Review Article

Impact of COVID-19 on Cardiovascular Outcomes in Patients with Chronic Heart Failure

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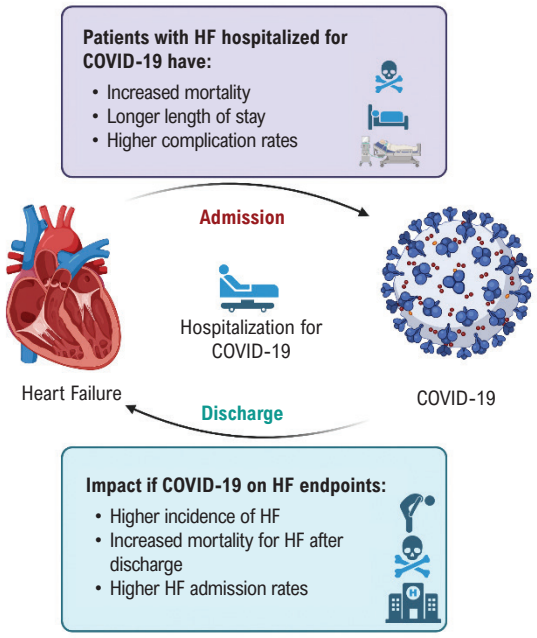
Central Illustration: Impact of COVID-19 on Cardiovascular Outcomes in Patients with Chronic Heart Failure



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COVID-19: coronavirus disease 2019; HF: heart failure.

Abstract

SARS-CoV-2 infection affects multiple organs and systems, including the cardiovascular system. Cardiovascular complications include myocarditis, pericarditis, acute coronary syndrome, acute heart failure (HF), pulmonary hypertension, right ventricular dysfunction, and arrhythmias. Inflammation is a mechanism that is present in both COVID-19 and HF. The mechanisms of myocardial injury due to COVID-19 include direct damage by the pathogen, hypoxemic stress, endothelial dysfunction with associated thromboembolic phenomena, and effects of systemic inflammatory syndrome. Impaired cardiopulmonary reserve makes patients with chronic HF more susceptible to decompensation during the course of COVID-19. Coronary and pulmonary vascular alterations lead to worse prognosis in relation to ischemic and thromboembolic events. These patients have longer hospital stays, increased risk of mechanical ventilation, and higher mortality.

COVID-19 increases the incidence of cardiovascular complications in the first 12 months after acute infection, including cerebrovascular, arrhythmic, thromboembolic, and ischemic events, in addition to inflammatory heart disease. An increased risk of developing HF with preserved ejection fraction has also been reported. During the 12 months following acute infection, myocardial injury has been associated with persistent symptoms, as well as increased rates of hospital readmission and mortality.

Keywords

Heart Failure; COVID-19; Prognosis.

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Therefore, HF is related to increased mortality, length of hospital stay, and incidence of complications in patients who are affected by SARS-CoV-2, and individuals with chronic HF who have COVID-19 show a higher rate of events during post-COVID follow-up.

Introduction

Coronavirus disease 2019 (COVID-19), which is caused by the SARS-CoV-2 virus, has resulted in more than 700 million people becoming ill, with approximately 6.9 million deaths worldwide, according to the World Health Organization. Illness due to SARS-CoV-2 infection affects multiple organs and systems, including the cardiovascular system. Acute cardiovascular complications of COVID-19 infection include myocarditis, pericarditis, acute coronary syndrome, acute heart failure (HF), pulmonary hypertension, right ventricular (RV) dysfunction, and arrhythmias.

Medium-term follow-up has shown that, even after recovery from acute infection, there is an increased incidence of many of these complications (arrhythmias, HF, coronary ischemic events), in addition to arterial hypertension, diabetes mellitus, and myocardial fibrosis.[1,2]

HF is among the leading causes of mortality and morbidity worldwide, and it is associated with high resource use and health care expenditures. Due to population aging and improved care and treatment, the prevalence of HF has rapidly increased. It is estimated that 26 million people suffer from this clinical syndrome. In Brazil, the prevalence of HF is approximately 2 million patients, and its incidence is 240,000 new cases per year.[3] HF is associated with increased mortality and increased incidence of complications in patients who become ill with SARS-CoV-2. Nonetheless, few studies have evaluated the long-term effects of COVID-19 on the progress of patients with chronic HF.

In this review, we will address the impact of HF as a comorbidity on the in-hospital progress of patients with COVID-19 and the impact of COVID-19 on the post-discharge progress of patients with chronic HF, as well as its impact on the incidence of HF.

Pathophysiology of myocardial injury in COVID-19

The mechanisms of myocardial injury due to COVID-19 include direct damage by the pathogen, hypoxemic stress related to lung injury, endothelial dysfunction with associated thrombotic and embolic phenomena, and effects of systemic inflammatory syndrome (Figure 1). Postmortem studies have demonstrated the main histological patterns in the lungs and heart. In the lungs, they include diffuse alveolar damage with extensive hyaline membrane, edema, and microthrombi in small vessels. In the heart, they include massive inflammatory infiltrates with myocarditis, pericarditis, vasculitis, as well as foci of necrosis and vascular fibrosis.[4]

Inflammation is a mechanism that is present in COVID-19 as well as in individuals with HF. This shared pathophysiological mechanism may, in part, explain the development of acute ventricular dysfunction in COVID-19 and the worse progress of patients with chronic HF who have COVID-19.[1] Rey et al. published a study with 3080 patients who sought emergency care with COVID-19 infection. Of these patients, 2.5% showed symptoms of acute HF at the time of admission, and

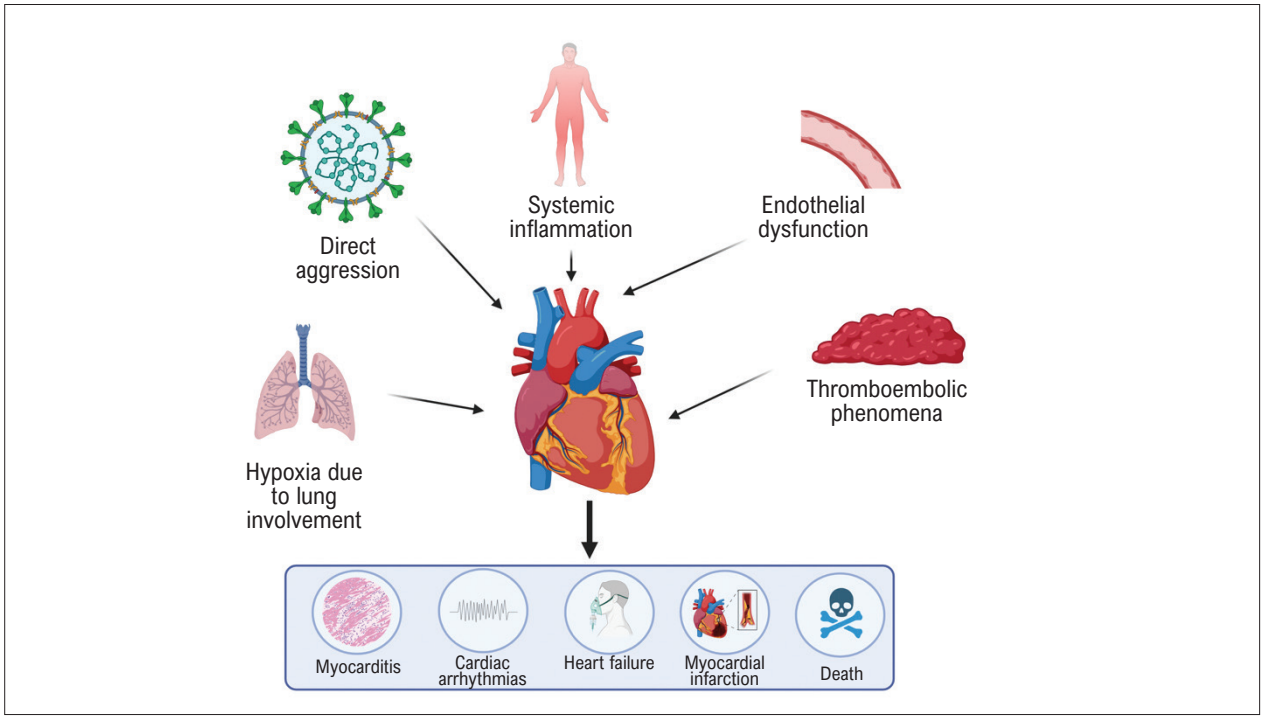

Figure 1 – *Mechanisms of myocardial injury in COVID-19 and their repercussions. Source: figure prepared by the authors.*

77.9% of acute HF cases were in patients who had no previous history of HF. Proportionally, patients with chronic HF had a greater percentage of acute HF (11.2% versus 1.8%).[5] These data may reflect the damage caused by SARS-CoV-2 to the myocardium and vascular endothelium, with the release of large amounts of pro-inflammatory chemokines, promoting a prothrombotic state. Other viral infections, such as the influenza virus, may also cause direct myocardial damage and damage due to inflammatory mechanisms. In these cases, an increase in cases of HF decompensation during influenza infection has been documented, but it had a lower mortality rate than that recorded in COVID-19 (1.8% in patients with HF who were not vaccinated against influenza and 1.4% in those who were vaccinated).

Cardiac arrhythmias, especially atrial tachyarrhythmias, have a higher incidence during viral infection. Patients with HF are even more susceptible to this complication, which is associated with increased hospital stay and mortality.[6] This increased incidence of cardiac rhythm disorders due to disturbances in myocyte cell membrane homeostasis is due both to the pathophysiological mechanisms of infection (inflammation and hypoxia) and to electrolyte disturbances related to renal dysfunction and muscle injury. Given that it is a prothrombotic state, episodes of atrial fibrillation/flutter are associated with an increased occurrence of events such as stroke.

Myocardial injury is a marker of poor prognosis, because it is associated with higher mortality. It has been identified in 12% of patients with COVID-19, in 41% of patients with severe forms, and in 76% of patients who progressed to death.[7] An increase in circulating endothelial cells was also associated with worse prognosis during COVID-19 infection. Patients requiring intensive care unit admission have shown an elevated number of circulating endothelial cells. These cells originate from the desquamation of the vascular endothelium stressed by inflammatory injury, and they indicate a correlation between endothelial injury and organic dysfunctions in severe COVID-19.[8]

Inflammatory infiltration of the myocardium accompanied by vasculitis and followed by vascular fibrosis and necrosis culminates in reduced tissue oxygen supply and, consequently, impaired myocardial relaxation. Szekely et al. analyzed the echocardiographic images of 100 patients hospitalized for COVID-19 and identified that left ventricular (LV) systolic function was preserved in most patients, but LV diastolic function and RV function were impaired. Myocardial injury and clinical deterioration during patient follow-up are associated with acute RV dysfunction, regardless of the presence of deep vein thrombosis. During follow-up, 20% of patients developed acute LV systolic dysfunction.[9] Similar findings were observed in a retrospective study from New York City including 105 patients with COVID-19 and similar age distribution. RV hypokinesia and moderate to severe tricuspid regurgitation were the most prevalent changes in patients hospitalized with COVID-19 and RV enlargement. Mortality was higher in patients with RV dysfunction when compared to patients without these echocardiographic alterations (41% versus 11%).[10]

Effects of HF as a comorbidity on the progress of patients with COVID-19

Patients hospitalized for HF in Brazil have shown a mortality rate of 12.6% according to data from the BREATHE study. This elevated value may increase when it is associated with COVID-19 as the cause of HF decompensation.[11]

Table 1 summarizes the main studies that have evaluated the effects of chronic HF on the outcomes of COVID-19. Impaired cardiopulmonary reserve makes individuals with chronic HF more susceptible to HF decompensation during the course of COVID-19. Vascular alterations in coronary and pulmonary circulation lead to worse prognosis in relation to ischemic and thromboembolic events. Garcia et al. studied a retrospective cohort with 6,439 patients admitted with COVID-19. Their analyses allowed them to conclude that patients with chronic HF and COVID-19 had a longer hospital stay (8 days versus 6 days; $p < 0.001$), increased risk of mechanical ventilation (22.8% versus 11.9%; adjusted odds ratio: 3.64; 95% confidence interval: 2.56 to 5.16; $p < 0.001$), and greater mortality (40.0% versus 24.9%; adjusted odds ratio: 1.88; 95% confidence interval: 1.27 to 2.78; $p = 0.002$).[12] Yonas et al. found similar data in a meta-analysis with 18 published studies, totaling 21,640 patients. HF was associated with hospitalization in COVID-19 (hazard ratio: 2.37 [1.48; 3.79; $p < 0.001$], with high heterogeneity [I^2 82%; $p < 0.001$]), worse outcomes (odds ratio: 2.86 [2.07; 3.95; $p < 0.001$], with high heterogeneity [I^2 80%; $p < 0.001$]), and greater mortality in patients with preexisting HF (odds ratio: 3.46 [2.52; 4.75; $p < 0.001$], with moderately high heterogeneity [I^2 77%; $p < 0.001$]).[13]

SARS-CoV-2 uses as its cell entry receptor angiotensin-converting enzyme type 2 (ACE-2), a molecule that is abundantly expressed on the surface of endothelial cells, kidneys, lungs, heart, and other organs. The use of medications such as angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) increases the expression of ACE-2 receptors in different tissues, including the lungs and heart. In spite of the hypothetical risk that the use of these medications could facilitate SARS-CoV-2 infection, there is evidence that patients with HF should continue using these medications, because the withdrawal of ACEI/ARB, beta-blockers, or aldosterone antagonists has been associated with a significant increase in in-hospital mortality.[5,12]

During the COVID-19 pandemic, there was a significant reduction in seeking health care for chronic diseases, which include some of the most important cardiovascular risk factors: hypertension, diabetes, and dyslipidemia. Dale et al. analyzed data from the United Kingdom during a 3-month period of the pandemic and identified a significant reduction in the dispensing of medications for these comorbidities.[14] It is difficult to estimate the direct impact of the reduction in care for these diseases and the increase in the incidence of cardiovascular complications, but they certainly potentiated the effects of the direct action of SARS-CoV-2 infection.

A retrospective cohort compared the number of hospital admissions due to HF, the average length of hospital stay, and the 30-day readmission rate before and during the COVID-19 pandemic. During the period evaluated, there was a reduction in the number of hospitalizations (774 versus

Table 1 – Effects of chronic HF as a comorbidity on the outcomes of COVID-19

Author	Number of participants	Main results
Alvarez-Garcia et al.[12]	6,439	• Patients with chronic HF and COVID-19 had longer hospital stays, increased risk of MV, and greater mortality. • Regardless of LVEF or use of ACEI/ARB, there was no difference in these outcomes.
Rey et al.[5]	3,080	• There was a higher incidence of acute HF during COVID-19 infection in patients with prior history of HF (11.2% versus 2.1%; p < 0.001). This increased incidence of acute HF was accompanied by higher NT-proBNP levels and higher mortality rates (48.7% versus 19.0%; p < 0.001). • Patients without prior history of HF who developed the acute form also had higher mortality compared to patients who did not develop this complication (46.8% versus 19.7%; p < 0.001). • The withdrawal of ACEI/ARB, beta-blockers, and aldosterone antagonists was associated with a significant increase in in-hospital mortality.
Yonas et al.[13]	21,640	• HF was associated with increased hospital and ICU admission rates, need for MV, and unfavorable outcomes (severe forms of COVID-19 and death). • Prior history of HF was associated with greater mortality.
Pimentel et al.[6]	241	• Atrial tachyarrhythmias were the most frequent. • Length of hospital stay was longer in those who presented cardiac arrhythmias. • The occurrence of cardiac arrhythmias was more frequent in men, patients on MV, and those with a history of HF.

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; COVID-19: coronavirus disease 2019; HF: heart failure; ICU: intensive care unit; LVEF: left ventricular ejection fraction; MV: mechanical ventilation; NT-proBNP: N-terminal pro-B-type natriuretic peptide.

864; p < 0.001), while the mean length of stay (6.05 versus 5.25 days; p < 0.001) and the readmission rate increased (20.6% versus 19.1%; p < 0.001).[15]

Effects of COVID-19 on exacerbation of pre-existing diseases correlated with chronic HF

COVID-19 symptoms can mimic HF decompensation. The 2021 update of the Brazilian Heart Failure Guideline recommends testing for SARS-CoV-2 by means of RT-PCR in both emergency room and outpatient settings.[16]

Some symptoms resulting from COVID-19 can persist for a long time after recovery from the acute infection, a condition known as long COVID. Huang et al. followed patients discharged after hospitalization for COVID-19 for 3 months. The team found that approximately 76% of cases persisted with at least one of the symptoms of fatigue, tiredness, myalgia, arthralgia, and insomnia.[17] Salah et al. found an even higher percentage, with 87% of patients persisting with complaints of dyspnea and fatigue.[18]

There is evidence that COVID-19 increases the incidence of cardiovascular complications in the first 12 months after acute infection. A prospective study demonstrated that the presence of myocardial injury was associated with the persistence of symptoms over the 12 months following acute infection, as well as increased rates of hospital readmission and mortality.[19] Xie et al. identified a significant increase in cerebrovascular, arrhythmic, thromboembolic, and ischemic events, in addition to inflammatory heart diseases.[20] Individuals who have recovered from COVID-19 have a 90% additional risk of developing HF within the first 9 months after acute infection. This risk is directly influenced by age and history of systemic arterial hypertension, and it is higher in the initial period of recovery from the acute infection phase. However, this risk is not restricted only to this initial phase, and it is necessary to consider individuals who have recovered from infection as a risk group for future development of HF.[1] The pathophysiological mechanism by which COVID-19 survivors are at an increased risk of HF has not yet been fully established. It is still unclear to what extent these complications are secondary to unique pathological processes, rather than direct consequences of acute infectious complications. Direct viral invasion of cardiomyocytes and subsequent cell death or infection of endothelial cells followed by complement-mediated dysfunction, coagulopathy, and microangiopathy may represent some of the possible triggers.[1]

The risk of developing HF, particularly with preserved ejection fraction, may be related to prior presence of subclinical HF with preserved ejection fraction with consequent progression to a symptomatic stage, but it may also be the result of the myocardial inflammatory and microvascular ischemic process. This inflammatory process caused by SARS-CoV-2 may continue after recovery from viral infection, with persistent tissue infiltration of inflammatory cells and, in some cases, development of areas of fibrosis. Survivors of the COVID-19 pandemic may represent a population at risk for the future development of complications involving LV diastolic dysfunction, pulmonary hypertension, and RV dysfunction, especially those recovered from severe forms with severe hypoxemia and thrombotic complications.[2]

Few studies have evaluated the immediate and long-term risk in patients with HF who developed COVID-19. Most registries limited participant inclusion to patients hospitalized with COVID-19, did not stratify associated clinical events, and developed a short follow-up period ending this phase before the end of the pandemic. There are reports of a substantial increase in mortality rates during the year following COVID-19 infection, with rates ranging from 1.3% to 12%.[17,21,22] These events are often attributed to sudden cardiovascular death due to likely arrhythmic or thrombotic mechanisms.

Bhatt et al.[23] conducted a prospective, multicenter, double-blind study with participants from 20 countries, titled DELIVER. The study included individuals with HF who had preserved or mildly reduced ejection fraction to evaluate the safety and efficacy of daily use of dapagliflozin 10 mg versus placebo. Approximately 9.4% of participants developed COVID-19 during the study period, with almost half of them requiring hospitalization for a prolonged period and 20% having a fatal outcome. Participants diagnosed with COVID-19 were found to have higher all-cause mortality after infection. This increased mortality remained high even 3 to 6 months after diagnosis. The study excluded immediately fatal COVID-19 events and adjusted for covariates. Dapagliflozin demonstrated benefits in the treatment of HF, even during treatment for COVID-19.[23]

In a retrospective cohort including more than 90,000 individuals, Greene et al.[24] evaluated the impact of COVID-19 on patients with HF with reduced ejection fraction (HFrEF), in both hospital and outpatient settings. The main outcomes assessed were all-cause mortality at 30 and 90 days and the composite outcome of hospitalization and mortality. In these patients with HFrEF, a positive COVID-19 test was identified as an independent factor for increased risk of death and worsening HF in outpatients, when compared to patients with HFrEF and a negative COVID-19 test. In individuals with clinical criteria for HF stability at the time of the positive COVID-19 test, 33% had an unfavorable outcome (hospitalization or death) within 5 months of follow-up.[24]

Table 2 summarizes the main studies that have evaluated the incidence of HF and cardiovascular complications after COVID-19 infection.

Conclusions

HF is associated with increased mortality, length of hospital stay, and incidence of complications in patients who are affected by SARS-CoV-2. Individuals with chronic HF show a higher rate of exacerbation (acute HF) when they have COVID-19. There is likely an association between COVID-19 and HF through a shared pathophysiological mechanism centered on inflammation.

Even after recovery from acute COVID-19 infection, there is an increased incidence of complications such as arrhythmias, HF (especially HF with preserved ejection fraction), coronary ischemic events, arterial hypertension, diabetes mellitus, and myocardial fibrosis. There are reports of a substantial increase in the mortality rate in the year following COVID-19 infection, often attributed to sudden cardiovascular death due to probable arrhythmic or thrombotic mechanisms.

Few studies have evaluated the effects of COVID-19 on patients with chronic HF after recovery from acute infection, and they are limited to short-term follow-ups and post-hoc studies. Further studies are needed to expand knowledge of the long-term cardiovascular impact caused by the pandemic.

Author Contributions

Conception and design of the research, Analysis and interpretation of the data and Critical revision of the manuscript for content: Magalhães LFS, Villacorta H; Acquisition of data: Magalhães LFS, Sant'Anna GC, Figueira JCABA; Writing of the manuscript: Magalhães LFS, Sant'Anna GC, Figueira JCABA, Villacorta H.

Table 2 – Effects of COVID-19 on the incidence of HF and on the outcomes of patients with chronic HF

Author	Number of participants	Main results
Son et al.[25]	2,152	• Myocardial injury caused by COVID-19 has a more severe clinical course in patients with CAD and HF (worse prognosis). • There was no significant difference in relation to complications in patients who used ACEI/ARB and developed COVID-19.
Yonas et al.[13]	21,640	• There was an increase in the incidence of acute HF in patients without prior history.
Zuin et al.[1]	21,463.173	• Individuals who recovered from COVID-19 had a 90% additional risk of developing HF during the first 9 months after acute infection. This risk was directly influenced by age and history of systemic arterial hypertension.
Babapoor-Farrokhran et al.[15]	1,638	• A retrospective cohort study was conducted comparing the number of hospital admissions due to HF, the mean length of stay, and the 30-day readmission rate before and during the COVID-19 pandemic. During the period evaluated, there was a reduction in the number of hospitalizations (774 versus 864; $p < 0.001$), while the mean length of hospital stay (6.05 versus 5.25 days; $p < 0.001$) and the readmission rate increased (20.6% versus 19.1%; $p < 0.001$).
Bhatt et al.[23]	6,263	• Approximately 9.4% of participants developed COVID-19 during the study period, with almost half of them requiring prolonged hospitalization and 20% having a fatal outcome. • Participants diagnosed with COVID-19 had greater all-cause mortality after infection. • This increase in mortality remained elevated even 3 to 6 months after diagnosis.
Greene et al.[24]	90,052	• In patients with history of HFrEF, a positive test for COVID-19 was identified as an independent factor for increased risk of death and worsening HF in outpatients, when compared to patients with HFrEF and a negative COVID-19 test. In patients with clinical criteria for HF stability at the time of the positive COVID-19 test, 33% had an unfavorable outcome (hospitalization or death) within 5 months of follow-up.

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; CAD: coronary artery disease; COVID-19: coronavirus disease 2019; HF: heart failure; HFrEF: heart failure with reduced ejection fraction.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

References

1. Zuin M, Rigatelli G, Roncon L, Pasquetto G, Bilato C. Risk of Incident Heart Failure after COVID-19 Recovery: A Systematic Review and Meta-Analysis. Heart Fail Rev. 2023;28(4):859-64. doi: 10.1007/s10741-022-10292-0.
2. Freaney PM, Shah SJ, Khan SS. COVID-19 and Heart Failure with Preserved Ejection Fraction. JAMA. 2020;324(15):1499-1500. doi: 10.1001/jama.2020.17445.
3. Cestari VRF, Garces TS, Sousa GJB, Maranhão TA, Souza JD Neto, Pereira MLD, et al. Spatial Distribution of Mortality for Heart Failure in Brazil, 1996 - 2017. Arq Bras Cardiol. 2022;118(1):41-51. doi: 10.36660/abc.20201325.
4. Varga Z, Flammer AJ, Steiger P, Haberecker M, Andermatt R, Zinkernagel AS, et al. Endothelial Cell Infection and Endotheliitis in COVID-19. Lancet. 2020;395(10234):1417-8. doi: 10.1016/S0140-6736(20)30937-5.
5. Rey JR, Caro-Codón J, Rosillo SO, Iniesta ÁM, Castrejón-Castrejón S, Marco-Clement I, et al. Heart Failure in COVID-19 Patients: Prevalence, Incidence and Prognostic Implications. Eur J Heart Fail. 2020;22(12):2205-15. doi: 10.1002/ejhf.1990.
6. Pimentel M, Magalhães APA, Novak CV, May BM, Rosa LGBD, Zimerman LI. Cardiac Arrhythmias in Patients with COVID-19. Arq Bras Cardiol. 2021;117(5):1010-5. doi: 10.36660/abc.20200963.
7. Italia L, Tomasoni D, Bisegna S, Pancaldi E, Stretti L, Adamo M, et al. COVID-19 and Heart Failure: From Epidemiology During the Pandemic to Myocardial Injury, Myocarditis, and Heart Failure Sequelae. Front Cardiovasc Med. 2021;8:713560. doi: 10.3389/fcvm.2021.713560.
8. Guervilly C, Burtey S, Sabatier F, Cauchois R, Lano G, Abdili E, et al. Circulating Endothelial Cells as a Marker of Endothelial Injury in Severe COVID -19. J Infect Dis. 2020;222(11):1789-93. doi: 10.1093/infdis/jiaa528.
9. Szekely Y, Lichter Y, Taieb P, Banai A, Hochstadt A, Merdler I, et al. Spectrum of Cardiac Manifestations in COVID-19: A Systematic Echocardiographic Study. Circulation. 2020;142(4):342-53. doi: 10.1161/CIRCULATIONAHA.120.047971.
10. Argulian E, Sud K, Vogel B, Bohra C, Garg VP, Talebi S, et al. Right Ventricular Dilation in Hospitalized Patients with COVID-19 Infection. JACC Cardiovasc Imaging. 2020;13(11):2459-61. doi: 10.1016/j.jcmg.2020.05.010.
11. Albuquerque DC, Silva PGMB, Lopes RD, Hoffmann-Filho CR, Nogueira PR, Reis H, et al. In-Hospital Management and Long-term Clinical Outcomes and Adherence in Patients with Acute Decompensated Heart Failure: Primary Results of the First Brazilian Registry of Heart Failure (BREATHE). J Card Fail. 2024;30(5):639-50. doi: 10.1016/j.cardfail.2023.08.014.
12. Alvarez-Garcia J, Lee S, Gupta A, Cagliostro M, Joshi AA, Rivas-Lasarte M, et al. Prognostic Impact of Prior Heart Failure in Patients Hospitalized With COVID-19. J Am Coll Cardiol. 2020;76(20):2334-48. doi: 10.1016/j.jacc.2020.09.549.
13. Yonas E, Alwi I, Pranata R, Huang I, Lim MA, Gutierrez EJ, et al. Effect of Heart Failure on the Outcome of COVID-19 - A Meta Analysis and Systematic Review. Am J Emerg Med. 2021;46:204-11. doi: 10.1016/j.ajem.2020.07.009.
14. Dale CE, Takhar R, Carragher R, Katsoulis M, Torabi F, Duffield S, et al. The Impact of the COVID-19 Pandemic on Cardiovascular Disease Prevention and Management. Nat Med. 2023;29(1):219-25. doi: 10.1038/s41591-022-02158-7.
15. Babapoor-Farrokhran S, Alzubi J, Port Z, Sooknanan N, Ammari Z, Al-Sarie M, et al. Impact of COVID-19 on Heart Failure Hospitalizations. SN Compr Clin Med. 2021;3(10):2088-92. doi: 10.1007/s42399-021-01005-z.
16. Marcondes-Braga FG, Moura LAZ, Issa VS, Vieira JL, Rohde LE, Simões MV, et al. Emerging Topics Update of the Brazilian Heart Failure Guideline - 2021. Arq Bras Cardiol. 2021;116(6):1174-212. doi: 10.36660/abc.20210367.
17. Huang C, Huang L, Wang Y, Li X, Ren L, Gu X, et al. 6-month Consequences of COVID-19 in Patients Discharged from Hospital: A Cohort Study. Lancet. 2021;397(10270):220-32. doi: 10.1016/S0140-6736(20)32656-8.
18. Salah HM, Fudim M, O'Neil ST, Manna A, Chute CG, Caughey MC. Post-recovery COVID-19 and Incident Heart Failure in the National COVID Cohort Collaborative (N3C) Study. Nat Commun. 2022;13(1):4117. doi: 10.1038/s41467-022-31834-y.
19. Weber B, Siddiqi H, Zhou G, Vieira J, Kim A, Rutherford H, et al. Relationship between Myocardial Injury During Index Hospitalization for SARS-CoV-2 Infection and Longer-Term Outcomes. J Am Heart Assoc. 2022;11(1):e022010. doi: 10.1161/JAHA.121.022010.
20. Wang W, Wang CY, Wang SI, Wei JC. Long-term Cardiovascular Outcomes in COVID-19 Survivors among Non-vaccinated Population: A Retrospective Cohort Study from the TriNetX US Collaborative Networks. EClinicalMedicine. 2022;53:101619. doi: 10.1016/j.eclinm.2022.101619. E.
21. Ayoubkhani D, Khunti K, Nafilyan V, Maddox T, Humberstone B, Diamond I, et al. Post-covid Syndrome in Individuals Admitted To Hospital with Covid-19: Retrospective Cohort Study. BMJ. 2021;372:n693. doi: 10.1136/bmj.n693.
22. Maestre-Muñiz MM, Arias Á, Mata-Vázquez E, Martín-Toledano M, López-Larramona G, Ruiz-Chicote AM, et al. Long-Term Outcomes of Patients with Coronavirus Disease 2019 at One Year after Hospital Discharge. J Clin Med. 2021;10(13):2945. doi: 10.3390/jcm10132945.
23. Bhatt AS, Kosiborod MN, Claggett BL, Miao ZM, Vaduganathan M, Lam CSP, et al. Impact of COVID-19 in Patients with Heart Failure with Mildly Reduced or Preserved Ejection Fraction Enrolled in the DELIVER trial. Eur J Heart Fail. 2023;25(12):2177-88. doi: 10.1002/ejhf.3043.
24. Greene SJ, Lautsch D, Yang L, Tan XI, Brady JE. Prognostic Interplay between COVID-19 and Heart Failure with Reduced Ejection Fraction. J Card Fail. 2022;28(8):1287-97. doi: 10.1016/j.cardfail.2022.05.001.
25. Son M, Jo YS, Jo S, Park K. Impact of COVID-19 on heart Failure Patients in South Kprea: A Nationwide Population-Based Study. Int Heart J 2021; 62:1083-1090.