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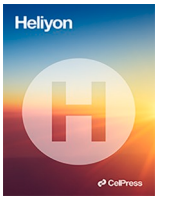

Research article

Comorbidities and mortality risk in COVID-19 patients with congestive heart failure: A comprehensive analysis

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ABSTRACT

The COVID-19 pandemic has posed unprecedented challenges to global healthcare systems, resulting in alarming incidence and mortality rates among patients with comorbidities, including heart failure. Understanding the characteristics of heart failure and other comorbidities during the COVID-19 pandemic is crucial for effective prevention and treatment. However, the current understanding of these characteristics among different racial groups remains incomplete. In this study, we investigated a cohort of 4711 patients, classifying them into congestive heart failure (CHF) and non-CHF groups. Biomarker analysis revealed noteworthy variations in blood urea nitrogen, aspartate aminotransferase, and white blood cell levels based on the presence or absence of CHF. Stratified by three racial groups, univariate logistic regression analysis identified significant differences in multiple variables, including CHF. Subsequent univariate Cox regression and Kaplan-Meier analysis demonstrated variations in mortality factors among distinct populations, with age and comorbidity playing prominent roles. This study utilized a large-scale database to investigate the characteristics of heart failure and related variables during the COVID-19 pandemic. The findings revealed distinctive mortality risk factors among various racial groups, emphasizing the significance of customized risk assessment and management approaches for diverse populations. These findings also provide a valuable resource for the development of targeted interventions and the promotion of equitable healthcare outcomes in the context of the COVID-19 pandemic.

1. Introduction

Global pandemics have significant impacts on economies and public health. The 2019 coronavirus pandemic is one of the most severe pandemics in recent times. Declared as a global pandemic by the World Health Organization on March 11, 2020, this epidemic presents an unprecedented global challenge with far-reaching consequences for the global economy and society. As of 30 October 2023, the SARS-COV-2 pandemic has caused more than 771,549,718 cases and 6,974,473 deaths worldwide [1]. Patients diagnosed with COVID-19 often experience complications, including the occurrence or worsening of heart failure. The presence of comorbidities

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increases the risk of disease progression and the development of more symptoms. Consequently, once infected, these patients often experience a severe and unfavorable clinical course [2–4]. This is because these patients already have cardiovascular disease and their immune system and health status are compromised, making them more susceptible to COVID-19. Some research investigate the prognostic value of plasma troponin levels and associated variables in predicting myocardial injury [5,6], as well as the increased risk of developing complications, including heart failure [7]. The research indicates that a history of atrial fibrillation (AF) is associated with an adverse clinical course, including a higher mortality rate [8], patients with heart failure are at an elevated risk. A recent study has examined the impact of COVID-19 on heart failure and revealed that individuals with heart failure in conjunction with COVID-19 have a significantly higher mortality rate compared to those without COVID-19 (21.8 % and 3.8 %, respectively) [9]. Hospitalized patients with concomitant heart failure and COVID-19 are at an elevated risk for in-hospital all-cause mortality. Furthermore, this risk remains significantly high even after discharge, up to six months [10].

Heart failure is a clinical syndrome that typically presents as insufficient cardiac output. It is an end-stage manifestation of a variety of cardiac diseases and places a significant burden on the healthcare system [11]. Heart failure is a prevalent cause of mortality on a global scale, resulting in a significant strain on healthcare resources. In the United States, approximately 6.2 million adults suffer from heart failure, and the economic cost of the disease to the nation was approximately $30.7 billion in 2012. In addition, heart failure varies geographically and contributes to many deaths [12]. Globally, the burden of heart failure has increased to approximately 23 million people and this number is still growing [13]. There is reason to believe that the prevalence of heart failure will continue to rise with an ageing population and unhealthy lifestyle changes. This poses a great challenge to the healthcare system and to society and the economy. The COVID-19 pandemic has worsened the prevalence of heart failure, underscoring the need for a comprehensive analysis study.

In the COVID-19, there are marked differences in morbidity and mortality rates by race. Part of this is due to long-standing economic and educational disadvantages that have led to decreased health care utilization by minorities and a general distrust of the health care system [14,15]. These factors contribute to the existence of racial health inequalities and are further magnified in the new coronavirus epidemic. The COVID-19 pandemic of 2019 has had a disproportionate impact on racial and ethnic minorities in the United States, who are more vulnerable to infection, hospitalization, and mortality [16].

Individuals afflicted with heart failure represent a notably susceptible cohort. Moreover, those with documented histories of both COVID-19 and heart failure face considerably worse prognostic outcomes. Furthermore, the healthcare resource utilization of these patients during hospitalization demonstrates a marked and substantial escalation [17]. A thorough investigation is essential to uncover the distinctive characteristics of COVID-19 patients with heart failure across diverse ethnic groups. This comprehensive inquiry has the potential to reveal varying factors among these populations, thus facilitating the development of tailored strategies for prevention and treatment.

The current research has not adequately addressed the comprehensive investigation of the impact of COVID-19 on heart failure within large-scale datasets. Moreover, there is an urgent need to deepen our understanding of how ethnicity influences the prognosis of COVID-19 patients with comorbidities and multiple biomarkers. To address these gaps, we adopted a classification methodology, categorizing ethnicity into Black patients, White patients, and other patients. Our study encompasses a wide range of analyses and discussions, significantly advancing our comprehension of the intricate interplay between COVID-19 and heart failure. Our analysis holds paramount significance as it not only unravels the complexities associated with ethnicity and heart failure but also delineates precision-based interventions for both prevention and treatment. By shedding light on these intricate dynamics, our findings provide critical insights for developing tailored strategies that can improve outcomes in COVID-19 patients with heart failure and comorbidities, while considering the influence of ethnicity.

2. Materials and methods

The data originates from the research conducted by Altschul et al. [18], who have made their data accessible at this link: (https://figshare.com/s/79827c396af7df42b3d7). The study included adult patients (above 18 years old) who tested positive for COVID-19 using RT-PCR and were admitted to four hospitals within the Montefiore Health System in New York City between March 1, 2020, and April 16, 2020. The data were collected on May 7, 2020. The outcome measure is in-hospital mortality, with 1 indicating occurrence and 0 indicating non-occurrence. In order to ensure complete data collection, individuals who were not admitted to the hospital or who died before admission were excluded. The variables were collected at admission, and mortality-related data were obtained through hospital death records or national death registration authorities. The data for this study were collected from a total of 4711 publicly available COVID-19 patients who were admitted to these hospitals. The dataset contains information on mortality, admission laboratory values, demographics, and comorbidities. It has been reviewed in terms of ethics and publicly available in anonymised form [18]. The database used in this study can be utilized for conducting secondary analyses based on various scientific hypotheses. The original research was granted exempt status by the Ethics Committee for Clinical Research of the Albert Einstein College of Medicine, Montefiore Medical Center [19], thereby waiving the requirement for obtaining informed consent. The main aim of this study is to identify and classify patients who are at an elevated risk of developing severe illness or facing mortality. This information will be valuable in aiding healthcare professionals to make well-informed clinical decisions.

To enhance comparability across studies, we employed a consistent framework, yielding deeper insights into the impact of the epidemic on ethnicity. Our classification of race into Black patients, White patients, and Other patients closely aligns with prior research. Typically, race is classified as White, Black, and “Other”, based on predefined definitions in the CRWD database [20]. These racial categories offer a straightforward means of investigating the connection between COVID-19 and heart failure within different racial groups. Our study comprised a cohort of 4711 patients. Among them, 715 individuals were aged over 80 years, 1019 fell within

the age group of 70–80 years, 1123 were in the 60–70 years age group, and 1854 were within the 0–60 years age range. Specifically, there were 1743 individuals identified as Black patients, 466 as White patients, and 2502 as Other patients under this classification. This classification approach provides a novel perspective for examining the risk of the pandemic among diverse racial demographics.

This study involved four experiments. Initially, a sample of 4711 individuals was classified into two groups: those with heart failure and those without. Chi-square tests were then used to examine the differences between these two groups of data. Subsequently, a univariate logistic regression analysis was performed to assess variations in racial classifications across three distinct ethnic groups. Next, a univariate Cox regression analysis was employed in conjunction with the length of stay to further explore these differences. Univariate logistic regression analysis was conducted to determine the risk factors for in-hospital mortality odds ratio (OR), while univariate Cox regression was used to evaluate the hazard ratio (HR) during the follow-up period. A 95 % confidence interval (CI) was applied, with statistical significance set at $P < 0.05$. Then, Kaplan-Meier (KM) survival curves, with time units measured in days, were generated for the categorized heart failure population, and KM curves were also plotted for the significant factors identified during the analysis. In summary, a four-step investigation was executed to evaluate the impact of heart failure and ethnicity on survival and survival time among COVID-19 patients. This study was carried out according to STROBE guidelines. The findings, analyzed using Python and R programming languages, have implications for the development of personalized prevention and treatment strategies, while also serving as a reference for healthcare practitioners.

3. Results

With our primary research objective being the exploration of the relationship between diverse patient populations and heart failure, and motivated by these initial observations, we conducted a comprehensive series of subsequent analyses. We employed population segmentation as a valuable tool to further investigate these findings.

Table 1
Stratified outcomes of the study population based on heart failure history.

Variables	Total (N = 4711)	Non-CHF (n = 4170; 88.5 %)	CHF (n = 541; 11.5 %)	P Value
Age, year				0.287
0–60	1854 (39.4 %)	1660 (39.8 %)	194 (35.9 %)	–
>60	1123 (23.8 %)	993 (23.8 %)	130 (24.0 %)	–
>70	1019 (21.6 %)	893 (21.4 %)	126 (23.3 %)	–
>80	715 (15.2 %)	624 (15.0 %)	91 (16.8 %)	–
Comorbidities				
MI	201 (4.27 %)	74 (1.77 %)	127 (23.5 %)	< **0.001**
PVD	848 (18.0 %)	631 (15.1 %)	217 (40.1 %)	< **0.001**
CVD	506 (10.7 %)	329 (7.89 %)	177 (32.7 %)	< **0.001**
DEMENT	372 (7.90 %)	250 (6.00 %)	122 (22.6 %)	< **0.001**
COPD	265 (5.63 %)	173 (4.15 %)	92 (17.0 %)	< **0.001**
DM Complicated	495 (10.5 %)	328 (7.87 %)	167 (30.9 %)	< **0.001**
DM Simple	686 (14.6 %)	422 (10.1 %)	264 (48.8 %)	< **0.001**
Renal Disease	833 (17.7 %)	480 (11.5 %)	353 (65.2 %)	< **0.001**
All CNS	607 (12.9 %)	523 (12.5 %)	84 (15.5 %)	0.06
Pure CNS	488 (10.4 %)	418 (10.0 %)	70 (12.9 %)	**0.044**
Stroke	58 (1.23 %)	53 (1.27 %)	5 (0.92 %)	0.631
Seizure	38 (0.81 %)	34 (0.82 %)	4 (0.74 %)	1
OldSyncope	88 (1.87 %)	78 (1.87 %)	10 (1.85 %)	1
OldOtherNeuro	145 (3.08 %)	123 (2.95 %)	22 (4.07 %)	0.2
OtherBrnLsn	27 (0.57 %)	21 (0.50 %)	6 (1.11 %)	0.118
Biomarkers				
O2 Sat <94, %	1862 (39.5 %)	1629 (39.1 %)	233 (43.1 %)	0.081
Temp >38, °C	854 (18.1 %)	746 (17.9 %)	108 (20.0 %)	0.263
MAP <70, mmHg	339 (7.20 %)	294 (7.05 %)	45 (8.32 %)	0.325
D-Dimer >3, mg/ml	1151 (24.4 %)	1022 (24.5 %)	129 (23.8 %)	0.776
INR >1.2	1151 (24.4 %)	1022 (24.5 %)	129 (23.8 %)	0.776
BUN >30, mg/dL	1285 (27.3 %)	1113 (26.7 %)	172 (31.8 %)	**0.014**
Sodium <139 or >154, mmol/L	592 (12.6 %)	536 (12.9 %)	56 (10.4 %)	0.113
Glucose <60 or >500, mmol/L	114 (2.42 %)	108 (2.59 %)	6 (1.11 %)	0.05
AST >40, U/L	2121 (45.0 %)	1854 (44.5 %)	267 (49.4 %)	**0.035**
ALT >40, U/L	1292 (27.4 %)	1130 (27.1 %)	162 (29.9 %)	0.179
WBC <1.8 or >4.8, per mm^3	3891 (82.6 %)	3469 (83.2 %)	422 (78.0 %)	**0.003**
Lymphocytes <1, per mm^3	2124 (45.1 %)	1859 (44.6 %)	265 (49.0 %)	0.059
IL6 > 150, pg/ml	291 (6.18 %)	258 (6.19 %)	33 (6.10 %)	1
Ferritin >300, μg/L	2561 (54.4 %)	2248 (53.9 %)	313 (57.9 %)	0.091
C-Reactive. Prot >10, mg/L	1853 (39.3 %)	1629 (39.1 %)	224 (41.4 %)	0.317
Procalcitonin >0.1, ng/ml	1724 (36.6 %)	1510 (36.2 %)	214 (39.6 %)	0.141
Troponin >0.1, ng/ml	450 (9.55 %)	386 (9.26 %)	64 (11.8 %)	0.066

3.1. Contrasting outcomes between CHF patients and non-CHF individuals: a comparative analysis

Table 1 gives a summary of the clinical characteristics of the study population, divided into two groups: those with Congestive Heart Failure (CHF) and those without. The total sample size is 4711 individuals, with 4170 individuals without CHF and 541 with CHF. Chi-square tests were conducted to investigate the relationship between CHF and different variables, as well as comorbidities. The study uncovered significant differences ($p < 0.05$) between the two groups in terms of Myocardial Infarction (MI), Peripheral Vascular Disease (PVD), Cardiovascular Disease (CVD), Dementia (DEMENT), Chronic Obstructive Pulmonary Disease (COPD), Diabetes Mellitus (DM) Complicated, DM Simple, Renal Disease, and Pure Central Nervous System (Pure CNS) conditions. Additionally, biomarker analysis revealed significant differences ($p < 0.05$) in Blood Urea Nitrogen (BUN) levels >30, Aspartate Aminotransferase (AST) levels >40, and White Blood Cell (WBC) levels <1.8 or >4.8 between the two groups based on the presence or absence of CHF. The study revealed significant differences between CHF and various clinical attributes and biomarkers.

3.2. Univariate logistic regression analysis investigating the role of race in predicting mortality outcomes in COVID-19 patients

This section employed univariate logistic regression analysis to examine the independent risk factors for mortality among diverse ethnic groups as presented in Table 2. The results revealed unique mortality risk factors among varying ethnicities, with age and comorbidities playing a significant role in each group. For Black patients, the mortality risk factors included age, PVD, COPD, All CNS, and Pure CNS. For White patients, the mortality risk factors were age and Renal Disease. Among other patients, the mortality risk factors were age, PVD, CHF, Renal Disease, All CNS, Pure CNS, and Stroke.

In addition, biomarker analysis using univariate logistic regression identified various biomarkers associated with mortality risk in different ethnic groups. The biomarkers associated with mortality risk in black patients were Oxygen saturation (O2 Sat) (<94), Mean arterial pressure (MAP) (<70), D-Dimer (>3), International normalized ratio (INR) (>1.2), BUN (>30), Sodium (<139 or >154), AST

Table 2
Race-based univariate logistic regression analysis findings.

Variable	Black patients		White patients		Other patients	
	OR (95 % CI)	p-value	OR (95 % CI)	p-value	OR (95 % CI)	p-value
Age(<60 -reference)						
>60	2.68(1.93,3.75)	< **0.001**	3.38(1.61,7.70)	**0.001**	2.51(1.92,3.28)	< **0.001**
>70	4.23(3.07,5.86)	< **0.001**	5.37(2.63,12.0)	< **0.001**	4.32(3.33,5.63)	< **0.001**
>80	5.27(3.72,7.52)	< **0.001**	9.00(4.38,20.2)	< **0.001**	6.79(5.13,9.01)	< **0.001**
Comorbidities						
MI	0.82(0.46,1.37)	0.456	0.73(0.23,1.92)	0.547	1.57(1.00,2.42)	0.05
PVD	0.58(0.41,0.81)	**0.001**	0.52(0.31,0.87)	**0.011**	0.53(0.40,0.68)	< **0.001**
CHF	**1.05(0.75,1.46)**	**0.766**	**1.06(0.58,1.87)**	**0.85**	**1.40(1.06,1.85)**	**0.02**
CVD	1.05(0.73,1.48)	0.807	1.15(0.64,2.02)	0.631	1.16(0.86,1.55)	0.336
DEMENT	1.31(0.89,1.89)	0.17	0.96(0.46,1.90)	0.915	1.20(0.84,1.68)	0.309
COPD	1.58(1.04,2.38)	**0.034**	0.78(0.25,2.06)	0.636	1.35(0.91,1.97)	0.134
DM Complicated	0.72(0.48,1.05)	0.089	1.36(0.73,2.49)	0.328	1.01(0.74,1.36)	0.933
DM Simple	0.85(0.61,1.16)	0.318	1.22(0.69,2.11)	0.478	1.09(0.84,1.40)	0.521
Renal Disease	1.12(0.85,1.46)	0.426	2.04(1.24,3.33)	**0.005**	1.28(1.01,1.63)	**0.045**
All CNS	1.72(1.28,2.30)	< **0.001**	1.65(0.93,2.90)	0.087	1.95(1.50,2.52)	< **0.001**
Pure CNS	1.77(1.29,2.41)	< **0.001**	1.50(0.80,2.73)	0.197	1.90(1.42,2.54)	< **0.001**
Stroke	2.42(0.92,6.09)	0.073	6.93(0.80,200)	0.08	3.01(1.52,5.93)	**0.002**
Seizure	1.52(0.47,4.26)	0.462	–	–	1.00(0.32,2.59)	0.998
OldSyncope	0.89(0.35,1.98)	0.793	–	–	0.96(0.46,1.85)	0.918
OldOtherNeuro	1.41(0.77,2.49)	0.253	2.23(0.75,6.46)	0.142	1.19(0.69,1.98)	0.516
OtherBrnLsn	0.37(0.01,1.95)	0.284	1.32(0.04,16.4)	0.839	0.97(0.21,3.25)	0.966
Biomarkers						
O2 Sat <94, %	1.66(1.33,2.08)	< **0.001**	2.53(1.68,3.83)	< **0.001**	2.16(1.79,2.60)	< **0.001**
Temp >38, °C	0.99(0.74,1.32)	0.968	0.91(0.51,1.57)	0.732	1.59(1.28,1.98)	< **0.001**
MAP <70, mmHg	21.1(12.8,36.7)	< **0.001**	31.1(12.1108)	< **0.001**	22.6(15.2,34.6)	< **0.001**
D-Dimer >3, mg/ml	2.56(2.02,3.24)	< **0.001**	1.95(1.25,3.03)	**0.003**	2.11(1.72,2.59)	< **0.001**
INR >1.2	2.56(2.02,3.24)	< **0.001**	1.95(1.25,3.03)	**0.003**	2.11(1.72,2.59)	< **0.001**
BUN >30, mg/dL	2.96(2.35,3.73)	< **0.001**	2.64(1.74,4.02)	< **0.001**	2.55(2.09,3.11)	< **0.001**
Sodium <139 or >154, mmol/L	1.54(1.11,2.12)	**0.011**	1.29(0.72,2.25)	0.391	1.68(1.31,2.15)	< **0.001**
Glucose <60 or >500, mmol/L	1.29(0.66,2.37)	0.436	0.46(0.02,2.81)	0.446	1.23(0.67,2.18)	0.489
AST >40, U/L	2.03(1.62,2.54)	< **0.001**	2.42(1.61,3.67)	< **0.001**	1.88(1.56,2.26)	< **0.001**
ALT >40, U/L	1.31(1.03,1.68)	**0.03**	0.94(0.58,1.51)	0.806	0.96(0.78,1.17)	0.68
WBC <1.8 or >4.8, per mm^3	1.37(1.02,1.86)	**0.034**	1.66(0.95,3.05)	0.075	1.59(1.22,2.11)	**0.001**
Lymphocytes <1, per mm^3	1.60(1.28,1.99)	< **0.001**	1.35(0.90,2.03)	0.146	1.74(1.45,2.10)	< **0.001**
IL6 > 150, pg/ml	5.01(3.30,7.67)	< **0.001**	3.06(1.47,6.43)	**0.003**	2.86(2.06,3.95)	< **0.001**
Ferritin >300, μg/L	1.19(0.95,1.49)	0.126	1.02(0.68,1.53)	0.914	1.68(1.39,2.03)	< **0.001**
C-Reactive. Prot >10, mg/L	2.59(2.07,3.25)	< **0.001**	2.39(1.58,3.61)	< **0.001**	2.48(2.06,2.99)	< **0.001**
Procalcitonin >0.1, ng/ml	2.63(2.10,3.31)	< **0.001**	3.07(2.02,4.67)	< **0.001**	2.67(2.22,3.23)	< **0.001**
Troponin >0.1, ng/ml	2.44(1.77,3.34)	< **0.001**	2.83(1.56,5.11)	**0.001**	3.28(2.45,4.37)	< **0.001**

(>40), Alanine aminotransferase (ALT) (>40), WBC (<1.8 or >4.8), Lymphocytes (<1), Interleukin-6 (IL6) (>150), C-reactive protein (C-Reactive. Prot) (>10), Procalcitonin (>0.1), and Troponin (>0.1). For white patients, the biomarkers associated with mortality risk were O2 Sat (<94), MAP (<70), D-Dimer (>3), INR (>1.2), BUN (>30), AST (>40), IL6 (>150), C-Reactive. Prot (>10), Procalcitonin (>0.1), and Troponin (>0.1) (OR>1, $p < 0.05$). Among other patients, the biomarkers affecting mortality were O2 Sat (<94), Temperature (Temp) (>38), MAP (<70), D-Dimer (>3), INR (>1.2), BUN (>30), Sodium (<139 or >154), AST (>40), WBC (<1.8 or >4.8), Lymphocytes (<1), IL6 (>150), Ferritin (>300), C-Reactive. Prot (>10), Procalcitonin (>0.1), and Troponin (>0.1).

It is important to highlight that within the context of CHF, the p-value for Black patients was 0.766, for White patients it was 0.85, and for other patients, it was 0.02. The obtained values highlight a significant impact that varies among different racial groups. Consequently, we conducted further investigations to gain deeper insights into these observations. The identified findings emphasize the existence of distinct mortality risk factors among diverse ethnicities.

3.3. Univariate Cox regression analysis sheds light on predictive factors for mortality in COVID-19 patients

This section utilized Univariate Cox regression analysis to identify potential predictors in the study. Analysis of the three racial groups (as shown in Table 3) revealed distinct risk factors influencing mortality rates. For Black patients, age, DM Complicated (HR 0.65) and seizure (HR 2.48) were significant risk factors. In White patients, age, renal disease (HR 1.51), and stroke (HR 3.64) were associated with higher mortality rates. Among other patients, age and All CNS diseases (HR 1.25) were identified as risk factors for mortality.

Furthermore, biomarker analysis using univariate Cox regression showed various biomarkers associated with mortality risk in different racial groups. For Black patients, O2 Sat, MAP, D-Dimer, INR, BUN, AST, WBC, Lymphocytes, IL6, Ferritin, C-Reactive. Prot, Procalcitonin, and Troponin were significant biomarkers. In White patients, O2 Sat, MAP, BUN, AST, Ferritin, C-Reactive. Prot, Procalcitonin, and Troponin were identified as significant biomarkers. Among other patients, O2 Sat, MAP, D-Dimer, INR, BUN,

Table 3
Examining the impact of race through univariate Cox regression.

Variable	Black patients		White patients		Other patients	
	HR (95 % CI)	p-value	HR (95 % CI)	p-value	OR (95 % CI)	p-value
Age(<60 -reference)						
>60	2.00(1.48,2.71)	< **0.001**	3.07(1.50,6.30)	**0.002**	1.81(1.42,2.31)	< **0.001**
>70	2.94(2.20,3.93)	< **0.001**	5.20(2.61,10.4)	< **0.001**	3.21(2.55,4.04)	< **0.001**
>80	3.87(2.85,5.25)	< **0.001**	5.71(2.89,11.3)	< **0.001**	4.57(3.61,5.78)	< **0.001**
Comorbidities						
MI	0.83(0.51,1.34)	0.441	0.77(0.32,1.89)	0.574	1.16(0.81,1.67)	0.420
PVD	0.95(0.69,1.30)	0.749	0.86(0.54,1.36)	0.513	0.91(0.71,1.16)	0.448
CHF	**1.04(0.78,1.40)**	**0.768**	**0.95(0.58,1.54)**	**0.831**	**1.16(0.92,1.47)**	**0.214**
CVD	1.16(0.85,1.58)	0.347	1.03(0.64,1.67)	0.891	1.09(0.84,1.40)	0.527
DEMENT	1.23(0.89,1.70)	0.201	0.94(0.52,1.70)	0.835	1.15(0.86,1.54)	0.358
COPD	1.38(0.98,1.94)	0.066	1.09(0.45,2.68)	0.847	1.05(0.76,1.45)	0.779
DM Complicated	0.65(0.46,0.93)	**0.017**	1.38(0.84,2.28)	0.203	1.02(0.78,1.33)	0.883
DM Simple	0.81(0.61,1.08)	0.144	1.32(0.83,2.09)	0.239	1.08(0.86,1.35)	0.519
Renal Disease	1.02(0.81,1.29)	0.868	1.51(1.02,2.22)	**0.038**	1.20(0.97,1.47)	0.090
All CNS	1.06(0.83,1.35)	0.640	1.20(0.76,1.89)	0.430	1.25(1.01,1.54)	**0.040**
Pure CNS	1.06(0.82,1.38)	0.644	1.10(0.67,1.79)	0.712	1.25(0.99,1.58)	0.062
Stroke	1.41(0.70,2.83)	0.341	3.64(1.15,11.5)	**0.028**	1.46(0.90,2.36)	0.127
Seizure	2.48(1.03,6.02)	**0.044**	–	0.994	0.90(0.37,2.18)	0.821
OldSyncope	0.95(0.45,2.01)	0.897	–	0.995	1.42(0.78,2.59)	0.248
OldOtherNeuro	0.73(0.44,1.19)	0.207	1.50(0.70,3.21)	0.300	0.79(0.50,1.23)	0.291
OtherBrnLsn	0.21(0.03,1.50)	0.121	1.94(0.27,14.0)	0.509	0.70(0.22,2.17)	0.533
Biomarkers						
O2 Sat <94, %	1.32(1.08,1.60)	**0.005**	1.67(1.18,2.36)	**0.004**	1.38(1.17,1.62)	< **0.001**
Temp >38, °C	0.85(0.66,1.10)	0.225	0.65(0.40,1.05)	0.077	1.18(0.98,1.42)	0.075
MAP <70, mmHg	3.64(2.88,4.59)	< **0.001**	3.31(2.27,4.84)	< **0.001**	3.54(2.95,4.25)	< **0.001**
D-Dimer >3, mg/ml	1.59(1.30,1.93)	< **0.001**	1.18(0.82,1.69)	0.367	1.29(1.09,1.52)	**0.004**
INR >1.2	1.59(1.30,1.93)	< **0.001**	1.18(0.82,1.69)	0.367	1.29(1.09,1.52)	**0.004**
BUN >30, mg/dL	2.34(1.92,2.84)	< **0.001**	1.97(1.41,2.77)	< **0.001**	2.13(1.80,2.51)	< **0.001**
Sodium <139 or >154, mmol/L	1.30(0.99,1.71)	0.056	1.14(0.71,1.82)	0.586	1.39(1.13,1.71)	**0.002**
Glucose <60 or >500, mmol/L	1.23(0.72,2.10)	0.440	0.42(0.06,3.01)	0.388	1.39(0.85,2.29)	0.190
AST >40, U/L	1.40(1.15,1.70)	**0.001**	1.70(1.21,2.39)	**0.002**	1.47(1.25,1.73)	< **0.001**
ALT >40, U/L	1.18(0.96,1.46)	0.117	0.95(0.63,1.42)	0.797	0.93(0.78,1.11)	0.424
WBC <1.8 or >4.8, per mm^3	1.37(1.05,1.80)	**0.020**	1.32(0.79,2.20)	0.282	1.35(1.05,1.73)	**0.018**
Lymphocytes <1, per mm^3	1.24(1.02,1.51)	**0.029**	1.00(0.71,1.42)	0.994	1.24(1.06,1.46)	**0.008**
IL6 > 150, pg/ml	1.99(1.49,2.64)	< **0.001**	1.30(0.78,2.17)	0.320	1.40(1.10,1.79)	**0.007**
Ferritin >300, μg/L	0.74(0.60,0.90)	**0.002**	0.60(0.42,0.86)	**0.005**	1.07(0.91,1.27)	0.395
C-Reactive. Prot >10, mg/L	1.43(1.18,1.74)	< **0.001**	1.45(1.03,2.04)	**0.033**	1.43(1.22,1.69)	< **0.001**
Procalcitonin >0.1, ng/ml	1.34(1.10,1.64)	**0.003**	1.51(1.07,2.12)	**0.020**	1.37(1.17,1.62)	< **0.001**
Troponin >0.1, ng/ml	1.55(1.21,2.00)	**0.001**	1.83(1.18,2.84)	**0.007**	2.12(1.71,2.62)	< **0.001**

Sodium, AST, WBC, Lymphocytes, IL6, C-Reactive. Prot, Procalcitonin, and Troponin were significant biomarkers.

In conclusion, this study emphasizes the importance of considering diverse risk factors and biomarkers in relation to mortality among different racial groups. Age consistently emerged as a significant risk factor across all groups, highlighting its universal impact on mortality rates. Additionally, specific comorbidities and biomarkers demonstrated varying levels of influence on mortality, emphasizing the crucial requirement for personalized risk assessment and management strategies tailored to different populations.

3.4. Examining survival rates in heart failure and related incidents: a KM analysis of in-hospital mortality

To assess the impact of heart failure and related events on in-hospital mortality, Kaplan-Meier (KM) survival curves were constructed to track the time to in-hospital death. The graphs depict the KM survival curves for the presence or absence of heart failure among Black patients, White patients, and other patients, arranged from left to right in Fig. 1. The p-value obtained was 0.768 for Black patients (Figs. 1A), 0.831 for White patients (Figs. 1B), and 0.214 for other patients (Fig. 1C). The results reveal discrepancies in survival rates among the three racial groups in both datasets, manifesting as distinct curves based on the presence or absence of heart failure.

Additionally, the graph showing the differential survival rates in the Black population (Fig. 2) highlights the impact of two co-morbidities, DM Complicated and seizure, on survival. As depicted in the graph on the left (Fig. 2A), patients with DM Complicated had a higher survival rate of 0.017 ($p < 0.05$) compared to patients without DM Complicated. In contrast, patients with seizure had a lower survival rate of 0.044 ($p < 0.05$) (Fig. 2B). Slightly diverging from this, the graph illustrating the two co-morbidities affecting survival in White patients (Fig. 3) reveals that patients with stroke 0.028 ($p < 0.05$) (Fig. 3A) and renal disease 0.038 ($p < 0.05$) (Fig. 3B) experienced lower survival rates. Finally, the graph showing the co-morbidities affecting other patients (Fig. 4) indicates that patients with All CNS had a lower survival rate of 0.040 ($p < 0.05$).

In summary, the findings of this study provide critical insights into racial disparities in survival rates in patients with heart failure and related incidents, highlighting the impact of racial background, diabetes, renal disease, stroke, and other comorbidities. The results underscore the significance of personalized interventions in reducing racial disparities in survival rates and offer preliminary evidence for the effectiveness of such interventions.

4. Discussion

The COVID-19 has created a global crisis, highlighting the imperative for comprehensive research to comprehend the health conditions that can impact mortality rates. While some studies suggest that ethnicity and specific health conditions may increase the risk of succumbing to the novel coronavirus, additional research is needed to elucidate the underlying mechanisms. Hence, further studies are needed to assess the impact of race and health status on coronavirus infection and mortality, aiming to develop more effective prevention and treatment strategies.

This study focus on hospitalized patients and investigates the interplay between heart failure, the novel coronavirus, and various racial backgrounds. To the best of our knowledge, it is the first to investigate the influence of comorbidities exacerbated by COVID-19 and underlying health conditions across black, white, and other racial groups. The findings reveal disparities in mortality rates attributed to heart failure among different racial groups, as well as different outcomes for other underlying conditions. These variations may be attributed to disparities in racial healthcare, community structures, and other factors. Conducting a thorough analysis using patient data is essential to achieve a comprehensive understanding of this phenomenon.

We believe that factors influencing mortality rates differ across different populations. Statistical results obtained after population

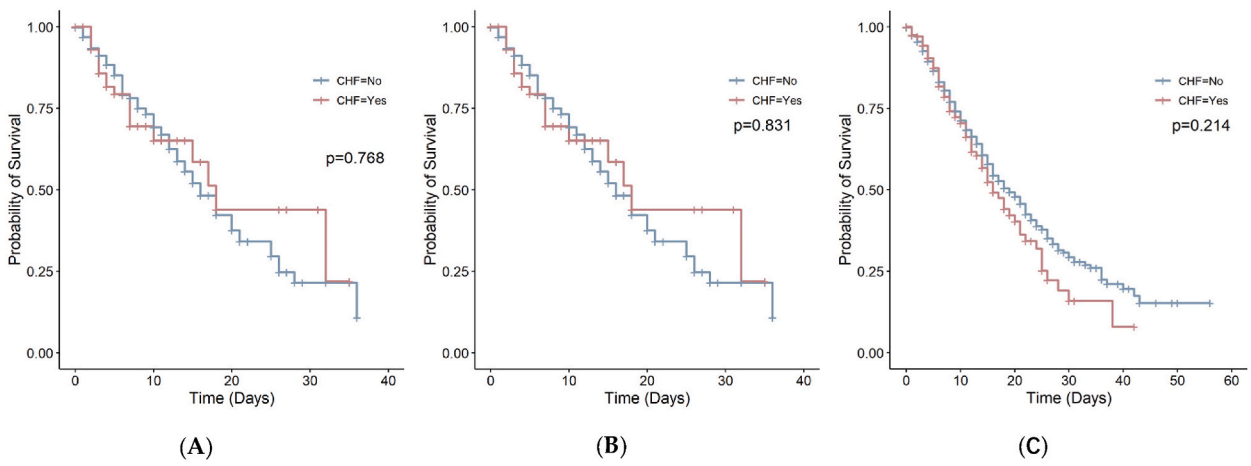

Fig. 1. KM survival curves of in-hospital mortality among different racial groups with and without CHF. (A) Survival analysis of Black patients, (B) Survival analysis of White patients, (C) Survival analysis of other patients. Abbreviations: KM, kaplan-meier; CHF, congestive heart failure.

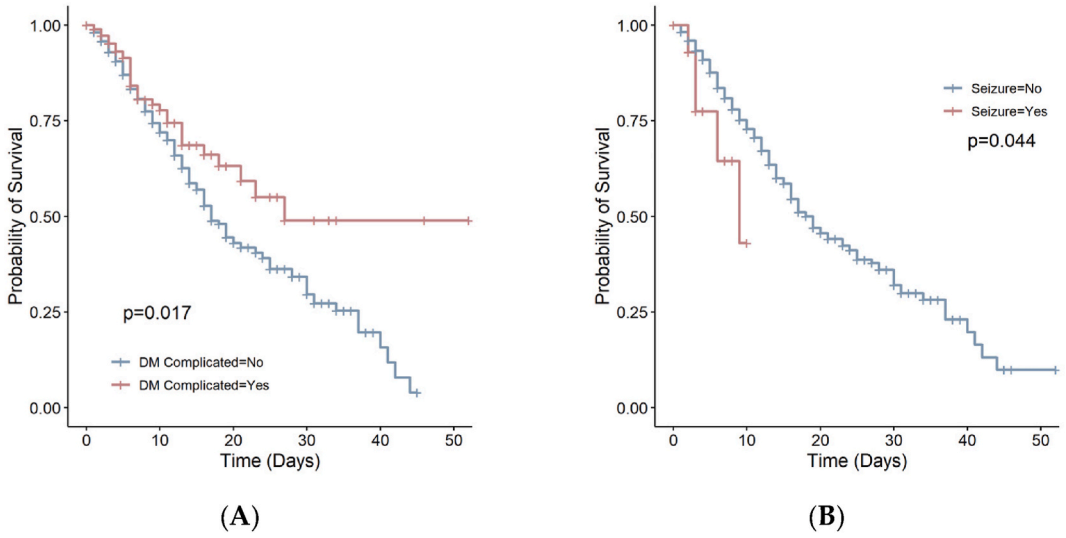

Fig. 2. Survival rate disparities in Black Population with differential diseases: Impact of DM Complicated and seizure. (A) Survival analysis of patients with DM Complicated, (B) Survival analysis of patients with seizure. Abbreviations: DM Complicated, diabetes mellitus complicated.

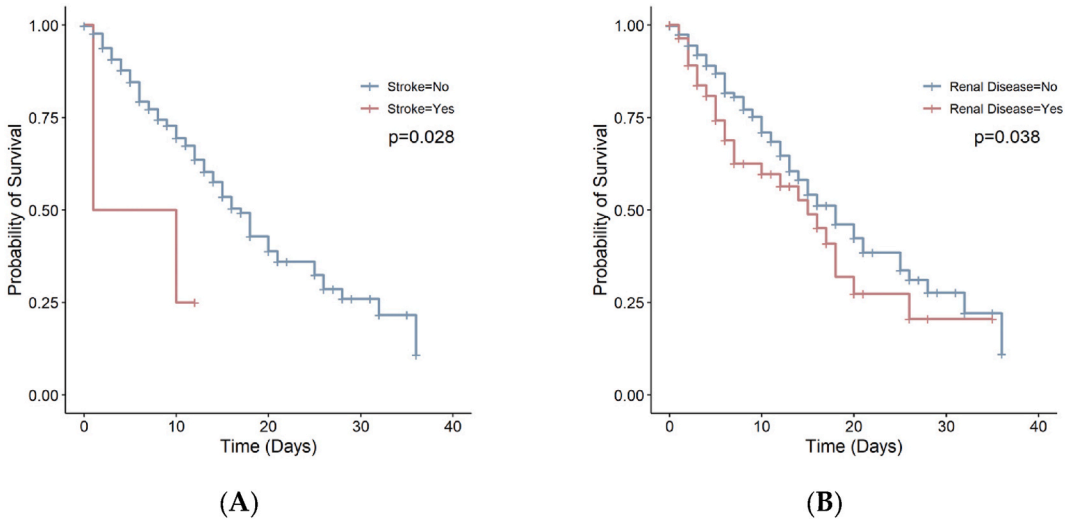

Fig. 3. Survival rate disparities in White population with differential diseases: Impact of stroke and renal disease. (A) Survival analysis of patients with stroke, (B) Survival analysis of patients with renal disease.

segmentation will be more meaningful, showing greater differences in both heart failure and other characteristics. Under population segmentation, we conducted logistic regression analysis and found that CHF had statistical significance among other patients, which is somewhat consistent with some studies. Isath et al. [21] found that logistic regression analysis was utilized to identify independent predictors associated with increased odds of in-patient mortality, revealing that heart failure was correlated with heightened mortality risk. Tomasoni et al. [22] found that in-hospital mortality was higher in patients with a history of heart failure (41.1 %) compared to those without (20.9 %), and heart failure history independently predicted in-hospital mortality. Castagna et al. [23] reported that patients with heart failure had a notably higher in-hospital mortality rate ($p < 0.001$), this association remained statistically significant even after adjusting for various factors and propensity score matching. Furthermore, in other research, the study findings demonstrate a significant 21.2 % readmission rate among patients diagnosed with both COVID-19 and acute heart failure. This highlights the urgent need for addressing this issue and implementing effective interventions to reduce readmissions, The relationship between COVID-19 and heart failure is currently being studied [24]. COVID-19 patients with heart failure have an elevated risk of in-hospital mortality. The incidence of worsening heart failure or new-onset acute heart failure during hospitalization is high and associated with a further increase in in-hospital mortality [25]. Although a number of previous studies have indicated an association between heart failure and an increased risk of mortality, there are also studies suggesting that the risk of death within 60 days may not be high, despite a high

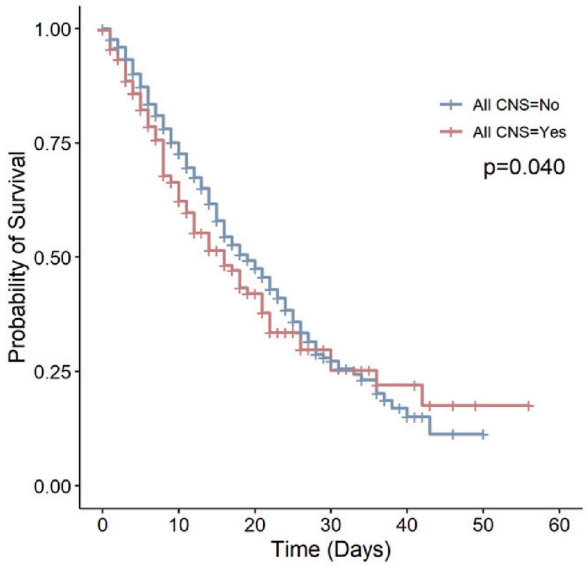

Fig. 4. Impact of comorbidities on survival in other patients with all CNS: A descriptive analysis using KM survival curves. Abbreviations: CNS, central nervous system; KM, kaplan-meier.

incidence rate [26]. Some research has found a reduction in hospital admissions for heart failure during the COVID-19 pandemic [27]. Furthermore, it has been observed that during the COVID-19 pandemic, the in-hospital mortality rate for heart failure decreased by 29 %, while the at-home mortality rate increased by 31 % [28]. Therefore, there is a certain complexity in the relationship between COVID-19 and heart failure, which may be attributed to regional variations, differences in data quantity or collection methods, or other potential factors. However, further observations have revealed that the risk of heart failure varies among different population groups, as evidenced by the significant value of 0.02 observed for "Other patients" in Table 2. Additionally, differences in comorbidities such as COPD and renal disease among different population groups have been identified, further demonstrating the rationale and effectiveness of population stratification.

Similar findings have been reported in other studies, the subsequent research incorporated racial factors in its analysis. COVID-19 hospitalization disparities persist among racial/ethnic groups in the USA, emphasizing the need for targeted interventions in high-risk populations [29]. The study examines the impact of racial and gender identity on COVID-19 hospitalization outcomes. Its findings reveal that Black men face the highest risk of acute kidney injury (aOR 2.0, CI 1.9–2.0), while Asian/Pacific Islander women have the highest risk of stroke (aOR 1.5, $p = 0.001$) [30]. Similarly, in our study, we discovered that DM complicated may have a protective effect specifically among Black individuals. This impact may involve genetic predisposition. Over the years, it has been recognized that there are differences in the prevalence of diabetes among different racial groups, with a more significant impact on certain populations [31]. One significant reason for these racial disparities is genetics, with over 400 genetic variants potentially associated with diabetes already identified [32]. Similar studies have found that certain genes in black patients with diabetes may have a protective effect against diabetes [33], and genetic research has shown that alleles like HLA, when negative, can lower the hospitalization death rate for African Americans (Blacks) with COVID-19 [34]. Given the complexity involved, previous studies have indicated that there is still uncertainty regarding the increased risk of diabetes, necessitating more detailed investigations and research [35]. However, we believe that there are even more complex factors at play, as recent research based on a cohort study of 181,280 individuals revealed an increased risk of developing diabetes after contracting the COVID-19 virus [36]. Therefore, in light of our findings on diabetes complications, collecting more comprehensive data may help to further elucidate this intricate mechanism.

The aforementioned points, such as the high mortality rate of heart failure and the decreased hospitalization rate during the pandemic, as well as the genetic protective effects of diabetes and its induction by COVID-19, may appear contradictory. However, they ultimately contribute to a more comprehensive understanding of the complex physiological responses triggered by COVID-19, which also share certain commonalities, such as the urgency caused by the high mortality rate of hospitalized heart failure patients. This highlights the importance of conducting extensive research to understand the health conditions that may influence mortality rates. A comprehensive, race-stratified study becomes particularly important in this regard. During the epidemic, the reduced number of heart failure patients being hospitalized makes it crucial to focus on risk control for patients at home. Precise clinical care is essential for heart failure, and telemedicine can provide effective primary and secondary preventive measures [37]. Considering the potential for complex comorbidities and viral variations associated with COVID-19, our strategy of tailored research based on ethnicity or comorbidities holds promise for further elevating the level of home care, ensuring that patients receive optimal care and treatment. It is crucial to acknowledge that the COVID-19 is constantly spreading and undergoing genetic mutations. Thus, it is vital to continuously monitor data statistics and stay updated on advancements in this field. The insights obtained from this study are of great value in enhancing our understanding of the implications of the novel coronavirus and in developing improved measures to

safeguard public health.

5. Advantages and limitations

Our study has several strengths. Firstly, we conducted a comprehensive analysis of publicly available large-scale datasets and designed a series of experiments to analyze the relationship between COVID-19, heart failure, and other variables. This approach allowed us to consider a wide range of factors and ensure the comprehensiveness of our analysis. Additionally, our study included racial group classification experiments, providing valuable insights into the impact of these variables within different populations. Furthermore, we utilized a substantial amount of data and employed rigorous analysis methods, enhancing the robustness of our findings. Overall, our study contributes to a deeper understanding of the COVID-19 pandemic, particularly regarding the effects of heart failure and other variables.

However, we acknowledge several limitations in our research. Despite our efforts to select ideal datasets, the generalizability of our findings may be limited. The results obtained from specific cohorts might not be applicable to broader populations or different healthcare systems. Additionally, while we presented the results of all influencing factors in an intuitive manner, it is possible that our methods did not capture the full complexity of interactions. This is especially relevant to the analysis of biomarkers, where further exploration and potential discoveries may exist.

6. Conclusions

The COVID-19 pandemic has presented unprecedented challenges to healthcare systems worldwide, especially for patients with comorbidities such as heart failure. This study explores the relationship between COVID-19 and heart failure, investigating risk factors and biomarkers linked to mortality in diverse racial groups. Our analysis reveals distinct mortality risk factors for each racial group, underscoring the importance of personalized risk assessment and management strategies for diverse populations. The study contributes to our understanding of the complex interactions between COVID-19, risk factors, and racial disparities in mortality rates. Continual monitoring of data and genetic mutations of the virus is recommended to inform future interventions and public health measures. This study serves as a valuable resource for developing targeted disease management strategies, promoting equitable healthcare outcomes, and mitigating the impact of the COVID-19 pandemic on affected populations.

Data availability statement

We analyzed the public dataset in this study. It is available at https://figshare.com/s/79827c396af7df42b3d7.

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Ethical approval

Ethical review and approval were waived for this study, due to the reason that this study only makes use of publicly available data.

Abbreviations

AF	atrial fibrillation	OR	odds ratio
HR	hazard ratio	CI	confidence interval
MI	myocardial infraction	OtherBrnLsn	other brain lesions
PVD	peripheral vascular disease	O2 Sat	oxygen saturation
CVD	cardiovascular disease	Temp	temperature
DEMENT	dementia	MAP	mean arterial pressure
COPD	chronic obstructive pulmonary disease	INR	international normalized ratio
DM Complicated	diabetes mellitus complicated	BUN	blood urea nitrogen
DM Simple	diabetes mellitus simple	AST	aspartate aminotransferase
All CNS	all central nervous system	ALT	alanine aminotransferase
Pure CNS	pure central nervous system	WBC	white blood cells
OldSyncope	old syncope	IL6	Interleukin-6
OldOtherNeuro	old other neurologic disorders	C-Reactive. Prot	C-reactive protein

CRediT authorship contribution statement

Yi Liu: Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization, Data curation, Software. **Dengao Li:** Writing – review & editing, Supervision, Software, Funding acquisition, Conceptualization, Methodology, Project administration, Resources, Validation. **Yuchen Liang:** Writing – original draft, Investigation, Formal analysis, Conceptualization, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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