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ORIGINAL RESEARCH

Long-Term Effects of COVID-19 on Chronic Obstructive Pulmonary Disease

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Background: Research has demonstrated that chronic obstructive pulmonary disease (COPD) is a negative prognostic factor for patients with the coronavirus disease 2019 (COVID-19). The long-term complications of COVID-19 among patients with COPD remain poorly understood due to limited studies.

Methods: This retrospective study included patients with COPD who underwent regular follow-ups in a medical center between January 1, 2020, and December 31, 2022. The patients were categorized into COVID-19 and non-COVID-19 groups. Comparative analyses were conducted to assess clinical demographics, characteristics, acute exacerbations of COPD (AECOPD), and survival rates between the two groups. Subgroup analysis was performed based on inpatient and outpatient status within the COVID-19 group.

Results: Of the 696 patients with COPD, 86 (12.4%) were included in the COVID-19 group, while 610 (87.6%) were included in the non-COVID-19 group. Patients in the COVID-19 group were significantly older (age: 75.0 ± 8.8 years versus 72.0 ± 9.0 years, $p = 0.004$), exhibited higher mortality rates (4.6% versus 0%, $p < 0.001$), and increased annual times of AECOPD (0.17 versus 0.08, $p = 0.018$) than those in the non-COVID-19 group after COVID-19. Multivariate analysis revealed that COVID-19 infection is an independent risk factor for increased AECOPD incidence (adjusted odds ratio: 1.74; 95% confidence interval [CI]: 1.07–2.83, $p = 0.024$). Within the COVID-19 group, the inpatient subgroup exhibited a higher prevalence of heart failure comorbidity (20% versus 2.8%, $p = 0.035$) and lower forced vital capacity than the outpatient subgroup (2.03 ± 0.60 L versus 2.56 ± 0.72 L, $p = 0.016$).

Conclusion: Age is a significant risk factor for COVID-19 infection among patients with COPD. After COVID-19, these patients exhibit an increased frequency of severe exacerbations and a high risk of mortality. Notably, the susceptibility to severe exacerbations persists regardless of whether the patients receive inpatient or outpatient care.

Keywords: Coronavirus disease, COVID-19, SARS-CoV-2, COPD, outcome

Introduction

Since its emergence in late 2019, the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has caused a global pandemic, posing severe threats to public health and prompting various lifestyle modifications. Current statistics indicate 770 million confirmed cases and approximately 7 million deaths, as reported by the World Health Organization, designating it as a global health crisis.[1] The impact of coronavirus disease 2019 (COVID-19) surpasses the immediate clinical ailment associated with infection; therefore, investigating the long-term complications in patients is imperative.

Chronic obstructive pulmonary disease (COPD) is a leading cause of chronic morbidity and mortality globally. Patients with COPD commonly experience airway symptoms, including dyspnea, chest tightness, and cough, and often suffer exacerbations that hinder their ability to perform routine daily activities. This condition worsens economic and social burdens.[2]

A previous study demonstrated that patients with COPD may experience a slightly increased risk of severe COVID-19 outcomes compared to those without obstructive lung diseases.[3] Recent studies have increasingly demonstrated that COPD is a significant negative prognostic factor for patients with the novel coronavirus.[3–9] Singh et al reported that

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various factors increase the susceptibility to SARS-CoV-2 infection in patients with COPD. These include elevated levels of angiotensin-converting enzyme 2 (ACE2), weakened antiviral defenses, and compromised immune responses.[10] Several studies have demonstrated an association between COPD and COVID-19; however, the mechanisms underlying the exacerbation of clinical outcomes in patients with COPD remain unclear. ACE2, the primary receptor facilitating SARS-CoV-2 cellular entry, is upregulated in the small airway epithelium and alveoli of patients with COPD.[11–13] Furthermore, patients with COPD exhibit pre-existing systemic inflammation, which may exacerbate the hyperinflammatory response elicited by COVID-19. This exacerbation may lead to cytokine storms and severe respiratory failure.[14,15]

Although the COVID-19 pandemic has subsided, long-term complications among patients with COPD remain a significant concern. This study aims to investigate the long-term impact of COVID-19 on COPD patients, focusing on rates of acute exacerbations and mortality following infection. Unlike previous research, this study provides a comprehensive analysis of these outcomes within the COPD population, addressing a gap that has been inadequately explored to date.

Methods

Study Design and Participants

This retrospective study included patients with COPD who registered in the COPD management system of the medical center of Far Eastern Memorial Hospital between January 1, 2020, and December 31, 2022. All registered patients with COPD met the criteria established by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guideline. COPD is defined as a post-bronchodilator ratio of forced expiratory volume in 1 second (FEV1)/forced vital capacity (FVC) <70%, confirmed using pulmonary function tests, according to the GOLD guideline.[2]

COVID-19 diagnosis was confirmed using the SARS-CoV-2 antigen test or polymerase chain reaction test. The patients were categorized into the SARS-CoV-2-infected group (COVID-19) and the SARS-CoV-2-non-infected group (non-COVID-19), and their outcomes were analyzed within this COPD population.

This study was conducted in accordance with the Declaration of Helsinki and approved by the Research Ethics Review Committee of Far Eastern Memorial Hospital (FEMH-112131-E). The requirement for informed consent was waived because of the retrospective nature of this study.

Clinical and Demographic Data Collection

The demographic data were obtained from the medical records of our COPD management system. Comorbidities, including diabetes mellitus, hypertension, heart failure (HF), coronary artery disease, previous pulmonary tuberculosis infection, chronic kidney disease (CKD), end-stage renal disease, and cancer, were verified using the International Classification of Diseases, 10th Revision coding, and associated medical records. CKD is an estimated glomerular filtration rate of <45 mL/min/1.73 m^2, maintained for >3 months. Furthermore, previous studies demonstrate that vaccines effectively lower the risk of severe or critical cases of COVID-19, correlating with significant declines in hospitalizations and fatalities associated with COVID-19.[16–20] Therefore, information regarding COVID-19 vaccination and booster status was obtained from the outpatient department system records to clarify the potential bias of follow-up duration.

The medical records included the medications for COPD management, including long-acting β2-agonists, long-acting muscarinic antagonists, inhaled corticosteroids, oral corticosteroids (OCS), and acetylcysteine. The administration of OCS was defined as being prescribed continuously for >3 months.

The index date was designated as the date of COVID-19 infection. In the COVID-19 group, the pulmonary function tests before COVID-19 were defined as the latest test before the index date in the past 3 years. However, the pulmonary function tests for the non-COVID-19 group were defined as the latest tests conducted between 2019 and 2022.

Furthermore, we used medical records to examine the COPD status of patients with acute exacerbation among our enrolled patients. An acute exacerbation is an event marked by increased dyspnea and/or cough and sputum that deteriorates within 14 days, potentially accompanied by tachypnea and/or tachycardia.[2] A mild COPD exacerbation is

defined as one that is treated exclusively with short-acting bronchodilators. A moderate exacerbation requires treatment with short-acting bronchodilators and OCS. A severe exacerbation is defined as one that necessitates hospitalization or a visit to the emergency department.[2]

In the COVID-19 group, we analyzed the incidence of COPD with acute exacerbations (AE) in these patients within 1 year before and after the index date, which indicated the exacerbations that occurred before and after the onset of COVID-19 (pre-COVID AE versus post-COVID AE). Additionally, the epidemic prevention policy implemented by the Taiwan Centers for Disease Control against COVID-19 led to the pandemic outbreak of COVID-19 in Taiwan in 2022.[21] Therefore, in the non-COVID-19 group, we analyzed the incidence of COPD exacerbations before COVID-19 in 2021 and after COVID-19 in 2023.

We performed a survival duration analysis until March 31, 2024, comparing the COVID-19 group (from the index date) and the non-COVID-19 group (from January 1, 2021).

Furthermore, we performed a subgroup analysis of inpatients and outpatients within the COVID-19 group. All inpatients were classified as having severe COVID-19 infections, as evidenced by using peripheral capillary oxygen saturation (SpO_2 level of ≤94%) while breathing ambient air and requiring supplemental oxygen.[22]

Statistical Analysis

IBM Statistical Package for the Social Sciences software (version 19) was utilized for all statistical analyses. Categorical and continuous variables were analyzed utilizing the chi-square and independent sample *t*-test. Aside from univariable analysis, variables with $p < 0.10$ were included in the multivariable analysis, utilizing the forward input method for the logistic regression model. Statistical significance was set as $p < 0.05$. Mortality and survival analyses were performed between January 1, 2021, and March 1, 2024, utilizing the Kaplan–Meier method.

Results

Clinical Demographic Characteristics

Of the 696 enrolled patients, 86 (12.4%) patients were included in the COVID-19 group and 610 (87.6%) patients were included in the non-COVID-19 group. Statistically significant differences were observed in the mean ages of patients in the COVID-19 and non-COVID-19 groups (mean age:75.0 ± 8.8 years versus 72.0 ± 9.0 years [$p = 0.004$]). The remaining basic demographic profiles, including sex, body mass index, and smoking status, exhibited statistically non-significant differences between the two groups. No statistically significant differences were observed between the groups regarding comorbidities, COPD medications, or COVID-19 vaccination status (Table 1). Regarding OCS medication, the dose of OCS equivalent to prednisolone among our patients was 7.3 ± 3.8 mg daily (Table 1). Among the 29 patients who were prescribed OCS, 3 patients administered the medication for more than one year, and 1 patient developed adrenal insufficiency. Regarding pulmonary function tests, including FEV_1, FEV_1%, FVC, FVC%, and FEV1/FVC before COVID-19, no statistically significant difference was observed between the two groups.

Table 1 Demographic and Clinical Characteristics of the Study Population

	Total (n = 696)	COVID-19 (n = 86) (12.4%)	Non-COVID-19 (n = 610) (87.6%)	*p*
Sex (male/female)		80/6	556/54	0.684
Age		75.0 ± 8.8	72.0 ± 9.0	0.004*
BMI		23.7 ± 4.46	23.9 ± 4.16	0.685
Smoking (nil/active/quit)		14/16/56	75/174/361	0.129

(*Continued*)

Table 1 (Continued).

	Total (n = 696)	COVID-19 (n = 86) (12.4%)	Non-COVID-19 (n = 610) (87.6%)	*p*
Comorbid conditions				
DM		21 (24%)	120 (20%)	0.317
HTN		43 (50%)	272 (45%)	0.357
CAD		22 (26%)	119 (20%)	0.198
HF		5 (5.8%)	30 (4.9%)	0.791
Cancer		8 (9.3%)	52 (8.5%)	0.837
CKD		4 (4.6%)	31 (5%)	1.00
ESRD		0	4 (1%)	1.00
Old CVA		7 (8.1%)	31 (5%)	0.305
Old PTB		6 (6.9%)	44 (7.2%)	1.00
COPD medication				
ICS		38 (44%)	274 (45%)	0.908
LAMA		14 (16%)	85 (14%)	0.620
LABA		5 (5.8%)	18 (2.9%)	0.188
Dual BD		29 (34%)	262 (43%)	0.129
ICS-LABA-LAMA		30 (35%)	203 (33%)	0.807
OCS[a]		7 (8.1%)	22 (3.6%)	0.156
Acetylcysteine		33 (38%)	258 (42%)	0.560
Vaccination		78 (90%)	534 (88%)	0.482
Vaccine≧3 (booster)		66 (77%)	488 (80%)	0.477
Baseline PFT				
FEV1 (L)		1.47 ± 0.51	1.46 ± 0.54	0.992
FEV1 (% predicted)		61.6 ± 18.7	58.5 ± 18.2	0.188
FVC (L)		2.46 ± 0.73	2.46 ± 0.76	0.988
FVC (% predicted)		79.0 ± 19.9	75.8 ±18.6	0.190
FEV1/FVC		59.7 ± 11.8	59.5 ± 11.9	0.979
Severe AE COPD (times) Pre-COVID AE Post-COVID AE		 0.15 0.17	 0.10 0.08	 0.323 0.018*
Mortality		4 (4.6%)	0	<0.001*

Notes: *Statistically significant; [a]OCS dose:7.3mg ±3.8 mg daily. Only three patients took oral steroid for more than one year, and one of them was for adrenal insufficiency.

Abbreviations: AE, acute exacerbation; BD, bronchodilator; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DM, diabetes mellitus; ESRD, end stage renal disease; HTN, hypertension; HF, heart failure; ICS, inhaled corticosteroids; LABA, long-acting beta-agonist; LAMA, long-acting muscarinic antagonist; OCS, oral corticosteroids; PFT, pulmonary function test; PTB, pulmonary tuberculosis.

Rate of Acute Exacerbation in COPD, Mortality, and Survival Analysis

Before the SARS-CoV-2 outbreak, no significant difference was observed in the rate of severe exacerbations between the groups. However, after the infection, patients in the COVID-19 group experienced a significant increase in severe exacerbations (0.17 versus 0.08, $p = 0.018$) (Table 1 and Figure 1). Furthermore, the mortality rate of patients in the COVID-19 group was higher than that of patients in the non-COVID-19 group ($p < 0.001$). The survival analysis revealed that patients with COPD in the non-COVID-19 group experienced a higher survival rate than those in the COVID-19 group (log-rank $p < 0.001$) (Figure 2).

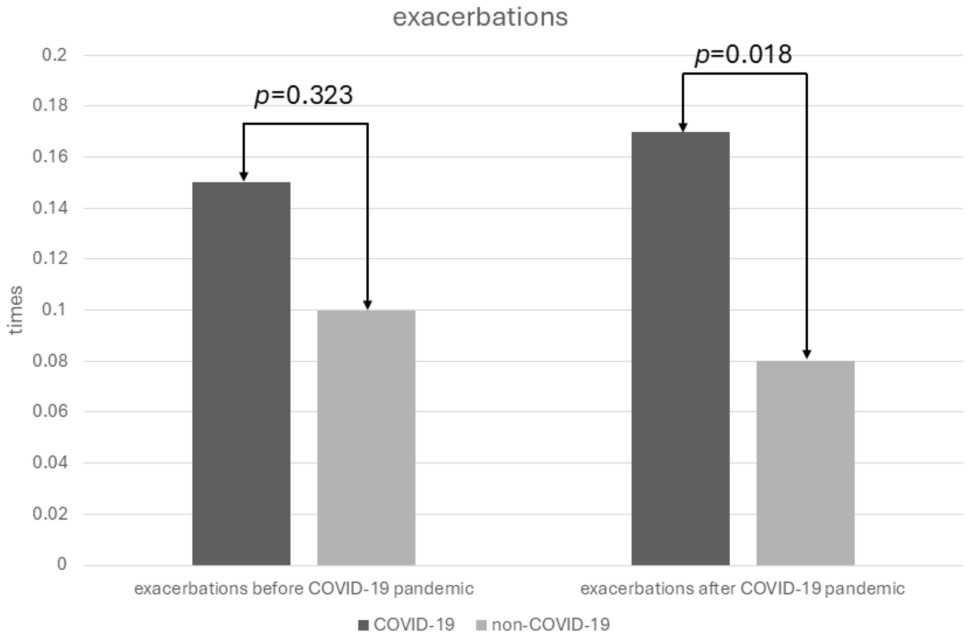

Figure 1 Severe exacerbations before and after COVID-19 pandemic between COVID-19 versus non-COVID-19 groups.

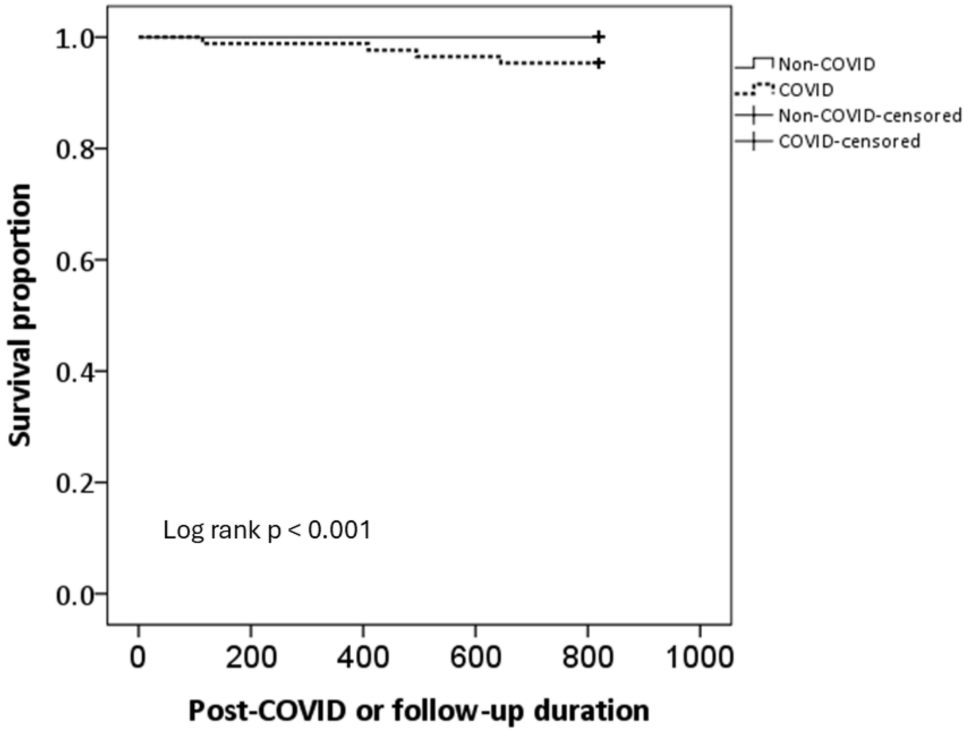

Figure 2 Kaplan–Meier method for the COVID-19 group versus the non-COVID-19 group.

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Multivariate Analysis

Multivariate analysis revealed that age is a significant risk factor for COVID-19 infection, with an adjusted odds ratio of 1.037 (1.011-1.064, p = 0.004) (Table 2). Additionally, patients in the COVID-19 group experienced a higher incidence of severe exacerbations than those in the non-COVID-19 group after SARS-CoV-2 infection, indicated by an adjusted odds ratio of 1.743 (1.074 - 2.827, p = 0.024).

Inpatients Versus Outpatients

We performed a subgroup comparison of demographic and clinical characteristics and pulmonary function tests between outpatients and inpatients in our analysis of patients in the COVID-19 group. Within the COVID-19 group, inpatients were significantly older than outpatients (mean age: 79.9 ± 9.7 years versus 74.0 ± 8.4 years, $p = 0.019$; Table 3). Additionally, patients

Table 2 Univariate and Multivariate Analyses

	Crude OR	p	Adjusted OR	*p*
Age	1.037 (1.011–1.063)	0.004	1.037 (1.011–1.064)	0.004*
AE after COVID	1.734 (1.074–2.800)	0.024	1.743 (1.074–2.827)	0.024*

Note: *Statistically significant.
Abbreviations: AE, acute exacerbation; OCS, oral corticosteroids; OR, odds ratio.

Table 3 Demographic and Clinical Characteristics of COVID-19 Inpatients Versus COVID-19 Outpatients

	Total (n = 86)	COVID-19 inpatients (n = 15) (17%)	COVID-19 outpatients (n = 71) (83%)	*p*
Sex (M/F)		14/1	66/5	1.000
Age		79.9 ± 9.7	74.0 ± 8.4	0.019*
BMI		22.3 ± 3.6	24.0 ± 4.5	0.179
Comorbid conditions				
DM		5 (33%)	16 (23%)	0.508
HTN		10 (67%)	33 (46%)	0.255
CAD		4 (27%)	18 (25%)	1.0
HF		3 (20%)	2 (2.8%)	0.035*
Cancer		2 (13%)	7 (9.8%)	0.747
CKD		2 (13%)	2 (2.8%)	0.139
ESRD		0	0	NA
CVA		1 (6.7%)	6 (8.5%)	1.00
Old PTB		0	6 (8.5%)	0.585
Baseline PFT				
FEV1 (L)		1.28 ± 0.45	1.51 ± 0.52	0.146
FEV1 (% predicted)		58.2 ± 18.9	62.4 ± 18.8	0.473
FVC (L)		2.03 ± 0.60	2.56 ± 0.72	0.016*
FVC (% predicted)		69.1 ± 18.6	81.3 ± 19.6	0.046*
FEV1/FVC		62.7 ± 13.0	59.0 ± 11.5	0.307

(*Continued*)

Table 3 (Continued).

	Total (n = 86)	COVID-19 inpatients (n = 15) (17%)	COVID-19 outpatients (n = 71) (83%)	*p*
Severe AE COPD (times)				
Pre-COVID AE		0.41	0.12	0.211
Post-COVID AE		0.32	0.17	0.846

Note: *Statistically significant.
Abbreviations: AE, acute exacerbation; BD, bronchodilator; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DM, diabetes mellitus; ESRD, end stage renal disease; HTN, hypertension; HF, heart failure; PFT, pulmonary function test; PTB, pulmonary tuberculosis.

in the inpatient subgroup exhibited a greater prevalence of HF, among other clinical characteristics. The pulmonary function tests indicated that patients in the inpatient group exhibited lower values for FVC and FVC% than those in the outpatient group (2.03 ± 0.60 L versus 2.56 ± 0.72 L, $p = 0.016$ and 69.1 ± 18.6% versus 81.3 ± 19.6%, $p = 0.046$, respectively). However, the severe acute exacerbation rate after COVID-19 infection did not differ significantly between the inpatient and outpatient subgroups.

Discussion

This study demonstrated that age is the primary risk factor that increases the susceptibility of patients with COPD to SARS-CoV-2 infection, and patients in the COVID-19 group exhibited a higher rate of severe exacerbations of COPD and a higher mortality rate after COVID-19 than those in the non-COVID-19 group. A previous comprehensive systemic review by Vanesa Bellou et al[9] identified age as a prognostic factor associated with hospitalization and mortality in patients with COVID-19. Our research highlights its significant prognostic relevance in the patients with COPD population.

According to the extensive cohort study of Aveyard et al,[4] patients with a clinical diagnosis of COPD experience poor clinical outcomes from COVID-19. Subsequent studies demonstrated that COVID-19 infection correlates with an increased rate of exacerbation and mortality among patients with COPD.[7,23] Our research revealed similar results, demonstrating that patients with COPD who contract COVID-19 subsequently experience increased rates of severe AE. Furthermore, this group of patients with COPD experience a higher mortality rate than those without COVID-19. A previous meta-analysis conducted by Caifang Zheng et al concluded that the COVID-19 vaccines are highly protective against SARS-CoV-2-related diseases in real-world settings.[18] Therefore, we must emphasize the significance of COVID-19 vaccination for these vulnerable patients with COPD in our standard care practices. The GOLD report and research by Solberg RB et al recommended masking to prevent COPD exacerbation and as a preventive measure for COVID-19.[2,24,25] Furthermore, Chan KPF et al reported that shielding methods effectively alleviated respiratory symptoms and lowered AECOPD during the COVID-19 pandemic.[25]

Our analysis revealed that inpatients exhibited a more significant decline in baseline FVC than outpatients. Kwok et al reported this phenomenon in a recent cohort study of 328 patients with COPD in Hong Kong.[26] In contrast to our findings regarding the risk of severe exacerbations, they reported that patients with severe COVID-19 experience a significantly higher risk of frequent severe exacerbations than those with mild to moderate COVID-19. These findings are inconsistent with those of our study. The probable rationale for the statistically insignificant difference in post-COVID AECOPD between inpatients and outpatients was presumably considered to be the limited sample size within our COVID-19 group, suggesting that further research with a more extensive sample size is necessary. We must emphasize the importance of receiving the COVID-19 vaccine to reduce the risk of hospitalization from severe illness.[16,18] Alongside vaccination, we recommend early pulmonary rehabilitation for patients with COVID-19 in the hospital, as a systematic review of 13 randomized controlled trials demonstrated that it can decrease mortality rates, shorten hospital stays, and reduce readmission rates when administered during hospitalization or within four weeks post-discharge.[27] A multinational task force has proposed early bedside rehabilitation for patients with severe COVID-19.[28]

Furthermore, we observed that patients with comorbid COPD and HF are particularly susceptible to hospitalization upon COVID-19 infection. HF is a common comorbidity in patients with COPD, and exacerbations of COPD can

exacerbate heart failure, and HF can similarly exacerbate COPD.[2] COPD is characterized by chronic inflammation affecting the lung parenchyma and peripheral airways, resulting in irreversible and progressive airflow limitation. Beyond the localized lung inflammation, COPD is associated with chronic systemic inflammation, a key mechanism contributing to the increased incidence of HF.[29–32] Consequently, patients with comorbid COPD and HF experiencing severe COVID-19 may have a hyperinflammatory response, termed a cytokine storm, induced by SARS-CoV-2, which may significantly exacerbate COPD and HF. Therefore, prioritizing these vulnerable patients comorbid with COPD and HF is imperative. A retrospective cohort study by Hassan et al underscores the importance of COVID-19 vaccination for patients with COPD and cardiovascular disease.[33]

Long-term administration of OCS can weaken the immune system, thereby increasing susceptibility to infections. An international registry study by Erica J. Brenner demonstrated a correlation between corticosteroids and severe COVID-19 in patients with inflammatory bowel diseases.[34] Herein, the prevalence of OCS prescription was higher in the COVID-19 group than in the non-COVID-19 group; however, the difference was not statistically significant ($p = 0.156$). This phenomenon in our study may be attributed to the administration of low-dose OCS (7.3 mg), which meets the criteria of the Global Initiative for Asthma guidelines (≤ 7.5 mg/day prednisolone equivalent),[35] and a small proportion of prolonged administration of OCS. Some patients with COPD exhibit an asthmatic component known as asthma-COPD overlap (ACO), with a global prevalence of 2.0%.[36] This disease is significantly associated with frequent acute exacerbations. In addition to the triple therapy for COPD, it is noteworthy that the majority of biological agents that have received approval for the management of severe asthma lack robust evidence to substantiate their efficacy in treating COPD. Consequently, this situation compels clinicians to continue their dependence on prolonged OCS therapy for a limited subset of COPD patients. Close observation of patients receiving extended courses of OCS is essential because of the possible adverse effects associated with their administration.

This study has some limitations. First, it was conducted with registered patients with COPD from a single medical center, which did not represent the wider population. Second, respiratory infections are the leading causes of exacerbations in COPD. The present study revealed an increased risk of exacerbation following COVID-19. However, since this cohort study did not assess the effects of viruses other than SARS-CoV-2, further research is warranted to investigate the impact of infections caused by other respiratory viruses on exacerbation rates. Third, the follow-up results and survival analysis for these patients with COPD were assessed only until March 1, 2024, suggesting that additional studies are required to investigate long-term outcomes.

In conclusion, this study demonstrated that age is a significant risk factor for the susceptibility of patients with COPD to COVID-19. Furthermore, age is a critical determinant in assessing whether patients comorbid with COPD and COVID-19 require hospitalization for treatment. We observed an increase in severe exacerbations and a higher mortality rate among patients with COPD after COVID-19 infection, emphasizing the need to prioritize this post-COVID population. Emphasizing the importance of COVID-19 prevention is essential in the daily care of patients with COPD, particularly older adults with comorbid heart failure, by ensuring they receive regular vaccinations and use masks for protection. Furthermore, early pulmonary rehabilitation should be implemented after COVID-19 infection among patients with COPD.

Abbreviations

AECOPD, Acute exacerbation of COPD; COPD, Chronic obstructive pulmonary disease; CAT, COPD assessment test; COVID-19, Coronavirus disease-2019; FEV_1, Forced expiratory volume in one second; FVC, Forced vital volume.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author, [P.H.W], upon reasonable request.

Ethics Approval and Consent to Participate

The Institutional Review Board of Far Eastern Memorial Hospital approved this study (IRB 112131-E). Informed consent was waived because of the retrospective nature of this study. The data will only be accessible to the research team, and

electronic data will be password-protected and kept strictly confidential. After obtaining relevant information, the identifiable information will be hidden, there should be no risk of privacy leakage, and the research content will not harm/label groups after publication. This research adhered to the ethical principles outlined in the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors declare no conflicts of interest in this work.

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