ± 13.66	0.672
Sex [n%]			
Male	34(59.6)	13(39.4)	0.064
Female	23(40.4)	20(60.6)	
Diabetes mellitus [n%]	8 (14.0)	10 (30.3)	0.063
Hypertension [n%]	9 (15.8)	8 (24.2)	0.324
Heart rate (beat/min) (mean ± SD)			
Day1	95.12 ± 13.14	103.6 ± 19.36	0.031*
Day3	89.11 ± 8.73	95.64 ± 12.53	0.011*
Temperature (°C) (mean ± SD)			
Day1	38.54 ± 0.81	39.30 ± 0.88	<0.001*
Day3	38.04 ± 0.59	38.49 ± 0.71	0.002*
Respiratory rate (breath/min) (mean ± SD)			
Day1	20.95 ± 2.86	22.48 ± 2.84	0.016*
Day3	19.23 ± 2.64	20.76 ± 4.07	**0.002***
Mean arterial blood pressure (mmHg) (mean ± SD)			
Day1	82.79 ± 9.93	74.09 ± 9.82	**<0.001***
Day3	84.30 ± 7.86	66.91 ± 13.40	**<0.001***
Vasopressor (Norepinephrine)			
Need for it [n%]	6 (10.5)	16 (48.5)	**<0.001***
Day of beginning (mean ± SD)	2.50 ± 0.84	3.06 ± 1.12	**0.207**
Duration [days] (mean ± SD)	3.0 ± 0.89	5.25 ± 1.98	**0.020***
Inotropes			
Need for it [n%]	2 (3.5)	8 (24.2)	**0.004***
Day of beginning (mean ± SD)	3.50 ± 0.71	3.25 ± 0.71	**0.665**
Duration [days] (mean ± SD)	3.50 ± 0.71	4.50 ± 1.85	**0.425**
D. Dimer (mg/l) (mean ± SD)			
Day1	0.89 ± 0.14	1.10 ± 0.19	**<0.001***
Day3	0.54 ± 0.12	0.92 ± 0.27	**<0.001***
Interleukin-6 (pg/ml) (mean ± SD)			
Day1	77.16 ± 80.4	154.55 ± 249.65	0.002*
Day3	42.37 ± 35.81	70.79 ± 83.42	<0.001*
CRP (mg/l) (mean ± SD)			
Day1	65.74 ± 66.33	79.0 ± 78.37	0.008*
Day3	34.84 ± 37.66	69.03 ± 55.48	<0.001*
PH (mean ± SD)	7.40 ± 0.05	7.38 ± 0.06	0.041*
PCO_2 (mmHg) (mean ± SD)	36.25 ± 6.21	38.61 ± 6.40	0.089
PaO_2/FiO_2 (mean ± SD)	141.02 ± 35.37	122.88 ± 42.80	0.006*
HCO_3 (mEq/L) (mean ± SD)	23.09 ± 2.15	23.09 ± 2.15	0.046*
RVFAC (%) (mean ± SD)			
Day1	45.95 ± 13.23	34.33 ± 9.12	<0.001*
Day3	47.12 ± 11.55	47.12 ± 11.55	<0.001*
SPAP (mm Hg) (mean ± SD)			
Day1	36.81 ± 12.22	56.27 ± 5.84	<0.001*
Day3	35.07 ± 10.63	56.85 ± 5.93	<0.001*
RV diameter (mm) (mean ± SD)			
Day1	35.07 ± 5.07	40.09 ± 5.38	<0.001*
Day3	33.33 ± 4.54	41.48 ± 4.74	<0.001*
RV wall thickness (mm) (mean ± SD)			
Day1	2.99 ± 0.71	2.49 ± 0.50	<0.001*
Day3	3.50 ± 0.88	2.42 ± 0.57	<0.001*
TAPSE (mm) (mean ± SD)			
Day1	19.54 ± 3.75	13.73 ± 3.08	<0.001*
Day3	20.39 ± 3.44	11.88 ± 2.76	<0.001*
Invasive mechanical ventilation			
Yes [n%]	7 (12.3)	33 (100)	
No [n%]	50 (87.7)	0 (0.0)	<0.001*
Duration (days) (mean ± SD)	7.29 ± 1.80	5.76 ± 1.58	0.035*
Length of ICU stay(days) (mean ± SD)	12.91 ± 3.26	7.64 ± 3.06	

Note: CRP:C reactive protein, HCO_3: serum bicarbonate, ICU: intensive care unit, PCO_2: partial pressure of carbon dioxide, PaO_2/FiO_2: partial pressure oxygen, RVFACL: Right ventricular fractional area change, RV: Right ventricle, SPAP: Systolic Pulmonary Artery Pressure, TAPSE: Tricuspid annular plane systolic excursion, *: Statistically significant at $p \leq 0.05$.

Table 2. Correlation between RV parameters with different parameters in total sample (n = 90).

		PaO_2/FiO_2		Mechanical ventilation days		Average MAP	
RV parameters		rs	p	rs	p	r	p
RVFAC	day 1	0.248*	0.018*	−0.003	0.983	0.240*	0.023*
	day 3	0.263*	0.012*	−0.025	0.881	0.287*	0.006*
SPAP	day 1	−0.277*	0.008*	−0.221	0.171	−0.458*	<0.001*
	day 3	−0.268*	0.011*	−0.223	0.167	−0.497*	<0.001*
RV. D	day 1	−0.342*	0.001*	0.086	0.600	−0.278*	0.008*
	day 3	−0.275*	0.009*	−0.016	0.921	−0.420*	<0.001*
RV. WT	day 1	0.204	0.054	−0.039	0.810	0.204	0.053
	day 3	0.273*	0.009*	0.033	0.840	0.393*	<0.001*
TAPSE	day 1	0.249*	0.018*	−0.008	0.960	0.409*	<0.001*
	day 3	0.210*	0.047*	0.180	0.267	0.468*	<0.001*

RV: right ventricle, RVFAC: Right ventricular fractional area change, RV. D: Right ventricular diameter, RV. WT: Right ventricular wall thickness, SPAP: Systolic Pulmonary Artery Pressure, TAPSE: Tricuspid annular plane systolic excursion r: Pearson coefficient, rs: Spearman coefficient, *: Statistically significant at $p \leq 0.05$.

mortality. So TAPSE plus SPAP were the highest statistically significant independent predictor for mortality (Table (3a)). Additionally, on the 3rd day RVFAC followed by SPAP were independently associated with mortality ($p < 0.05$). So RVFAC was the highest statistically significant independent predictor for mortality (Table (3b)).

On ROC curve analysis to predict the 1st day mortality. The most specific RV function parameter to predict mortality was TAPSE then RV wall thickness then SPAP ($p < 0.001$). Whereas the highest sensitivity to predict mortality was SPAP with then RV diameter ($p < 0.001$) (Table 4a and Figure 1). Also, on the 3rd day, the most specific RV function parameter to predict mortality was TAPSE then RV wall thickness, then RVFAC ($p < 0.001$). Whereas SPAP had the highest sensitivity to predict mortality, then RVFAC ($p < 0.001$) (Table 4b and Figure 2).

4. Discussion

Research has found biological indicators of cardiac injury with good prediction accuracy for in-hospital events that are easy to use [15–18]. Notwithstanding these efforts, COVID-19's cardiac pathophysiological processes must be clarified. Early results showed that cardiac imaging can predict and explain cardiac involvement [19–22].

The main aim of this study was to identify transthoracic echocardiography (TTE) parameters of RV dysfunction that can be used to predict outcomes in patients with severe COVID-19 pneumonia.

Group 2 had more adverse events and a higher heart rate on days 1 and 3. A recent systematic review and meta-analysis of COVID-19 mortality predictors by Shi et al. indicated that a higher heart rate was strongly associated with death [23]. On days 1 and 3, group 2 patients had higher temperatures. Yu et al. discovered that COVID-19 in-hospital mortality was independently associated with body temperature >37.3°C [24]. Mammen et al. also discovered a strong mortality-fever correlation [25].

On days 1 and 3, group 2 showed considerably higher respiratory rates. Shi et al.'s systematic review and meta-analysis also identified a substantial link between COVID-19 respiratory rate and death [23]. Liu et al. also discovered that COVID-19 patients' breathing rates substantially correlated with death [26].

Group 1 had a higher MAP on days 1 and 3. Liu et al. reported comparable relationships between MAP and COVID-19 mortality, validating our findings [26].

Table 3. (A) Univariate and multivariate analyses for the parameters affecting mortality on day 1. (b) Univariate and multivariate analyses for the parameters affecting mortality on day 3.

	Univariate		Adjusted multivariate	
Day 1	p	OR (95%C. I)	p	AOR# (95%C. I)
RVFAC	<0.001*	0.909* (0.863–0.957)	0.001*	0.886*(0.823–0.953)
SPAP	<0.001*	1.192*(1.106–1.284)	<0.001*	1.233*(1.105–1.376)
RV. D	<0.001*	1.184*(1.084–1.293)	0.001*	1.222*(1.087–1.375)
RV. WT	0.001*	0.253*(0.110–0.582)	0.008*	0.217*(0.071–0.667)
TAPSE	<0.001*	0.606*(0.491–0.749)	<0.001*	0.602*(0.467–0.775)
Day 3	Univariate		Adjusted multivariate	
	p	OR (95%C.I)	p	AOR# (95%C.I)
RVFAC	<0.001*	0.839* (0.778–0.904)	<0.001*	0.837*(0.758–0.924)
SPAP	<0.001*	1.284*(1.147–1.438)	0.008*	1.559*(1.121–2.169)
RV. D	<0.001*	1.368 (1.211–1.545)	0.633	0.919 (0.649–1.300)
RV. WT	0.001*	0.137* (0.058–0.324)	0.167	8.089 (0.416–157.251)
TAPSE	<0.001*	0.483*(0.357–0.653)	0.142	0.612 (0.318–1.178)

RV: right ventricle, RVFAC: Right ventricular fractional area change, RV. D: Right ventricular diameter, RV. WT: Right ventricular wall thickness, SPAP: Systolic Pulmonary Artery Pressure, TAPSE: Tricuspid annular plane systolic excursion #: Adjusted OR with PaO_2/FiO_2, MAP, IL6, Need for vasopressors, and D. dimer admission, OR: Odd`s ratio, C.I: Confidence interval, *: Statistically significant at $p \leq 0.05$.

Table 4. (A) validity (AUC, sensitivity, specificity) for different parameters to predict mortality in 1st day. (b) validity (AUC, sensitivity, specificity) for different parameters to predict mortality in 3rd day.

1st day	AUC	p	95% C. I	Cut off#	Sensitivity	Specificity	PPV	NPV
RVFAC	0.762	<0.001*	0.663–0.860	**≤38**	72.73	64.91	54.5	80.4
SPAP	0.890	<0.001*	0.822–0.957	**>46**	93.94	77.19	70.5	95.7
RV. D	0.784	<0.001*	0.688–0.879	**>34#**	81.82	68.42	60.0	86.7
RV. WT	0.692	0.002*	0.582–0.803	**≤2.3#**	51.52	78.95	58.6	73.8
TAPSE	0.890	<0.001*	0.817–0.964	**≤15**	81.82	82.46	73.0	88.7
3rd day	AUC	p	95% C.I	Cut off#	Sensitivity	Specificity	PPV	NPV
RVFAC	0.907	<0.001*	0.836–0.979	**≤33**	87.88	84.21	76.3	92.3
SPAP	0.949	<0.001*	0.908–0.990	**>44**	96.97	77.19	71.1	97.8
RV. D	0.883	<0.001*	0.812–0.954	**>38**	81.82	84.21	75.0	88.9
RV. WT	0.845	<0.001*	0.762–0.929	**≤2.9#**	78.79	87.72	78.8	87.7
TAPSE	0.962	<0.001*	0.921–1.003	**≤13**	87.88	100.0	100.0	93.4

AUC: Area Under a Curve value, CI: Confidence Intervals, NPV: Negative predictive value, PPV: Positive predictive value RV: right ventricle, RVFAC :Right ventricular fractional area change, RV.D: Right ventricular diameter RV.WT: Right ventricular wall thickness, SPAP: Systolic Pulmonary Artery Pressure, *p* value: Probability, TAPSE: Tricuspid annular plane systolic excursion, *: Statistically significant at $p \leq 0.05$, #Cut off was choose according to Youden index.

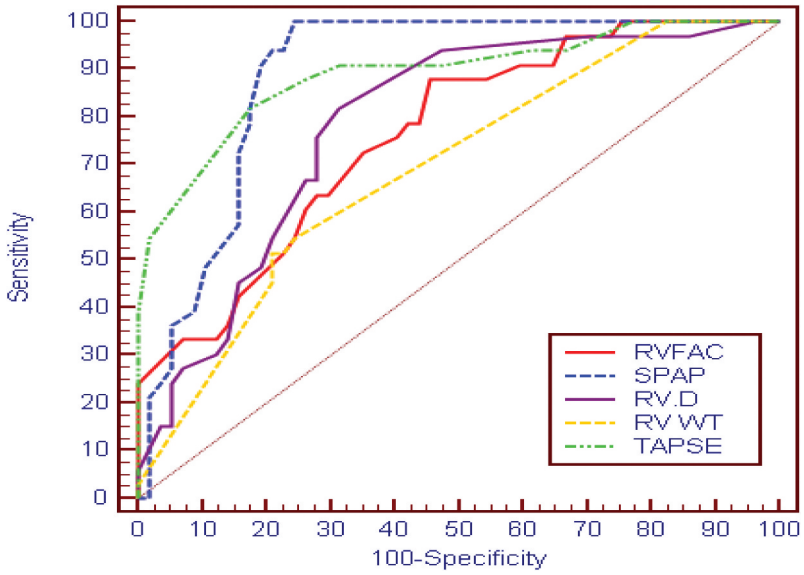

Figure 1. ROC curve for different parameters to predict mortality in 1st day.

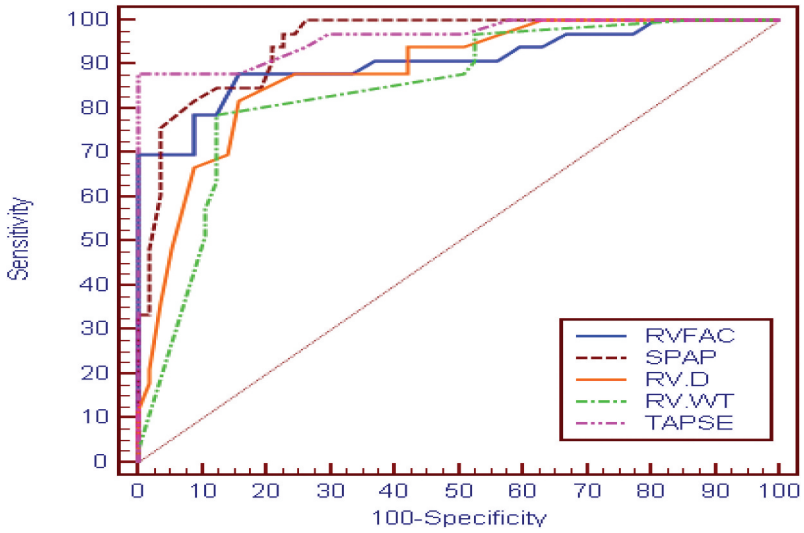

Figure 2. ROC curve for different parameters to predict mortality in 3rd day.

Group 2 had higher concentrations of D. Dimer, IL-6 and CRP on days 1 and 3. D-dimer >1 mg/L and ferritin >500 ng/mL increased mortality risk, according to Mammen et al. [25]. Also non-survivors had higher admission IL-6 levels than survivors.

Chidambaram et al. D-dimer-CRP's mortality relationship validated our findings [27]. Interleukin-6 did not affect mortality or illness severity. Shi et al. confirmed that dead patients had higher D-dimer, CRP, and IL-6 levels than survivors [23].

Group 1 had much greater PH, PaO2/FiO2, and HCO3 than Group 2. Paternoster et al. showed that 3 of 9 PH investigations in 446 individuals supported our findings. Seventy-two of 136 (52.9%) PH patients and 46 of 310 (14.8%) non-PH patients died [28].

Mammen et al. discovered a substantial correlation between mortality and PaO2/FiO2 ratio, supporting our findings [25]. Grasselli et al. found that ICU entrance high FiO2, high PEEP, or low PaO2:FiO2 ratio were independent mortality risk factors [29]. Shi et al. identified PaO2, PaCO2, and PaO2/FiO2 differences between survivors and non-survivors [23].

On days 1 and 3, groups differed in RVFAC, SPAP, RV diameter, RV wall thickness, and TAPSE. Rath et al. connected right-ventricular dysfunction to death, supporting our findings [30]. RVLS, RVFAC, and TAPSE increased COVID-19 mortality, according to Li et al [31]. Li et al. discovered RVFAC and TAPSE increased COVID-19 mortality [32]. Zhang et al. also found that RVLS and RVFAC increased COVID-19 mortality [33].

Groups 1 and 2 had very varied mechanical ventilation days. Mammen et al. related invasive mechanical ventilation to mortality. Invasive mechanical breathing increased mortality [25]. Taylor et al. linked death to admission mechanical ventilation [34].

Manzur-Sandoval et al. found significant relationships between PaO2/FiO2, TAPSE, RV S wave, TAPSE/PASP ratio, but not RV basal diameter or RV FS [35].

Around 10% of hospitalized patients had RV dysfunction and dilatation, and 60% had elevated PASP levels. The absence of linkage between RV alterations and PaO2/FiO2 levels shows that, in addition to hypoxemia, hypercapnia, and elevated intra-thoracic pressure, PH and RV afterload are associated to pulmonary infection severity and high mechanical ventilation parameters in this group of patients.

ROC curves predicted first-day mortality. SPAP with >46 mmHg was the most sensitive right ventricular function metric to predict death, whereas TAPSE with 15 mm was the most specific. While in third day SPAP with >44 mmHg was the most sensitive right ventricular function metric to predict death, whereas TAPSE with 13 mm was the most specific.

Univariate analysis showed that increased HR, temperature, RR, MAP, vasopressors, inotropes, D-Dimer, IL-6, CRP, PH, PaO2/FiO2, RVFAC, SPAP, RV diameter, RV wall thickness, TAPSE, and days of mechanical ventilation were associated with higher death rates. Research shows this.

An adjusted multivariable regression analysis revealed that on day 1, the most significant independent predictors of mortality were TAPSE in addition to SPAP. The most significant independent predictor on day 3 was RVFAC.

Despite the fact that our study's sample size is larger than that of previous research, a larger sample size is necessary to raise the study's power and reduce the error margin. In addition, our study was conducted at a single center, which may diminish the external validity necessary to justify widespread practice improvements.

5. Conclusions

With high sensitivity plus specificity, TTE parameters of the RV might be utilized to predict death in patients with severe COVID-19 pneumonia.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

[1] Valencia DN. Brief Review on COVID-19: The 2020 Pandemic Caused by SARS-CoV-2. Cureus. 2020 Mar 24;12(3):e7386. doi:10.7759/cureus.7386

[2] Dong E, Du H, Gardner L. An interactive web-based dashboard to track COVID-19 in real time. Lancet Infect Dis. 2020;20(5):533–534.

[3] Zhang S, Diao M, Yu W, et al. Estimation of the reproductive number of novel coronavirus (COVID-19) and the probable outbreak size on the diamond princess cruise ship: a data-driven analysis. Int J Infect Dis. 2020;93:201–204.

[4] Guo T, Fan Y, Chen M, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020;5(7):811–818. DOI:10.1001/jamacardio.2020.1017

[5] Fried JA, Ramasubbu K, Bhatt R, et al. The variety of cardiovascular presentations of COVID-19. Circulation. 2020;141(23):1930–1936. DOI:10.1161/CIRCULATIONAHA.120.047164

[6] Clerkin KJ, Fried JA, Raikhelkar J, et al. COVID-19 and cardiovascular disease. Circulation. 2020;141(20):1648–1655. DOI:10.1161/CIRCULATIONAHA.120.046941

[7] Zochios V, Parhar K, Tunnicliffe W, et al. The right ventricle in ARDS. Chest. 2017;152(1):181–193.

[8] Lazzeri C, Bonizzoli M, Batacchi S, et al. Echocardiographic assessment of the right ventricle in COVID-related acute respiratory syndrome. IEM. 2021;16(1):1–5.

[9] Nishiga M, Wang DW, Han Y, et al. COVID-19 and cardiovascular disease: from basic mechanisms to clinical perspectives. Nat Rev Cardiol. 2020;17(9):543–558.

[10] Azevedo RB, Botelho BG, Hollanda JVG, et al. Covid-19 and the cardiovascular system: a comprehensive review. J Hum Hypertens. 2021;35(1):4–11. DOI:10.1038/s41371-020-0387-4

[11] Baigent C, Windecker S, Andreini D. ESC guidance for the diagnosis and management of cardiovascular disease during the COVID-19 pandemic: part 2—care pathways, treatment, and follow-up. Eur Heart J. 2022;43(11):1059–1103.

[12] Long C, Xu H, Shen Q, et al. Diagnosis of the coronavirus disease (COVID-19): rrt-PCR or CT? Eur J Radiol. 2020;126:108961.
[13] Jones N, Burns AT, Prior DL. Echocardiographic assessment of the right ventricle–state of the art. Heart Lung Circ. 2019;28(9):1339–1350.
[14] Martha JW, Pranata R, Wibowo A, et al. Tricuspid annular plane systolic excursion (TAPSE) measured by echocardiography and mortality in COVID-19: a systematic review and meta-analysis. Int J Infect Dis. 2021;105:351–356.
[15] Parohan M, Yaghoubi S, Seraji A. Cardiac injury is associated with severe outcome and death in patients with Coronavirus disease 2019 (COVID-19) infection: a systematic review and meta-analysis of observational studies. Eur Heart J Acute Cardiovasc Care. 2020;9(6):665–677.
[16] Stefanini GG, Chiarito M, Ferrante G, et al. Early detection of elevated cardiac biomarkers to optimise risk stratification in patients with COVID-19. Heart. 2020;106(19):1512–1518. DOI:10.1136/heartjnl-2020-317322
[17] Gao C, Gao C, Cai Y, et al. Association of hypertension and antihypertensive treatment with COVID-19 mortality: a retrospective observational study. Eur Heart J. 2020;41(22):2058–2066. DOI:10.1093/eurheartj/ehaa433
[18] Ruscica M, Macchi C, Iodice S, et al. Prognostic parameters of in-hospital mortality in COVID-19 patients—an Italian experience. Eur J Clin Invest. 2021;51(9).
[19] Argulian E, Sud K, Vogel B, et al. Right ventricular dilation in hospitalized patients with COVID-19 infection. JACC Cardiovasc Imaging. 2020;13(11):2459–2461. DOI:10.1016/j.jcmg.2020.05.010
[20] Huang L, Zhao P, Tang D, et al. Cardiac involvement in patients recovered from COVID-2019 identified using magnetic resonance imaging. JACC Cardiovasc Imaging. 2020;13(11):2330–2339. DOI:10.1016/j.jcmg.2020.05.004
[21] Churchill TW, Bertrand PB, Bernard S, et al. Echocardiographic features of COVID-19 illness and association with cardiac biomarkers. J Am Soc Echocardiogr. 2020;33(8):1053–1054. DOI:10.1016/j.echo.2020.05.028
[22] Mahmoud-Elsayed HM, Moody WE, Bradlow WM, et al. Echocardiographic findings in patients with COVID-19 pneumonia. Can J Cardiol. 2020;36(8):1203–1207. DOI:10.1016/j.cjca.2020.05.030
[23] Shi C, Wang L, Ye J, et al. Predictors of mortality in patients with coronavirus disease 2019: a systematic review and meta-analysis. BMC Infect Dis. 2021 Dec;21(1):1–5.
[24] Yu Z, Ke Y, Xie J, et al. Clinical characteristics on admission predict in-hospital fatal outcome in patients aged≥ 75 years with novel coronavirus disease (COVID-19): a retrospective cohort study. BMC Geriatr. 2020 Dec;20(1):1–2.
[25] Mammen JJ, Kumar S, Thomas L, Kumar G, Zachariah A, Jeyaseelan L, Kumar VS. Factors associated with mortality among moderate and severe patients with COVID-19 in India: a secondary analysis of a randomised controlled trial. BMJ Open. 2021;11(10):e050571.
[26] Liu Y, Du X, Chen J, et al. Neutrophil-to-lymphocyte ratio as an independent risk factor for mortality in hospitalized patients with COVID-19. J Inf Secur. 2020;81(1):e6–12. DOI:10.1016/j.jinf.2020.04.002
[27] Chidambaram V, Tun NL, Haque WZ, et al. Factors associated with disease severity and mortality among patients with COVID-19: a systematic review and meta-analysis. PLoS ONE. 2020 Nov 18;15(11):e0241541.
[28] Paternoster G, Bertini P, Innelli P, et al. Right ventricular dysfunction in patients with COVID-19: a systematic review and meta-analysis. J Cardiothorac Vasc Anesth. 2021 Nov 1;35(11):3319–3324.
[29] Grasselli G, Greco M, Zanella A, et al. Risk factors associated with mortality among patients with COVID-19 in intensive care units in Lombardy, Italy. JAMA Intern Med. 2020 Oct 1;180(10):1345–1355.
[30] Rath D, Á PU, Avdiu A, et al. Impaired cardiac function is associated with mortality in patients with acute COVID-19 infection. Clin Res Cardiol. 2020 Dec;109(12):1491–1499.
[31] Li Y, Li H, Zhu S, et al. Prognostic value of right ventricular longitudinal strain in patients with COVID-19. Cardiovascular Imaging. 2020 Nov 1;13(11):2287–2299.
[32] Li Y, Fang L, Zhu S, et al. Echocardiographic characteristics and outcome in patients with COVID-19 infection and underlying cardiovascular disease. Front Cardiovasc Med. 2021 Mar 16;8:127.
[33] Zhang Y, Sun W, Wu C, et al. Prognostic value of right ventricular ejection fraction assessed by 3D echocardiography in COVID-19 patients. Front Cardiovasc Med. 2021 Feb 9;8:61. DOI:10.3389/fcvm.2021.641088.
[34] Taylor EH, Marson EJ, Elhadi M, et al. Factors associated with mortality in patients with COVID-19 admitted to intensive care: a systematic review and meta-analysis. Anaesthesia. 2021 Sep;76(9):1224–1232.
[35] Manzur-sandoval D, García-cruz E, Gopar-nieto R, et al. Right ventricular dysfunction and right ventricular–arterial uncoupling at admission increase the in-hospital mortality in patients with COVID-19 disease. Echocardiography. 2021 Aug;38(8):1345–1351.