

Original Article

COVID-19 worsens quality of life in elderly heart failure patients: a clinical study

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Abstract: Background: The coronavirus disease 2019 (COVID-19) pandemic has significantly worsened the health and quality of life of vulnerable populations, particularly elderly patients with heart failure. This study aimed to assess the effect of COVID-19 infection on the quality of life in elderly patients with heart failure during the pandemic. Methods: This retrospective case-control study included elderly heart failure patients admitted to the Second People's Hospital of Lanzhou between December 2022 and December 2023, all of whom were diagnosed during the ongoing COVID-19 pandemic. All patients underwent COVID-19 nucleic acid testing upon admission. Among the 96 heart failure patients who tested positive for COVID-19 and the 68 who tested negative, multiple validated instruments were used to assess both physical and mental health quality of life. These instruments included the Minnesota Living with Heart Failure Questionnaire (MLHFQ), 36-Item Short Form Health Survey physical component summary score (SF-36 PCS), 6-Minute Walking Test (6MWT), Geriatric Depression Scale-15 (GDS-15), Self-Rating Anxiety Scale (SAS) Total, Pittsburgh Sleep Quality Index (PSQI), Mini Nutritional Assessment-Short Form (MNA-SF), and the Fatigue, Resistance, Ambulation, Illness, and Loss of weight (FRAIL) Scale. Results: Heart failure patients who tested positive for COVID-19 exhibited significantly lower blood pressure, SF-36 scores, and 6MWT distances compared to those who tested negative ($P<0.05$). Additionally, the COVID-19-positive group had higher MLHFQ scores, older average age, a greater proportion of patients in NYHA class III-IV, more frequent electrolyte imbalances, elevated D-dimer, C-reactive protein (CRP), and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, and longer hospital stays ($P<0.05$). These patients also exhibited higher levels of anxiety (SAS total), poorer sleep quality (PSQI), and greater frailty (FRAIL Scale) compared to their COVID-19-negative counterparts ($P<0.05$). In addition, heart failure patients with COVID-19 infection reported more severe symptoms of dyspnea and fatigue ($P<0.05$). Both age and COVID-19 infection were identified as significant factors negatively affecting the quality of life in this patient population. Conclusion: COVID-19 infection significantly exacerbates the decline in quality of life in elderly patients with heart failure. This highlights the urgent need for strengthened, comprehensive treatment and targeted mental health support for this vulnerable group.

Keywords: COVID-19, heart failure, elderly patients, quality of life

Introduction

The Coronavirus 2019 (COVID-19) pandemic has had far-reaching global consequences, affecting various aspects of society. While the immediate public health crisis is well-documented, it is equally important to recognize the challenges it presents to vulnerable populations, particularly individuals with pre-existing health conditions. Among these groups, elderly patients with heart failure represent a particularly high-risk cohort due to their compromised immune systems and pre-existing conditions. Heart failure patients infected with COVID-19

often experience significant changes in dietary habits, which can result in complications such as electrolyte imbalances and malnutrition. Furthermore, the mental health consequences of the pandemic should not be underestimated. The heightened anxiety surrounding health concerns can precipitate psychological distress, including depression and anxiety [1]. Social isolation, a key public health strategy to prevent viral transmission, has further compounded these issues. Measures such as home confinement and reduced social interaction have contributed to increased feelings of loneliness and unmet social needs, which may have had a det-

rimental effect on the overall well-being of these already vulnerable patients.

In addition, the presence of anxiety and depression among patients can significantly increase the risk of developing heart failure, thereby further compromising their quality of life [2]. In recent years, there has been growing interest in investigating the relationship between COVID-19 and cardiovascular conditions, particularly in elderly populations. For instance, Metkus unveiled a notable increase in mortality rates among individuals with both COVID-19 and heart failure [3-5]. Similarly, Khajehpoor noted a decline in the quality of life of heart failure patients infected with COVID-19 [6]. These studies highlight the detrimental effects of COVID-19 in individuals with pre-existing heart failure.

Given these findings, we sought to understand the effect of COVID-19 on the quality of life in elderly heart failure patients. This study aims to investigate how COVID-19 affects the quality of life of this vulnerable population, focusing on the long-term consequences of the virus and providing insight to inform clinical practice and interventions. By assessing the effects of the pandemic on the daily lives, mental health, and overall well-being of elderly heart failure patients, this study underscores the importance of recognizing these impacts to enhance our response to the pandemic [7, 8], enabling us to improve management strategies for current and future health crises, and ultimately mitigating the adverse effects of pandemics [9, 10].

Materials and methods

Study population

This retrospective case-control study was conducted among elderly patients with heart failure who were admitted to the Second People's Hospital of Lanzhou between December 2022 and December 2023. The inclusion criteria were patients aged 65 years or older, diagnosed with chronic heart failure according to the Chinese diagnostic criteria, who were fully conscious and capable of participating in the relevant surveys. The exclusion criteria included patients with brain injuries, speech impairments, communication difficulties, severe cerebrovascular diseases, cognitive dysfunction, or

chronic conditions such as tumors, mental illnesses, or motor dysfunctions. Additionally, patients who died during hospitalization were excluded from the study. A total of 1584 patients were included in the study. On the day of admission, all patients underwent COVID-19 nucleic acid testing. Of these, 288 patients tested positive for COVID-19 and 1296 tested negative. Among those who tested positive for COVID-19, 96 had heart failure, while 68 patients who tested negative had heart failure. All heart failure patients were admitted due to recent exacerbations of their condition. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Second People's Hospital of Lanzhou. The rights and privacy of the participants were fully respected, and their personal information was kept confidential. Prior to participation, the study's purpose, procedures, potential risks, and benefits were explained to all participants, either orally or in writing.

Research methods

Demographic data collected in this study included age and gender. Clinical data encompassed a range of factors, including body mass index (BMI), smoking history, hypertension, diabetes, coronary heart disease, renal insufficiency, chronic obstructive pulmonary disease, and other related diseases. Additionally, key clinical indicators were measured upon admission, including blood pressure, NYHA function classification, heart rate, and oxygen saturation. Laboratory and examination results were thoroughly recorded, including blood lipid profiles, renal function, red blood cell counts, D-dimer levels, N-terminal pro-brain natriuretic peptide (NT-proBNP), and C-reactive protein (CRP). Medication use after admission was also meticulously documented. Three months after discharge, the patient's quality of life was assessed, considering both physical and psychological aspects.

Measurement tools

The Minnesota Living with Heart Failure Questionnaire (MLHFQ) [11] and 36-Item Short Form Health Survey (SF-36) scale were used to assess the quality of life of the patients [12]. The 6-minute walk test was used to evaluate exercise tolerance. Negative emotions were

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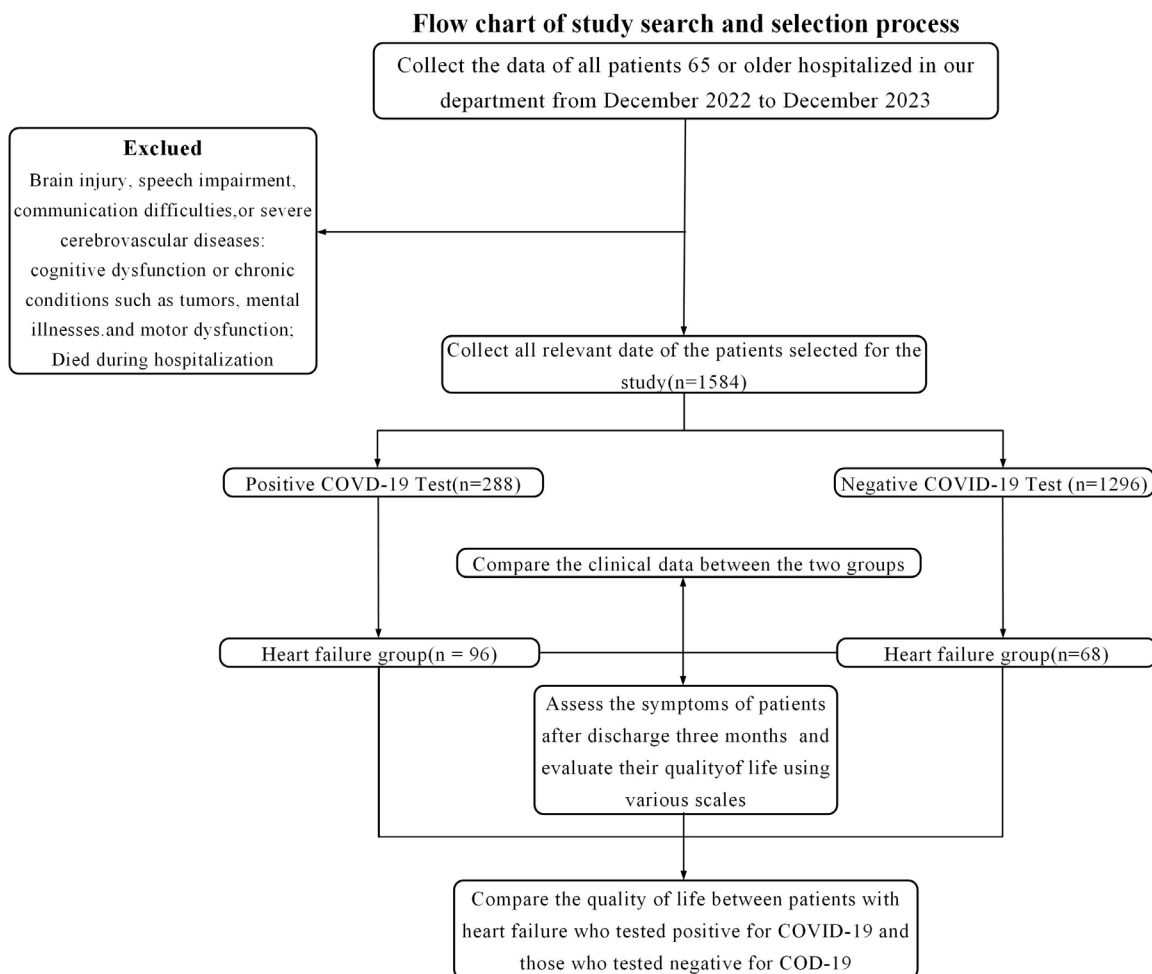


Figure 1. Flowchart of study search and selection process. Note: COVID-19, Coronavirus disease 2019.

evaluated using the Self-rating Anxiety Scale (SAS) and Geriatric Depression Scale-15 (GDS-15) [13, 14]. Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI) [15], while nutritional status was evaluated with the Mini Nutritional Assessment-Short Form (MNA-SF) [16]. Frailty status was assessed using the FRAIL Scale [17]. Clinical data were thoroughly compared between COVID-19-positive and COVID-19-negative groups of heart failure patients, examining the differences between those with and without COVID-19. Follow-up evaluations and quality-of-life assessments were performed at discharge. Based on prior clinical findings, the MLHFQ is used to evaluate the quality of life in patients with heart failure. It is widely recognized for its high reliability and stability, making it valuable for evaluating QOL, providing therapeutic guidance, facilitating clinical research, demonstrating robust validity

and reliability, and promoting self-management among heart failure patients. Given that comorbid conditions such as hypertension, diabetes, and chronic kidney disease - along with clinical indicators like heart rate, blood pressure, cardiac function status, and NT-proBNP - can significantly influence the quality of life of elderly heart failure patients, the MLHFQ plays a pivotal role in capturing these variables. This helps inform clinical decision-making processes [18-20]. The MLHFQ score was selected as the dependent variable, while independent variables included age, gender, comorbidities (e.g., COVID-19, hypertension, diabetes), blood NT-proBNP levels, ejection fraction (EF), frailty status (FS), and other relevant clinical indicators. Univariate and multivariate regression analyses were conducted to investigate the factors that significantly affect the quality of life in elderly patients with heart failure (**Figure 1**).

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Table 1. Baseline characteristics of COVID-19 patients

Variable	Positive COVID-19 Test (n=288)	Negative COVID-19 Test (n=1296)	P Value
Age (years)	75.80±6.80	69.00±8.60	<0.001
Gender (Female), n (%)	158 (54.86%)	786 (60.88%)	0.070
Hypertension, n (%)	120 (41.67%)	545 (42.05%)	0.904
Diabetes, n (%)	36 (12.50%)	172 (59.72%)	0.726
Dyslipidaemia, n (%)	56 (19.44%)	286 (22.07%)	0.328
Smoking Habit, n (%)	66 (22.92%)	311 (24.00%)	0.697
Obesity, n (%)	38 (13.19%)	191 (14.74%)	0.501
Ischemic Stroke, n (%)	32 (11.11%)	165 (12.73%)	0.451
Coronary Artery Disease, n (%)	22 (7.64%)	65 (5.01%)	0.077
Atrial Fibrillation/Flutter, n (%)	20 (6.94%)	72 (5.56%)	0.362
Chronic Heart Failure, n (%)	96 (33.33%)	68 (5.25%)	<0.001
COPD, n (%)	76 (26.39%)	216 (16.67%)	<0.001
Kidney Failure, n (%)	24 (8.33%)	97 (7.48%)	0.624
Electrolyte Imbalance, n (%)	83 (28.82%)	136 (10.49%)	<0.001
SBP, mmHg, mean ± SD	132.13±22.89	149.22±20.07	<0.001
Heart Rate, bpm, mean ± SD	86.96±18.70	85.22±17.49	0.132
First Oxygen Saturation, %, mean ± SD	90.79±5.76	91.22±5.47	0.232
Median Hemoglobin, g/dL	138.88±20.72	141.11±19.39	0.082
Highest D-dimer, ng/mL	0.97±0.48	0.82±0.41	<0.001
Highest NT-proBNP, pg/mL	1034.51±511.74	986.89±475.84	0.130
Highest CRP (mg/L)	21.51±10.72	19.91±9.95	0.015
Hospital Stay (day)	12.04±3.48	8.39±3.02	<0.001

Note: COVID-19, Coronavirus Disease 2019; COPD, Chronic obstructive pulmonary disease; SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein.

Statistical analysis

Data analysis was conducted utilizing SPSS statistical software. Quantitative data that followed a normal distribution were presented as mean ± standard deviation (mean ± SD). Independent sample t-tests were performed for group comparisons. Counted data were represented as percentages and analyzed using chi-square tests. Multivariate linear regression analysis was employed to identify factors associated with the quality of life in elderly patients with heart failure. Before conducting the regression analysis, the assumptions for multiple linear regression were thoroughly tested. The results indicated that the continuous independent variables were linearly correlated with the dependent variable, the residuals met the assumptions of independence and normal distribution, and the data satisfied the homogeneity of variance assumption. The data were deemed appropriate for multiple linear regression analysis. A significance level of 0.05 was set for all statistical tests.

Results

Comparison of clinical data between COVID-19 positive and negative patients

A total of 1584 patients were included in the study, consisting of 288 COVID-19-positive patients and 1296 COVID-19-negative patients. The COVID-19-positive group exhibited a significantly higher incidence of heart failure, as well as increased average age, a greater prevalence of chronic obstructive pulmonary disease (COPD), electrolyte imbalances, elevated D-dimer levels, higher CRP levels, and longer hospital stays. Additionally, this group had significantly lower blood pressure upon admission compared to the the COVID-19-negative group ($P<0.05$) (**Table 1**).

Comparison of clinical data between COVID-19 positive and negative heart failure patients

Among the 164 patients with heart failure, 96 were COVID-19-positive and 68 were COVID-

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Table 2. Baseline data of patients with heart failure

Variable	Positive COVID-19 Test (n=96)	Negative COVID-19 Test (n=68)	P Value
Age (years)	76.55±4.25	69.33±8.45	<0.001
Gender (Female), n (%)	56 (58.33%)	43 (63.40%)	0.527
Hypertension, n (%)	51 (53.13%)	40 (58.82%)	0.469
Diabetes, n (%)	18 (18.75%)	14 (0.59%)	0.770
Dyslipidemia, n (%)	21 (21.88%)	16 (23.53%)	0.803
Smoking Habit, n (%)	30 (31.25%)	26 (38.24%)	0.353
Obesity, n (%)	15 (15.63%)	17 (0.25%)	0.136
Ischemic Stroke, n (%)	11 (11.45%)	11 (16.18%)	0.382
Coronary Artery Disease, n (%)	15 (15.63%)	10 (14.71%)	0.872
Atrial Fibrillation/Flutter, n (%)	24 (25.00%)	14 (20.59%)	0.509
COPD, n (%)	40 (41.67%)	27 (39.71%)	0.801
Kidney Failure, n (%)	8 (8.33%)	4 (5.89%)	0.553
Electrolyte Imbalance, n (%)	24 (25.00%)	5 (7.35%)	0.004
NYHA FC III-IV	83 (86.46%)	43 (63.24%)	0.001
SBP, mmHg, mean ± SD	126.15±26.12	151.88±19.55	<0.001
Heart Rate, bpm, mean ± SD	83.46±20.10	78.94±10.88	0.094
First Oxygen Saturation, %, mean ± SD	89.69±9.01	92.22±8.67	0.074
Median Haemoglobin, g/dL	138.15±16.57	142.38±16.41	0.108
Highest D-dimer, ng/mL	1.14±0.52	0.91±0.43	0.003
Highest NT-proBNP, pg/mL	3520.69±1089.21	2764.33±940.32	<0.001
Highest CRP, mg/L	61.41±30.53	22.21±11.03	<0.001
Hospital Stay (day)	13.54±3.51	9.52±1.82	<0.001
Therapeutic Anticoagulation	65 (67.70%)	8 (11.80%)	<0.001
Antiplatelet	75 (78.10%)	58 (85.20%)	0.248
ACEI or ARB	56 (58.30%)	37 (54.40%)	0.618
Beta-blocker	28 (29.10%)	23 (33.80%)	0.618
Diuretics	87 (90.60%)	62 (91.20%)	0.904
SGLT2 Inhibitors	13 (13.50%)	8 (11.80%)	0.737
Digoxin	7 (7.20%)	5 (7.30%)	0.988
Statin	67 (69.80%)	42 (61.80%)	0.283
EF (%)	41.31±7.96	43.22±7.29	0.119
FS (%)	19.38±3.18	19.06±2.58	0.494
Hospital Stay (day)	14.38±3.41	9.83±1.76	<0.001

Note: COVID-19, Coronavirus disease 2019; COPD, chronic obstructive pulmonary disease; NYHA, New York Heart Association Functional Class; SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; SGLT2, sodium-glucose co-transporter 2; EF, Ejection Fraction; FS, Fractional Shortening.

19-negative. The COVID-19-positive heart failure group had significantly higher average age, prevalence of NYHA class III-IV, frequency of electrolyte imbalances, D-dimer levels, CRP levels, NT-proBNP levels, length of hospital stays, and rates of anticoagulant therapy, along with significantly lower blood pressure levels ($P<0.05$) (**Table 2**).

Comparison of quality of life between COVID-19 positive and negative heart failure patients

A comparison of quality of life between heart failure patients with and without COVID-19 revealed that the COVID-19-positive group had significantly higher MLHFQ scores, SAS scores, PSQI scores, FRAIL scores, as well as higher

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Table 3. Quality of life in heart failure patients with COVID-19 compared to those with negative COVID-19 tests

Variable	Positive COVID-19 Test (n=96)	Negative COVID-19 Test (n=68)	P Value
MLHFQ			
Physical Domain	55.49±14.21	48.37±14.28	0.002
Emotional Domain	52.16±9.94	48.67±9.19	0.024
Other Domain	53.74±10.02	47.34±8.18	<0.001
Total Score	54.61±10.56	48.46±8.57	<0.001
6 Minute Walk (feet)	281.91±118.56	335.56±112.43	0.004
SF-36 PCS	57.06±15.92	49.09±15.13	0.002
GDS-15	7.55±3.47	6.72±2.81	0.105
SAS Total	55.45±14.77	48.72±12.81	0.003
PSQI	8.82±4.41	7.11±2.56	0.005
MNA-SF	8.86±3.38	9.56±3.03	0.175
FRAIL Scale	3.81±1.84	2.94±1.74	0.003
Symptom			
Dyspnea	45 (46.88%)	21 (30.88%)	0.040
Cough	25 (26.04%)	19 (27.94%)	0.787
Abdominal Distension	21 (21.88%)	13 (19.12%)	0.668
Fatigue	59 (61.46%)	28 (41.18%)	0.010
NYHA class			
I-II	75 (78.13%)	62 (91.18%)	0.026

Note: COVID-19, Coronavirus disease 2019; MLHFQ, Minnesota Living with Heart Failure Questionnaire; SF-36 PCS, the 36-item Short-Form Health Survey Physical Component Summary; GDS-15, Geriatric Depression Scale-15; SAS Total, Self-Rating Anxiety Scale Total; PSQI, Pittsburgh Sleep Quality Index; MNA-SF, Mini Nutritional Assessment - Short Form; FRAIL Scale, Fatigue, Resistance, Ambulation, Illness, and Loss of weight scale; NYHA class, New York Heart Association Class.

frequencies of dyspnea and fatigue. In contrast, the COVID-19-positive group had significantly lower SF-36 scores and 6MWT distances compared to patients without COVID-19 ($P<0.05$) (Table 3).

Univariate linear regression

Univariate linear regression analysis identified significant correlations between COVID-19, age, NT-proBNP levels, systolic blood pressure, and MLHFQ scores (Table 4). COVID-19 was positively correlated with MLHFQ scores, with the strongest association observed (regression coefficient =17.489, $R^2=0.075$, $P<0.001$). Age also showed a positive correlation with MLHFQ scores (regression coefficient =1.052, $R^2=0.011$, $P<0.001$), indicating that older age was associated with poorer quality of life. NT-proBNP levels showed a robust positive correlation with MLHFQ scores ($R^2=0.105$, $P<0.05$), suggesting that higher levels of this biomarker, reflecting greater heart failure

severity, significantly affect the quality of life. Conversely, systolic blood pressure was negatively correlated with MLHFQ scores ($R^2=0.008$, $P<0.05$), implying that lower blood pressure may be associated with worse quality of life (Figures 2-4).

Multivariate linear regressions

To account for multiple confounding factors, a multivariate linear regression analysis was conducted to evaluate the determinants of quality of life in elderly patients with chronic heart failure. The MLHFQ total score was designated as the dependent variable, while age, gender, blood pressure, NT-proBNP, EF, FS, and comorbidities (hypertension, diabetes, kidney failure), heart rate, and COVID-19 status were included as independent variables (Figure 5). The analysis indicated that COVID-19 infection was positively correlated with MLHFQ scores (regression coefficient =11.128, $P<0.05$),

suggesting that COVID-19 infection is associated with poorer quality of life. Additionally, age was positively correlated with MLHFQ scores, implying that advancing age is associated with an increase in MLHFQ scores and a decline in quality of life ($B=0.570$, $P<0.05$) (Table 4).

Discussion

This study investigates the effect of COVID-19 on the quality of life in elderly patients with heart failure, providing a comprehensive analysis of the research results. With a sample size of 1584 patients, this study supports the generalizability of the results. However, since all participants were recruited from hospitals within the same geographical region, the findings may not be fully applicable to populations in other regions or healthcare systems. Despite this limitation, the results of this study highlight the negative impact of COVID-19 on the quality of life in elderly patients with heart failure, providing a foundation for clinicians to optimize treatment strategies and improve patient care.

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Table 4. Univariate linear regression and multivariate linear regression analysis of factors influencing MLHFQ score in elderly patients with heart failure

Factor	Univariate Linear Regression					Multivariate Linear Regression						
	β	95% CI	P-value	R Square	Adjusted R Square	B	SE	β	t	P value	R Square	Adjusted R Square
Age	1.052	[0.743, 1.36]	0.000	0.011	-0.003	0.570	0.270	0.341	2.113	0.039	0.436	0.419
Gender (Male)	3.191	[-3.899, 10.281]	0.378	0.396	0.387	-1.301	3.138	-0.43	-0.414	0.680		
Hypertension	2.496	[-4.356, 9.349]	0.475	0.007	-0.007	-0.120	2.947	-0.004	-0.041	0.968		
Diabetes	3.750	[-3.760, 11.260]	0.328	0.014	-0.001	7.159	3.775	0.225	1.897	0.063		
Kidney Failure	-19.529	[-39.505, 0.446]	0.055	0.051	0.037	7.218	7.021	0.194	1.028	0.313		
Heart Rate	-0.083	[-0.298, 0.131]	0.448	0.369	0.360	-5.333	4.767	-0.210	-1.119	0.273		
COVID-19	17.489	[12.052, 22.926]	0.000	0.075	0.061	11.128	5.240	0.386	2.124	0.038		
SBP	-0.157	[-0.288, -0.026]	0.019	0.008	-0.006	0.047	0.078	0.115	0.602	0.552		
NT-proBNP	0.005	[0.001, 0.008]	0.005	0.105	0.092	0.000	0.002	-0.18	-0.797	0.685		
EF	-0.259	[-0.864, 0.347]	0.403	0.010	-0.004	0.416	0.378	0.207	1.100	0.281		
FS	-0.38	[-1.634, 0.875]	0.553	0.005	-0.009	1.052	0.746	0.262	1.410	0.170		

Note: COVID-19, Coronavirus disease 2019; SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide; EF, Ejection Fraction; FS, Fractional Shortening; MLHFQ, Minnesota Living with Heart Failure Questionnaire.

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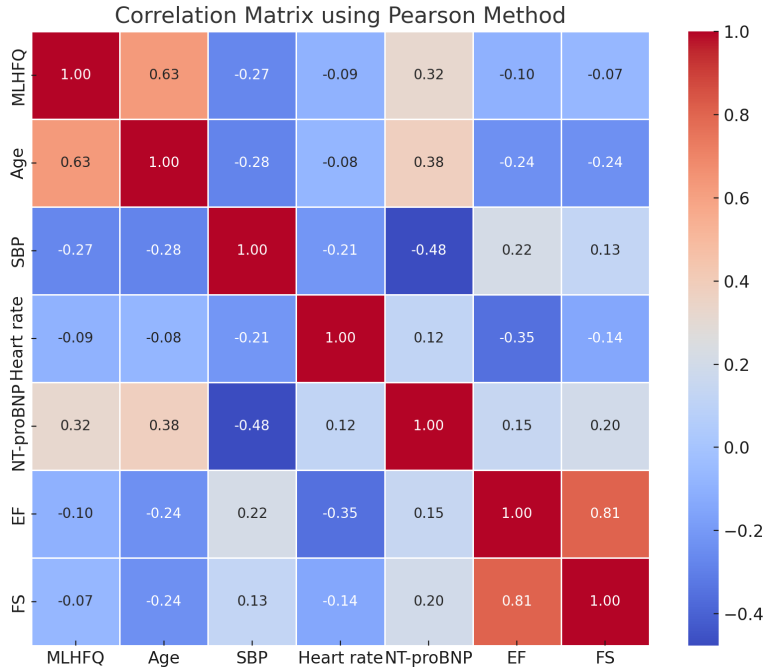


Figure 2. Correlation matrix using the Pearson method. Note: MLHFQ, Minnesota Living with Heart Failure Questionnaire; SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide; EF, Ejection Fraction; FS, Fractional Shortening.

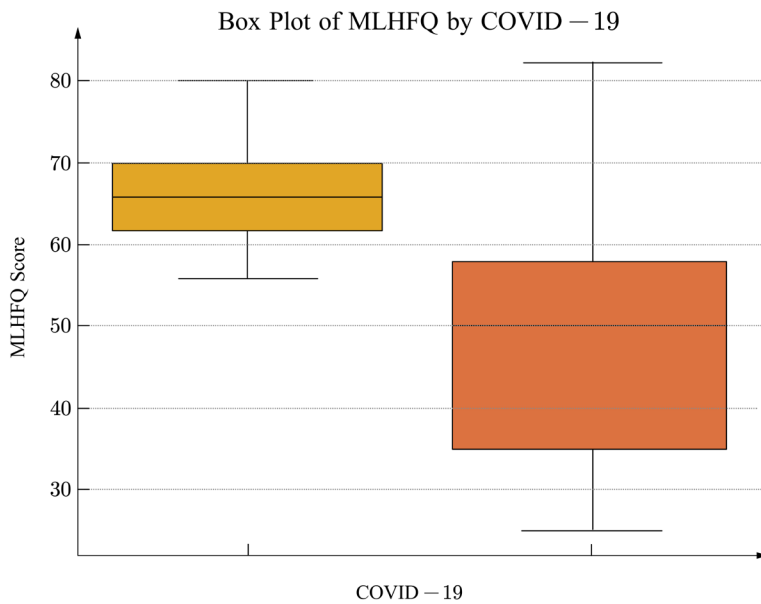


Figure 3. Box plot of MLHFQ scores by COVID-19 status. Note: MLHFQ, Minnesota Living with Heart Failure Questionnaire; COVID-19, Coronavirus disease 2019.

Elderly patients are particularly susceptible to COVID-19 due to age-related decline in physiologic functions, including weakened respiratory

defenses and diminished lung function. This study found a significant increase in the average age of patients testing positive for COVID-19, alongside evidence of immune system deterioration, such as reduced T and B cell counts and decreased antibody levels [21, 22]. Moreover, elderly patients often suffer from multiple chronic comorbidities, such as cardiovascular and cerebrovascular diseases, chronic lung conditions, and diabetes, which can exacerbate the severity of COVID-19 infections and increase the risk of complications. COVID-19-positive patients also exhibit elevated levels of CRP and D-dimer, which are biomarkers of systemic inflammation and coagulation abnormalities. Inflammatory reactions in COVID-19 patients trigger the release of various cytokines, leading to an acute-phase reaction that raises CRP levels. The uncontrolled release of inflammatory cytokines and chemokines leads to elevated D-dimer levels [23]. As the disease progresses, COVID-19 can induce hypercoagulability, blood stasis, and endothelial injury, further increasing the risk of venous thromboembolism (VTE) and exacerbating D-dimer levels [24-26]. These findings indicate that the combination of age-related physiological decline and pre-existing comorbidities significantly heightens the risk and severity of COVID-19 in elderly patients.

Recent studies have demonstrated an association between COVID-19 and heart failure, with the pandemic substantially elevating the incidence of heart failure and adversely affecting patients'

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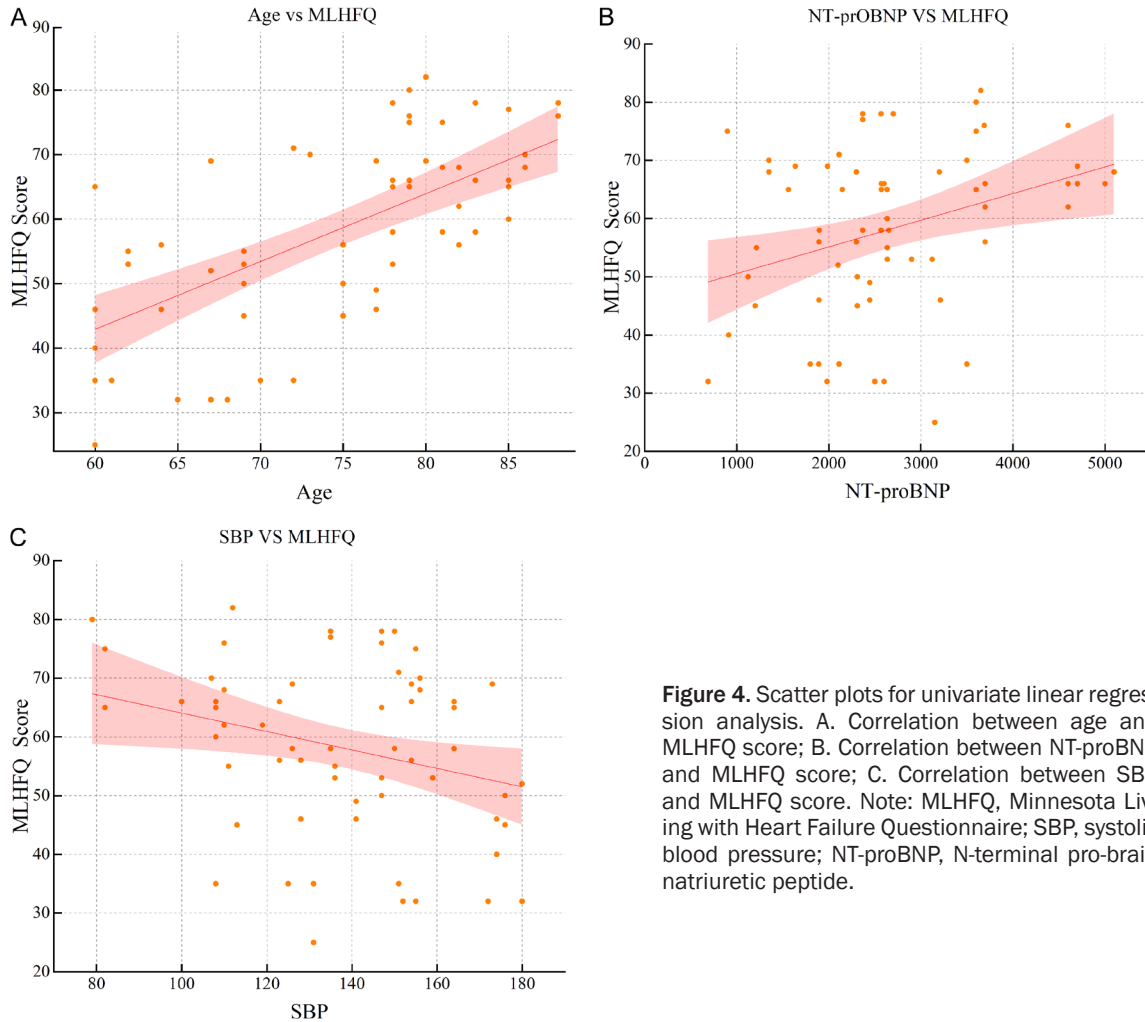


Figure 4. Scatter plots for univariate linear regression analysis. A. Correlation between age and MLHFQ score; B. Correlation between NT-proBNP and MLHFQ score; C. Correlation between SBP and MLHFQ score. Note: MLHFQ, Minnesota Living with Heart Failure Questionnaire; SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide.

quality of life [27, 28]. The virus enters cells through ACE2 receptors, which can lead to direct myocardial injury, thereby increasing the risk of heart failure [29, 30]. Inflammatory responses, particularly cytokine storms, may occur in infected patients, triggering excessive immune activation that harms the heart and contributes to myocardial injury, arrhythmia, and heart failure. Additionally, lung damage and subsequent hypoxia can further strain the cardiovascular system, particularly in severe cases, leading to ventricular dysfunction and, ultimately, heart failure. This study further elucidates the impact of COVID-19 on heart failure in elderly patients. The results indicate that COVID-19 infection significantly exacerbates the severity of heart failure, posing additional challenges in treatment. Elderly patients with heart failure who contract COVID-19 experience more severe dyspnea, elevated NT-proBNP levels, and an increased propensity for compli-

cations such as electrolyte imbalances and hypotension. These factors complicate the therapeutic management of these patients, resulting in prolonged hospitalization and poorer clinical outcomes. The multifaceted effect of COVID-19 on the cardiovascular system, especially in elderly patients with pre-existing heart failure, underscores the need for targeted interventions and comprehensive management strategies to improve patient outcomes.

This study details the effects of COVID-19 on patients with heart failure, highlighting the direct repercussions of the virus on their quality of life. Lung damage and hypoxia increase the cardiac workload, impairing myocardial function and significantly reducing quality of life. Cytokine storms can cause systemic inflammatory reactions, leading to additional cardiac damage, and the severity of the disease aggravates conditions such as myocarditis, arrhyth-

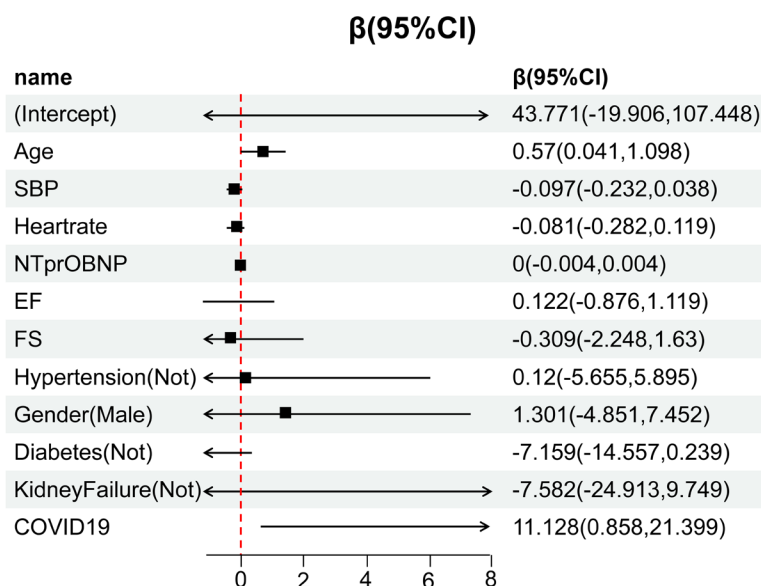


Figure 5. Forest plot illustrating effect sizes and confidence intervals of multivariate regression models. Note: SBP, systolic blood pressure; NT-proBNP, N-terminal pro-brain natriuretic peptide; EF, Ejection Fraction; FS, Fractional Shortening; COVID-19, Coronavirus disease 2019.

mias, and heart failure, all of which negatively affect the quality of life of elderly patients with heart failure [31]. Moreover, COVID-19 therapies, including antiviral medications, glucocorticoids, and immunosuppressants, may also adversely affect cardiac function, further complicating the management of these patients. From a physiological perspective, elderly patients experience a gradual decline in the function of multiple organ systems, which makes them particularly vulnerable to the effects of COVID-19. Upon infection, they are more susceptible to symptoms such as fatigue and dyspnea, which significantly impair their ability to perform daily activities, thereby reducing their independence. Additionally, the nutritional intake of these patients is often severely affected, leading to complications such as hypotension, electrolyte imbalances, and malnutrition. The increased complexity of treatment further detracts from their quality of life. In summary, the combined effects of age-related physiological decline, COVID-19-related complications, and intricate treatment regimens collectively reduce the quality of life in elderly heart failure patients.

The psychological stress induced by the COVID-19 pandemic cannot be overlooked. Upon contracting COVID-19, patients often experience

heightened concern about their health and recovery, which may trigger psychological distress. To prevent the spread of the virus, measures such as social isolation and home confinement have reduced people’s interactions with the external world, exacerbating feelings of loneliness and unmet social needs. These factors can contribute to the development of anxiety, depression, and sleep disorders in affected patients [32, 33]. As individuals age, their mobility typically decreases, further limiting their ability to engage in social activities outside the home. Consequently, opportunities for social interaction and communication are diminished, weakening their social support networks. This

reduction in social engagement can negatively affect mental well-being, impairing psychological adjustment. Additionally, anxiety and depression in these patients may exacerbate heart failure, worsening both their quality of life and prognosis [34, 35]. Thus, psychological stress and social isolation significantly worsen the mental health and overall quality of life in elderly patients with heart failure.

Limitations

The limitations of this study primarily stem from the restricted sample size and geographical scope, which may limit the generalizability and applicability of the findings. Moreover, the research design may not have fully accounted for all potential confounding factors, which could introduce bias and affect the accuracy of the results. Future research should focus on elucidating the differential impact of the COVID-19 pandemic on patients with heart failure across various age groups and severity levels, exploring the lasting consequences of the infection, and devising tailored intervention strategies to enhance the quality of life and resilience of elderly patients.

Despite these limitations, the findings of this study provide important insight into the effects

of COVID-19 on elderly patients with heart failure. The results offer valuable references for clinicians in optimizing treatment strategies and can inform policymakers in formulating appropriate healthcare policies. Furthermore, these findings lay the groundwork for future research in this area, facilitating further exploration of the long-term effects of COVID-19 on heart failure patients.

Conclusions

This study highlights the significant negative impact of COVID-19 on the quality of life of elderly patients with heart failure. The results show that elderly heart failure patients infected with COVID-19 experience more severe symptoms of dyspnea, elevated NT-proBNP levels, and a higher incidence of complications, including electrolyte imbalances and hypotension. These factors contribute to the increased complexity of their treatment, leading to prolonged hospital stays and poorer patient outcomes. Additionally, the psychological stress and social isolation associated with the pandemic have further compromised both the physical and mental health of these patients. Therefore, providing comprehensive care that addresses both the medical and psychological needs of this vulnerable group is crucial for improving patient outcomes and quality of life.

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Disclosure of conflict of interest

None.

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