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Prevalence of Heart Failure Symptoms in COVID-19 Survivors: A Cross-Sectional Study at IMSS General Hospital No. 20, Puebla, Mexico

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Abstract
Background: The COVID-19 pandemic has left a wide range of long-term sequelae, particularly affecting the cardiovascular system. Heart failure symptoms have been reported among survivors, yet their prevalence remains poorly documented in Mexico.
Objective: To determine the prevalence of heart failure symptoms in COVID-19 survivors.
Methods: An observational, descriptive, retrospective, cross-sectional, single-center study was conducted at IMSS General Hospital No. 20, Puebla, Mexico. Clinical records of patients with confirmed SARS-CoV-2 infection between January 2020 and July 2024 were reviewed. Heart failure symptoms were identified according to the Framingham criteria. Sociodemographic and clinical data were analyzed using descriptive statistics with SPSS v30.
Results: Ninety-three patients were included (median age 64 years; range 19–99). Of these, 17 patients (18.3%) presented symptoms consistent with heart failure. The most frequent symptoms were exercise intolerance (94.1%), orthopnea (88.2%), nocturnal cough (82.4%), and bendopnea (76.5%). Heart failure symptoms were observed across all levels of COVID-19 severity, with a higher frequency in patients with severe infection (47.1%). Most affected patients were older adults (median 80 years) and predominantly male (64.7%). According to the NYHA classification, 70.6% of symptomatic patients had mild functional limitation (Class I–II), while 29.4% presented marked or severe limitation (Class III–IV).
Conclusion: The study demonstrates a higher prevalence of heart failure symptoms among COVID-19 survivors in this Mexican population compared to international reports. These findings highlight the need for long-term cardiovascular follow-up and preventive strategies in post-COVID care.

Keywords: COVID-19 survivors; Heart failure symptoms; Prevalence; Framingham criteria; Post-COVID syndrome; NYHA classification

Introduction

Since the first case of coronavirus disease 2019 (COVID-19) was identified in December 2019, the infection caused by SARS-CoV-2 has spread rapidly worldwide, reaching an estimated 779 million confirmed cases worldwide and over 193 million in the Americas by the end of September 2025 [1, 2]. In Mexico alone, official records documented over 7.7 million confirmed cases by mid-2025 [2], with over 226,000 reported in the state of Puebla. Although the World Health Organization announced the end of the public health emergency in May 2023 [3], the consequences of the pandemic did not cease with the resolution of acute infection. On the contrary, an increasing number of survivors are presenting with persistent sequelae that compromise different organ systems, raising concerns about the true burden of COVID-19 in the medium and long term [4, 5].

Cardiovascular complications are among the most relevant post-COVID manifestations, given their strong association with morbidity, mortality, and diminished quality of life [6]. COVID-19 has been linked to a spectrum of cardiovascular outcomes, including arrhythmias, myocarditis, thromboembolic events, myocardial infarction, and ultimately heart failure [7]. Heart failure is not a single disease but a clinical syndrome characterized by typical symptoms—such as dyspnea, orthopnea, and fatigue—along with signs or objective evidence of cardiac dysfunction [7]. From a hemodynamic perspective, it represents the inability of the heart to pump sufficient blood to meet the body's demands or the need to do so only at the cost of elevated intracardiac filling pressures. Because of its chronic course, frequent hospitalizations, and poor prognosis, heart failure is a condition of special concern when evaluating long-term COVID-19 complications.

The mechanisms by which COVID-19 can contribute to the development or exacerbation of heart failure are multifactorial. SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2) receptors, which are expressed in cardiomyocytes and vascular endothelial cells, providing a direct pathway for viral entry into cardiac tissue [8]. Histopathological analyses have revealed viral localization in the myocardium of patients with cardiogenic shock associated with COVID-19, confirming the potential for direct cytopathic effects [9]. Moreover, COVID-19 triggers a systemic inflammatory response characterized by cytokine storm, with elevated levels of interleukin-6, ferritin, and D-dimer, which further contribute to myocardial injury and ventricular dysfunction [10, 11]. In addition, hypoxia secondary to severe pneumonia, endothelial injury with microvascular thrombosis, and the imbalance between increased metabolic demand and reduced oxygen supply generate a vicious cycle that predisposes to cardiac decompensation [12, 13]. These complex processes, acting in concert, explain why even patients without preexisting cardiovascular disease may develop new-onset heart failure after recovering from COVID-19.

The clinical significance of this association has been explored in several studies across different countries. In a systematic review of 220 studies including more than 29,000 patients, Pellicori *et al.* reported a pooled prevalence of heart failure of 6.8% among individuals with COVID-19, though the distinction between prevalent and incident cases was not always clear [14]. Similarly, Rey *et al.* evaluated 3,080 patients and found that 2.5% developed new-onset heart failure following COVID-19, with a median age of 61 years and a predominance of male patients [15]. However, the diagnosis in that study relied largely on imaging and biomarkers, rather than on the systematic identification of specific clinical symptoms. Giustino *et al.* reported that between one-third and one-half of hospitalized patients showed evidence of myocardial injury, which in many cases evolved into heart failure, although diagnosis was again based primarily on elevated troponin levels [16]. Lala *et al.* also described a high prevalence of cardiovascular complications in 2,736 patients hospitalized in New York, with myocardial injury detected in nearly one-third of cases, underscoring the vulnerability of this population [17]. These findings illustrate the variability in reported prevalence, which depends on study design, diagnostic criteria, and population characteristics.

Beyond the pathophysiological and diagnostic aspects, the epidemiological relevance of heart failure after COVID-19 also lies in its sociodemographic implications. In many countries, older adults represent the majority of those affected, and advanced age itself is a major risk factor for both severe COVID-19 and heart failure [14, 15]. Male sex has also been consistently associated with higher rates of cardiovascular complications, possibly due to hormonal influences, genetic predisposition, or a greater burden of comorbidities such as hypertension and diabetes [16, 17]. Furthermore, lower educational attainment and limited social support networks may reduce adherence to medical follow-up and delay access to healthcare, exacerbating outcomes in vulnerable groups. These social determinants of health are especially relevant in Latin American contexts, where disparities in healthcare access are frequent and may amplify the long-term consequences of COVID-19.

Despite the abundance of international research, most studies have been conducted in hospitalized populations in high-income countries, and their conclusions may not be generalizable to community settings or to health systems with different characteristics. Evidence from Latin America is particularly scarce, and Mexico lacks data on the prevalence of heart failure symptoms in COVID-19 survivors [2]. Considering that the Instituto Mexicano del Seguro Social (IMSS) provides healthcare coverage to more than half of the national population, understanding the burden of post-COVID cardiovascular sequelae in this system is crucial for designing effective prevention and follow-up strategies.

In this context, the present study was designed to determine the prevalence of heart failure symptoms among survivors of COVID-19 treated at IMSS General Hospital No. 20. By systematically evaluating clinical records according to established criteria, this study aims to provide local evidence that contributes to the global understanding of COVID-19 cardiovascular sequelae and supports the development of strategies for long-term care in Mexican patients.

Materials and Methods

Study Design

This was an observational, descriptive, cross-sectional, and single-center study. The design was chosen because it allows for the estimation of the prevalence of symptoms compatible with heart failure in a well-defined population of COVID-19 survivors at a specific point in time [18, 19]. By using a cross-sectional framework, the study ensured feasibility within the clinical setting and provided an adequate snapshot of post-COVID cardiac sequelae in the selected population.

Study Area

The study was carried out at the Hospital General de Zona No. 20 "La Margarita," Instituto Mexicano del Seguro Social (IMSS), located in Puebla, Mexico. This hospital is an institution that provides specialized care to a large number of patients in the region and has been a referral center during the COVID-19 pandemic. The healthcare setting offered access to a diverse patient base recovering from SARS-CoV-2 infection, thereby ensuring that the study sample adequately reflected the population commonly affected by long-term cardiovascular complications associated with the disease [20].

Population and Sample

The population consisted of patients with a confirmed diagnosis of COVID-19 infection, treated at General Hospital Zone No. 20. From this population, a final sample of 93 individuals was included, determined using a stratified probabilistic sampling method to ensure representativeness of the broader patient base. The sample size was calculated with a 95% confidence interval and a margin of error of 5%, to ensure statistical reliability [21]. This rigorous process helped to minimize sampling bias and provided a robust dataset for the analysis of post-COVID heart failure symptoms.

Inclusion and Exclusion Criteria

Patients were included if they were over 18 years of age, had a confirmed diagnosis of COVID-19 through PCR testing, and were in follow-up at the hospital after the acute phase of the infection.

Exclusion criteria included patients with incomplete medical records and those diagnosed with chronic heart failure prior to COVID-19 infection. These criteria were applied to ensure that the symptoms recorded were associated with the post-COVID condition and not confounded by prior chronic cardiological diagnoses [22].

Data Collection and Variables

Information was collected through systematic review of medical records. The principal tool was a standardized data collection sheet specifically designed for this study, which guided the extraction of relevant variables and guaranteed homogeneity across the dataset. The main variables included sociodemographic factors (age and sex) and symptoms associated with heart failure (Exercise intolerance, orthopnea, nocturnal cough, bendopnea, exertional dyspnea, paroxysmal nocturnal dyspnea, tachycardia, bilateral malleolar edema, and palpitations). For symptom definition, the Framingham criteria were adopted, as they represent an internationally recognized standard for the clinical diagnosis of heart failure [23]. This systematic approach minimized subjectivity and ensured comparability with other studies published in the field.

Statistical Analysis

All data were processed using the Statistical Package for the Social Sciences (SPSS), version 30.0. Descriptive statistics were employed to summarize the variables, using frequencies and percentages for categorical data and means with standard deviations for continuous variables. The prevalence of heart failure symptoms was calculated as a proportion of the total sample, and stratified analyses were performed according to sex, age group, and comorbidities. Confidence intervals at 95% were reported where appropriate, in order to strengthen the interpretability and external validity of the findings [24].

Ethical Considerations

The study adhered to the principles of the Declaration of Helsinki and national regulations for research involving human subjects. Approval was obtained from the Local Research and Ethics Committee of the Instituto Mexicano del Seguro Social (IMSS), with the registration number R-2024-2108-039. Ethical rigor was maintained throughout the process to ensure respect for the rights of participants and the integrity of the research, protecting confidentiality by anonymizing their personal data [25].

Results

This study evaluated the onset of heart failure-related symptoms in a cohort of patients with SARS-CoV-2 infection hospitalized at IMSS General Hospital No. 20 in Puebla, Mexico. Ninety-three patients aged 19 to 99 years were included, with a mean age of 64 years. Of these, 17 (equivalent to 18.3% of the total number of patients) presented symptoms of heart failure according to the New York Heart Association (NYHA) classification, while 76 patients (equivalent to 81.7%) did not report symptoms suggestive of this pathology, as shown in Tables 1 and 2.

Table 1: Table with representation of age with minimum, maximum and median of the total number of patients studied and of patients with symptoms of heart failure.

	Total	Minimum age	Maximum age	Median
Total number of patients	93 patients	19 years old	99 years	64 years old
Patients with symptoms suggestive of heart failure	17 patients	56 years old	99 years	80 years

Table 2: Representation of frequency and prevalence of patients who presented symptoms of heart failure among the total number of participants.

Prevalence of heart failure symptoms in patients with SARS COV-2 infection	
	Number of patients (Prevalence)
Present	17 (18.3)
Absent	76 (81.7)
Total	93 (100)

Regarding the gender distribution of the 93 patients, it was found that 51 patients (54.8%) were male, and 42 patients (45.2%) were female, a trend that continued in patients who presented with symptoms suggestive of heart failure, since 11 patients (64.7%) were male and 6 patients (35.3%) were female, as shown in Table 3.

Table 3: Gender representation of total patients and patients with heart failure symptoms by frequency and percentage.

	Total number of patients	Total number of patients with symptoms of heart failure
Males (%)	51 (54.8)	11 (64.7)
Female (%)	42 (45.2)	6 (35.3)
Total (%)	93 (100)	17 (100)

Severity of Sars-Cov-2 Infection

Of the total 93 patients studied, 54 patients (58.1%) had mild SARS COV-2 infection, followed by 27 patients (29%) who had moderate infection and 12 patients (12.9%) who had severe infection.

When these data were stratified according to the severity of the initial SARS-CoV-2 infection, a clear gradient emerged, since of the patients who presented data suggestive of heart failure, the SARS COV-2 infection they presented was classified as severe in 8 patients (47.1%), followed by moderate infection with 6 patients (35.3%) and 3 patients (17.6%) had an infection classified as mild, showing that the greater the severity of the infection, the more frequently the symptoms of heart failure occurred, as shown in Table 4.

Table 4: Representation of the severity of SARS-COV-2 infection according to its frequency and percentage of the total number of patients included in the study and of patients with symptoms of heart failure.

Severity of infection by SARS COV-2	Total number of patients	Total number of patients with symptoms of heart failure
	Frequency (%)	Frequency (%)
MILD	54 (58)	3 (17.6)
MODERATE	27 (29)	6 (35.3)
SERIOUS	12 (13)	8 (47.1)
TOTAL	93 (100)	17 (100)

Symptoms suggestive of heart failure

A closer look at the symptom profile among the 17 affected individuals revealed a heterogeneous but consistent picture. As shown in Table 5, exercise intolerance was the most prevalent manifestation, reported by 16 symptomatic patients (94.1%). Symptoms presented as follows: orthopnea in 15 patients (88.2%), nocturnal cough in 14 patients (82.4%), bendopnea in 13 patients (76.5%), dyspnea on exertion in 11 patients (64.7%), paroxysmal nocturnal dyspnea in 6 patients (35.3%), tachycardia in 3 patients (17.6%), bilateral ankle edema, and palpitations in 2 patients (11.8%), respectively.

Table 5: Representation of symptoms and percentage presented by patients among the total number of participants.

	Exercise intolerance	Orthopnea	Nighttime cough	Bend-pnea	Exertional dyspnea	Paroxysmal nocturnal dyspnea	Tachycardia	Bilateral malleolar edema	Palpitations
Present (%)	16 (94.1)	15 (88.2)	14 (82.4)	13 (76.5)	11 (64.7)	6 (35.3)	3 (17.6)	2 (11.8)	2 (11.8)
Absent (%)	1 (5.9)	2 (11.8)	3 (17.6)	4 (23.5)	6 (35.3)	11 (64.7)	14 (82.4)	15 (88.2)	15 (88.2)
Total	17	17	17	17	17	17	17	17	17

Taken together, these data reveal that, although the spectrum of manifestations is broad, a core group of exercise intolerance, orthopnea, nocturnal cough, and bendopnea predominate in the clinical presentation, similar to classic early heart failure syndromes.

Severity Of Heart Failure In Patients With Sars Cov-2 Infection

To further assess the functional impact of these symptoms, all symptomatic patients were classified according to the New York Heart Association (NYHA) functional scale.

Of the total number of patients who presented symptoms of heart failure (Table 6), 7 patients (41.2%) met the criteria to classify them as Class I (No limitation in their activities), followed by 5 patients (29.4%) with Class II (Slight limitation), 3 patients (17.6%) were classified as Class IV (Total inability to perform any physical activity without symptoms), and only 2 patients (11.8%) presented criteria for Class III (Marked limitation), which shows that of the total number of patients with symptoms of post-COVID heart failure, 70.6% present symptoms of heart failure without limitation or with a slight limitation in their daily activities, and only about a third (29.4%) presented a marked or total limitation in carrying out their activities.

Table 6: Representation of patients with frequency and percentage according to the severity of heart failure according to the NYHA classification.

Severity of heart failure in patients with SARS COV-2 infection	
NYHA classification of heart failure	Frequency (%)
CLASS I	7 (41.2)
CLASS II	5 (29.4)
CLASS III	2 (11.8)
CLASS IV	3 (17.6)
TOTAL	17 (100)

Discussion

The present study highlights an important and underexplored dimension of the COVID-19 pandemic: the long-term cardiovascular consequences manifesting as symptomatic heart failure.

Our findings show that nearly one-fifth of COVID-19 survivors evaluated at a hospital in Mexico presented with symptoms consistent with heart failure according to the Framingham criteria, a prevalence significantly higher than that reported in international literature. This discrepancy underscores the heterogeneous nature of post-COVID cardiovascular sequelae, influenced by demographic, clinical, and healthcare system characteristics that vary across populations. By focusing on a Mexican cohort, this research fills a critical gap in knowledge, providing evidence that may guide regional strategies for long-term care and prevention.

Several mechanisms may account for the high prevalence of symptoms observed. SARS-CoV-2 has demonstrated tropism for cardiomyocytes and vascular endothelial cells through the ACE2 receptor, facilitating direct viral injury [8]. Histopathological analyses confirm viral localization in the myocardium, reinforcing the plausibility of direct cytotoxic effects [9]. Beyond this, the systemic inflammatory response associated with COVID-19, characterized by cytokine storm, endothelial dysfunction, and microvascular thrombosis, creates an environment that predisposes to both systolic and diastolic dysfunction [10, 11]. Hypoxemia during severe pneumonia, coupled with increased metabolic demand, adds further stress to the myocardium, creating a vicious cycle that culminates in decompensation [12, 13]. These mechanisms explain the functional abnormalities observed in our population, and correlate with the abnormalities emphasized in international studies, including reduced ejection fraction, diastolic dysfunction and left atrial enlargement, as well as arrhythmias, mainly atrial fibrillation [16, 17].

The relationship between the severity of acute COVID-19 and subsequent cardiovascular sequelae is another dimension of interest. Although heart failure symptoms were documented across all levels of initial severity, their prevalence was clearly higher among patients with severe disease requiring hospitalization or oxygen support. This gradient supports the hypothesis that systemic inflammation and hypoxemia during the acute phase increase the risk of subsequent myocardial compromise. Nevertheless, the presence of symptomatic patients even among those with moderate disease indicates that post-COVID cardiovascular surveillance should not be limited exclusively to severe cases. Similar observations have been reported by Rey *et al.* and Giustino *et al.*, who identified cardiovascular dysfunction in patients without critical illness [15, 16]. This suggests that SARS-CoV-2 may act as a trigger for cardiac remodeling regardless of the initial severity of infection. Sociodemographic characteristics of the symptomatic group add another layer of interpretation. In our cohort, the median age of patients with symptoms of heart failure was 80 years, well above the mean ages reported in other international cohorts, which typically range between 60 and 65 years [14, 15], reinforcing advanced age as a well-established risk factor for heart failure, possibly due to reduced cardiac reserve and the accumulation of comorbidities.

Furthermore, the predominance of male patients (64.7%) aligns with prior research associating male sex with higher rates of cardiovascular complications, possibly due to hormonal differences, immune modulation, and a greater burden of cardiometabolic risk factors [16, 17]. These demographic patterns suggest that older men in particular may represent a priority group for post-COVID cardiovascular surveillance. Moreover, the Mexican context—characterized by high prevalence of diabetes, obesity, and hypertension—may exacerbate this vulnerability [5, 4, 6]. Such interactions create a syndemic effect in which COVID-19 amplifies existing epidemics of chronic noncommunicable diseases [7].

The functional classification of patients according to the New York Heart Association (NYHA) further illustrates the clinical significance of our findings. Although most symptomatic individuals were classified as Class I–II, approximately one-third fell into Class III–IV, denoting marked or severe limitation of physical activity. These data emphasize that COVID-19 not only precipitates cardiovascular pathology but also translates into tangible disability that restricts independence and increases healthcare demands. While prior studies have focused primarily on biomarkers or imaging findings [14, 15], fewer have provided detailed accounts of functional limitation. Our study therefore contributes by showing that post-COVID heart failure is not merely a biochemical or imaging-based diagnosis but also a syndrome with profound repercussions on quality of life.

The integration of clinical findings reinforces the validity of the observed associations, which reflect patterns similar to those described in viral myocarditis and dilated cardiomyopathy. These parallels support the hypothesis that COVID-19 may act as a triggering factor for chronic structural heart disease [6]. Together, the results delineate a multifaceted syndrome involving structural, functional, electrical, and biochemical alterations of the myocardium. Importantly, even patients without traditional cardiovascular risk factors developed heart failure symptoms, underscoring the independent role of SARS-CoV-2 infection as a primary driver of cardiac dysfunction. However, the cumulative effect of comorbidities can magnify both their prevalence and severity, reinforcing the need for integrated management of chronic conditions in post-COVID care, an approach that resonates with contemporary heart failure guidance [7].

Our study also carries methodological strengths worth noting. The systematic use of the Framingham criteria ensured that the identification of heart failure symptoms was grounded in internationally recognized clinical standards [23]. This contrasts with many prior studies that relied predominantly on biomarkers or imaging, which, while valuable, may underestimate the prevalence of symptomatic disease. By adopting a clinical approach, our findings reflect the lived reality of patients and highlight the burden of recognizable symptoms that impair daily life. However, some limitations must also be acknowledged. First, the retrospective nature of medical record review may have introduced underreporting of symptoms or inconsistencies in documentation. Second, the cross-sectional design precludes causal inference, limiting our conclusions to prevalence rather than incidence or prognosis [18, 19]. Third, the lack of imaging or biochemical evaluations may underestimate the prevalence of subclinical abnormalities. Finally, being a single-center study, external generalizability is limited, particularly for rural or under-resourced healthcare settings in Mexico.

Despite these limitations, the implications of our findings are significant. Clinically, they call for structured follow-up programs for COVID-19 survivors, particularly targeting older adults and patients with comorbidities. Routine cardiovascular screening, along with imaging and biochemical studies (echocardiography and cardiac biomarkers), may be justified in high-risk groups. Rehabilitation programs focusing on functional recovery may mitigate the disability burden observed among patients in NYHA Classes III–IV. From a public health perspective, these findings highlight the urgency of designing integrated, long-term strategies that bridge infectious disease management and chronic disease care. In countries like Mexico, where cardiometabolic disease is highly prevalent, such approaches are indispensable to prevent a surge of post-COVID cardiovascular morbidity.

Conclusion

This study highlights a substantial burden of heart failure symptoms among COVID-19 survivors treated at IMSS General Hospital No. 20 in Puebla, Mexico. Nearly one in five patients developed conditions compatible with heart failure, with exercise intolerance, orthopnea, nocturnal cough, bendopnea, and exertional dyspnea being the most common complaints.

Sociodemographic characteristics—particularly advanced age and male predominance—underscore the existence of vulnerable subgroups requiring closer cardiovascular surveillance in the post-COVID setting. Importantly, the study also illustrates that cardiac sequelae can occur at all levels of severity from the initial infection, suggesting that even patients with mild disease remain at risk for long-term complications.

Compared with international reports, the prevalence observed in this Mexican cohort appears higher, pointing to possible regional factors such as the background burden of chronic noncommunicable diseases and disparities in access to healthcare. These findings emphasize the need for targeted follow-up strategies, multidisciplinary management, and preventive interventions designed to address both the direct effects of COVID-19 and its interaction with pre-existing comorbidities.

In summary, this research provides evidence that post-COVID heart failure is a relevant and growing concern within the Mexican population. Recognizing its prevalence and clinical spectrum is essential to inform healthcare planning, optimize resource allocation, and ultimately improve the quality of life and prognosis of survivors. Future studies should aim to validate these findings in larger, multicenter cohorts and to explore the mechanisms and therapeutic approaches that could mitigate the long-term cardiovascular impact of the pandemic.

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Conflict of Interest

Not available

Financial Support

Not available

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