Table 1 Impact of COVID-19 pandemic on diagnosis and outcomes of newborns diagnosed with SVCHD

	Prepandemic group (*n* = 53)	Pandemic group (*n* = 29)	*P* value
Demographics			
Ethnicity			**.06**
Caucasian	22 (43)	18 (62)	
Hispanic	22 (43)	9 (31)	
Other non-Caucasian	9 (14)	2 (7)	
Gender, male	31 (58)	16 (55)	NS
Maternal health insurance type			**.03**
Private	13 (26)	12 (52)	
State	36 (73)	11 (47)	
Maternal age, y	27 ± 5.2	28 ± 5.4	NS
Diagnosis			
Prenatal diagnosis	49 (92)	24 (83)	NS
Gestational age at prenatal diagnosis, wk	25 ± 5.4	26 ± 5.2	NS
Birth weight, g	2,918 ± 117	3,102 ± 126	NS
Genetic anomaly			NS
Genetic testing done/available	41 (77)	6 (79)	
Significant anomaly	10 (24)	3 (13)	
Variant of unknown significance	2 (5)	5 (17)	
Clinical outcomes			
Hypoplastic left heart syndrome	47 (89)	21 (72)	**.07**
Restrictive atrial septum	5 (10)	4 (19)	NS
Pre–stage 1 inotropic support	14 (26)	5 (17)	NS
Pre–stage 1 mechanical ventilation	19 (36)	10 (34)	NS
Time to stage 1 surgery, d	7 (6-11)	7 (5-15)	NS
Death before stage 1 surgery	9 (17)	4 (14)	NS
Stage 1 surgery outcomes			NS
Alive at stage 2	39 (87)	22 (88)	
Death/transplantation before stage 2	6 (13)	3 (12)	

Data are expressed as number (percentage), mean ± SEM, or median (interquartile range). *P* values in boldface type denote statistical significance.

mothers were on state or public insurance, which is an indirect indicator of lower socioeconomic status.

Our study demonstrates a nonsignificant decrease in prenatal diagnosis of SVCHD during the COVID-19 pandemic restrictions, which appears to have disproportionately affected certain ethnic and socioeconomic groups.

It has been reported that mothers in lower socioeconomic quartiles have decreased prenatal detection of SVCHD.[5] The COVID-19 pandemic appears to have amplified this disparity. The diagnosis of SVCHD declined in patients who were on public insurance (an indirect indicator of lower socioeconomic status). We also noted a trend in decreased prenatal diagnosis of patients self-identifying as non-Caucasian, which has not traditionally been reported in SVCHD. This represents potential geographic and demographic variation in access to care.

We acknowledge limitations of the study, including a single-center experience and small volume of patients in the pandemic group. Despite the small cohort, these findings are important, as they highlight the impact of the COVID-19 pandemic restrictions on the prenatal diagnosis of SVCHD.

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Association of Right Ventricular Dilation on Echocardiogram With In-Hospital Mortality Among Patients Hospitalized With COVID-19 Compared With Bacterial Pneumonia

Right ventricular (RV) abnormalities are the most common echocardiographic findings in acute COVID-19.[1,2] While RV dysfunction is

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Table 1 Baseline characteristics of individuals hospitalized with COVID-19 or bacterial pneumonia who underwent a clinically indicated TTE

Variable	COVID	Bacterial pneumonia	*P* value
N	225	257	
Age, years, mean ± SD	62.9 ± 16.7 (*n* = 225)	67.5 ± 17.4 (*n* = 257)	.003
Sex:			.07
Male	148 (65.8)	148 (57.6)	
Female	77 (34.2)	109 (42.4)	
Race/ethnicity:			<.001
Hispanic/Latinx	85 (38.1)	29 (11.3)	
Native American/Alaska native	3 (1.3)	1 (0.4)	
Non-Hispanic Black/African American	22 (9.9)	25 (9.7)	
Non-Hispanic White	42 (18.8)	110 (42.8)	
Asian	44 (19.7)	79 (30.7)	
Native Hawaiian or Pacific Islander	7 (3.1)	2 (0.8)	
Other Non-Hispanic	20 (9.0)	11 (4.3)	
BMI, mean ± SD	29.4 ± 8.1	26.7 ± 7.8	<.001
Hypertension	100 (44.4)	107 (41.6)	.53
Diabetes	53 (23.6)	52 (20.2)	.38
Dyslipidemia	40 (17.8)	55 (21.4)	.32
Heart failure prior to admission	14 (6.2)	23 (8.9)	.26
Prior coronary artery bypass graft	1 (0.4)	1 (0.4)	.92
Prior heart attack	4 (1.8)	2 (0.8)	.32
Prior percutaneous coronary intervention	5 (2.3)	1 (0.4)	.07
Prior atrial fibrillation	12 (5.3)	31 (12.1)	.01
Prior stroke/transient ischemic attack	18 (8.0)	17 (6.6)	.56
Cancer	37 (16.4)	82 (31.9)	<.001
Prior deep vein thrombosis	5 (2.2)	11 (4.3)	.21
Prior pulmonary embolism	4 (1.8)	17 (6.6)	.009
Chronic kidney disease	25 (11.1)	30 (11.7)	.89
On dialysis prior to admission	5 (2.2)	3 (1.2)	.37
Solid organ transplant	9 (4.0)	8 (3.1)	.6
HIV	4 (1.8)	3 (1.2)	.71
Chronic obstructive pulmonary disease	8 (3.6)	33 (12.8)	<.001
Asthma	13 (5.8)	24 (9.3)	.14
Patient managed in intensive care unit	170 (75.6)	97 (37.7)	<.001
Mechanical ventilation during hospitalization	121 (53.8)	38 (14.8)	<.001
Initiation of hemodialysis/continuous renal replacement therapy	21 (9.3)	3 (1.2)	<.001
Deep vein thrombosis or pulmonary embolism during hospitalization	57 (25.3)	29 (11.3)	<.001
Length of stay in days, median (interquartile range)	20.0 (10.0, 34.0)	8.0 (5.0, 13.0)	<.001
Days to echo, median (interquartile range)	2.0 (1.0, 5.0)	2.0 (1.0, 3.0)	.50
Intubated at echo	70 (31.1)	22 (8.6)	<.001
LV ejection fraction, mean ± SD	60.4 ± 12.7 (*n* = 203)	59.7 ± 12.2 (*n* = 232)	.56
Reduced LV function (<50%)	30 (14.8)	36 (15.5)	.83
LV longitudinal strain, mean ± SD	−18.2 ± 4.0 (*n* = 30)	−17.7 ± 4.1 (*n* = 21)	.66
RV volume:			.99
Normal	164 (77.7)	183 (77.2)	
Borderline	9 (4.3)	9 (3.8)	
Mildly reduced	19 (9.0)	25 (10.5)	

(*Continued*)

Table 1 (*Continued*)

Variable	COVID	Bacterial pneumonia	*P* value
Mild to moderately reduced	5 (2.4)	6 (2.5)	
Moderately reduced	9 (4.3)	8 (3.4)	
Moderate to severely reduced	1 (0.5)	1 (0.4)	
Severely reduced	4 (1.9)	5 (2.1)	
RV dilated:			.91
Normal RV size	165 (77.8)	183 (77.2)	
Dilated RV	47 (22.2)	54 (22.8)	
RV function:			.078
Normal	187 (87.4)	200 (87.0)	
Borderline	3 (1.4)	0 (0.0)	
Mildly reduced	17 (7.9)	23 (10.0)	
Mild to moderately reduced	3 (1.4)	7 (3.0)	
Moderately reduced	3 (1.4)	0 (0.0)	
Moderate to severely reduced	1 (0.5)	0 (0.0)	
Reduced RV function:			1
Normal RV function	187 (87.4)	200 (87.0)	
Reduced RV function	27 (12.6)	30 (13.0)	
Tricuspid annular plane systolic excursion, cm, mean ± SD	2.3 ± 1.0 (*n* = 199)	2.3 ± 1.4 (*n* = 251)	.74
RV S' velocity, mean ± SD	14.3 ± 4.2 (*n* = 92)	13.6 ± 3.8 (*n* = 121)	.2
TR peak gradient, mean ± SD	31.5 ± 13.5 (*n* = 103)	35.3 ± 15.5 (*n* = 164)	.020
TR peak gradient elevated	32 (35.2)	85 (51.8)	.01
Estimated RV systolic pressure, mm Hg, mean ± SD	35.7 ± 14.4 (*n* = 89)	40.6 ± 16.3 (*n* = 164)	.02
Estimated right atrial pressure, mm Hg, mean ± SD	4.6 ± 3.6 (*n* = 119)	5.3 ± 4.1 (*n* = 192)	.09

Data in parentheses are percentages unless otherwise specified. *T* tests were used for normally distributed continuous variables, chi-squared for categorical, and Wilcoxon rank-sum for skewed continuous data. *LV* = Left ventricular.

associated with in-hospital mortality in acute COVID-19,[3,4] the prognostic implications of RV dilation alone are less clear. Similarly, little is known about the clinical significance of RV dilation and dysfunction among patients hospitalized with acute COVID-19 compared with other respiratory illnesses, such as bacterial pneumonia. We studied the association of RV dilation and dysfunction with in-hospital mortality in acute COVID-19 compared with bacterial pneumonia.

A retrospective cohort study design was utilized to compare 225 consecutive adults admitted for acute COVID-19 between March 2020 and February 2021 at UCSF Health and Zuckerberg San Francisco General Hospital with 257 control hospitalizations for bacterial pneumonia. To be included, all patients had to have a clinically ordered transthoracic echocardiogram (TTE) during hospitalization. COVID-19 was diagnosed by polymerase chain reaction, while cases of bacterial pneumonia were included based on International Classification of Diseases codes. Qualitative measurements of RV size and function were extracted from the echo report of the first TTE performed after admission. We constructed logistic regression models to estimate associations between RV dilation, RV dysfunction, and in-hospital mortality while adjusting for age, sex, body mass index (BMI), medical history, admission estimated glomerular filtration rate, hemoglobin, and presence of mechanical ventilation and used postestimation to estimate adjusted relative risks. Fine and Gray competing-risks regression models treating discharged alive as a competing risk were implemented to estimate the effect of RV dilation on mortality.

Individuals in the COVID-19 cohort were younger, and more identified as Hispanic compared with the bacterial pneumonia group (Table 1). Body mass index was higher in the COVID-19 group, and there was a higher prevalence of cancer, chronic obstructive pulmonary disease, and atrial fibrillation in the bacterial pneumonia group. Compared with those hospitalized with bacterial pneumonia, patients with acute COVID-19 were more likely to be managed in the intensive care unit, require mechanical ventilation, be initiated on renal replacement therapy, develop a venous thromboembolism, and have a longer length of stay.

There was a similar prevalence of qualitative RV dilation (22.2% vs 22.8%, $P = .91$) and RV dysfunction (12.6% vs 13.0%, $P = 1.00$) in both groups. Similarly, the mean tricuspid annular plane systolic excursion and RV s′ were comparable between the 2 groups. Peak tricuspid regurgitation (TR) gradient and estimated RV systolic pressure were higher among those with bacterial pneumonia, despite higher use of mechanical ventilation among those with COVID-19.

Those hospitalized with COVID-19 had a higher risk of inpatient mortality compared with the bacterial pneumonia group (adjusted relative risk = 2.00; 95% CI, 1.28-3.14). Compared with those with bacterial pneumonia and normal RV size (Figure 1), the adjusted relative risk for in-hospital mortality among those with bacterial

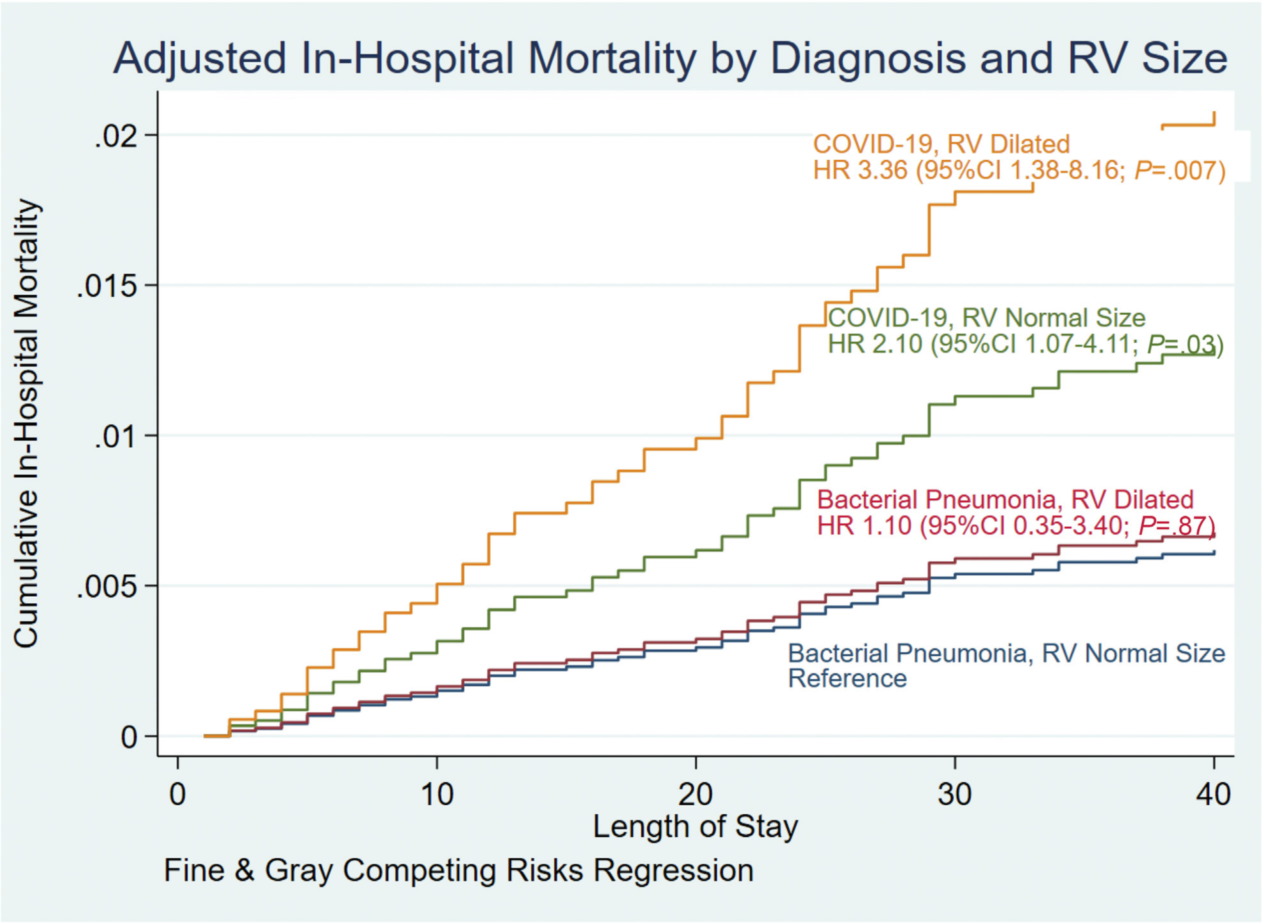

Figure 1 Fine and Gray competing-risks regression model adjusted for age, sex, BMI, medical history, admission estimated glomerular filtration rate, hemoglobin, and presence of mechanical ventilation. The reference group used was those with bacterial pneumonia with normal RV size.

pneumonia and RV dilation was 1.09 (95% CI, 0.47-2.57; $P = .84$); COVID-19 and normal RV size, 1.88 (1.05-3.37; $P = .03$); and COVID-19 with RV dilation, 3.02 (95% CI, 1.52-6.01; $P = .002$).

To our knowledge, this study is one of the first to compare the clinical significance of RV dilation in acute COVID-19 compared with other respiratory illnesses. We found that RV dilation was associated with in-hospital mortality among patients hospitalized with acute COVID-19 but not among those hospitalized with bacterial pneumonia. Consistent with our findings, in prior studies of adults admitted for H1N1 influenza, RV dilation and dysfunction were common echo findings, but neither was associated with mortality.[5,6] Our finding that RV dysfunction is not associated with mortality is discordant with the literature.[7]

Right ventricular dilation may be associated with mortality in COVID-19 due to the severity of the pulmonary pathology or due to SARS-CoV-2 infection independent of its effects in the lungs. Although the COVID-19 group had a higher mortality rate than the bacterial pneumonia group, there was a similar prevalence of RV dilation and RV dysfunction in both groups, which points toward an unidentified culprit for higher mortality beyond acute RV stress from hypoxic vasoconstriction. Interestingly, those with bacterial pneumonia had a higher TR peak gradient and estimated RV systolic pressure despite lower rates of pulmonary emboli and mechanical ventilation compared with the COVID-19 group, suggesting that the etiology of RV dilation may not correlate directly to elevated pulmonary artery pressures in hypoxic pulmonary disease. Differences in the inflammatory response to SARS-CoV-2 resulting in shunt physiology could increase RV volume overload, or perhaps the RV is more sensitive to microvascular dysfunction from coronary endotheliitis.

Limitations of this largely retrospective study include using electronic health records, which carries a risk of misclassification and confounding. Sampling bias likely occurred as we only included patients who received a clinically indicated echo; thus a sicker patient population may have been selected. While we mainly focused on qualitative measurements of RV size and function, manual measurement of RV was technically limited in clinical echocardiograms obtained in the critically ill. We did not classify the severity of illness in either group. Finally, this cohort reflects the inpatient experience of earlier variants among largely unvaccinated individuals.

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