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Impact of Preinfection Left Ventricular Ejection Fraction on Outcomes in COVID-19 Infection

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Abstract: Coronavirus disease 2019 (COVID-19) has high infectivity and causes extensive morbidity and mortality. Cardiovascular disease is a risk factor for adverse outcomes in COVID-19, but baseline left ventricular ejection fraction (LVEF) in particular has not been evaluated thoroughly in this context. We analyzed patients in our state's largest health system who were diagnosed with COVID-19 between March 20 and May 15, 2020. Inclusion required an available echocardiogram within 1 year prior to diagnosis. The primary outcome was all-cause mortality. LVEF was analyzed both as a continuous variable and using a cutoff of 40%. Among 396 patients (67 ± 16 years, 191 [48%] male, 235 [59%] Black, 59 [15%] LVEF ≤40%), 289 (73%) required hospital admission, and 116 (29%) died during 85 ± 63 days of follow-up. Echocardiograms, performed a median of 57 (IQR 11-

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122) days prior to COVID-19 diagnosis, showed a similar distribution of LVEF between survivors and decedents (P = 0.84). Receiver operator characteristic analysis revealed no predictive ability of LVEF for mortality, and there was no difference in survival among those with LVEF ≤40% versus >40% (P = 0.49). Multivariable analysis did not change these relationships. Similarly, there was no difference in LVEF based on whether the patient required hospital admission (56 ± 13 vs 55 ± 13, P = 0.38), and patients with a depressed LVEF did not require admission more frequently than their preserved-LVEF peers (P = 0.87). A premorbid history of dyspnea consistent with symptomatic heart failure was not associated with mortality (P = 0.74). Among patients diagnosed with COVID-19, pre-COVID-19 LVEF was not a risk factor for death or hospitalization. (Curr Probl Cardiol 2021;46:100845.)

Introduction

Coronavirus disease 2019 (COVID-19) is highly infectious and has caused extensive global morbidity and mortality.[1] However, the clinical presentation of COVID-19 infection can vary widely from asymptomatic, to mild symptoms, to critical illness and death. While improvements in detection and treatment have resulted in decreased case fatality rates, COVID-19 was the third leading cause of death in the United States for the year 2020.[2] Many investigators have studied the epidemiologic characteristics of this pandemic, and have identified variables such as age, race, and various comorbidities as important factors influencing the rate of adverse outcomes.[3-5] Cardiovascular disease, diabetes mellitus, and obesity have been identified as risk factors for poor outcomes in COVID-19.[6-9] However, the impact of left ventricular ejection fraction (LVEF), in particular, on COVID-19 prognosis has not been evaluated fully. A depressed LVEF could be expected to portend a poor outcome because it indicates a vulnerable myocardial status, or because reduced systolic function indicates that the patient may have less "reserve" to enable survival following the multiple organ dysfunction that can result from COVID-19. We hypothesized that lower baseline LVEF correlates with poorer outcomes. Therefore, we assessed the impact of LVEF assessed pre-COVID-19 on COVID-19 outcomes.

Methods

Study Design, Setting, and Population

This study was approved by the Ochsner Medical Center Institutional Review Board. Patients were accrued through clinical care at Ochsner Health, which is Louisiana's largest healthcare system, consisting of 40 hospitals and over 100 health centers and urgent care centers. In this retrospective cohort study, we assessed patients diagnosed with COVID-19 via qualitative polymerase chain reaction assay at an Ochsner Health facility between March 20 and May 15, 2020. Inclusion required an available echocardiogram to assess LVEF within one year prior to diagnosis. The most recent echocardiogram prior to COVID-19 diagnosis was used. The primary outcome was all-cause mortality occurring in any setting (ie, in-hospital or out-of-hospital). Hospital admissions and mortality were assessed via automated and manual review of the electronic medical record (EMR).

Data Collection

Clinical data were extracted from our health system's EMR system, Epic, with the use of an enterprise data warehouse, and also manually as required. The data extraction included the following: demographic characteristics (age, sex, patient-reported race); chronic conditions documented through diagnosis codes linked to ambulatory primary care and specialty care visits; body-mass index (BMI, the weight in kilograms divided by the square of the height in meters) recorded within the previous 12 months; smoking status; selected medications (including typical guideline-directed medical therapy for myocardial systolic dysfunction, as well as the once-common COVID therapies azithromycin and hydroxychloroquine); and vital signs (at first contact following COVID diagnosis) and medications linked to inpatient encounters. Preinfection dyspnea that could be attributed to cardiac dysfunction was assessed by review of EMR records from the date the echocardiogram was ordered, and were codified according to NYHA classification. Follow-up time was calculated manually through review of the medical record, and included all time between COVID diagnosis and the latest date the patient was known to be alive.

Statistical Analysis

Analyses were conducted using SPSS v27 (SPSS Inc., Chicago, IL). Categorical variables are presented as n (%), and continuous data are presented as mean±standard deviation (SD) or median and interquartile

range (IQR). All statistical tests were two-tailed. Values of $P \leq 0.05$ were considered significant.

Associations between LVEF, other clinical variables, and the outcome of mortality were assessed with chi-square tests, Student t-tests, or Mann-Whitney *U* tests as appropriate. Time-dependent relationships between variables and outcomes were assessed using Cox proportional-hazards analysis. Standard univariable analysis of baseline laboratory values was performed. The current study's variable of particular interest, LVEF, was evaluated both as a continuous variable and as a dichotomous variable stratified at a cutoff of 40% (ie, the cutoff for "moderate systolic dysfunction"). For multivariable analyses, in the first model, LVEF was adjusted for age, sex, body mass index (BMI), and race. Next, LVEF and all characteristics associated with endpoints in univariable analyses ($P < 0.1$) were used to build a fully adjusted model. All hazard ratio (HR) results are presented with 95% confidence intervals. Receiver operator curve analysis was used as an additional method of assessing the utility of LVEF as a screening test for predicting mortality. Event-free survival over time was illustrated using Kaplan-Meier curves, and any difference in survival between LVEF groups was compared with the log-rank test.

Results

Patient Characteristics and Hospitalizations

The characteristics of the 396 patients with COVID-19 and a recent echocardiogram are presented in Table 1. The mean age was 67 years, with 48% males. Patients of African-American race made up 59% of the study population. Patients' echocardiograms had been performed a median of 57 (IQR 11-122) days prior to COVID-19 diagnosis. The most common indications for echocardiography were congestive heart failure (n = 208, 27%), arrhythmia (n = 48, 12%), shortness of breath (n = 43, 11%), and chest pain (n = 30, 8%). The population's average LVEF was 55% ± 13%, and the 59 patients with LVEF ≤40% comprised 15% of the population. Within the ≤40% group, the mean LVEF was 28% ± 9%.

During the 85 ± 63 days of follow-up after COVID-19 diagnosis, 289 (73%) required at least one hospital admission. Among those hospitalized at least once, 1.7 ± 1.0 separate admissions were required during follow-up, with 84 (29%) patients requiring mechanical ventilation.

TABLE 1. Clinical features of patients with COVID-19 infection and a recent echocardiogram prior to infection. Characteristics of the population, with additional stratification by vital status and LVEF group

	Total Population (n = 396)	Survivors (n = 280; 71%)	Decedents (n = 116; 29%)	*P* (alive vs dead)	LVEF >40% (n = 337; 85%)	LVEF ≤40% (n = 59; 15%)	*P* (LVEF >40% vs ≤40)
Age, years	67 ± 16	64 ± 16	73 ± 14	<0.001	67 ± 16	64 ± 17	0.17
Sex, male/female	191 (48%) / 205 (52%)	115 (41%) / 165 (59%)	76 (66%) / 40 (34%)	<0.001	155 (46%) / 182 (54%)	36 (61%) / 23 (39%)	0.04
BMI, kg/m^2	31 ± 10	32 ± 10	30 ± 8	0.07	31 ± 9	30 ± 12	0.48
Black race	235 (59%)	176 (63%)	59 (51%)	0.03	198 (59%)	37 (63%)	0.67
Current smoker	89 (23%)	59 (21%)	30 (26%)	0.29	78 (23%)	11 (19%)	0.50
LVEF, %	55±13	55±13	55±14	0.84	60±6	28±9	na
LVEF ≤40%	59 (15%)	40 (14%)	19 (16%)	0.64	0 (0%)	59 (100%)	na
NYHA Class	1.8±1.0	1.8±1.0	1.8±0.9	0.61	1.7±0.9	2.5±0.8	<0.001
NYHA ≥2	186 (47%)	130 (46%)	56 (48%)	0.74	137 (41%)	49 (83%)	<0.001
Chronic kidney disease	119 (30%)	75 (27%)	44 (38%)	0.03	101 (30%)	18 (31%)	1.0
COPD	51 (13%)	35 (13%)	16 (14%)	0.74	46 (14%)	5 (9%)	0.40
Coronary Artery Disease	58 (15%)	40 (14%)	18 (16%)	0.76	43 (13%)	15 (25%)	0.02
Diabetes	123 (31%)	90 (32%)	33 (28%)	0.55	105 (31%)	18 (31%)	1.0
ESRD / HD	30 (8%)	17 (6%)	13 (11%)	0.10	24 (7%)	6 (10%)	0.42
Hypertension	229 (58%)	168 (60%)	61 (53%)	0.18	195 (58%)	34 (58%)	1.0
SLE	6 (2%)	4 (1%)	2 (2%)	1.0	4 (1%)	2 (3%)	0.22
ACE/ARB	229 (58%)	166 (59%)	63 (54%)	0.37	186 (55%)	43 (73%)	0.02
Aldosterone blocker	3 (1%)	2 (1%)	1 (1%)	1.0	0 (0%)	3 (5%)	<0.01
Azithromycin	48 (12%)	21 (8%)	26 (22%)	<0.001	32 (10%)	15 (25%)	<0.01
Beta blocker	287 (73%)	190 (68%)	97 (84%)	0.001	237 (70%)	50 (85%)	0.03
Heparin/LMWH	8 (2%)	5 (2%)	3 (3%)	0.70	4 (1%)	4 (7%)	0.02
Hydroxychloroquine	147 (37%)	78 (28%)	69 (60%)	<0.001	127 (38%)	20 (34%)	0.66

(*continued on next page*)

TABLE 1. (*continued*)

	Total Population (n = 396)	Survivors (n = 280; 71%)	Decedents (n = 116; 29%)	*P* (alive vs dead)	LVEF >40% (n = 337; 85%)	LVEF ≤40% (n = 59; 15%)	*P* (LVEF >40% vs ≤40)
SBP, mm Hg	135 ± 27	136 ± 28	132 ± 26	0.18	136 ± 27	128 ± 27	0.06
DBP, mm Hg	74 ± 16	76 ± 15	72 ± 17	0.04	75 ± 15	70 ± 21	0.08
Heart rate, bpm	95 ± 22	94 ± 22	95 ± 23	0.82	95 ± 22	95 ± 24	0.88
Peak TnI, ng/mL	0.46 ± 3.2	0.46 ± 3.7	0.47 ± 1.2	0.98	0.31 ± 2.0	1.3 ± 6.7	0.26
Required hospitalization	289 (73%)	179 (64%)	110 (95%)	<0.001	245 (73%)	44 (75%)	0.87

ACE, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure. ESRD, end stage renal disease; HD, hemodialysis; LMWH, low molecular weight heparin. LVEF, left ventricular ejection fraction; na, Not applicable; SBP,systolic blood pressure; SLE, systemic lupus erythematosus; TnI, peak troponin level.

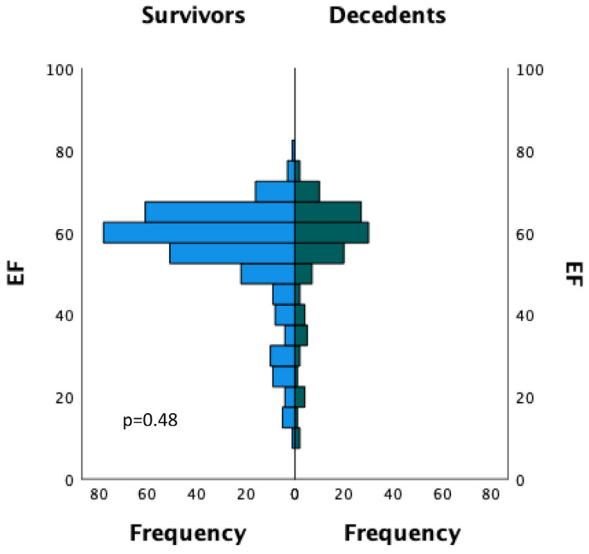


FIG 1. Distribution of left ventricular ejection fraction. There was no significant difference in LVEF when stratified by vital status at the end of follow-up (Mann-Whitney *U* test, $P = 0.42$). EF, ejection fraction.

Factors Associated With Endpoints and LVEF

Table 1 also stratifies patients by vital status at the end of follow-up, as well as by LVEF group.

First, we compared those who survived following COVID-19 diagnosis (n = 280; 71%) to those who died during follow-up (n = 116, 29%). Compared to survivors, decedents were older, more likely to be male, and less likely of African-American race. Those who died had lower diastolic blood pressure (DBP) and were more likely to have a history of chronic kidney disease. Analysis of medication use during the follow-up period showed that decedents were more likely to use beta blockers, with significantly more common use of hydroxychloroquine or azithromycin as well. As seen in Figure 1, there was a similar distribution of LVEF between survivors and decedents (55% ± 13% vs 54% ± 14%; Mann-Whitney *U* test $P = 0.48$), and there was no significant mortality-based difference in the proportion of patients with LVEF ≤40% ($P = 0.64$).

As seen in Figure 2, receiver operator characteristic analysis revealed that LVEF had no significant predictive ability for mortality ($P = 0.49$).

Similarly, there was no difference in LVEF based on whether the patient required hospital admission (56 ± 13 vs 55 ± 13, $P = 0.38$). Lastly, the distribution of pre-COVID NYHA class was similar between

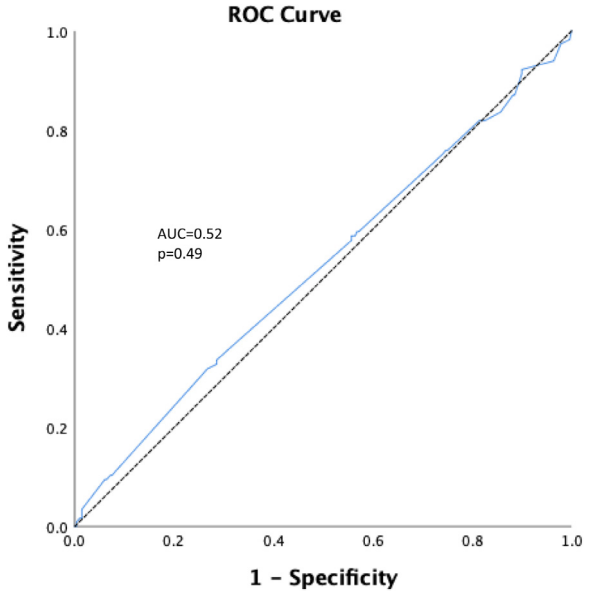


FIG 2. ROC Curve. Receiver operating characteristic curve for pre-COVID-19 LVEF as a predictor of all-cause mortality, revealing no significant predictive value (AUC 0.53, $P = 0.43$).

survivors and decedents (1.8 ± 1.0 vs 1.8 ± 1.0, $P = 0.61$), and there was no survival-based difference in the proportion of patients with symptoms consistent with NYHA ≥ 2 (46% vs 48%, $P = 0.74$).

Factors Associated With Reduced LVEF

We then compared the baseline characteristics of patients with LVEF $>40\%$ (n = 337, 85%) versus those with LVEF $\leq 40\%$ (n = 59, 15%). Compared to those with preserved LVEF, patients with reduced LVEF were more likely male, more likely to have coronary artery disease, and more likely to use medicines typical of guideline-directed medical therapy for reduced LVEF (ie, angiotensin converting enzyme inhibitors or angiotensin receptor blockers, beta blockers, and aldosterone antagonists). They were also more likely to be treated with heparin (though only a small minority received this therapy) or azithromycin. There was no difference between LVEF groups in whether hospitalization was required (73% vs 75% for preserved- vs low-LVEF, respectively; $P = 0.87$). Patients with a preserved LVEF tended to have a lower pre-COVID NYHA class (1.7 ± 0.9 vs 2.5 ± 0.8, $P < 0.001$), and these patients were

less likely to have had any heart failure symptoms pre-COVID (41% vs. 83%, $P < 0.001$).

Time-Dependent Analysis of Mortality

We then assessed the unadjusted relationships between predictor variables and death over time, using Cox analyses (Table 2).

When analyzed as a continuous variable, prediagnosis LVEF demonstrated no ability to predict death, and as seen in Figure 3, there was no difference in survival between patients with LVEF ≤40% versus those with LVEF >40% ($P = 0.49$). To further evaluate the graphically worse early survival among patients with a low LVEF, we examined the LVEFs of those who died during the first 10 days after COVID diagnosis. There was no difference in LVEF between those who died before or after 10 days in the entire population (55% ± 13% vs 54±14%, $P = 0.48$) or within the subpopulation with LVEF ≤40% (27% ± 9% vs 30±9%, $P = 0.40$).

Similarly, there was no difference in survival between those without pre-COVID HF symptoms (ie, NYHA Class I) and those with NYHA ≥II symptoms ($P = 0.91$), as depicted in Figure 4.

Univariable predictors of death included older age, male sex, lower BMI, non-African-American race, absence of hypertension, lower DBP, and use of beta blockers, azithromycin, or hydroxychloroquine.

Multivariable Cox analysis was then performed to test whether other predictors had any effect on the ability of LVEF to predict death. In a model including LVEF, age, sex, BMI, and race, premorbid LVEF ≤40% remained not associated with subsequent death (HR 0.82 [95% CI 0.48-1.38], $P = 0.45$). In a fully adjusted model accounting for all univariable predictors of death with $P < 0.1$ (age, gender, BMI, race, CKD, hypertension, diastolic blood pressure, and medication use), the lack of association between LVEF and death persisted (HR 0.87 [0.50-1.52], $P = 0.63$).

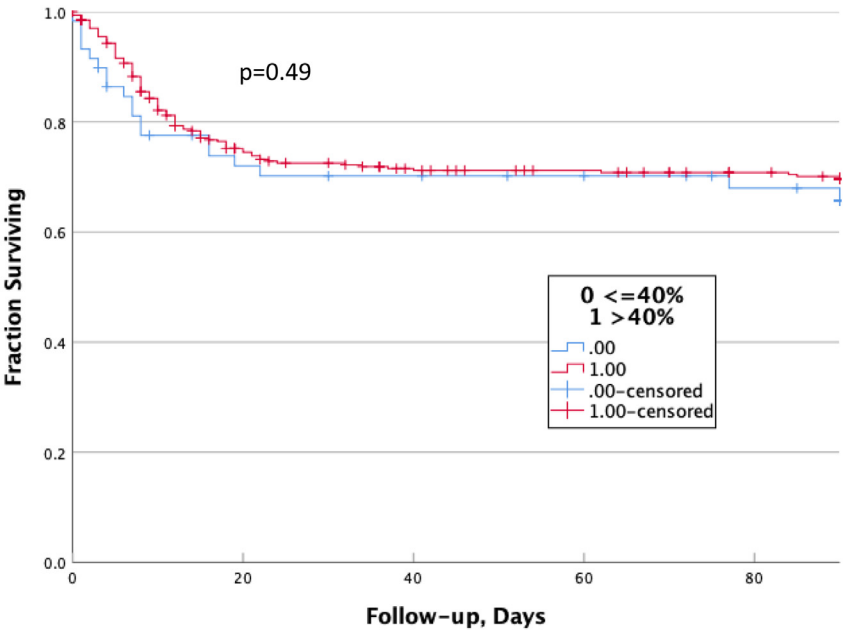
Subsequent Assessment of LVEF

During follow-up, 61 (15%) of patients had a repeat echocardiogram, which was performed a median of 90 (IQR 48-132) days after the diagnosis of COVID-19. There was no trend found in changes between pre- and post-COVID LVEF: median 0.0% (IQR −5.0% to +5.0%). Among the 11 patients whose LVEF showed an absolute decrease of ≥10%, 3 (27%) died ($P = 0.88$ for the comparison with the rest of the population).

TABLE 2. Relationships between variables and mortality

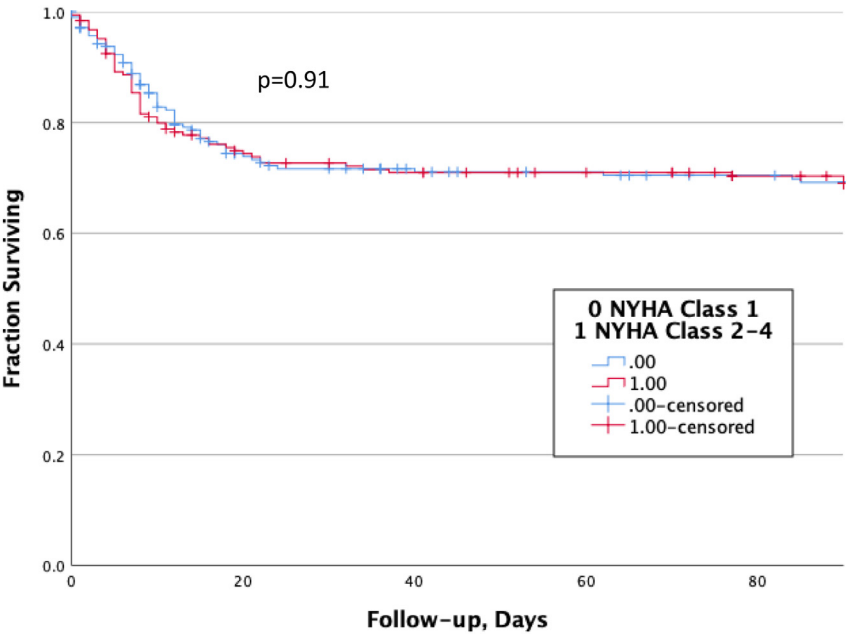
	Univariable HR (95% CI)	*P*	Multivariable Model 1 HR (95% CI)	*P*	Multivariable Model 2 HR (95% CI)	*P*
Age, per year	1.03 (1.02-1.05)	<0.001	1.03 (1.02-1.05)	<0.001	1.03 (1.01-1.05)	0.001
Male sex	2.35 (1.60-3.45)	<0.001	2.42 (1.60-3.66)	<0.001	2.30 (1.51-3.48)	<0.001
BMI, per kg/m^2	0.98 (0.96-1.00)	0.04	0.99 (0.97-1.02)	0.44	0.98 (0.96-1.01)	0.17
Black race	0.66 (0.46-0.95)	0.03	0.70 (0.47-1.04)	0.08	0.70 (0.46-1.06)	0.09
Current smoker	1.29 (0.85-1.95)	0.24				
LVEF, per % increase	1.00 (0.99-1.01)	0.99				
LVEF ≤40%	1.19 (0.73-1.95)	0.49	0.82 (0.48-1.38)	0.45	0.87 (0.50-1.52)	0.63
NYHA ≥2	1.02 (0.71-1.47)	0.91				
Chronic kidney disease	1.42 (0.97-2.06)	0.07			1.37 (0.88-2.14)	0.17
COPD	1.03 (0.61-1.74)	0.92				
Coronary artery disease	1.05 (0.64-1.74)	0.85				
Diabetes	0.79 (0.53-1.18)	0.25				
ESRD / HD	1.57 (0.88-2.80)	0.13				
Hypertension	0.69 (0.48-1.0)	0.048			0.58 (0.38-0.87)	0.01
SLE	0.93 (0.23-3.77)	0.92				
ACE/ARB	0.81 (0.57-1.17)	0.27				
Aldosterone blocker	1.16 (0.16-8.29)	0.88				
Azithromycin	2.46 (1.59-3.11)	<0.001			1.35 (0.83-2.20)	0.23
Beta blocker	2.06 (1.26-3.37)	<0.01			1.08 (0.64-1.84)	0.77
Heparin/LMWH	1.32 (0.42-4.17)	0.63				
Hydroxychloroquine	2.75 (1.89-3.98)	<0.001			1.81 (1.21-2.71)	<0.01
SBP, per mmHg	0.99 (0.99-1.00)	0.13				
DBP, per mmHg	0.99 (0.98-1.00)	0.02			0.99 (0.98-1.00)	0.18
Heart rate, per bpm	1.00 (0.99-1.01)	0.96				
Peak TnI, per ng/mL	1.00 (0.95-1.06)	0.98				

ACE, angiotensin converting enzyme inhibitor; ARB. angiotensin receptor blocker; BMI, body mass index; CI, confidence interval; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; HD, hemodialysis; ESRD, end stage renal disease; HR, hazard ratio; LMWH, low molecular weight heparin; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association class; SBP, systolic blood pressure; SLE, systemic lupus erythematosus; TnI, troponin I.



Number at Risk					
LVEF >40%	337	230	208	199	188
LVEF ≤40%	59	39	37	35	31

FIG 3. Survival stratified by groups of preinfection LVEF. Unadjusted Kaplan-Meier curves of estimated survival following diagnosis of COVID-19, stratified by LVEF ≤40% vs >40%, revealing no difference in survival ($P = 0.56$).



Number at Risk					
NYHA I	210	136	121	116	110
NYHA II-IV	186	133	124	118	109

FIG 4. Survival stratified by groups of pre-COVID NYHA class. Unadjusted Kaplan-Meier curves of estimated survival following diagnosis of COVID-19, stratified by pre-COVID NYHA Class I vs ≥II, revealing no difference in survival ($P = 0.91$).

Discussion

In this cohort study of patients who had an echocardiogram within the year prior to COVID-19 diagnosis, we found that premorbid LVEF had no significant impact on mortality. This lack of association between LVEF and death was shown on several modalities of assessment, and it persisted even after multivariable adjustment. Similarly, premorbid LVEF did not predict the need for hospitalization following COVID-19 infection. In addition, in contrast to a prior investigation in patients who required hospital admission, preinfection symptoms of dyspnea on exertion were not associated with death in our population of COVID-positive patients in any care setting.

A recent report of Ochsner Health's early experience with COVID-19 examined predictors of hospitalization and in-hospital mortality, focusing on patient race as a predictor, and found that African-Americans were disproportionately affected by the disease.[3] Our population corroborates the disproportionate effect of COVID-19 upon patients of African-American race, and, like the prior investigation, we found a trend toward superior survival among Black patients. Our findings of higher risk among older patients and males are consistent with prior evaluations of risk for in-hospital mortality.[6,10,11] However, while other studies have shown that higher BMI predicts death in COVID-19, following multivariable analysis we found no such relationship.[12] We evaluated mortality events both during the index hospitalization as well as in the outpatient setting, though as depicted in Figure 3 most deaths did occur early following infection. This is an important point, as during the early COVID-19 epidemic, hospital admissions for decompensated heart failure declined, while in-hospital HF-related mortality increased.[13]

As it may indicate a vulnerable myocardial status and reduced myocardial function, a pre-existing depressed LVEF could be expected to portend a poor outcome among patients with COVID-19. These patients may have less "reserve" to enable them to survive the multiple organ dysfunction that can result from COVID.[14] As with many acute illnesses, during COVID-19 infection there may be perturbations in endothelial function, electrolyte imbalances, increased inflammation, and hypercoagulability.[15] The extreme cytokine storm seen in severe COVID-19 illness can cause further decompensation of an already weakened myocardium. Direct cytotoxic damage may also play a role.[16] Several cases of COVID-related fulminant myocarditis have been reported.[17–19]

Earlier in 2020, Alvarez-Garcia et al. examined a large cohort of COVID-positive patients who required hospital admission, focusing on the preinfection

presence of diagnosed HF and its impact on outcomes.[9] In that retrospective study, compared to those without HF, patients with a prior diagnosis of HF had a longer length of stay, higher probability of mechanical ventilation, and higher in-hospital mortality. However, the diagnosis of heart failure, and its extraction from the EMR, can be subject to some error. As entry into the Alvarez-Garcia study was based on ICD-9/10 codes in the EMR, some patients may have been misclassified. Furthermore, the timing of the diagnosis of HF, including whether HF was first diagnosed during the index hospitalization, was not reported. The rigor with which the diagnosis of HF was made also was not reported: it is unclear whether these diagnoses resulted from evaluation by a cardiologist, and whether the diagnosis was supported by objective quantitative modalities (eg, Doppler echocardiography and/or right heart catheterization). The point of entry for the Alvarez-Garcia study was admission to the hospital, and the outcome was mortality during the index hospitalization. In contrast, we found that neither preinfection LVEF nor preinfection symptoms consistent with NYHA $\geq$2 correlated with mortality, and our population included both inpatients and outpatients, who were followed for months following COVID diagnosis, including during subsequent hospital admissions. This information is additive, especially given the emergence of cases in which patients who acutely recover from COVID still suffer lingering effects.

More recently, Matsushita et al. used a single-center registry of patients with a history of acute coronary syndrome to examine the correlation between baseline LVEF and the combined endpoint of COVID-related hospitalization or death.[20] Only a small minority (4%) of these patients underwent a COVID diagnostic test. Although a higher proportion of patients with low LVEF met the combined endpoint than among those with preserved LVEF, the study was limited: the incidence of COVID testing was much higher among low-LVEF patients, there were only 18 total events, and the endpoint was driven entirely by hospitalization, not death. Although Matsushita's results are intriguing, further investigation in populations with more complete testing data would be required to draw firm associations between reduced LVEF and the risk of COVID infection.

Clinical Implications and Recommendations for Further Study

Unless a very effective treatment becomes available or effective population vaccination is quickly accomplished, ongoing "waves" of COVID infection are expected in the coming months. Ahead of such waves, advance identification of high-risk markers could be important for clinicians working in resource-limited environments, so as to enable appropriate targeting of those resources toward patients who may benefit from

more intense care. We were interested in evaluating pre-infection LVEF as a predictor, and we found that LVEF did not predict adverse outcomes. Based on these data, known baseline LVEF should not be used as a risk stratifier for poor outcomes, or as a basis for resource allocation, following COVID infection. In addition, our patients' pre-COVID severity of symptoms did not impact outcomes, which argues for decision-making based on the immediate clinical presentation rather than on patients' historical symptom status.

In the case of other infections, prior evaluations of premorbid LVEF as a predictor of outcomes are quite limited, and the available literature shows mixed results regarding the utility of echocardiography during sepsis.[21-23] There is some evidence that echocardiography performed after the diagnosis of COVID may be valuable for prognostication, especially in the setting of high troponin I levels.[24-26] More study is needed to define the optimal use of repeated LVEF assessment, perhaps using ultraportable ultrasound units, following COVID infection.[27,28]

Limitations

There are several limitations to this study. In this retrospective analysis, not every patient diagnosed with COVID-19 had a prior echocardiogram available for evaluation, resulting in a modest sample size. However, including only patients with prior echocardiograms ensures that all members of the population had established health care prior to infection, reducing any related differences that may exist between patients. The negative impact of our modest sample size is mitigated by the population's unfortunately high rate of mortality, allowing analysis of a considerable number of outcomes. Ejection fraction alone does not characterize completely cardiac function. However, LVEF offers a single number that is accessible to a wide variety of clinicians and policymakers. In addition, especially during the early period of the COVID-19 epidemic, clinicians' exposure to infected patients was often minimized as possible. Thus, serial changes in LVEF following infection were not commonly available. Our investigation concentrated on known pre-COVID-19 LVEF as a predictor of poor outcomes, rather than on the development of new cardiac dysfunction. However, we did include a description of changes in LVEF in the minority of patients who had a repeat echocardiogram following COVID-19, and found no trend in LVEF changes. We cannot eliminate the possibility that some patients received subsequent care outside of the Ochsner system, leading to missed data, or that some patients died after leaving Ochsner Health care.

Lastly, with growing clinical experience with the care of COVID-19-infected patients, improvements in care, and perhaps changing demographics in the infected population, COVID-19-related mortality may improve over time. Results of examinations of the early COVID-19-positive population may be not generalizable to currently or future infected patients.

Conclusions

In patients with PCR-diagnosed COVID-19 infection, pre-COVID-19 LVEF does not predict death following COVID-19 infection.

Author Contributions

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