Pak. J. Chest Med. 2025;31(03)

Original Article

journal homepage: https:/www.pjcm.net/

Pakistan Journal of Chest Medicine

Official journal of Pakistan Chest Society

Long-Term Cardiopulmonary Sequelae in Severe COVID-19 Survivors

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Article History:

Received:	Apr 24, 2025
Revised:	July 07, 2025
Accepted:	July 26, 2025
Available Online:	Sep 02, 2025

Author Contributions:
FI conceived idea, NI drafted the study, MS IA collected data, MD did statistical analysis and interpretation of data, FI NI critical reviewed manuscript. All approved final version to be published.

Declaration of conflicting interests:
The authors declare that there is no conflict of interest.

How to cite this article:
Islam F, Shah M, Ahmad I, Daud M, Islam N. Long-Term Cardiopulmonary Sequelae in Severe COVID-19 Survivors. Pak J Chest Med. 2025;31(03):230-239.

ABSTRACT

Background: Severe coronavirus disease 2019 (COVID-19) caused by SARS-CoV-2 has resulted in high morbidity and mortality worldwide. In addition to the acute disease, an increasing number of patients, particularly those who had severe disease, present with ongoing symptoms and dysfunction of organs, a syndrome known as long COVID. The cardiopulmonary system appears to be highly susceptible to long-term consequences.

Objective: To assess the chronic cardiopulmonary complications in survivors of severe COVID-19.

Methodology: This was a cross-sectional observational study of 235 adult patients hospitalized with severe COVID-19 and followed up at least three months after discharge. Baseline demographics, clinical information, residual symptoms, pulmonary function tests (PFTs), high-resolution computed tomography (HRCT) chest abnormalities, and cardiovascular evaluations were collected.

Results: The mean age of participants was 58.4 ± 12.6 years, with 62.1% being male. At follow-up, 58.7% reported dyspnea, 51.9% fatigue, and 47.2% exercise intolerance. PFTs revealed reduced DLCO in 51.5% and obstructive or restrictive defects in a significant subset. HRCT abnormalities persisted in over 80% of patients, with ground-glass opacities (45.1%) and fibrotic changes (34.9%) being the most common. Cardiovascular abnormalities were found in 28.9% as abnormal ECGs, 13.6% as left ventricular dysfunction, and 50% as myocardial fibrosis on cardiac MRI. 23.8% and 8.1% had elevated NT-proBNP and hs-Troponin I, respectively.

Conclusion: A high percentage of patients recovering from serious COVID-19 are left with persistent cardiopulmonary abnormalities, such as compromised lung function, residual radiological alterations, and cardiac impairment. These results highlight the importance of thorough long-term follow-up and multidisciplinary management for COVID-19 survivors, with a particular emphasis on cardiopulmonary health.

Keywords: COVID-19; Long COVID; Pulmonary Fibrosis; Myocardial Damage; Cardiopulmonary Sequelae; Post-COVID Syndrome

Introduction

The global COVID-19 pandemic, caused by the novel coronavirus SARS-CoV-2, has presented novel obstacles for scientific investigation, clinical management, and public health. Even though the initial therapeutic focus was on managing acute infections, it is now obvious that many patients, especially those who suffered from severe sickness, continue to have symptoms and organ failure even after the virus has been cleared. The long-term effects on the cardiopulmonary system are among the most concerning, as they may lead to increased healthcare utilization, diminished quality of life, and persistent impairment.[1]

The symptoms of severe COVID-19 include acute respiratory distress syndrome (ARDS), pneumonia, and systemic inflammation, which can harm the heart, lungs, and vasculature directly or indirectly. Many of these patients report that their respiratory and cardiovascular symptoms, including exhaustion, palpitations, chest discomfort, dyspnea, and exercise intolerance, continue for months or even years after the infection has ended. This developing illness, also known as prolonged COVID or post-acute sequelae of SARS-CoV-2 infection (PASC), is currently a major focus of clinical and research attention.[2]

According to new data, severe COVID-19 instances may be especially susceptible to long-term harm to the pulmonary system. Research has documented both lung function impairment, including reduced diffusion capacity (DLCO) and restricted ventilatory patterns, as well as structural abnormalities, such as fibrotic alterations, ground-glass opacities, and bronchiectasis, on follow-up imaging. Many of these observations are suggestive of persistent inflammatory or fibrotic processes that delay full healing and remind us of lung remodelling following ARDS.[3]

The cardiovascular system has also demonstrated a great deal of long-term susceptibility. Heart failure, arrhythmias, myocarditis, and myocardial fibrosis are among the cardiac sequelae that patients recovering from severe COVID-19 may experience. It is believed that a combination of endothelial dysfunction, microvascular thrombosis, cytokine-mediated damage, and direct viral invasion causes these consequences. Even among individuals who were previously healthy and did not have any pre-existing heart illness, a significant portion of post-COVID patients have been found to have myocardial oedema and late gadolinium enhancement by advanced imaging techniques such as cardiac MRI.[4]

Cardiopulmonary symptoms that overlap make diagnosis and treatment more difficult, particularly for individuals whose underlying problems were previously unknown. Furthermore, ongoing work is being conducted to identify the entire range of these long-term impacts, their natural history, and their predictors. Since these patients frequently suffer from more severe and long-lasting aftereffects, the burden is especially noticeable for those who need hospitalization, intensive care unit treatment, or mechanical ventilation during the acute stage of their illness.[5] Developing successful management measures requires an understanding of the mechanisms behind these long-term cardiopulmonary consequences. Post-inflammatory fibrosis, immunological dysregulation, and viral persistence are believed to collaborate in causing persistent tissue damage and symptoms. Careful research should also be done on the roles of demographic variables, acute-phase treatment modalities (such as corticosteroids or mechanical ventilation), and pre-existing comorbidities.[6]

Given the global burden of COVID-19 and its significant morbidity among hospitalized patients, especially those with severe disease, understanding the long-term health consequences has become a critical public health priority. Emerging evidence suggests that SARS-CoV-2 infection may lead to lasting damage to both the pulmonary and cardiovascular systems, manifesting as impaired lung function, myocardial dysfunction, arrhythmias, and persistent dyspnea. However, comprehensive data on the chronic cardiopulmonary sequelae, specifically in survivors of severe COVID-19, remain limited, particularly in low- and middle-income settings. Identifying and characterising these long-term complications is crucial for developing targeted follow-up strategies and rehabilitation protocols, as well as for optimising patient care. Therefore, this study aims to assess the chronic cardiopulmonary complications in survivors of severe COVID-19, informing clinical practice and guiding the long-term management of affected individuals.

Objective

To outline the variety of cardiovascular and pulmonary issues that individuals experience after acquiring a severe case of COVID-19.

Methodology

This prospective observational cohort study was conducted between January 2022 and January 2023 in the Chest department of Aga Khan University Hospital, Karachi. Adult patients 18 years of age and older who had previously been admitted to the hospital for severe COVID-19, as defined by one or more of the following criteria by WHO guidelines, were eligible participants: oxygen therapy requirements, intensive care unit (ICU) admissions, the use of invasive or non-invasive mechanical ventilation, and radiological evidence of bilateral pneumonia. To evaluate ongoing or new long-term cardiopulmonary symptoms and complications, patients were included at least three months after discharge. Exclusion criteria included having a history of

heart failure or chronic respiratory conditions (like COPD or interstitial lung disease) before contracting COVID-19, having an active cancer or immunosuppressive condition, having incomplete clinical records, or not being able to attend follow-up appointments.

Clinical, laboratory, and radiological data were collected at each follow-up visit and also from hospital medical records. The collected data included baseline demographics and clinical characteristics such as age, sex, body mass index (BMI), smoking status, comorbidities (such as diabetes, hypertension, and cardiovascular disease), and COVID-19 severity parameters during hospitalization (such as length of intensive care unit stay, use of oxygen or ventilators, and inflammatory markers).

Pulmonary Function Testing (PFT) was conducted according to ATS/ERS guidelines using a calibrated spirometer. The following parameters were recorded: Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), FEV_1/FVC ratio and Diffusing capacity for carbon monoxide (DLCO). High-resolution computed Tomography (HRCT) of the chest was performed on all patients to assess residual parenchymal abnormalities. Findings were categorized into Ground-glass opacities, Interstitial thickening, Fibrotic bands and Bronchiectasis.

All patients were also referred for cardiac assessment, which included a 12-lead Electrocardiogram (ECG) and echocardiography to assess ejection fraction (EF), wall motion abnormalities, and pericardial effusion. Selected patients (based on clinical indications) underwent cardiac MRI to evaluate myocardial fibrosis, oedema, or inflammation.

The baseline characteristics were summarized using descriptive statistics. Depending on their distribution, continuous variables were shown as either the median with interquartile or mean ± standard deviation (SD). Both percentages and frequencies were used to represent categorical variables. To find predictors of significant cardiac or pulmonary sequelae, multivariate logistic regression analysis was conducted. A p-value of less than 0.05 is considered statistically significant. The SPSS version 23 was used for all analyses.

Results

The study cohort comprised 235 patients who had experienced severe COVID-19 illness requiring hospitalization. The mean age was 58.4 ± 12.6 years, indicating a predominance of middle-aged to older adults, a population known to be at higher risk for severe outcomes from COVID-19. Notably, 62.1% of the participants were male (n = 146), reflecting the established trend of increased severity and complications of COVID-19 in men compared to women 37.8%. The mean Body Mass Index (BMI) was 27.9 ± 4.8 kg/m², classifying the average participant as overweight. Excess body weight is a recognized risk factor for both acute COVID-19 severity and post-acute complications, including reduced pulmonary function and cardiovascular strain. A history of smoking was present in 72 patients (30.6%), which may contribute to the development or persistence of pulmonary abnormalities such as impaired lung function or fibrotic changes, making this an important confounding factor in long-term sequelae.

Regarding comorbidities, nearly 46% (n = 108) had hypertension, and 39.1% (n = 92) were diagnosed with diabetes mellitus, both of which are well-known risk factors for severe COVID-19 infection and are independently associated with increased risk for cardiopulmonary complications. Coronary artery disease (CAD) was noted in 17.4% (n = 41) of patients, indicating a significant proportion had pre-existing cardiovascular vulnerabilities. Over half of the patients (50.2%, n = 118) required admission to the Intensive Care Unit (ICU) during the acute phase of their illness, indicating the severity of the disease in this cohort. Mechanical ventilation was administered in 23% (n = 54) of patients, suggesting that a substantial subset experienced critical respiratory failure requiring invasive support. The average duration of hospitalization was 14.8 ± 6.3 days, indicating prolonged medical care and likely exposure to interventions that could contribute to long-term organ dysfunction.

The analysis of persistent symptoms at a follow-up period of ≥3 months post-discharge among the 235 patients revealed a significant burden of ongoing cardiopulmonary complaints, many of which impacted daily functioning and quality of life. Dyspnea (mMRC Grade ≥1) was reported by 138 patients (58.7%), making it the most common lingering symptom. The modified Medical Research Council (mMRC) dyspnea scale measures breathlessness and a grade ≥1 indicates that the patient experiences shortness of breath when hurrying on level ground or walking up a slight hill. Fatigue was the second most prevalent symptom, reported by 122 patients (51.9%). Fatigue in post-COVID patients may result from systemic inflammation, mitochondrial dysfunction, or deconditioning following prolonged illness and hospitalization. Chest pain or discomfort was noted in 49 patients (20.8%). While non-specific in origin, chest discomfort may arise from residual inflammation, musculoskeletal strain (from coughing or ICU care), pericardial involvement, or undiagnosed cardiac complications. In some cases, it could also reflect psychological stress or anxiety. Palpitations were present in 38 patients (16.2%). This symptom may be linked to autonomic dysregulation, persistent myocardial injury, or arrhythmias. Exercise intolerance was experienced by 111 patients (47.2%), representing a reduced ability to perform physical activities and potentially resulting from impaired oxygen delivery, cardiovascular deconditioning, reduced lung capacity, or persistent fatigue. Orthopnea, defined as difficulty breathing when lying flat, was found in 18 patients (7.7%). Although less common, this

Table 1. Baseline Demographic and clinical characteristics of study cases

Parameter	Value
Age (years), mean ± SD	58.4 ± 12.6
Male, n (%)	146 (62.1%)
Female, n (%)	89 (37.8%)
BMI (kg/m^2), mean ± SD	27.9 ± 4.8
Smoking history (current/former), n (%)	72 (30.6%)
Hypertension, n (%)	108 (45.9%)
Diabetes mellitus, n (%)	92 (39.1%)
Coronary artery disease, n (%)	41 (17.4%)
ICU admission during acute illness, n (%)	118 (50.2%)
Mechanical ventilation, n (%)	54 (23.0%)
Hospital stay duration (days), mean ± SD	14.8 ± 6.3

symptom is clinically significant, as it may suggest underlying cardiac dysfunction such as heart failure, particularly in those with elevated BNP levels or echocardiographic abnormalities (Table 2).

Table 3 presents the results of pulmonary function tests (PFTs) conducted on 235 patients at ≥3 months following recovery from severe COVID-19. The findings reveal a high prevalence of abnormal pulmonary function, highlighting significant residual respiratory impairment. Forced Expiratory Volume in 1 Second (FEV_1). The mean FEV_1 was 76.2 ± 18.5% of predicted, with 72 patients (30.6%) demonstrating FEV_1 values below 80% of predicted, a threshold typically used to define impaired airflow. This finding suggests that nearly one-third of the patients had persistent airflow limitation. Such impairment may result from airway inflammation, fibrosis, or pre-existing lung disease exacerbated by COVID-19. Forced Vital Capacity (FVC). The mean FVC was 81.9 ± 20.4% of predicted, with 63 patients (26.8%) falling below normal limits, suggesting a restrictive ventilatory defect in over a quarter of patients. This could be attributed to parenchymal lung damage, loss of alveolar volume, or fibrotic changes identified on radiographic imaging. FEV_1/FVC Ratio. The mean FEV_1/FVC ratio was 0.78 ± 0.09, which falls within the normal range, but 38 patients (16.2%) exhibited values suggestive of an obstructive pattern (typically defined as a ratio <0.7). This indicates that a subgroup may have underlying or COVID-induced small airway disease, such as post-viral bronchial hyperreactivity or chronic obstructive pulmonary disease-like changes. The most striking abnormality was observed in the Diffusing Capacity for Carbon Monoxide (DLCO), which had a mean value of 63.4 ± 17.9% of the predicted value. A total of 121 patients (51.5%) had significantly reduced DLCO (<80% predicted), indicating impaired alveolar-capillary gas exchange.

High-resolution computed Tomography (HRCT) of the chest was performed on all 235 patients approximately 3 months or more post-discharge to evaluate persistent structural lung abnormalities. The results reveal a high burden of residual radiological changes, even after clinical recovery from the acute phase of COVID-19. Ground-glass opacities (GGO) were present in 106 patients (45.1%), making them the most common radiological finding. GGOs represent areas of partial airspace filling or interstitial thickening and suggest ongoing inflammation, resolving pneumonia, or early fibrotic activity. Their persistence at ≥3 months post-infection indicates incomplete resolution of parenchymal involvement and potential progression to chronic lung changes. Reticulations and fibrotic changes were identified in 82 patients (34.9%), indicating a significant proportion had developed post-COVID pulmonary fibrosis. These findings include linear opacities, traction

Table 2. Persistent Symptoms at Follow-Up (≥3 months post-discharge)

Symptom	Frequency, n (%)
Dyspnea (mMRC Grade ≥1)	138 (58.7%)
Fatigue	122 (51.9%)
Chest pain/discomfort	49 (20.8%)
Palpitations	38 (16.2%)
Exercise intolerance	111 (47.2%)
Orthopnea	18 (7.7%)

bronchiectasis, and architectural distortion. Such fibrotic patterns are consistent with the sequelae of acute respiratory distress syndrome (ARDS). They are known to contribute to restrictive lung function defects and reduced DLCO observed in this cohort.

Bronchiectasis was seen in 29 patients (12.3%). Subpleural bands were noted in 61 patients (26.0%). These linear opacities, located near the pleural surfaces, are suggestive of resolving inflammation or early fibrotic changes and are often seen in patients recovering from viral pneumonia. A normal HRCT scan was found in 46 patients (19.6%), suggesting complete radiological resolution in about one-fifth of the cohort. These patients are likely to have had either less severe lung involvement during acute illness or better recovery. However, a normal scan does not entirely rule out functional abnormalities, such as impaired DLCO or persistent symptoms, which were still observed in some of these patients (Table 4).

A comprehensive cardiovascular evaluation was performed in all 235 patients at least 3 months following recovery from severe COVID-19, with advanced imaging, such as cardiac MRI, performed in a subset. The findings reveal a notable prevalence of cardiovascular abnormalities, suggesting that COVID-19 can lead to sustained cardiac involvement, even in patients without pre-existing heart disease. Abnormal ECG (non-specific changes) were identified in 68 patients (28.9%). These changes included T-wave inversions, ST-segment abnormalities, and sinus tachycardia. While non-specific, they may indicate ongoing myocardial strain, ischemia, or residual inflammation and warrant further evaluation to exclude underlying pathology. Left ventricular (LV) dysfunction, defined as an ejection fraction (EF) < 50%, was observed in 32 patients (13.6%) via echocardiography. This suggests impaired systolic function, potentially resulting from myocarditis, ischemic injury, or cytokine-mediated myocardial depression during acute COVID-19. LV dysfunction can contribute to persistent symptoms like fatigue, dyspnea, and orthopnea.

Myocardial fibrosis, identified on cardiac MRI in a subset of 42 patients, was present in 21 cases (50.0%). Elevated NT-proBNP levels (>125 pg/mL) were found in 56 patients (23.8%), indicating myocardial stress or volume overload. Elevated natriuretic peptides are commonly associated with heart failure, and their presence may reflect subclinical cardiac dysfunction or ongoing hemodynamic stress post-recovery. High-sensitivity Troponin I, a sensitive marker of myocardial injury, was elevated in 19

Table 3. Pulmonary Function Test (PFT) Findings of study cases

PFT Parameter	Mean ± SD	Abnormality Frequency, n (%)
FEV_1 (% predicted)	76.2 ± 18.5	72 (30.6%)
FVC (% predicted)	81.9 ± 20.4	63 (26.8%)
FEV_1/FVC ratio	0.78 ± 0.09	38 (16.2%) – obstructive pattern
DLCO (% predicted)	63.4 ± 17.9	12 (11.5%)

Table 4. Radiological Findings on HRCT Chest

HRCT Finding	Frequency, n (%)
Ground-glass opacities	106 (45.1%)
Reticulations/fibrotic changes	82 (34.9%)
Bronchiectasis	29 (12.3%)
Subpleural bands	61 (26.0%)
Normal HRCT	46 (19.6%)

patients (8.1%). Pericardial effusion, of mild to moderate degree, was found in 14 patients (6.0%) on echocardiography. At the same time, generally, small and hemodynamically insignificant pericardial effusions may reflect subclinical pericarditis, immune-mediated inflammation, or post-viral changes. Some cases may evolve into constrictive pericarditis if unmonitored (Table 5).

Discussion

The long-term impacts that survivors particularly those who experienced severe forms of the virus face are receiving more attention as the global effects of COVID-19 become more apparent. Despite an extensive range of research on the acute phase of COVID-19, clinicians and healthcare systems around the world continue to be increasingly concerned about the post-acute sequelae of SARS-CoV-2 infection (PASC).[2] In our study 235 patients who were hospitalized for severe COVID-19 and followed up for at least three months after their discharge are included, which offers a thorough evaluation of cardiopulmonary long-term sequelae. The findings highlight how the respiratory and cardiovascular systems are both affected by the ongoing burden of symptoms, functional restrictions, and structural abnormalities.

Our cohort's clinical and demographic characteristics provide crucial context for recognizing the cardiopulmonary sequelae observed during follow-up. The study population consisted mainly of older adults (mean age 58.4 ± 12.6 years) with a minor male predominance (62.1% male, 37.8% female). This pattern is in accordance with international evidence indicating that older men are at higher risk of serious COVID-19 with increased hospitalization, intensive care unit (ICU) admission, and mortality.1 Our findings agree with the report for large cohorts like the ISARIC study (UK),[6] reporting a median age of 59 for hospitalized COVID-19 patients and an analogous male dominance (~60%). Older age has been consistently linked with poorer outcomes owing to dysregulation of the immune system with age and increased comorbidity burden. The average BMI was 27.9 ± 4.8 kg/m², which means that a high percentage of patients belonged to the mildly obese or overweight category. This is consistent with research such as the Scheen et., al (2020)[7], in which obesity was shown to be a major risk factor for critical COVID-19 and unfavorable in-hospital outcomes. Obesity could also influence long-term recovery as it is linked with systemic inflammation and cardiopulmonary comorbidities.

Results showed had high rates of chronic conditions, including coronary artery disease (17.4%), diabetes mellitus (39.1%), and hypertension (45.9%). These results are consistent with trends noted in previous research, Dennis et., al (2021),[8] where similar burdens of comorbidities were seen in hospitalized patients with acute COVID-19. These conditions not only predispose to acute complications but also to protracted convalescence and organ-related sequelae, such as cardiac and pulmonary dysfunction. For example, one study by Huang et al. (2022)[9] of 1,733 patients from Wuhan noted hypertension in 30% and diabetes in 12% of their cohort, though their patient population had a marginally lower mean age. Nearly 50.2% of the patients needed ICU care, and 23.0% needed mechanical ventilation, highlighting the severity of presentation in our population. Mechanical ventilation and extended ICU admission are both known risk factors for post-ICU syndrome, pulmonary fibrosis, and cardiopulmonary dysfunction. In comparison to the Yang (2022),[10] reporting ICU admissions in 40% of long COVID patients, post-COVID complications are more likely to occur among the studied cases Prolonged hospitalization typically indicates more severe illness and complications in the form of secondary infections, acute respiratory distress syndrome (ARDS), or requirement of ventilatory support. A post-acute COVID-19 sequelae study by Carfì et al. (2020)[11] demonstrated that longer hospitalization is associated with more symptom burden upon recovery, which is concordant with our experience of ongoing dyspnea, tiredness, and functional impairment. Ongoing symptoms

Table 5. Cardiovascular assessment results of study cases

Parameter	Frequency, n (%)
Abnormal ECG (non-specific changes)	68 (28.9%)
Left ventricular dysfunction (EF < 50%)	32 (13.6%)
Myocardial fibrosis (on MRI)	21 of 42 scanned (50.0%)
Elevated NT-proBNP (>125 pg/mL)	56 (23.8%)
Elevated hs-Troponin I	19 (8.1%)
Pericardial effusion (mild/moderate)	14 (6.0%)

after COVID-19 inpatient admission are an emerging public health issue. Our data indicate a substantial burden of long-term cardiopulmonary and systemic symptoms, the most common among survivors at ≥3 months post-discharge being dyspnea, fatigue, and exercise intolerance. The findings are in according with an emergent international literature on post-acute sequelae of SARS-CoV-2 infection (PASC), or so-called long COVID. Dyspnea, (138 (58.7%)) particularly exertional, was the most common symptom in our population. Similar prevalence (26.0%) was reported by Huang et al. (2022)[9] with lower rates of ICU admission. Conversely, González et al. (2021)[12] reported 53% dyspnea at 12 weeks post-discharge in a Spanish population, which closely matches our rate of 58.7%.

Fatigue is one of the most frequent and impairing symptoms in long COVID sufferers. The present study showed that 122 (51.9%) patients experienced fatigue. Carfì et al. (2020),[11] also reported that 53.1% of patients complained of fatigue at two months from symptom onset. Likewise, Lopez-Leon et al. (2021)[13] performed a systematic review and meta-analysis from 15 nations and reported 58% of people having fatigue as the most frequent long-term symptom of COVID-19 worldwide. Rinaldo et al. (2021)[14] identified decreased functional capacity in more than 40% of patients by 3 months, even in the presence of some normal pulmonary function, implying intrinsic muscle deconditioning or cardiovascular stress. Chest Pain/Discomfort was found among 49 (20.8%) patients Dennis A et al. (2021)[8] in their study also found that 29% of the cases was associated with myocardial inflammation or fibrosis in certain individuals. Puntmann et al. (2020)[15] also documented cardiac compromise in convalesced COVID-19 patients, with 78% having cardiac abnormalities and 60% chest symptoms, proposing a cardiac cause for this post-viral chest pain. In the Mount Sinai COVID-19 Recovery Clinic (New York), Sudre et al. (2021)[16] noted that 11% of long COVID patients had palpitations, usually related to postural orthostatic tachycardia syndrome (POTS) or persistent myocardial irritation. Although less common, orthopnea in 18 patients (7.7%) may indicate cardiac involvement, particularly left ventricular dysfunction. This is in line with our results which showed that 13.6% of patients had a lower ejection fraction (EF < 50%). Mandal et al. (2021)[17] highlighted that even mild reductions in cardiac function can manifest as nocturnal dyspnea or orthopnea, especially when fluid balance is altered post-ICU.

FEV_1 (predicted percentage): 76.2 ± 18.5, Results showed that FEV_1 was found abnormal in 30.6% of cases. After COVID, nearly one-third of our patients have low FEV_1 values, which indicates mild airflow limitation. This result agrees with Mo et al. (2020),[18] who reported abnormal FEV_1 in 25% of cases discharged at 4 weeks. Huang et al. (2022)[9] reported that 22–29% of recovered patients had decreased FEV_1 at 6 months. These abnormalities are likely due to small airway inflammation, residual bronchial reactivity, or mechanical ventilation-induced damage in ICU-treated subjects. The mean predicted FVC value was 81.9 ± 20.4, which is abnormal at 26.8. The observed decrease in FVC also suggests restrictive ventilatory patterns, which may be caused on by post-viral fibrotic changes. Among a cohort of survivors of COVID-19, Guler et al. (2021)[19] noted decreased FVC in 20.3% of patients at the 4-month follow-up. Patients with this finding frequently had extensive parenchymal alterations on imaging. In our study, the relatively well-preserved FVC in comparison to DLCO indicates vascular remodeling or parenchymal damage rather than outright restriction. Obstructive pattern was found in 16.2%, and FEV_1/FVC ratio was 0.78 ± 0.09. A decrease in the FEV_1/FVC ratio was observed in 16.2% of patients, suggesting an obstructive pattern. This is consistent with the observations made by Lerum et al. (2021),[20] who found obstructive patterns in 15% patients at 3 months follow-

up. These were more frequent in smokers or those with a history of respiratory disease, although COVID-19 can trigger underlying airway hyperreactivity. Also, mechanical ventilation and high-flow oxygen therapy could lead to airway remodeling among ICU survivors. In 121 patients (51.5%), the DLCO (predicted percentage) was abnormal at 63.4 ± 17.9. With over half (51.5%) of our group exhibiting abnormalities, DLCO was the most compromised parameter. This is consistent with studies like Huang et al. (2022),[9] which discovered that 56% of patients had a decreased DLCO at 6 months, and Zhao et al. (2020),[21] which found DLCO abnormalities in 52% of non-ICU patients and 65% of ICU patients at 3 months. This recurrent finding confirms that DLCO is a sensitive measure of lung damage following COVID, most likely due to pulmonary microangiopathy, interstitial fibrosis, vascular thrombosis, or damage to the alveolar-capillary membrane. Correlation of decreased DLCO with persistent dyspnea/exercise intolerance in our patients validates its clinical significance. High-resolution computed tomography (HRCT) has been a crucial imaging modality in the evaluation of pulmonary sequelae following COVID-19.

Normal scans were present in only 19.6% of the patients, suggesting that radiologic abnormalities remain in a high percentage of survivors of severe COVID-19. In Ground-Glass Opacities (GGO), 106 (45.1%) patients were the most common radiological abnormality in our population. These opacities probably reflect sites of chronic alveolar inflammation or unresolved pneumonia, as organizing pneumonia patterns. Our results agree with other research such as Han et al. (2021)[22], reporting GGOs in 44% of 114 patients at 6-month follow-up. Zhao et al. (2020)[21] reported similar long-term persistence of GGOs in 39% of patients 3 months from discharge and Sonnweber et al. (2021)[23] also observed GGOs in about 40% of cases at 100 days post-infection. These results emphasize that GGO is still one of the most frequent imaging findings among convalescent COVID-19 patients, especially with initial moderate-to-severe disease. Among study cases, 82 patients (34.9%) had reticulations or fibrotic changes. Post-inflammatory fibrotic remodeling is indicated by reticulations, interlobular septal thickening, and architectural distortion, which are fibrotic changes on HRCT. Because they have the potential to result in long-term functional impairment, these are clinically relevant. Like Guler et al. (2021),[19] who observed fibrotic markers in 34% of COVID-19 patients at 4 months, and Han et al. (2021),[22] who reported fibrotic-like alterations in 35% of patients, with a higher rate of intensive care unit (ICU) patients, our incidence of fibrotic alterations (34.9%) is comparable as well. The similarities in studies point to a close correlation between severe COVID-19 and the predisposition to post-COVID pulmonary fibrosis, and long-term monitoring is indicated. In Bronchiectasis, 29 patients (12.3%) the finding of bronchiectasis in 12.3% of patients most probably indicates structural airway injury, perhaps as a result of chronic inflammation, Severe infection, mechanical ventilation, or superimposed bacterial infection. Similar results also reported by Pan et al. (2020),[24] who reported the bronchial dilatation was found in 11% of patients at 3-month follow-up. Despite being less frequent than GGOs or fibrosis, post-COVID lung bronchiectasis can predispose to recurrent symptoms and chronic infection and therefore is clinically relevant. In Subpleural Bands, 61 patients (26.0%), indicative of residual scarring or focal atelectasis and are often seen in organizing pneumonia. In our study, they were seen in over a quarter of patients, suggesting structural remodeling following viral insult. Zhao et al. (2020)[21] reported subpleural lines or bands in 23% of survivors. These findings are characteristic of other interstitial patterns of lung disease and can persist for months, particularly in patients with more extensive initial lung involvement. In our study, normal HRCT ,46 (19.6%) patients had completely normal HRCT scans, suggesting most individuals with severe COVID-19 progress to some form of permanent radiological abnormality. This is consistent with Sonnweber et al. (2021),[23] with normal HRCT in 21% of moderate/severe COVID-19 patients while Huang et al. (2022)[9] reported 25–30% of patients had complete radiologic resolution at 6 months.

These figures emphasize that radiological abnormalities are prevalent even after the acute stage, especially in those who need ICU-level care or oxygen therapy. SARS-CoV-2 infection affects not only the respiratory system but also profoundly influences the cardiovascular system by direct viral damage, systemic inflammation, endothelial dysfunction, and pro-thrombotic states. In the present study, several cardiovascular abnormalities were observed on follow-up, indicating extensive long-term cardiac involvement. In abnormal ECG (non-specific changes), 68 patients (28.9%), a large proportion of patients (28.9%) exhibited ECG abnormalities such as sinus tachycardia, ST-T wave changes, and premature beats. These could indicate residual myocardial inflammation, disturbances in conduction, or autonomic dysfunction. Comparable results have also been observed in another study by Radin et al. (2021),[25] which reported that ECG abnormalities in around 30% of recovered COVID-19 patients, such as repolarization abnormalities and arrhythmias. These observations highlight the value of ECG in identifying active subclinical cardiac involvement in post-COVID patients. In Left Ventricular Dysfunction (EF < 50%), 32 patients (13.6%), Echocardiographic assessment found LV systolic dysfunction in 13.6% of our population. This indicates either new cardiac cardiomyopathy or exacerbation of previous cardiac disease, perhaps due to viral myocarditis or stress-induced cardiomyopathy (Takotsubo syndrome). This finding is also documented by Puntmann

et al. (2020)[15] who identified LV dysfunction in 15% of recovered patients on cardiac MRI. This finding suggests that there is a subgroup of patients who have chronic myocardial impairment that requires longitudinal cardiac surveillance. In myocardial fibrosis (on MRI), 21 out of 42 patients (50.0%) had myocardial fibrosis, which indicates a history of myocarditis or myocardial damage. Late gadolinium enhancement (LGE) on MRI signifies irreversible scarring and has the potential to predispose patients to arrhythmias or heart failure. This rate is like to Puntmann et al. (2020)[15] who had fibrosis or LGE in 60% of recovered patients, even in those with mild or asymptomatic COVID-19 and Huang et al. (2022)[9] had cardiac MRI abnormalities in 58% of patients 3–6 months following infection. The high rate of fibrosis necessitates regular post-COVID cardiac imaging in symptomatic patients or those with abnormal biomarkers. Increased NT-proBNP (>125 pg/mL), 56 patients (23.8%), elevation in NT-proBNP indicates myocardial strain, heart failure, or volume overload. In our study, 23.8% of the patients had elevated NT-proBNP levels, indicating subclinical ongoing ventricular dysfunction. Referencing literature encompasses van den Heuvel et al. (2021)[26], who identified increased NT-proBNP in 22% of survivors of COVID-19 with persistent dyspnea. This observation supports the incorporation of biomarker monitoring in cardiovascular follow-up of convalescent patients. Increased hs-Troponin I, 19 patients (8.1%), Increased hs-Troponin I in 8.1% of patients indicates continuing myocardial damage. Though troponin rise is usual in acute illness, post-recovery persistent elevation can suggest subacute myocarditis or unresolving cardiac stress. Comparative study by Kotecha et al. (2021)[27] also showed myocardial inflammation with raised troponin in 7–8% of MRI scans after COVID. Persistent troponin rise should be a pointer for additional cardiac imaging and cardiology referral. Mild/moderate pericardial effusion, 14 patients (6.0%), pericardial effusions were seen in 6.0% of patients, most likely as a result of pericarditis or ongoing inflammation. Although usually clinically quiet, they can produce chronic chest pain or tamponade complications in unusual instances. This agrees with Puntmann et al. (2020)[15] noted pericardial involvement in 6.7% of their population by MRI. Early identification is critical, especially in chest pain and ECG abnormality patients.

Conclusion

A significant portion of individuals who recover from severe COVID-19 nonetheless have long-lasting cardiovascular problems, including heart dysfunction, lingering radiological changes, and impaired lung function. These findings emphasize how crucial comprehensive long-term monitoring and interdisciplinary care, with a focus on cardiopulmonary health, are for COVID-19 survivors.

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