

Single centre study of pulmonary outcome in post-covid syndrome

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Abstract

The novel coronavirus SARS-CoV-2 is responsible for the COVID-19 pandemic, which has jeopardized people's health worldwide. This pandemic posed a hazard that had never been seen before. Although the acute phase of COVID-19 and its immediate respiratory difficulties have received much attention (which is understandable), there is growing concern regarding the chronic symptoms that some individuals continue to experience long after recovering from the initial infection. These symptoms can often be debilitating and can last for a long time after the initial infection has been cleared up.

Aim: As a result, this study aimed to investigate the clinical characteristics and outcomes of COVID-19-infected patients hospitalized at our establishment.

Materials & methods: Fifty COVID-19 patients hospitalized at the SCB Medical College's sole facility in Cuttack participated in this study. Fifty hospitalized individuals developed lung fibrosis within the designated time range due to COVID-19. **Results:** Following shortness of breath as the symptom that was reported the most often by COVID-19 patients diagnosed with pulmonary fibrosis on admission (49), cough (45), fever (23), sputum (10) and chest discomfort (8) were the next most frequently reported symptoms. In addition, a few patients developed diarrhoea (3), and one lost their sense of smell (1). Dyspnea was the symptom of COVID-19 reported most often by patients. Treatment in an intensive care unit was required for 83 per cent of patients diagnosed with COVID-19 pulmonary fibrosis. Patients who participated in the COVID-19 research with lung fibrosis, smoking, asthma, or acute renal impairment had a significantly increased risk of being admitted to the intensive care unit.

Conclusion: The COVID-19 pandemic has the potential to be lethal due to lung fibrosis. This may result in mortality or a lifelong decline in lung function. The probability that an issue may be avoided or, at the very least, its progression can be slowed down improves with early discovery.

Keywords: COVID-19; Post-COVID Syndrome; SARS-CoV-2; Acute Respiratory Distress Syndrome; Long COVID.

INTRODUCTION:

The pandemic brought on by COVID-19, which was kicked off by SARS-CoV-2, represents a threat to public health on a magnitude that has never been seen before. Chronic COVID-19 symptoms, which usually result in incapacity and have received far less attention, despite the fact that acute COVID-19 and its accompanying respiratory symptoms have received much attention, which is justified, are becoming a major cause for concern [1]. This condition, also called "Post-COVID Syndrome" or "Long COVID," is characterized by a wide range of symptoms that manifest even after an infection has been successfully treated [2].

The key cause for concern about this illness is a post-COVID syndrome, namely how it will affect the patient's lung health. SARS-CoV-2 can potentially induce pneumonia, ARDS, and substantial impairment of respiratory function in the early stages of the illness [3-5]. The SARS-CoV-2 virus primarily attacks the respiratory system as its primary objective. Many people can recover from the acute phase of the disease; nevertheless, many people never fully recover and end up with lung issues and repercussions as a result of the disease [6]. As a consequence of these difficulties, the affected persons may experience a severe decline in their quality of life and their capacity to function.

It is a difficult and ever-evolving problem, which presents challenges not only for those living with Post-COVID Syndrome but also for the medical experts who treat them. Some patients eventually fully recover from this multi-system illness that manifests itself in various symptoms. In contrast, others continue to struggle with symptoms that substantially impact their day-to-day lives [7]. Research into the pulmonary repercussions of post-COVID syndrome is absolutely required to enhance patients' overall quality of life, grasp the mechanisms at play, and develop viable treatments [8]. Several possible variables can impact post-COVID pulmonary outcomes, including lung damage during the acute phase of the infection, the length of time that inflammation lasted, and patient characteristics [9].

It is vital to establish a comprehensive strategy for studying, identifying, and treating post-COVID syndrome, particularly concerning its influence on pulmonary outcomes, as the COVID-19 pandemic continues to expand [10]. As a result of

continuous research and increasing public awareness, it is fair to believe that effective medicines and therapies will eventually be developed to alleviate the agony of individuals burdened with this puzzling and demanding sickness. This will be the case since successful medications and treatments will be in high demand. As a consequence of this, the objective of this study was to explore the clinical features and outcomes of COVID-19-infected patients who were hospitalized at our place.

MATERIALS & METHODS:

At the SCB Medical College hospital in Cuttack, fifty COVID-19 inpatients were evaluated for this research project. In the allotted time, lung fibrosis developed in fifty of the nineteen hospitalized COVID patients. With this primary objective in mind, we compared the clinical characteristics of post-COVID-19 pulmonary fibrosis in the two groups by comparing 25 patients who had survived (cases) and 25 patients who had died (controls). The ratio of cases to rules was 1:1. Because the total fatalities were not our primary concern, we came to this conclusion. Those patients who completed the first phase of treatment continued to be monitored for an additional half year. Only participants who had received a post-COVID-19 diagnosis of pulmonary fibrosis within the allotted time limit were considered for inclusion in our research. This is a result of the exceptionally low incidence of post-COVID-19 pulmonary fibrosis. Nasopharyngeal samples were tested using real-time reverse transcription polymerase chain reaction to validate COVID-19 after viral RNA was found and isolated from these samples. The procedure was carried out under the recommendations made by the ICMR. The appropriate university committee gave their stamp of approval to the study's ethical conduct. The principles outlined in the Helsinki Declaration were adhered to throughout the study. Because retrieving secondary data that has already been anonymized is done retroactively, getting consent is not essential in this case.

In the study, the researchers took into consideration the participants' ages, sexes, smoking statuses, comorbidities, clinical symptoms, laboratory test results, radiographic findings, ICU admission, the use of mechanical ventilation, the length of hospital stay, the occurrence of complications, and the outcome (recovery or death). The most common comorbidity and chronic illnesses were diabetes, hypertension, cardiovascular disease, renal disease, obesity, tuberculosis, chronic obstructive pulmonary disease, and asthma. Most patients also reported experiencing other symptoms, including fever, cough, sputum, hemoptysis, dyspnea, chest pain, diarrhoea, and loss of smell. The COVID-19 clinical symptoms are described here. ESR, c-reactive protein, ferritin, d-dimer, and total blood count were evaluated as part of the laboratory investigation. Radiological examination included a variety of procedures, including chest X-rays and CT scans. Hospitalization and death dates associated with COVID-19 were also documented.

The severity of the condition was rated under the criteria established by the WHO [10]. A patient was considered to have a severe case when their oxygen saturation was 90% or below, and their respiratory rate was less than 30 breaths per minute. At the moment of admission, the patient was considered a hospital inpatient, and this status remained in effect until either the patient recovered or passed away. The length of time an individual was sick was determined by counting the number of days beginning with the start of clinical COVID-19 symptoms and ending with either recovery or passing away. The recommendations that the ICMR developed served as the basis for the criterion for the rally. After ten or three days, recovery was thought to have taken place if fever and respiratory symptoms had decreased and two negative polymerase chain reaction results had been received within twenty-four hours of each other.

Participants in this research were found to have a positive COVID-19 test and also exhibited lung fibrosis. After the acute infection had been cleared up, the patients had difficulty breathing due to lung fibrosis, which was evaluated using high-resolution CT scans. [11] This was seen in individuals who had no previous history of ILD. The trial participants were restricted to those who were pregnant or did not have ILD.

Participants were scanned in the prone position using 64-channel multidetector CT scanners while the chest was examined. During this examination, no intravenous contrast was given. When reconstruction is complete, the resolution of the transverse pictures ranges from 0.55 to 0.75 millimetres. CT scans were performed on survivors just after they were discharged from the hospital and one, three, and six months later as part of the survivors' follow-up care. The CT score was created [12] to quantify lung involvement at the time of discharge and throughout follow-up. They were dissected to score the lungs properly and then divided into five lobes. A score between 0 and 5 was assigned to each afflicted lobe, with 0 indicating no engagement, 1 showing involvement between 5% and 25%, three showing involvement between 26% and 49%, and 4 showing participation between 50% and 75%. The overall CT score for each patient, which can range from 0 to 25, was determined by adding up the results for each lobar segment individually. A radiologist unfamiliar with the situation looked at the CT images.

Statistical analysis:

The Social Sciences Statistical Package was utilized to perform the tasks of data entry and analysis. Continuous variables are frequently described by the mean value in addition to the standard deviation (SD) when the data have a normal distribution and by the mean value in addition to the standard error of the mean when the data do not have a normal distribution. Mean and standard deviation are two measures utilized to illustrate the distribution of continuous data. On the other hand, percentages are used to demonstrate the distribution of categorical variables. To identify links between data

categories supported by statistical evidence, we carried out a chi-square analysis. The Pearson correlation coefficient was utilized as the analytical tool of choice to determine the degree of similarity between two continuous variables. We compared the means of normally and nonnormally distributed variables between groups using the independent samples t-test and the Mann-Whitney U test while considering age, gender, and body mass index as control factors. We calculated the average difference between the two measurements by employing a t-test designed for paired samples (before and after). Using Cox regression analysis, we determined the hazard ratios (HRs) and the confidence intervals (CIs) for each occurrence. This was achieved after accounting for several characteristics, including age, gender, and body mass index (BMI). When $P < 0.05$ was met, it was determined that the results were statistically significant.

RESULTS:

Patients with pulmonary fibrosis who participated in the COVID-19 study had their comorbidities and demographic information classified according to their survival status. Those under the age of 65 had a higher mortality rate (48%) than those above 65 (24%). In the COVID-19 patients who had pulmonary fibrosis, there was no correlation between age and an increased risk of mortality ($P = .368$). The ages of the people who participated in the study ranged from 21 to 85, with 57 being the mean age and 11.8 being the standard deviation. Patients who passed away at 60 (with a standard deviation equal to 12.2) were, on average, nine years younger than those who survived the illness. When infected with COVID-19, males had a 75 per cent chance of developing lung fibrosis.

In contrast, females had a 25% chance of developing the condition. Patients diagnosed with COVID-19 had a mortality rate of 64 per cent higher in males than females. For COVID-19 trial participants with pulmonary fibrosis, smoking was associated with a significantly higher risk of death ($P < 0.05$). Following high blood pressure and diabetes as the most frequent comorbidities were obesity (40%) and asthma (13%), followed by coronary heart disease (8%), renal disease (1%), tuberculosis (1%), and COPD (2%). Both of these conditions had a prevalence rate of 64%. During the COVID-19 experiment, patients with asthma and pulmonary fibrosis were more than twice as likely to pass away as patients with other comorbidities (odds ratio = 3.6, $P = 0.002$).

Table 1: Pulmonary fibrosis in COVID-19 patients, as manifested by their symptoms and findings in the clinic.

Symptoms and signs	Overall (n=50)	Survived (n=24)	Deceased (n=26)	P Value
Difficulty breathing	49	23	26	0.441
Cough	45	22	23	0.341
Sputum	10	10	0	< 0.05
Hemoptysis	1	1	0	0.765
Fever	23	5	20	0.592
Chest pain	8	16	19	0.887
Diarrhea	3	1	2	0.156
Loss of smell	1	1	1	0.232
Low SpO2 (<95%)	33	15	0	0.342
ICU admission	25	14	19	< 0.05
Mechanical ventilation	21	4	26	< 0.05
High Flow nasal cannula	11	10	0	< 0.05

Shortness of breath (49 patients), cough (45 patients), fever (23 patients), sputum (ten patients), and chest pain (eight patients) were the most prevalent symptoms described by patients admitted to COVID-19 with a diagnosis of pulmonary fibrosis. A smaller group of people (3) were impacted by diarrhoea, and one patient lost their sense of smell due to the illness. Dyspnea was the COVID-19 symptom that was reported the most frequently. Table 1 demonstrates that 83 per cent of the COVID-19 patients diagnosed with pulmonary fibrosis needed to be transferred to the intensive care unit. Twenty-one of the hospitalized patients who had been given a diagnosis of COVID-19 pulmonary fibrosis required mechanical ventilation, and an additional eleven of these patients required high-flow nasal cannulas. Each twenty-six patient who ultimately passed away was given an intubation and brought into the intensive care unit (Table 1). The average length of recovery after discharge was 22 days, whereas the median length of hospitalization before death was 16 days. Patients with COVID-19 pulmonary fibrosis who reported having no disease-related concerns made up 52% of the total. It was determined that mortality was caused by critical complications such as acute renal damage (42%), sepsis (54%), and respiratory failure (90%). ($P < 0.05$).

Table 2: Results of a clinical lab examination, taken into account of the patient's age, gender, and body mass index.

Parameter	Overall (n=50)	Survived (n=24)	Deceased (n=26)	P value
Haemoglobin (g%)	12.4 ± 1.6	12.2 ± 1.6	8.7 ± 2.7	< 0.05
White blood cell	8.2 ± 4.0	7.9 ± 3.8	22.1 ± 11.1	< 0.05
Neutrophils (%)	6.4 ± 3.9	6.1 ± 3.9	83.1 ± 8.9	< 0.05
Lymphocytes (%)	1.4 ± 1.9	1.7 ± 2.6	9.5 ± 9.9	< 0.05
C-reactive protein (mg/L)	7.4 ± 4.9	8.0 ± 6.2	4.7 ± 4.9	0.215
ESR (mm/h)	73.1 ± 29.1	74.1 ± 28.2	62.5 ± 61.7	0.667
D-dimer (mcg/mL)	2.9 ± 7.3	2.5 ± 6.3	7.9 ± 6.6	< 0.05
Serum ferritin (pmol/L)	751 ± 654	723 ± 623	1786 ± 1679	< 0.05

The clinical laboratory results for COVID-19 patients who were admitted with a diagnosis of pulmonary fibrosis are shown in Table 2. Haemoglobin concentrations were much lower in those who eventually died than in those who recovered from their illness. Compared to survivors, individuals who eventually died had considerably higher amounts of leukocytosis and neutrophilia (Table 2). Furthermore, compared to patients who were able to recover, the levels of ferritin and D-dimer were discovered to be considerably higher in patients who finally passed away (Table 2).

Table 3: Scores of computed topographies in COVID-19 survivors with pulmonary fibrosis during follow-up.

Parameter		At discharge	1st month	3rd month	6 th month
Bilateral lung infiltrations	No	10.5 ± 1.8	6.2 ± 1.2	2.6 ± 1	1.4 ± 0.6
	Yes	11.6 ± 0.9	7.9 ± 0.6	3.1 ± 0.6	2.4 ± 0.3
	P value	0.268	0.378	0.787	0.268
Ground-glass opacity	No	11.5 ± 0.8	7.5 ± 0.6	2.8 ± 0.5	1.8 ± 0.2
	Yes	18.5 ± 2.7	9.1 ± 1.6	6.8 ± 1.9	5.1 ± 2.5
	P value	0.78	0.433	0.069	< 0.05
Honeycomb	No	11.2 ± 1.1	6.5 ± 0.6	1.8 ± 0.4	1.1 ± 0.2
	Yes	15.7 ± 0.5	11.2 ± 0.6	7.1 ± 0.6	5.4 ± 0.5
	P value	0.138	< 0.05	< 0.05	< 0.05
Consolidation	No	12.7 ± 0.9	7.3 ± 0.6	2.6 ± 0.4	1.6 ± 0.2
	Yes	16.1 ± 1.0	11.1 ± 0.5	9.1 ± 0.05	7.1 ± 0.5
	P value	> 0.05	> 0.05	< 0.05	< 0.05
ICU admission	No	8.8 ± 1.4	4.4 ± 0.4	1.3 ± 0.4	0.5 ± 0.3
	Yes	15.6 ± 0.7	9.7 ± 0.6	4.4 ± 0.7	3.1 ± 0.4
	P value	< 0.05	< 0.05	< 0.05	< 0.05
Mechanical ventilation	No	11.6 ± 0.9	6.4 ± 0.5	2.3 ± 0.4	1.4 ± 0.2
	Yes	17.2 ± 1.1	11.5 ± 1.3	6.2 ± 1.3	4.4 ± 0.8
	P value	< 0.05	< 0.05	< 0.05	< 0.05
High Flow nasal cannula	No	12.6 ± 1.3	7.3 ± 0.9	2.9 ± 0.7	2.2 ± 0.5
	Yes	13.5 ± 1.0	8.6 ± 0.7	3.1 ± 0.7	2.1 ± 0.4
	P value	0.888	0.645	0.981	0.994
Long-term oxygen therapy	No	11.9 ± 1.3	5.9 ± 0.6	1.8 ± 0.6	1.4 ± 0.3
	Yes	14.1 ± 1.1	9.5 ± 0.9	4.3 ± 0.7	2.9 ± 0.5
	P value	0.276	< 0.05	< 0.05	0.067

On CT scans and chest X-rays, bilateral lung infiltrates were observed in 92% of the COVID-19 patients with pulmonary fibrosis who fully recovered. This finding was consistent across all three imaging modalities. CT scans revealed that 89% of patients had "ground glass" opacity, 23% had "honeycomb" lung, and 7% had pulmonary consolidation. After being followed up on over six months after their hospital release, it was discovered that the patients' average CT scores had dropped from 12.9 to 2.4. At the 6-month follow-up, the CT scores of patients who had "ground glass" opacity, "honeycomb" lung, and pulmonary consolidation were all significantly higher than at the baseline (P 0.05, P 0.05, and P 0.051, respectively). These results can be seen in Table 4. Both at discharge and the 6-month follow-up, the CT scores of patients who required mechanical breathing or were hospitalized in the critical care unit were significantly higher (P 0.05 for both comparisons). We compared the patients' post-discharge CT ratings to various other factors, such as the length of their hospital stays, their dyspnea scales, and the outcomes of their laboratory tests. Patients with higher CT scores also had more severe dyspnea and needed to stay in the hospital longer (P 0.05, r = 0.498, 0.496). The results of the laboratory tests and the CT scans were in no way connected. Patients who made a full recovery had a dyspnea rating of 1.3 on average at the

6-month follow-up, significantly improving from the 2.4 rating at discharge. There was a significant correlation between having bilateral lung infiltrates and having a "honeycomb lung" and higher mean dyspnea scale ratings at six months (P 0.05). Patients in a severe ICU before being released were regarded as having significantly worse dyspnea than patients in a medical ward (P 0.05). Patients admitted to the hospital with severe dyspnea had a significantly increased risk of requiring continuous oxygen therapy after being released (P .05). The chance that a patient may require long-term oxygen treatment was enhanced considerably (P 0.05) in cases where honeycomb lung was present. Patients who participated in the COVID-19 research and had recovered entirely from pulmonary fibrosis used oxygen therapy 52 per cent of the time.

The multivariate Cox proportional hazards regression yielded statistically significant data (P.001). Tobacco use was associated with an increased risk of death. The mortality risk associated with smoking was significantly higher than that associated with not smoking (hazard ratio = 2.2; 95% confidence range = 1.4-5.1; P <.001). Those with asthma and pulmonary fibrosis had a mortality risk of more than three times as high as those who did not have either illness (hazard ratio = 2.9; 95% confidence range = 0.8-9.1; P =.001). People with COVID-19 who suffered from pulmonary fibrosis and were overweight were likelier to pass away. The presence of sepsis (hazard ratio = 2.1 [95% confidence range = 1.0-4.2]; P 0.05) and acute renal damage (hazard ratio = 4.4 [95% confidence interval = 2.0-9.2]; P 0.05) each amplified the chance of dying by 2.1 and 4.8 times, respectively. This was because both conditions were statistically significant. Among the people who participated in the COVID-19 study, acute renal damage, smoking, asthma, and lung fibrosis were strongly related to admission to the intensive care unit.

DISCUSSION:

People who have COVID-19 have an extremely low risk of developing the condition known as lung fibrosis, although it is possible. Infected patients with COVID-19 who had lung fibrosis and were receiving treatment at the Single Center of SCB Medical College in Cuttack were the focus of this study, which aimed to close a knowledge gap that had been identified. The COVID-19 study found a correlation between increasing age and increased lung fibrosis risk [14]. Patients diagnosed with severe acute respiratory syndrome had an increased risk of developing pulmonary fibrosis as they age. On average, patients who reacted well to the COVID-19 therapy for pulmonary fibrosis were 54 years old.

In contrast, patients who did not respond favourably were, on average, 60 years old. There was a correlation between sarcoidosis and viral pneumonia in older patients [15,16]. This includes instances of COVID-19 and cases of Middle East Respiratory Syndrome. The elevated immune responses that the patients had in response to the illness led to the development of lung fibrosis in some of them. Patients who were older when they received COVID-19 had worse outcomes and greater mortality rates [17]. Both our humoral immunity and our cell-mediated immunity weaken as we become older. This is true for both types of immunity.

Because of this reduction in reactivity, the inflammation can worsen, and the virus might get away from being controlled [18,19]. Recent investigations indicated that pulmonary fibrosis was present in 72% of male COVID-19 patients but only 28% of female COVID-19 patients. As a result of the illness, the death rate among male patients was much higher than the overall average of 62%. Those who are male significantly outnumber those who are female. The name given to this virus is COVID-19. According to the research findings, female patients had a statistically significant better probability of survival than male patients (P 0.05).

In response to testosterone, transmembrane serine transcription protease two is activated and upregulated [20]. This gene promotes viral fusion with the host cell. It inhibits the formation of antibodies directed against coronavirus 2, the infectious agent responsible for acute respiratory syndrome. When a person smokes, they put themselves at an increased risk of developing lung disorders such as COPD, pulmonary fibrosis, and CKD [21]. Patients with a history of smoking and COVID-19-related lung fibrosis were also at an elevated risk of developing the condition. This is what the most recent research has shown on the topic. According to the findings, smokers have a significantly greater chance of developing advanced lung fibrosis due to the treatment. The COVID-19 test predicts death more accurately in people currently smoking than those who have never smoked. Oxidative stress and mucosal thickness are both increased by smoking. When inflammation and cytokines act together, the consequence is a significant illness that manifests in various ways [22].

Several conditions, including high blood pressure, diabetes, and coronary artery disease, can make the symptoms of COVID-19 worse [23]. According to recent research, patients with COVID-19 who were also diagnosed with pulmonary fibrosis demonstrated neutrophilia, leukocytosis, and lymphopenia in the final 24 hours of their lives. Patients also exhibited lymphopenia. Over sixty-five per cent of the COVID-19 patients diagnosed with pulmonary fibrosis also had hypertension or diabetes. These findings are connected to a previous test that discovered a deterioration in the patient's health [23]. As a disease marker, elevated serum lactate dehydrogenase levels have been investigated in more than one study [24]. Around fifteen per cent of COVID-19 patients with pulmonary fibrosis also had bronchial asthma, and those patients had a considerably elevated mortality risk. Participants in this study included patients who were infected with COVID-19. In the COVID-19 study, a person's mortality risk was increased by a factor of two if they had asthma and pulmonary fibrosis. A disease known as airway wall thickening might make asthma symptoms and its prognosis worse. Most of the time, alveolar fibrosis is the root cause of pulmonary fibrosis [25].

Patients diagnosed with severe instances of COVID-19 may need to be hospitalized in an intensive care unit. Patients in the intensive care unit placed on mechanical ventilation for an extended time have an increased chance of developing pulmonary fibrosis. There is a correlation between using mechanical ventilation and developing pulmonary fibrosis [26]. According to the findings of this study, intubation was necessary for 62 per cent of COVID-19 patients who were hospitalized, and 81 per cent of those patients were admitted to the intensive care unit (ICU). Patients with no other option but to rely on mechanical ventilation commonly experience lung injury due to the ventilation equipment [27]. Proinflammatory cytokines are generated when severe lung damage is followed by prolonged automatic breathing [28]. This raises the risk of mortality as well as pulmonary fibrosis.

Many COVID-19 patients diagnosed with pulmonary fibrosis continued to have dyspnea six months after being discharged from the hospital, albeit at a milder level. This occurred although they had shown broad signs of improvement in their dyspnea. Previous research carried out in Italy indicated that after being discharged from the hospital, 43 per cent of patients suffered from dyspnea [29]. The findings support the validity of these inferences. Chest CT images were used in this study to monitor the progression of post-COVID-19 pulmonary fibrosis. "Ground glass" opacity, often known as "honeycomb lung," and pulmonary consolidation were the findings most frequently observed on CT scans. According to a study [30], "ground glass" opacity appears within the first week of a COVID-19 infection. Consolidation and fibrosis, on the other hand, appear later in the course of the infection (after week 7). According to the findings of this study, the average CT score of patients who had recovered increased from 2.4 at six months to 12.9 when they were discharged from the hospital. It effectively predicted patient outcomes and mortality risk among people with COVID-19 by using the considerable correlation between the CT score and the severity of the disease. The CT scores of patients in the intensive care unit who required mechanical breathing at discharge and six months were considerably higher than those of patients in the medical ward ($P < 0.05$ and $P < 0.05$). This was demonstrated by the fact that the patients in the intensive care unit required mechanical breathing. Both of these situations illustrate this point. These findings are consistent with those discovered in a prior research study [31]. New data suggests that dyspnea is connected to the length of a patient's hospital stay and CT score. Common radiographic abnormalities have been found to have a significant connection to steady reductions in lung function over time [32]. The length of time patients were exposed to COVID-19 was found to correlate with lung fibrosis [33]. According to the findings of a clinical investigation [34], individuals with more advanced types of pneumonia tended to have a more fibrous texture. In the current examination, prednisolone was the initial therapy of choice for post-COVID-19 lung fibrosis. The dosage of prednisolone was gradually decreased during the duration of the trial [35]. Patients going through the healing process reported fewer symptoms after using this medicine.

Patients diagnosed with fibroproliferative ARDS may benefit from treatment with anti-inflammatory corticosteroids [36,37]. They are still an integral part of the standard treatment for interstitial pneumonia. Patients diagnosed with post-pulmonary fibrosis and treated with COVID-19 may be able to tolerate corticosteroids and see a rapid improvement in their health [38]. The effectiveness of antifibrotic medications in controlling post-COVID-19 lung fibrosis has only been demonstrated in a limited number of case studies [39,40]. Patients diagnosed with pulmonary fibrosis following COVID-19 do not now have access to any therapies that the appropriate authorities have approved. Additional clinical tests are required to identify the treatment method that is most likely to be successful in treating this condition.

CONCLUSION:

One serious and perhaps lethal consequence of the COVID-19 pandemic is the development of lung fibrosis. This may result in mortality or a lifelong decline in lung function. When an issue is identified early on, there is a greater chance that it may be avoided or, at the very least, its progression is slowed down.

Conflict of interest:

The present study authors do not have a conflict of interest among themselves.

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