POST COVID PULMONARY SEQUELAE – ONE YEAR OBSERVATIONAL STUDY

By

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To,

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"POST COVID PULMONARY SEQUELAE – ONE YEAR OBSERVATIONAL STUDY AT KLES DR. PRABHAKAR KORE CHARITABLE HOSPITAL & MRC",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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LIST OF ABREVIATIONS

COVID-19	-Coronavirus disease 2019
SARS-CoV-2	- Severe acute respiratory syndrome coronavirus 2
SARS	- Severe acute respiratory syndrome
MERS	-Middle East respiratory syndrome
TLC	-Total lung capacity
HRCT	-High-resolution computed tomography
GGO	-Ground-glass opacifcation
FEV1	- Forced expiratory volume in one second
FVC	- Forced vital capacity
PEF	- Peak expiratory fow
MEF50	- Maximal expiratory fow at 50% of FVC
MEF25	- Maximal expiratory fow at 25% of FVC
MMEF75/25	-Maximal midexpiratory fow between 75 and 25% of FVC
MVV	-Maximum voluntary ventilation
RV	-Residual volume
SD	-Standard deviation
ANOVA	-One-way analysis of variance
BMI	-Body mass index
COPD	-Chronic obstructive pulmonary disease
NIPPV	-Noninvasive positive pressure ventilation
LDH	- Lactate dehydrogenase
IPF	-Idiopathic pulmonary fbrosis.
DAMPs	-damage-associated molecular patterns

ABSTRACT:

Background: Persistent impairment of pulmonary function ranging from months to even years after discharge has been reported in other corona virus infections, such as SARS and MERS. Post covid-19 pulmonary sequelae aspects are not well understood.

Objective: To assess the prevalence of post-COVID pulmonary sequelae & to compare radiological findings and lung function in post-COVID patients .

Methods: One year observational study including 108 post-COVID 19 patients, after discharge were followed up high resolution computed tomography (HRCT) thorax, PFT within months.

Results: Out of 108 follow up patients with HRCT thorax, 77 patients had some evidence of fibrosis with 63 patients among them showing restrictive lung function patterns which is statistically significant (P=0.001).

Conclusion: Hence post-COVID 19 pulmonary sequelae is of great concern in moderate to severe cases and these patients need regular follow up and need to investigate for benefits of antifibrotics in them .

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Keywords: Post-COVID pulmonary sequelae, Restrictive lung function.

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INTRODUCTION

Background of the Study

A novel coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is what causes the coronavirus illness 2019 (COVID-19) [1]. Since December 2019, it has quickly spread throughout China and across the globe. Global attention has been drawn to SARS-CoV-2 airborne rapid transmission, and significant steps have been taken to effectively contain the outbreak and treat COVID-19. The angiotensin-converting enzyme (ACE) plays a major role in facilitating SARS-CoV-2 entrance into human cells, which seem to be expressed by type 2 pneumocytes [2].

Lessons from the 2003 SARS epidemic, which was a severe acute respiratory syndrome (SARS) outbreak, showed that some of the survivors had persistent pulmonary fibrosis, likewise the severity of the disease has already been linked to radiological abnormalities and pulmonary function impairment in SARS-CoV-2 patients [3].

Similarly according to reports of COVID-19 survivors in hospitalized patients , radiographic abnormalities, physiological deficits, and chronic symptoms can be seen months after the initial illness [4 5].

Patients have experienced more TLC drop and 6MWD decline one year after discharge from the critical care unit, as well as a higher incidence of DLCO impairment has been seen[6]. Although independent clinical, biomarker, and genomic risk factors have not been identified. Cohort studies of COVID-19 survivors report that the severity of the initial illness is associated with a greater risk of persistent CT abnormalities, especially for patients who required supplemental oxygen and mechanical ventilation [78].

However, the knowledge about the sequelae of SARS-CoV-2 infection stays limited. The long-time follow-up of COVID-19 survivors has not been studied in detail.

Thinking that covid-19 will remain in the population for a longer period and its impact on the respiratory system is expected the most, this study has been taken up to know more in detail about post-covid pulmonary sequelae.

OBJECTIVES

1.To assess the prevalence of post-covid pulmonary sequelae.

2.To compare radiological findings and lung function in post-covid patients .

REVIEW OF LITERATURE

Coronaviruses, which are more often referred to as CoVs, have been linked to a number of important disease outbreaks that have taken place in East Asia and the Middle East over the course of the previous two decades. These outbreaks have affected millions of people. In the years 2002 SARS and in 2012 Middle East respiratory syndrome (MERS) were reported[9]. Both of these diseases affect the respiratory system. Recently, a novel coronavirus that is known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) emerged in late 2019, causing coronavirus disease 2019 (COVID-19), and it has posed a threat to the health of people all over the world, causing a pandemic that is still ongoing all over the world.

Coronaviruses that have recently undergone a genetic shift pose a significant threat to the health of people living in every region of the world[9] .This outbreak is the third instance of a COV virus to manifest in humans within the past two decades.

The COVID-19 virus was initially discovered in China, and from that location, it swiftly travelled over the remainder of China before moving on to other countries. The World Health Organization (WHO) issued a declaration of a global health emergency on the 31st of January, 2020. This was done because the severity of this outbreak and the potential that it will spread on a global scale both warranted such a proclamation. On March 11th, 2020, WHO announced that it was a pandemic crisis.

The vast majority of countries around the world are currently in the process of implementing preventative and control measures into place as part of their attempts to put a stop to the further spread of a virus that has the capacity to kill. It is believed that the presence of a high number of coronaviruses in their natural host kept the door open for the

development of new coronaviruses. This made it possible for new coronaviruses to emerge[10,11]. This is due to the fact that due to genetic recombination taking, which lead to newer strains of coronaviruses. Wei et al said the high rate of mutations that can be observed in CoVs is caused by several causes, including the inconsistency of RNA-dependent RNA polymerases as well as the increased frequency of homologous RNA recombination [12]. These mutations are responsible for the virus's immense genetic diversity as well as its capacity to infect a wide array of distinct host species [11]. It will be important for disease surveillance , the development of novel targeted treatments, and the prevention of subsequent outbreaks if the origin of SARS-CoV-2 as well as the course of the infection can be determined. The signs and symptoms that are most frequently associated with COVID-19 include a high-grade fever, cough, dyspnoea, headache, and myalgia, diarrhoea, haemoptysis .

To design effective responses, it is necessary to have an in-depth understanding of the virus, which is a novel agent at present; as a result, more studies are required. It was discovered that the spike protein's genetic code, which has a total length of 1,273 amino acids, had 27 amino acid changes when the genome of SARS-CoV-2 was compared to the genome of the closely related SARS/SARS-like CoV[13]. This was discovered when the genomes of both viruses were compared to one another. This was found when the genome of SARS-CoV-2 was compared to that of the SARS/SARS-like CoV.From some studies it was shown that the SARS-CoV-2 virus was produced from bat SARS-like coronaviruses (bat-SL-CoVZC45 and bat-SL-CoVZXC21) [14].

Bat-SL-CoVZC45 and bat-SL-CoVXC21 are the names given to these two different viruses . Of addition, SARS-CoV-2 is genetically distinct from both SARS-CoV, with which it shares 79% of its genetic similarity, and MERS-CoV, with which it shares around 50% of its genetic similarity. Both of these viruses are in the coronavirus family [15].

The behaviour of coronaviruses, a significant human pathogen, has been studied for almost a century. Previously known as 2019-nCoV, the human disease-causing coronavirus known as severe acute respiratory syndrome-2 (SARS-CoV-2). The World Health Organization changed its name to coronavirus disease 2019 (COVID-19) in February 2020. The most prevalent significant clinical symptom of COVID-19 is viral pneumonia, which is characterized by fever, cough, dyspnoea, hypoxemia, and bilateral infiltrates on chest radiography. A dry cough occurs more frequently than an active cough, with a median of 5 to 8 days passing before the onset of dyspnoea. A considerable percentage of people with ARDS experience severe hypoxemic respiratory failure[16].

Clinical indicators include hypoxemia, which frequently contrasts sharply with the patient's perception of dyspnoea . According to Gattinoni et al severe abnormalities in ventilation-perfusion (V/Q), matching may accompany shunt physiology, which involves the perfusion of unventilated respiratory units, with abnormal hypoxic vasoconstriction being an important factor [17]. In autopsy investigations of COVID-19 individuals, lungs were found to be engorged and gross examination revealed a patchy distribution of abnormalities[18]. Microscopically, diffuse alveolar damage (DAD) was seen to cause the formation of a hyaline membrane, pneumocyte activation, microvascular thrombi, lymphocytic inflammation, and proteinaceous oedema. Other autopsy data demonstrate vascular remodelling by intussusceptive angiogenesis in the presence of microvascular thrombi[18] . Another case series described haemorrhage and hemosiderin deposition along with complement complex deposition, particularly close to the alveolar capillaries, as well as deposition of fibrin and erythrocytes in the alveolar spaces and septa[19].

The airway epithelium functions as a barrier to pathogens and particles, preventing infection and tissue damage through the secretion of mucus and the action of mucociliary clearance while maintaining effective airflow .The respiratory tract epithelium is the main entry point for beta-coronaviridae, which includes SARS-CoV-2, MERSCoV, and SARS-CoV, into the human host [20,21] . Viral contact occurs in the nasal mucosa through binding of the spike protein to the ACE2 (angiotensin-converting enzyme-2) receptor, followed by cleavage of spike protein by TMPRSS2 .

Inhaled SARS-CoV-2 virus particles presumably infect many epithelial cell types on their approach to the distal lung (transmembrane serine protease 2), Within these cells, SARS-CoV-2 replication happens [22–24] . Angiotensin 2 is transformed into metabolites by the type I transmembrane metallocarboxypeptidase known as the ACE2 receptor. Many of these metabolites have vasodilatory effects or disrupt the renin-angiotensin-aldosterone system. There is debate about whether the ciliated airway epithelium expresses enough ACE2 to allow for viral entrance, despite in vitro findings from SARS-CoV indicating that this is the predominant site for viral infection [25]. The SARS-CoV-2 enters and replicates in the nasal mucosa before moving on to the airways, where it initiates an immunological and inflammatory response that results in the clinical signs and symptoms of COVID-19 [26]. The expression of inflammatory mediators like CXCL10 and interferons by infected epithelial cells is possible.

According to preliminary data, adults with COPD and current smokers exhibit higher small airway ACE2 expression, which suggest why patients with the underlying cardiopulmonary disease appear to be more susceptible to death from severe COVID-19 [27,28]. While SARS-CoV-2 infection frequently start in the upper airway epithelium, in a small number of patients, the virus diffusely infects or damages the alveolar epithelium,

leading to respiratory failure and noticeably decreased gas exchange. Through contact between the

viral S protein and ACE2, which causes the virion to be internalized into endosomes, infection is facilitated. The S protein is broken down by host proteases to produce a fusion protein that allows the virus to enter the cell cytoplasm [22,23]. Although both alveolar type I and alveolar type 2(AT2) cells express ACE2, the infection most likely affects AT2 cells primarily because they produce surfactant, as seen in SARS-CoV[29]. Viral particles produced by infected cells infect nearby epithelial, endothelial, and macrophage cells. Late-stage cases' pathology examinations reveal viral protein and a lack of obvious vasculitis and interstitial inflammation, which suggests that prolonged infection of the alveolar epithelium occurs in severe illness.

Alveolar epithelial cells that are infected are likely to undergo apoptosis, it is unknown how likely this is compared to other types of cell death, effector T cell killing, or survival. Virion generation is boosted by viral proteins that interfere with cellular processes like apoptosis and interferon release[30]. The fusion machinery that mediates viral entry facilitates the fusing of infected cells to form syncytia. Syncytium development encourages viral cell-to-cell dissemination and escape from immune surveillance.

Alveolar-capillary barriers that are permeable are left behind after infected cells separate. Loss of epithelium is associated with plasma exudation or bleeding, the production of hyaline membranes which contain fibrin, factor VIII, and a significant portion of the barrier function of the alveolar-capillary interface [31].

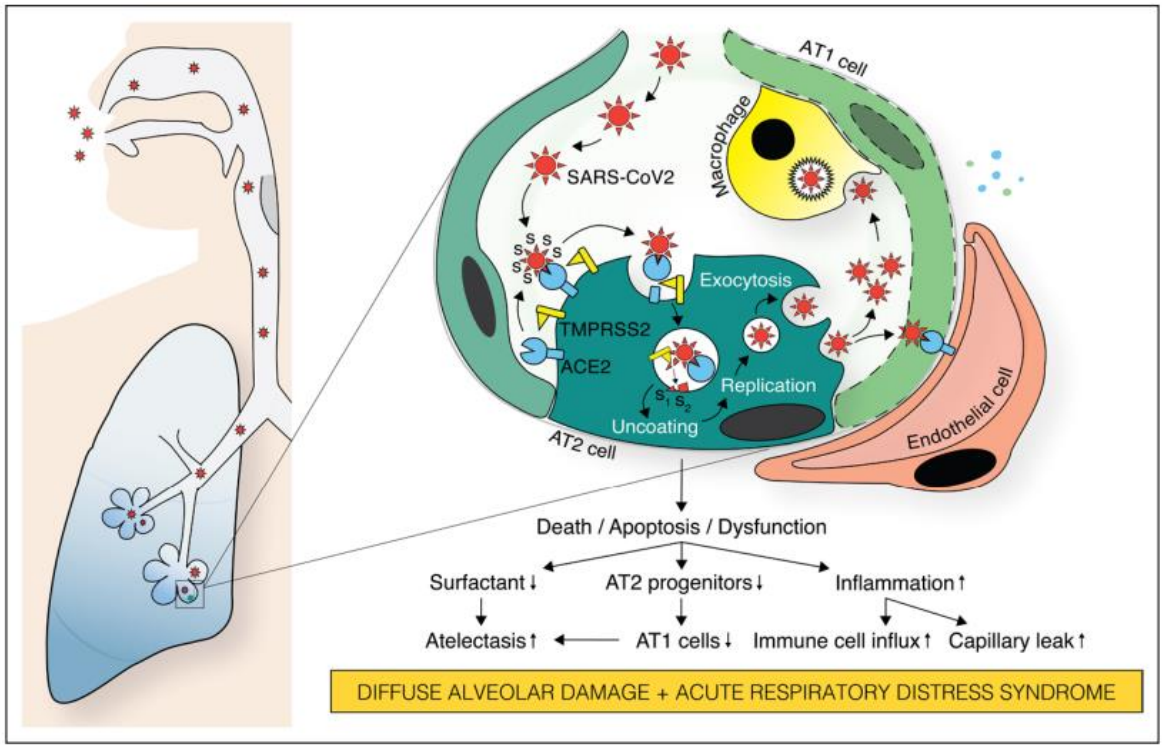


Figure 1. Pathobiological consequences of alveolar epithelial injury by severe acute respiratory syndrome coronavirus-2 (SARS-CoV2) infection.

SARS-CoV2 host entry through alveolar epithelium critically depends on expression of ACE2 (angiotensin-converting enzyme-2) and TMPRSS2 (transmembrane serine protease 2). First, coronavirus binds through one of its 4 structural proteins, glycoprotein S (spike) to ACE2 on alveolar type II (AT2) cells, initiating fusion of virus, and host cell membranes. Second, TMPRSS2 simultaneously cleaves ACE2, promoting cell surface clearance of ACE2, and the viral glycoprotein S into subunits S_1 and S_2, resulting in viral uncoating and release of viral genome into the cytoplasm. The virus is then replicated using both viral and host cell machinery, translation of the viral core proteins S, M, N, and E in the endoplasmatic reticulum (ER), assembly of virus particles in the ER-Golgi-intermediate compartment, and packaging into small wallet vesicles routed to the plasma membrane for exocytosis. SARS-CoV2 infection-induced AT2 dysfunction or loss is deleterious to the injured lung for several reasons: (1) decrease in surfactant increases the risk for alveolar collapse and atelectasis. (2) Decrease in AT2 progenitor cells causes impaired alveolar type I (AT1) cell replacement, affecting alveolar repair and likely promote fibrosis. (3) ACE2 downregulation drives geographically restricted overactivity of the ACE/Angiotensin II/AT1 receptor axis, worsening the tissue destructive effect of the inflammatory response. (4) Viral-induced cytokine release by AT1/AT2 cells results in capillary leak and alveolar interstitial immune cell infiltration.

The pathological finding of DAD is this process. Cytokines are produced by damaged or infected epithelium. In vitro, coronavirus- or influenza-infected alveolar epithelial cells release pro-inflammatory chemicals (eg, Interleukin-6, Interleukin -8, Interleukin -29, CCL5, CXCL9) [32] .Alveolar collapse is a result of a reduction in surfactant secretion caused by AT2 cell loss. Haemorrhage and fibrin deposition in the alveolar space are both signs of SARS-CoV-2 pathology, which suggests disturbances in coagulation and fibrinolysis. For ARDS of SARSCoV2 pneumonia to heal, reepithelialisation, removal of hyaline membrane , and regression of stromal cell and leukocyte accumulations are required. The remaining AT2 cells have the ability to regenerate epithelium because they multiply and develop into alveolar type I cells.

With COVID-19, vascular issues are a serious problem, while SARS-CoV 2 infection is primarily focused on the endothelium[33]. The pulmonary vascular endothelium reacts with vascular smooth muscle to induce hypoxic vasoconstriction via reversing the nitric oxide pathway and other mechanism to maintain ventilation & perfusion.Vascular endothelium as a barrier to the diffusion of water, solutes, and bigger molecules between plasma and the interstitium, it also regulates hemostatic and leukocyte movement into the interstitium.

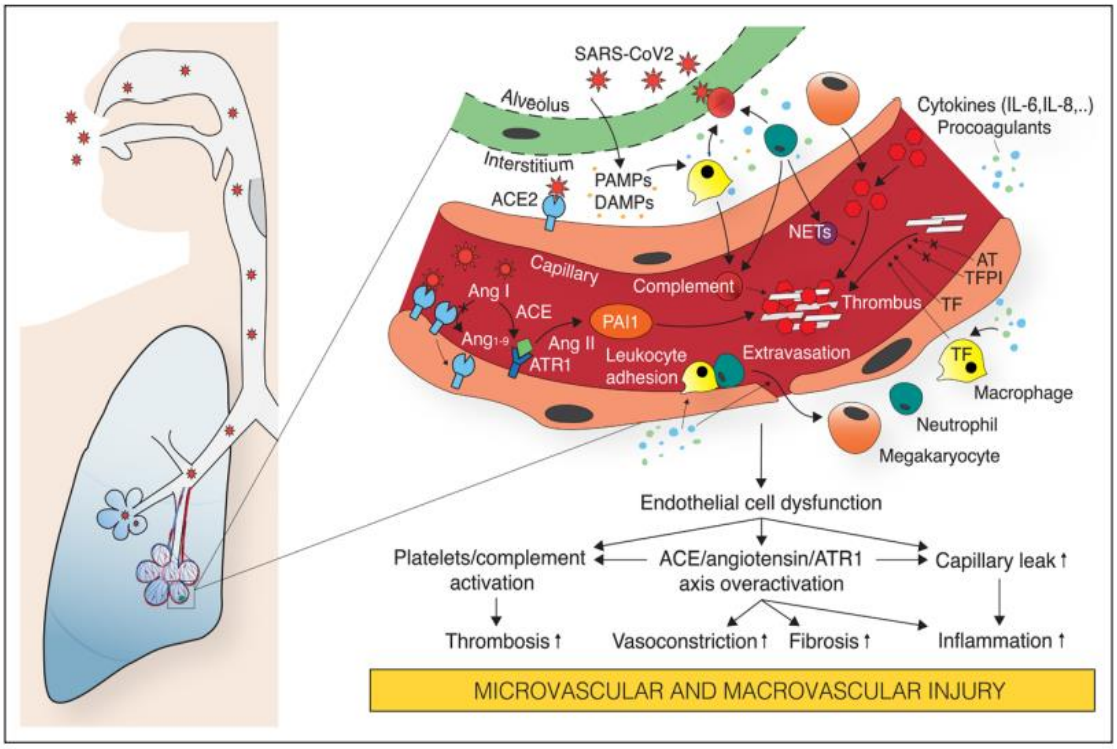


Fig :Pathobiological effects of (SARS-CoV2) infection on vascular endothelial damage.

SARS-CoV2 infection of endothelial cells, which might occur from luminal or alveolar interstitial side, triggers endothelial release of cytokines, which cause increased capillary permeability, thereby allowing adhesion and extravasation of neutrophils and monocytes into the alveolar interstitial space. Stimulated by PAMPs and DAMPs (pathogen-associated and damage-associated molecular patterns), neutrophils, and macrophages secret a multitude of cytokines, procoagulants, and complement, which promote viral attack and clearance but which induces further vascular injury enhancing the risk for thrombosis. Several factors might contribute to the prothrombotic environment, thereby promoting intravascular thrombus formation: (1) Neutrophil-mediated secretion of NETs (neutrophil extracellular traps) and complement enhances platelet aggregation. (2) Cytokine-triggered secretion of TF (tissue factor) by endothelial cells and macrophages stimulates the coagulation cascade and increases fibrin clot formation. (3) Endothelial damage decreases secretion of anticothrombotic mediators, such as AT (antithrombin) and TFPI (TF pathway inhibitor). (4) Lung residential megakaryocytes produce locally available platelets for aggregation. (5) Overactivation of the ACE (angiotensin-converting enzyme)/Ang II (angiotensin II)/AT_1 receptor axis due to virus-induced ACE2 downregulation increases production of PAI1 (plasminogen activator inhibitor 1), reducing plasmin activation and fibrinolysis. AT indicates antithrombin; ATR, angiotensin receptor; and IL, interleukin.

In COVID-19 all these functions are hampered apoptosis, pyroptosis, and lymphocytic inflammation of the endothelium in the lungs and other organs are all signs of direct viral infection of endothelial cells, according to several histopathologicinvestigations [34]. These histopathologic alterations can lead to tissue oedema, organ ischemia, and a procoagulant condition[35]. Small peripheral pulmonary arteries have an aberrant radiographic appearance, with vessel dilatation and tortuosity being two of the more noticeable findings [36]. These anomalies can cause aberrant pulmonary artery vasodilation in areas of hypoventilation, leading to loss of hypoxic vasoconstriction.

There will be direct endothelial infection, endothelial inflammation/oedema, and abnormal pulmonary vascular behaviour, resulting in hypoxemia and elevated physiological dead space that seems out of proportion to radiographic evidence of diseased lung parenchyma and relatively normal lung compliance [37]. Leukocytes produce inflammatory cytokines and other signalling molecules, which might cause the complement cascade to be activated and further spread endothelial damage [19]. In hospitalized COVID-19 patients, venous thromboembolic illness is common [38]. In COVID-19 patients, fibrinogen, von Willebrand factor, D-dimer and factor VIII reveal higher levels.

Activated partial thromboplastin time (aPTT) and platelet levels are frequently normal [39]. Increased dead space, a feature of both ARDS and COVID-19 pneumonia, would be caused by microvascular thrombosis in lung capillaries [37]. Hypercoagulability is brought on by an increase in the number of circulating neutrophils and monocytes, which is facilitated by cytokines and damage-associated molecular patterns(DAMPs)[40].

A perfect storm of excessive immobility, endothelial damage and a markedly hypercoagulable state of blood due to intense stimulation of endothelium and mononuclear cells to increase the release of factors, such as vWF, factor VIII, and TF, may be to blame for the apparent unusually high incidence of macroscopic and microvascular thrombosis. Diaphragm Disuse Atrophy-due to sedation, neuromuscular blockade, or excessive ventilator support, mechanical ventilation can unload the respiratory muscles and mute the respiratory centre in the brain stem. Strong evidence indicates that the primary cause of the diaphragm weakening during mechanical breathing is the inactivity of the diaphragm [41].

Severe COVID-19 results in impaired gas exchange, and severe respiratory failure due to significant alveolar injury and destruction of the pulmonary architecture. Although ARDS is the subject of several studies, 2 phenotypes of pulmonary fibrosis following COVID-19 were discussed in a recent article [42] . The distinguishing properties of these two phenotypes that are currently known . The first is the COVID-19 related ARDS (CARDS), characterized by the classical histopathological pattern of fibrotic diffuse alveolar damage or "fibrotic DAD" [43]. The exudative phase is the first phase of DAD,in which plasma fibrin leaks into the interstitium and alveolar space where it polymerizes and creates hyaline membranes. This is enhanced by strong alveolar inflammation caused by inflammatory cells infiltrating the alveoli [43]. The second phase is the proliferative phase, characterized by fibroblastic and myofibroblastic proliferation and extracellular matrix deposition resulting in fibrosis [43]. The squamous metaplasia further contributes to fibrosis [43]. Intubation is frequently necessary for patients with CARDS. These patients are often obese, which is linked to a higher risk of developing ARDS [44]. Advanced BMI is a risk factor for parenchymal fibrosis. Many people who survive this initial period,

particularly those whose illness lasts longer than three weeks, subsequently develop fibrosis [45]. Within the first year following COVID-19, these fibrotic changes remain[44].

Even in early ARDS survivors, this progressive fibrosis has the potential to be deadly. Post-COVID fibrosis(PCPF) is diagnosed using a combination of clinical, radiographic, and pathology information. Clinically, individuals with severe COVID-19 disease are more likely to have a restrictive ventilatory defect, which is associated with significantly decreased diffusing capacity in many weeks after hospital discharge .

There are fibrotic changes, reticular pattern, and traction bronchiectasis with or without honeycombing as radiological features of PCPF [45].The use of chest computed tomography (CT) as a diagnostic tool is advised early on.

In CARDS, pulmonary fibrosis in PCPF patients can develop at any stage of COVID-19 severity and occurs without a background of ARDS. Addressing the trigger event, pathophysiologic mechanism, prognosis of PCPF & IPF may not be the same. PCPF is not an idiopathic condition by definition because it is linked to COVID-19. Additionally, PCPF may be (partially) reversible [46].

Hafeda et al in a study of 84 patients found that 40 patients still

had unresolved CT findings [32]. Results from the follow-up HRCT indicate , diffuse involvement (65%) and a mix of central and peripheral lesions (75%) were the most prevalent types of lesions.

The most frequent findings were persistent ground glass opacities (85%) and reticular opacities (80%) . Additionally, because of the fibrotic adjacent changes, 35% of cases developed traction bronchiectasis.

The study showed that survivors of COVID-19 show permanent lung abnormalities on HRCT when cystic changes are present, >10 lung segments are involved, and the severity

score of the HRCT is >7. Patients who have distinct fibrotic changes after COVID-19 should be closely monitored.

Post Covid Pulmonary Sequelae

The National Institute for Health and Care Excellence (NICE) has provided the following terminology despite the fact that a precise definition of lengthy COVID-19 is still in the process of being defined. The names "Ongoing symptom" and "Post-COVID-19 syndrome" are included in this category[47].Post-COVID-19 syndrome is a name that refers to sequelae that occur during or after an infection with SARS-CoV-2 and remain for more than 12 weeks after the infection has cleared up[47]. No matter how severe the acute phase symptoms were, it has been documented in individuals with mild or severe COVID-19 [47] .Subjects report persisting symptoms at six months, including exhaustion, dyspnoea, anxiety, depression, and problems with focus, memory, attention, and sleep [48].Long COVID-19 syndrome is characterized by multiple contributing factors and extends beyond the acute phase of the infection to involve complications that affect multiple organs[48].

Long COVID-19 syndrome has a high probability of mortality. It can produce abnormalities in both the body and the mind, as well as restrictions in functional capacity and impairment in the ability to exercise, all of which can contribute to a decrease in quality of life. Additionally, it can produce restrictions in functional capacity and an impairment in the ability to exercise.

Studies have shown that as many as one-third of survivors may experience pulmonary symptoms at some point in their lives. In a study by Ivan et al on 43 symptomatic post-covid-19 patients,35% of patients showed a restrictive PFT pattern, and only 72.1% of participants completed the entire 6-min test [49].

Fajin et al in a study showed 29% of post covid -19 patients had pulmonary fibrosis at 7 months post-discharge and was positively associated with advanced age, longer hospital stay, use of mechanical ventilation therapy, higher quantitative CT parameters than those in the non-fibrosis group [50].

Considering the large number of COVID-19 cases, their long-term complications has to be studied in detail in order to treat them. Although there is evidence suggesting that patients who have persistent symptoms may benefit from a carefully designed pulmonary rehabilitation program, the effectiveness is still unknown[51]. This is due to the fact that patients with COVID-related conditions tend to have more than one of these symptoms.

Due to post COVID-19 pulmonary fibrosis, consequences might range from mild forms to serious types , requiring long-term oxygen therapy or even lung transplantation [42] . Post COVID-19, patients must have regular follow-ups, as few patients may develop complication which should be detected as early as possible.

Approximately 92 million people have been impacted by COVID-19 so far in the pandemic . Nearly 4.8 million people may have significant pulmonary involvement, even though the majority may had moderate to severe disease with ARDS. Even while the majority of patients will recover without lung injury, it's possible that a significant percentage of patients may have long-term complications [52].

From a fewer studies it is found that , use of anti-fibrotics in the early acute phase of severe disease with ARDS may minimize fibrosis, even if there is currently no fully effective treatment for post-COVID-19 pulmonary fibrosis [53].

CT severity score -is a score for the degree of lung affection based on dividing the lung into five lung lobes; each lobe affection was visually scored on a scale of 0–5, with 0 indicating no involvement, 1 indicating less than 5% involvement, 2 indicating 5–25%

involvement, 3 indicating 26–49% involvement, 4 indicating 50–75% involvement, and 5 indicating more than 75% involvement [549].The total CT score was the sum of the individual lobar scores and ranged from 0 (no involvement) to 25 (maximum involvement).

Rabab et al did a study in 210 post COVID-19 individuals, when a multivariate analysis was conducted ,the age of the patients, initial CT severity score, consolidation/crazy-paving score, and ICU admission were determined to be independent risk factors linked to the occurrence of post-COVID-19 fibrosis (P= 0.05) [50].Many post covid patients have persistent pulmonary function impairment . A study by Yiying et al showed severe covid -19 patients on follow-up had abnormal lung function testing results with significant changes in FVC, FEV1, FEV1/FVC ratio, TLC, and DLCO values[55].

METHODOLOGY

A one-year hospital based observational Study from January 2021 to December 2021 at KLE's Dr.Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi.

In this study, RT-PCR/CBNAAT / RAT Positive COVID-19 patients with the fulfilment of the inclusion criteria were included. All participants were given written informed consent prior to inclusion. This study was approved by the department of ethical clearance committee and college dissertation and research committee, J. N. Medical College, Belagavi. The patients were subjected to thorough history taking and clinical examination. pulmonary function test, and HRCT scan of the thorax of the patients were analyzed during the study.

Chest HRCT and image quantification. All participants underwent chest CT scan during the acute phase of the disease and during the follow-up .In the acute phase According to the percentage area occupied, each lobe was evaluated 0–5 points representing normal performance, lesions involving 75% of lobe respectively. Then the CT score was calculated by the addition of individual segmental scores.

To quantify lung lesions, each of the five lung lobes was reviewed for the lesions, such as ground-glass opacification (GGO), interstitial thickening, consolidation, bronchiectasis, irregular interfaces.

All participants underwent pulmonary function tests in the Pulmonary Function laboratory at KLE's Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi. American Thoracic Society/European Respiratory Society guidelines were followed. The assessment included: forced expiratory volume in one second (FEV1), forced vital

capacity (FVC), peak expiratory flow (PEF), maximal expiratory flow at 50% of FVC (MEF50), maximal expiratory flow at 25% of FVC (MEF25), maximal midexpiratoryflow between 75 and 25% of FVC (MMEF75/25), maximum voluntary ventilation (MVV), TLC, residual volume (RV).

Statistical analyses. Data were expressed as means±standard deviation (SD) when data were normally distributed or medians with interquartile range (IQR) when data were not normally distributed. Comparisons were determined by Student's t-test,

Mann–Whitney U test or Fisher exact tests as appropriate.

Multiple groups were compared using one-way analysis of variance (ANOVA) with a Bonferroni correction (normal data) or a Kruskal–Walli's test with a Dunn intergroup comparison (non-normal data). Spearman's rank correlation coefficient was used for correlation analyses. We used logistic regression analysis to explore the independent risk factor, Statistical significance was considered as p ,

p value <0.05 was considered as statistically significant. For the continuous quantitative variables mean and standard deviation was calculated. For the purpose of comparison, if the data was divided into groups with respect to certain qualitative characteristic, the continuous variables was compared using suitable tools of statistics like student's unpaired t test.

Discrete variables were represented by the median. The categorical data was expressed in terms of rates, ratios, and percentages. The association between the outcome, clinical and demographic characteristics was tested using the Chi-square test, test of proportion or Fisher's exact test. For discrete variables nonparametric tests are used, apart from the above suitable tools like ANOVA, and correlation.

Data was entered into a Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions.

Chi-square was used as test of significance. Pearson's Correlation was used to correlate between two quantitative variables. Pearson's Correlation Was used to correlate between two quantitative variables. p value <0.05 was considered as statistically significant.

SAMPLE SIZE CALCULATION

The sample size was estimated by using the proportion of post-Covid pulmonary sequelae detected by CT scan at 54.3% from the study by Yiying Huang et al[3]. using the formula P = 54.3 q = 45.7 d = 10% Using the above values at 95% Confidence level a sample size of 96 confirmed COVID-19 subjects was calculated and a total of 106 subjects were included in the study.

Inclusion criteria

1. Confirmed cases of SARS-COV 2 by RTPCR TEST /CBNAAT/RAT for covid 19.
2. HRCT/CT -covid 19 suspect.

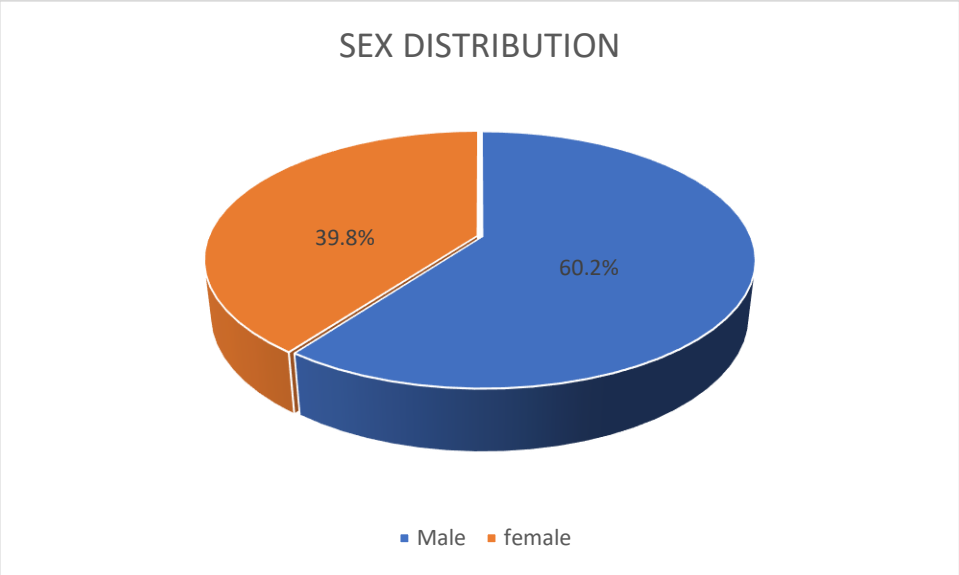
Exclusion criteria

Below 18 years of age.

RESULTS

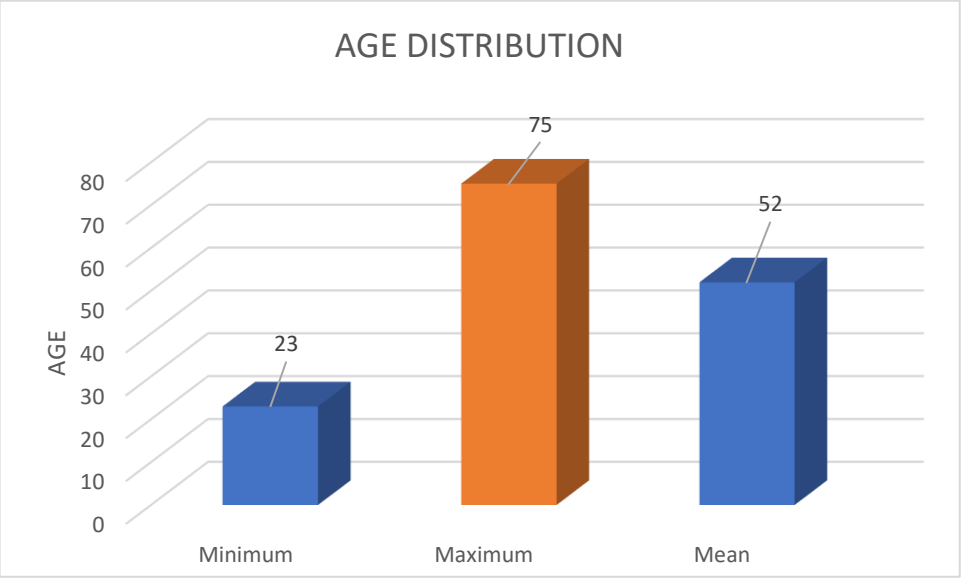
Total of 108 patients in this study met the eligibility criteria. Mean age was 52 years. Most of our follow-up patients were males,65 out of 108 patients (60.2%). Majority of our patients were males 65 out of 108 (60.2%).

Diagram :1



AGE DISTRIBUTION

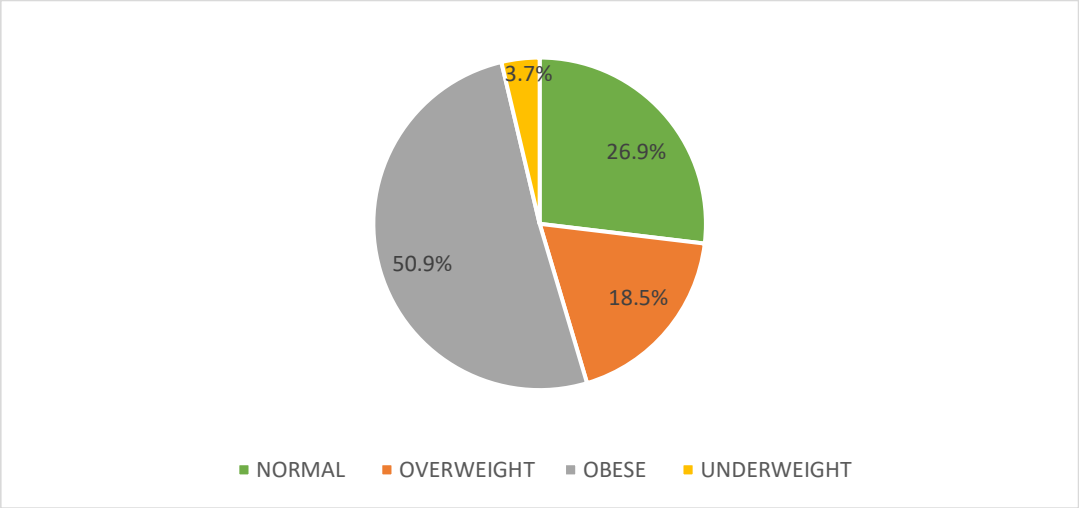
Diagram :2



The mean age of our study population was 52 years , with the maximum being 75 years and the minimum being 23 years.

BMI DISTRIBUTION

Diagram :3



Body mass index (BMI) of all the patients in the study population was calculated and was classified as underweight, normal, overweight, and obese with a BMI <18.5,18.5-24.9,25.9-29.9, >30 respectively.

Table :1 BMI DISTRIBUTION

Category	Number (n)
Underweight	4
Normal	29
Overweight	20
Obese	55

The average BMI of our study population was 25.4 with 4,29,20 ,55 patients in the underweight, normal, overweight, and obese category respectively.

Table :2 DISTRIBUTION OF SMOKING HISTORY

In this study 54 .6% of the patients were non smokers with 25 .9 % being smokers and 19.4 % being exsmokers.

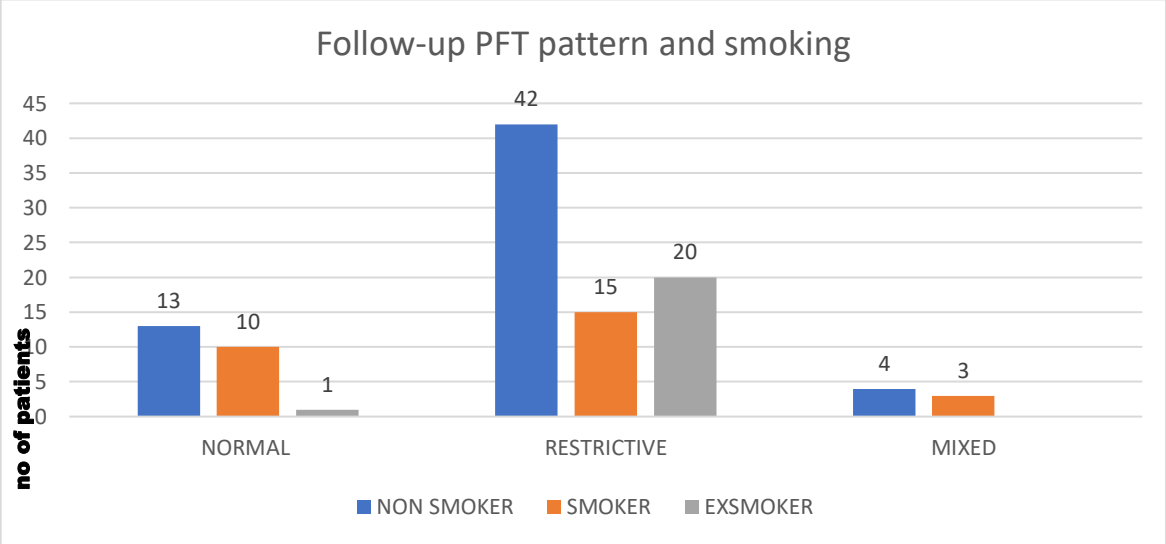
Factors	Number	Percentage
SMOKER	28	25.9%
EXSMOKER	21	19.4%
NON SMOKER	59	54.6%

Table :3 ASSOCIATION OF SMOKING WITH FOLLOW-UP LUNG FUNCTION TEST

	FOLOW-UP PFT PATTERN				P VALUE
	NORMAL	RESTRICTIVE	MIXED	Total	
NON SMOKER	13	42	4	59	0.097
SMOKER	10	15	3	28	0.025
EXSMOKER	1	20	0	21	0.99

ASSOCIATION OF SMOKING WITH FOLLOW-UP LUNG FUNCTION TEST

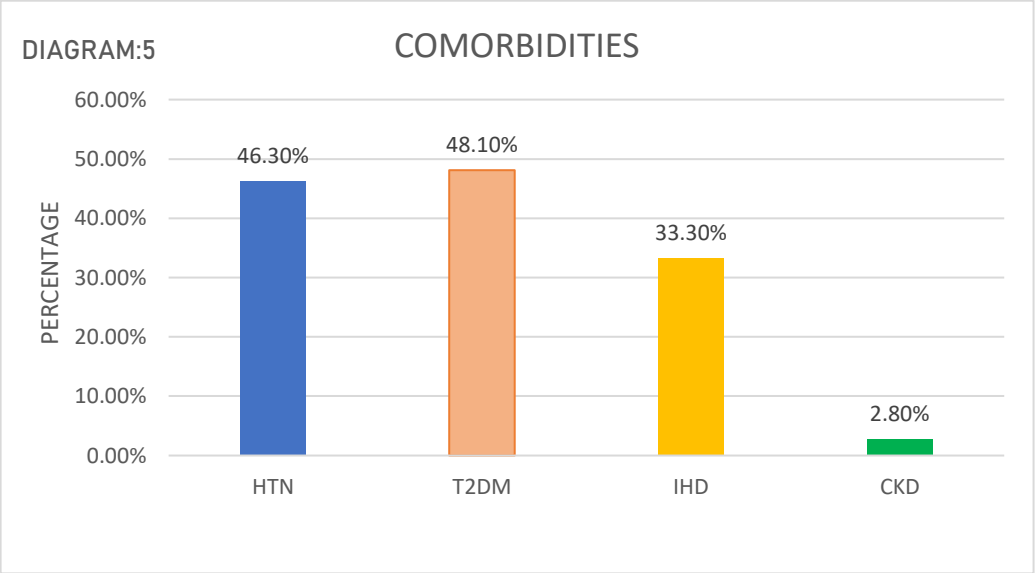
Diagram :4



In this study 42 out of 59 non-smokers (P=0.9) and 15 out of 28 active smokers (P=0.05) had restrictive PFT pattern during their follow-up but no statistical significance was associated, 20 out of 21 ex-smokers with significant smoking history had restrictive PFT pattern and was statistically significant (P=0.025).

Table : 4 DISTRIBUTION OF COMORBIDITIES

Factors	Number	Percentage
HTN	50	46.3%
T2DM	52	48.1%
IHD	36	33.3%
CKD	3	2.8%



In this study patients had T2DM, Hypertension, IHD as major co-morbidities with T2DM in 52 (48.1%) , IHD in 36 (33.3%), Hypertension seen in 50 (46.3%), and CKD in 3(2.8%) patients.

Table :5 ASSOCIATION OF COMORBIDITIES WITH FOLLOW-UP LUNG FUNCTION TEST

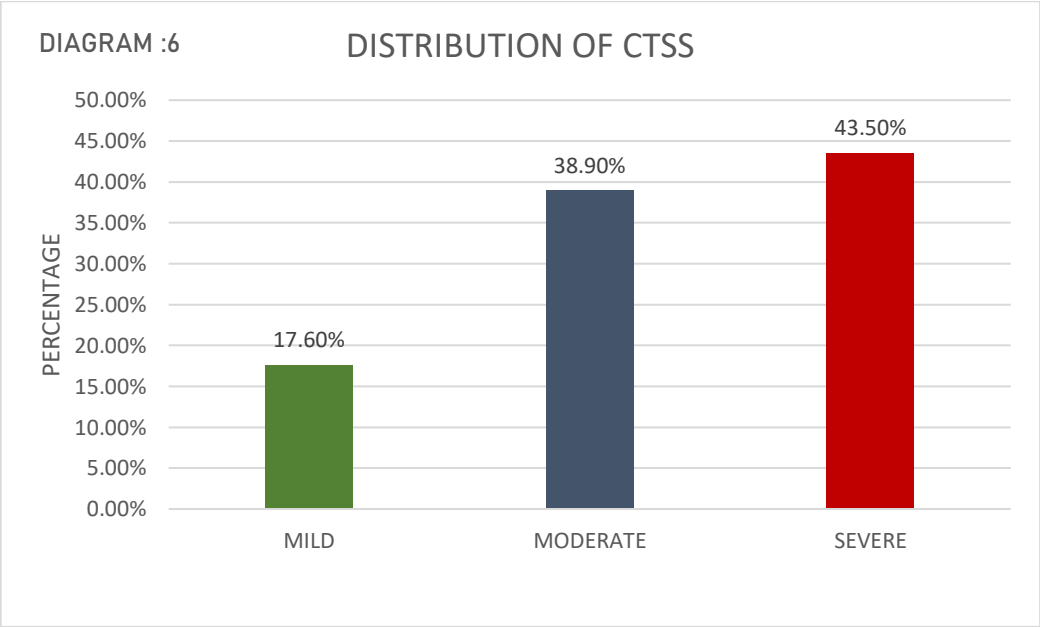
COMORBIDITIES	FOLLOW-UP PFT PATTERN			TOTAL	P value
	NORMAL	RESTRICTIVE	MIXED		
T2DM(n=52)	7	44	1	52	0.010
COPD(n=1)	0	0	1	1	0.001
CKD(n=3)	3	0	0	3	0.005
HTN(n=50)	10	37	3	50	0.845
IHD(n=36)	5	30	1	36	0.140

In this study, restrictive PFT pattern is Positively associated with comorbidities. 44 out of 52 T2DM patients had a restrictive PFT pattern, showing a statically significant association (P=0.01).

Whereas 37 out of 50 hypertensive patients had a restrictive PFT pattern(P=0.84) and 30 out of 36 IHD patients had a restrictive PFT pattern(P=0.14) but was not statically significant.

Table : 6 Classification According To CT Severity scores

Factors	Classification	Number	Percentage (%)
CTSS CATEGORY	**MILD**	19	17.6 %
	MODERATE	42	38.9 %
	SEVERE	47	43.5 %
	Total	108	100 %

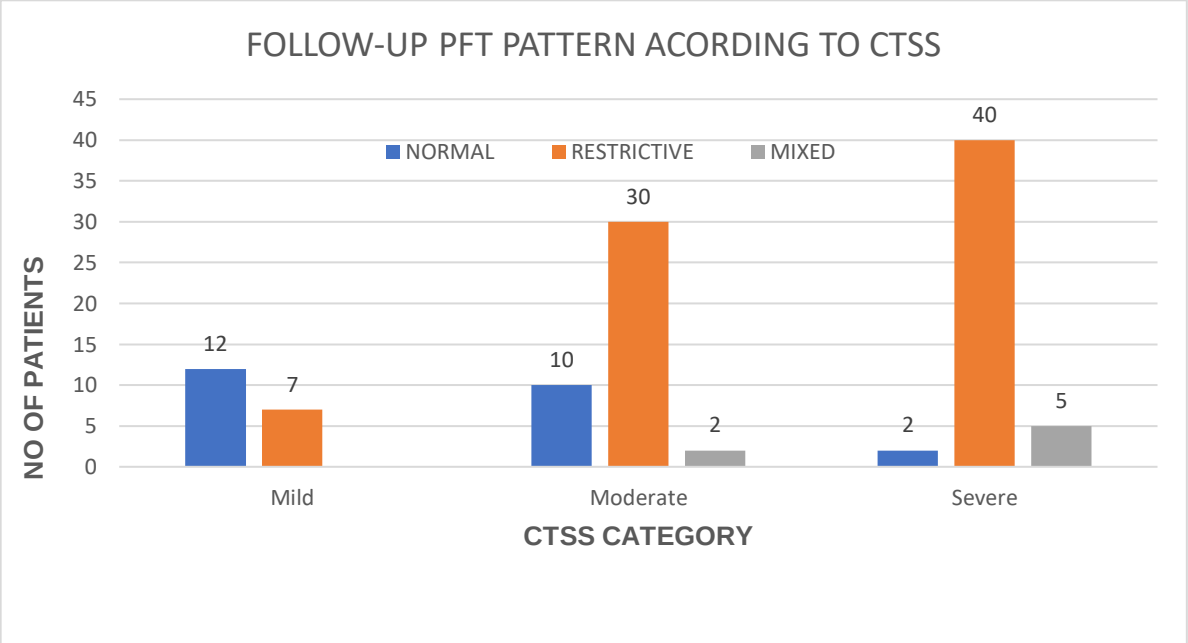


In this study ,with respect to the lung involvement ,out of 108 patients , 19(17.6%) patients had mild CTSS , 42 (38.9%) patients had moderate CTSS and 47 (43.5 %) had severe CT severity score, with a mean CTSS score of 14.7 out of 25 .

Table :7 ASSOCIATION OF CTSS CATEGORY WITH LUNG FUNCTION TEST DURING FOLLOW-UP

		FOLLOW-UP PFT PATTERN				P value
		NORMAL	RESTRICTIVE	MIXED	Total	
CTSS CATEGORY	Mild	12	7	0	19	
	Moderate	10	30	2	42	
	Severe	2	40	5	47	
Total		24	77	7	108	0.001

Diagram :7

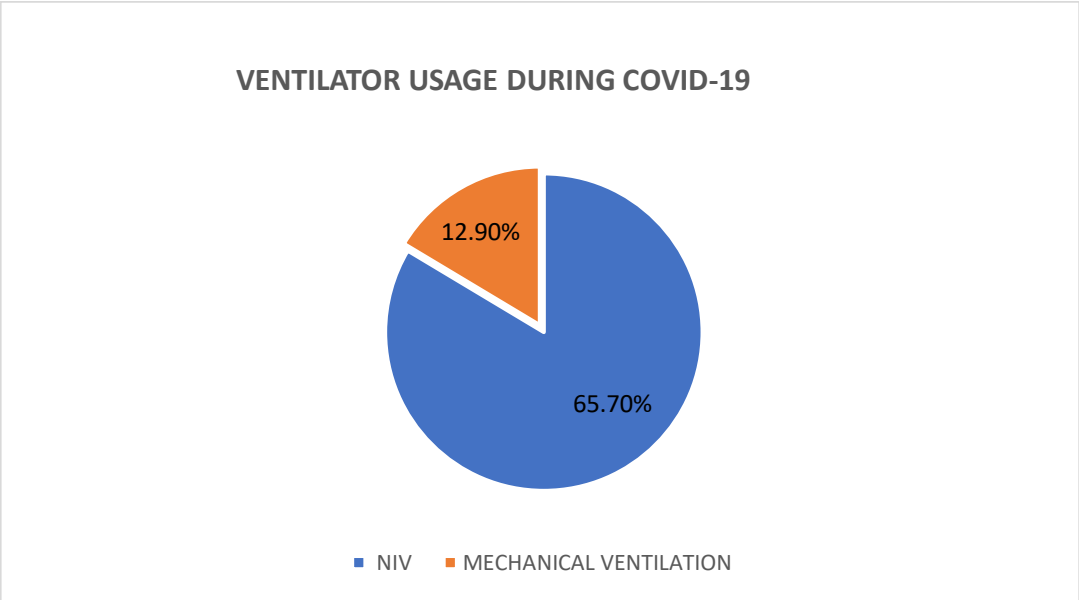


In this study, restrictive PFT pattern was seen more in the overall study population, which was significantly associated with the severity of the disease during covid-19 hospital admission . 2 out of 42 mild CTSS patients and 5 out of 47 severe CTSS patients had mixed pattern in PFT.7 out of 19 patients with mild CTSS,30 out of 42 patients with moderate CTSS and 40 out of 47 patients with severe CTSS had restrictive PFT and is statistically significant (P=0.0001).

Table : 8 VENTILATOR USAGE DURING COVID-19

VENTILATOR USAGE	NUMBER	PERCENT
NIV	71	65.7 %
MECHANICAL VENTILATION	14	12.9%

Diagram :8



In this study 71 (65.7%) patients were on NIV and 14(12.9%) patients were on mechanical ventilation during their covid -19 hospital admission.

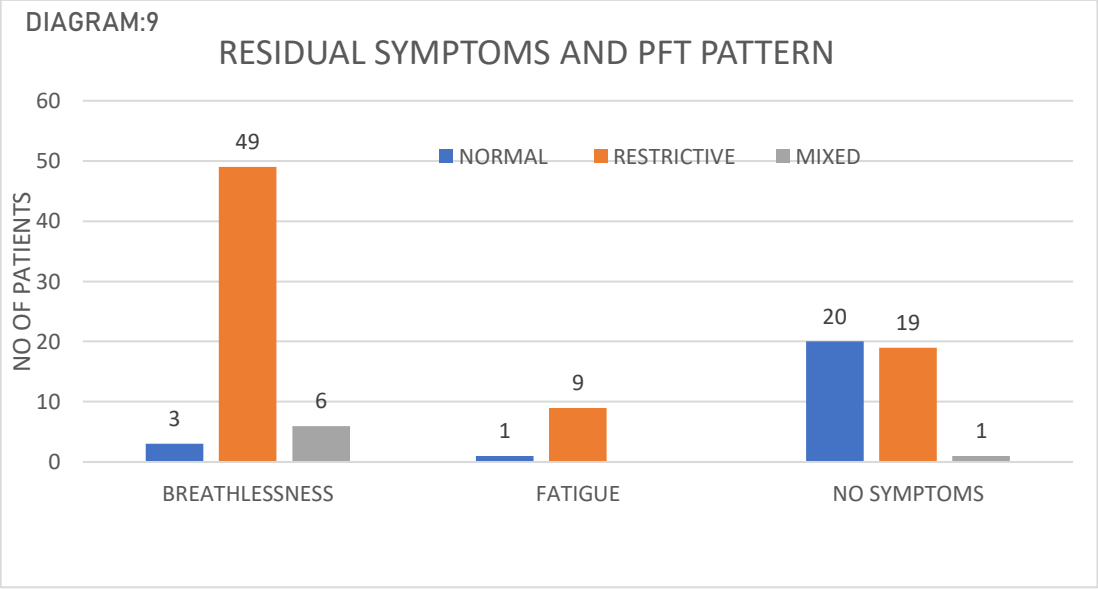
Table :9 ASSOCIATION OF VENTILATOR USAGE DURING COVID-19 WITH FOLLOW-UP LUNG FUNCTION TEST

VENTILATOR USAGE DURING COVID-19 ADMISSION	FOLLOW-UP PFT PATTERN				P value
	NORMAL	RESTRICTIVE	MIXED	TOTAL	
INVASIVE VENTILATION	2	11	1	14	0.74
NIV	5	60	6	71	0.001

In this study 71 patients had history of NIV usage and 14 patients had history on invasive ventilation during their covid-19 hospital admission. During the follow-up lung function test at 6 months ,60 patients had restrictive pattern, 6 patients had mixed pattern and 5 patients had normal PFT pattern among the NIV patients . 11 patients had restrictive pattern, 1 patient had mixed pattern and 2 patients had normal PFT pattern among the patients with history of invasive ventilation. Restrictive lung function test was significantly associated with the use of NIV during covid-19 hospital admission (P= 0.001).

Table : 10 FOLLOW-UP PFT PATTERNS ACCORDING TO THE RESIDUAL SYMPTOMS DURING FOLLOW-UP

		FOLLOW-UP PFT PATTERN			Total	P value
		NORMAL	RESTRICTIVE	MIXED		
MAIN COMPLAINTS AT FOLLOW UP	BREATHLESSNESS ON EXERTION	3	49	6	58	0.001
	FATIGUE	1	9	0	10	
	NO SYMPTOMS	20	19	1	40	
Total		24	77	7	108	



In this study, during the follow-up at 6 months ,58 patients had breathlessness on exertion and 10 patients had fatigue as main residual symptoms. During their follow-up lung function test ,49 out of this 58 patients with breathlessness on exertion and 9 out of 10 patients with fatigue had restrictive pattern in their lung function test and was statistically significant (p=0.001).

Table :11 CORRELATION OF FOLLOW-UP PFT PATTERN

WITH THE LENGTH OF HOSPITAL STAY ONE-WAY ANOVA ANALYSIS

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
NORMAL	24	6.04	2.926	0.597	4.81	7.28	4	16
RESTRICTIVE	77	9.44	3.370	0.384	8.68	10.21	3	17
MIXED	7	8.00	1.155	0.436	6.93	9.07	7	10
Total	108	8.59	3.462	0.333	7.93	9.25	3	17

Table :112 MULTIVARIENT ANOVA ANALYSIS

Multiple Comparisons

Dependent Variable:	Length of hospital stay					
Tukey HSD						
(I) PFT PATTERN		Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
NORMAL	RESTRICTIVE	-3.400*	0.746	0.000	-5.17	-1.63
	MIXED	-1.958	1.370	0.330	-5.22	1.30
RESTRICTIVE	NORMAL	3.400*	0.746	0.000	1.63	5.17
	MIXED	1.442	1.259	0.489	-1.55	4.43
MIXED	NORMAL	1.958	1.370	0.330	-1.30	5.22
	RESTRICTIVE	-1.442	1.259	0.489	-4.43	1.55

*. The mean difference is significant at the 0.05 level.

The average length of hospital stay in our study population was 8.59 days , with a maximum being 17 days and a minimum being 3 days.

Using statistical analysis there was a statistically significant difference between groups as determined by one-way ANOVA ($p = < 0.01$).

A Tukey post hoc test revealed that the length of hospital stay was statistically significantly higher with Restrictive PFT pattern during the follow up (9.44 ± 3.37 min, p = < 0.01) compared to the Normal PFT pattern (6.04 ± 2.9 min). There was no statistically significant difference between the normal and Mixed PFT patterns (p = 0.33).

Suggesting that the increase in the length of hospital stay was significantly associated with the restrictive lung function test.

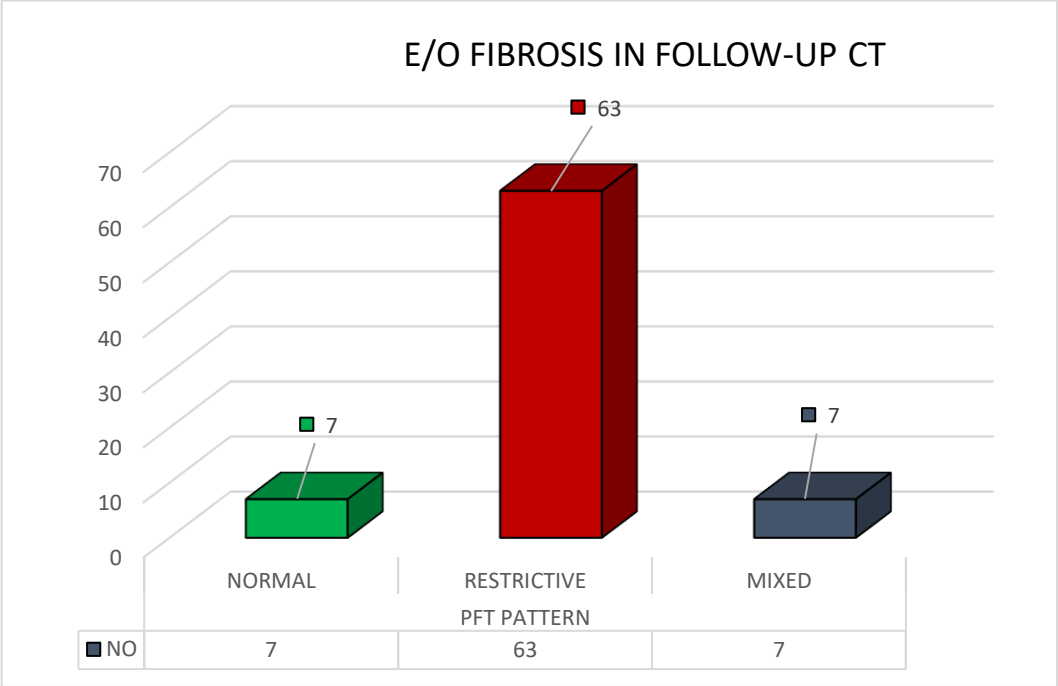
Table : 13 HRCT FINDINGS AT THE FOLLOW-UP

Ground-glass opacity	2
Reticulations	6
Traction bronchiectasis	9
Fibro parenchymal bands	50
Septal thickening	33
Total (n)	77

Table : 14 ASSOCIATION OF FIBROSIS WITH WITH FOLLOW-UP LUNG FUNCTION TEST

	PFT PATTERN				
FACTOR	**NORMAL**	**RESTRICTIVE**	**MIXED**	**Total**	**P value**
E/O FIBROSIS IN HRCT	**7**	**63**	**7**	**77**	**0.001**

Diagram : 10



Out of 108 follow-up patients with HRCT thorax, 77 patients had some evidence of fibrosis in the form of Fibroparenchymal bands in 50 patients (46%), septal thickening in 33 patients (30.5%) , evidence of traction bronchiectasis in 9 patients (8.3%) , reticulations in 6 patients(5.5%).Out of 77 patients ,63 patients had restrictive lung function pattern and 7 patients had normal and mixed PFT pattern ,which is statistically significant (P=0.001).

DISCUSSION

From November 2021 the pandemic of covid 19 started and the whole world was taken into stake. This pandemic has affected 672 million of people and taken many lives including younger and older. Being a novel viral disease, it became very difficult to form the guidelines about treatment , precautionary measures , vaccination and many things . It took everyone to understand its presentation, severity, virulence and behaviour. The disease started surfacing after the acute illness also leaving behind its impact largely on the society and that is how the term "post covid syndrome " came into the picture .

Post-COVID-19 syndrome is a name that refers to sequelae that occur during or after an infection with SARS CoV-2 and remain for more than 12 weeks after the infection has cleared up[47]. Post covid patients report persisting symptoms at six months including exhaustion, dyspnoea, anxiety, depression, and problems with focus, memory, attention, sleep ,Post-COVID fibrosis(PCPF) [53]. Post-COVID fibrosis is diagnosed using a combination of clinical, radiographic, and pathology information [53]. Clinically, individuals with severe COVID-19 disease (WHO Severity Grades 3 and 4) are more likely to have a restrictive ventilatory defect, which is associated with significantly reduced diffusing capacity for many weeks after hospital discharge. The commonly found CT findings are fibrotic changes, reticular pattern , and traction bronchiectasis with or without honeycombing as a features of PCPF [56].

COVID-19 has affected the worldwide with large number of population being involved , it is essential that long-term complications and sequelae should be known , studied and treated. Although there is evidence suggesting that the patients who have persistent symptoms may benefit from a carefully designed pulmonary rehabilitation program, but the effectiveness is still unknown. This might be due to the fact that the patients with COVID-related conditions tend to have many symptoms.

Post-COVID 19 pulmonary fibrosis of mild to serious forms ,may need long-term oxygen therapy or even lung transplantation in many of the patients [57].

In this study Post-COVID 19 patients who visited respiratory medicine outpatient department within 3 to 6 months after discharge were followed up with high-resolution computed tomography(HRCT) thorax, and lung function tests were done within 3 to 6 months post discharge.

In this study , the initial lung involvement were compared with the follow-up HRCT findings . The lung function test along with other variables like the severity of the disease, duration of hospital stay, use of mechanical ventilation, age, sex, the role of smoking history and other comorbidities were studied.

In this study average age of the patients were 52 years ,with the maximum being 75 years and the minimum being 23 years. overall male preponderance was seen among COVID-19 patients, similar male preponderance was observed in the present study as well. In this study majority of the patients were males 60.2% . This results were comparable to studies done by Han et al[58] and Gu et al [59] in post COVID patients .

Out of the 108 admitted COVID-19 patients the an average BMI of patients was 25.45 . According to the World Health Organization, 80% of SARS-CoV-2 infections are mild, 14% develop severe symptoms, and 6% will become critically ill. Factors associated with increased disease severity include comorbidities such as hypertension, diabetes, and coronary artery disease[60].

During the follow-up all 108 patients underwent thorough history taking and physical examination. None of the patients had any form of long-stand pre-existing parenchymal or airway problems in their medical records. No patient had pre-existing OSA, spine or chest wall deformity.

In this study patients had co-morbidities like T2DM in 52 (48%) , IHD in 36 (33%), Hypertension seen in 50 (46%), and CKD in 3(2.8%) patients. During the follow-up restrictive PFT pattern was Positively associated with comorbidities. 44 out of 52 T2DM patients had a restrictive PFT pattern, showing a statically significant association (P=0.01).Whereas 37 out of 50 hypertensive patients had a restrictive PFT pattern (P=0.84) and 30 out of 36 IHD patients had a restrictive PFT pattern (P=0.14) but was not statically significant.

Smoking has been linked to the pathogenesis of various lung diseases such as emphysema, chronic bronchitis, and pulmonary fibrosis. Epidemiological studies show a high incidence of familial and sporadic IPF in smokers when compared to nonsmokers[61] . Smoking is associated with chronic oxidative stress, increased expression of inflammatory cytokines, and interstitial lung fibrosis[62]. The injury associated with smoking continues even after cessation[61].

In this study 54 .6% (n=59) of the patients were non smokers with

25 .9 % (n=28) being smokers and 19.4 % (n=21) being exsmokers. During follow-up n the present study 42 out of 59 non-smokers (P=0.9) and 15 out of 28 active smokers (P=0.05) had restrictive PFT pattern during their follow-up with no statistical significance .20 out of 21 ex-smokers with significant smoking history had restrictive PFT pattern and was statistically significant (P=0.025). A study by Zhang et al in post-COVID patients showed history of smoking was positively associated with restrictive PFT pattern during the follow up [63].

In covid 19 patients CTSS shows the severity of the lung involvement which shows the extend of lung injury and it correlates with the fibroblastic response following the injury .In the present study Out of 108 patients, initial CT thorax showing a CT severity of mild in 19(17.6%) , moderate in 42 (38.9%), severe in 47 (43.5%) with a mean CTSS score of

14.7 / 25 .In this study, during the follow-up restrictive PFT pattern was seen more in the overall study population, which was significantly associated with the severity of the disease during covid-19 hospital admission. 2 out of 42 mild CTSS patients and 5 out of 47 severe CTSS patients had mixed pattern in PFT.7 out of 19 patients with mild CTSS,30 out of 42 patients with moderate CTSS and 40 out of 47 patients with severe CTSS had restrictive PFT and is statistically significant (P=0.0001).The results were comparable to a study by **Rasha et al** in 80 Post -COVID-19 patients which demonstrated a higher incidence of postCOVID-19 pulmonary fibrosis with severe CTSS group (42.8%) as compared to the moderate CTSS group[53].

In this study, during the follow-up at 6 months ,58 patients had breathlessness on exertion and 10 patients had fatigue as main residual symptoms. Among the patients who had breathlessness ,23 patients had MMRC grade 1 breathlessness, 18 patients had MMRC grade 2 breathlessness,15 patients had MMRC grade 3 breathlessness and among them 19 out of 23 patients with MMRC grade 1 ,15 out of 18 with MMRC grade 2, 13 out of 15 patients with MMRC grade 3 had a restrictive pattern in lung function which is statistically significant (p=0.002). suggesting that the patients with persistent symptoms, mainly breathlessness even after 3 months of discharge has a reduction in lung function mainly due to lung parenchymal fibrosis. This was in association with a study by **Jessica et al** on 41 severe COVID 19 patients also concluded a persistence of symptoms, in addition to a significant change in PFT in the post-discharge assessment of patients requiring hospitalization after admission for COVID-19 [64].

In the present study, the spirometric assessment done within 6 months after discharge showed a mean FVC of 69.72, a mean FEV1 of 68.32, with a mean FEV1 / FVC being 94.06, with a mean MVV of 64.7, mean FEF 25-75 being 72.76, with a mean VC max being 72.04.These values in the lung function test is suggestive of a restrictive pattern.

These result are consistent with one of the study by **Ivan et al** in 43 symptomatic Post-COVID-19 Patients where they concluded that a considerable proportion of follow-up patients presented with persistent symptoms and change in PFT, mainly a restrictive pattern and low DLCO [49].

In this study 71 patients had history of NIV usage and 14 patients had history on invasive ventilation during their covid-19 hospital admission. During the follow-up restrictive PFT pattern was associated with the use of invasive ventilation during COVID-19 hospital admission, with 60 out of 71 NIV patients and 11 out of 14 intubated patients had restrictive PFT pattern(P=0.001), suggesting that the patients who required NIV during the hospital stay were more at risk of developing a parenchymal insult leading to reduction in lung functions. These results are comparable to a study by **Rasha et al** on COVID-19 patients who had history of ventilatory support showed reduction in lung function during the follow-up [53].

In our study out of 108 follow-up patients with HRCT thorax, 77 patients had some evidence of fibrosis in the form of Fibroparenchymal bands in 50 patients (46%), septal thickening in 33 patients(30.5%) , evidence of traction bronchiectasis in 9 patients (8.3%) , reticulations in 6 patients(5.5) . This results were comparable with a study **Belen et al** on 481 patients of COVID-19, HRCT at 6 months follow up showed ground glass opacities and parenchymal bands in 68.35% of patients with severe disease [65]. In the present study among these 77 patients who had evidence of fibrosis in the follow-up HRCT thorax, 62 patients showed restrictive lung function pattern which is statistically significant (P=0.001).Suggesting that in the follow-up scan the evidence of fibrosis is associated with a reduction in the lung function of these patient and the presence of these patterns in the follow-up HRCT helps in the early pickup of these patients. The results was comparable to another study **by Hafeda et al** in 84 post-COVID patients, that concluded significant

positive association between these fibrotic-like changes in follow up HRCT and lung function [32]. The average length of hospital stay in our study population was 8.59. Multivariate ANOVA analysis revealed that the length of hospital stay was statistically significantly higher with Restrict PFT pattern (9.44 ± 3.37 min, p = < 0.01) compared to the normal PFT pattern (6.04 ± 2.9 min),suggesting that the increase

in the length of hospital stay was significantly associated with the restrictive lung function test. The results were comparable to a study by **Rabab et al** on 210 post-COVID 19 individuals also, concluded the length of hospital stay was significantly associated with the restrictive pattern in the lung function test [50].Our study results were comparable to outcomes of other similar studies and these patients need regular follow-ups, the benefits of antifibrotics in post-COVID patients to be studied in detail.

LIMITATIONS:

1.Major limitation of this study was a single centre study with only 108 participants.

2.DLCO and 6 minute walk test was not performed during the follow up.

3.Usage of antifibrotics in post COVID patients were not taken into consideration.

SUMMARY

- In this study in total of 108 patients, 42 out of 65 male patients and 35 out of 33 female patients had a restrictive pattern in PFT but was not associated with any statistical significance (P=0.097).
- In this study patients had an average BMI of 25.45,out of the 4 underweight patients, 24 out of the 29 normal patients,9 out of the 20 overweight patients and 41 out of the 55 obese patients had a restrictive pattern in lung function test and was not statistically significant (P=0.058).
- In the study population, the mean FVC of 69.72, mean FEV1 of 68.32, the mean FEV1 / FVC was 94.06 , the mean MVV of 64.7, the mean FEF 25-75 being 72.76, with a mean VC max being 72.04, these values in the lung function test is suggestive of a restrictive pattern.
- In this study 20 out of 21 ex-smokers with significant smoking history had restrictive PFT pattern and was statistically significant (P=0.025).
- In this study 44 out of 52 T2DM patients had a restrictive PFT pattern, showing a statistically significant association (P=0.01).
- With respect to hypertension and IHD , 37 out of 50 hypertensive patients had a restrictive PFT pattern(P=0.84) and 30 out of 36 IHD patients had a restrictive PFT pattern but was statically not significant (P=0.14).
- The restrictive pattern in lung function test was also associated with the use of invasive ventilation during the COVID-19 hospital admission with 60 out of 71 NIV patients and 11 out of 14 intubated patients having restrictive PFT pattern(P=0.001).

- In this study population 19 out of 23 patients with MMRC grade 1 ,15 out of 18 with MMRC grade 2, 13 out of 15 patients with MMRC grade 3 had a restrictive pattern in lung function which is statistically significant (p=0.002)
- Out of 108 follow up patients with HRCT thorax, 77 patients had some evidence of fibrosis with 62 patients among them showing restrictive lung function patterns which is statistically significant (P=0.001).
- A Tukey post hoc test revealed that the length of hospital stay was statistically significantly higher with Restrict PFT pattern (9.44 ± 3.37 min, p = < 0.01) compared to the Normal PFT pattern (6.04 ± 2.9 min). There was no statistically significant difference between the normal and Mixed PFT patterns (p = 0.33). Suggesting that the increase in the length of hospital stay was significantly associated with the restrictive lung function test.

CONCLUSION

From this study it is concluded that the prevalence of post COVID 19 pulmonary sequelae is seen higher than expected in the population, especially seen more among patients with comorbidities like T2DM, significant past history of smoking, with increased CT severity score suggesting of advanced lung involvement, use of ventilatory support during admission, with increased duration of hospital stay which showed statistical significance. Hence post-COVID pulmonary sequelae is of great concern and these patients need regular follow-up and pulmonary rehabilitation. The role of antifibrotics and pulmonary rehabilitation needs to be studied in detail.

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ANNEXURE 1

PROFORMA

CASE NO	
IP/ OP NO	
AGE	
SEX	
ADDRESS	
OCCUPATION	
MOBILE NO:	
BMI	

	YES	NO
CONTRAINDICATION FOR SPIROMETRY		
COMORBIDITIES		

Vitals during follow-up :

Temperature	
Pulse	
Respiratory Rate	
Blood Pressure	
SpO2	

AT ADMISSION	Covid status	HRCT/CT findings and Severity score

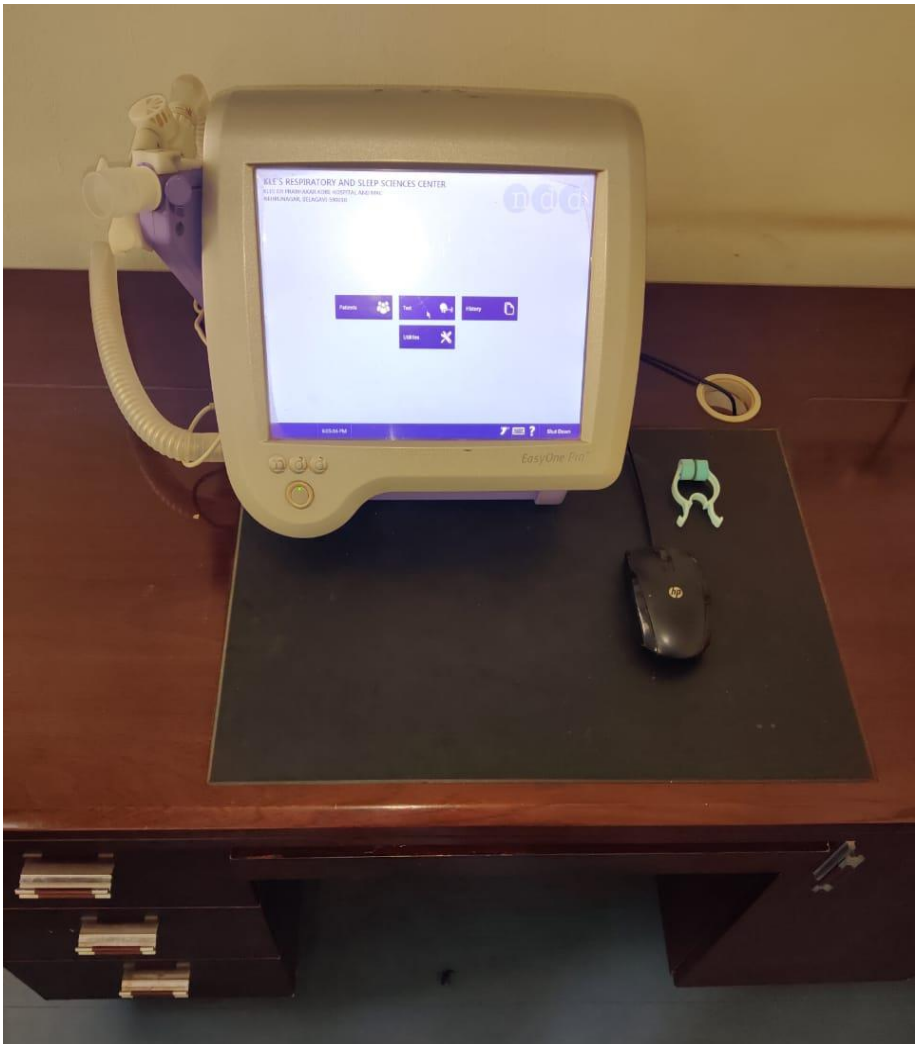
INVESTIGATIONS AT FOLLOW-UP:

HRCT/CT thorax **Findings**	**PFT STATUS**

COMPLAINTS AT FOLLOW-UP:

ANNEXURE II -PHOTOGRAPHS

PFT MACHINE



ANNEXUR-III KEY TO MASTER CHART

FEV1	-	Forced expiratory volume in one second
FVC	-	Forced vital capacity
PEF	-	Peak expiratory fow
MEF50	-	Maximal expiratory fow at 50% of FVC
MEF25	-	Maximal expiratory fow at 25% of FVC
MMEF75/25	-	Maximal midexpiratory fow between 75 and 25% of FVC
MVV	-	Maximum voluntary ventilation
RV	-	Residual volume
HTN	-	Hypertension
IHD	–	Ischemic heart disease
T2DM	-	Type 2 diabetes mellitus
COPD	–	Chronic Obstructive Pulmonary Disease
CTSS	–	CT severity score

ANNEXUR-IV-MASTERCHART

MASTER CHART

SL NO	AGE	SEX	BMI	SMOKER	EXSMOKER	NON SMOKER	HRCT THORAX CORADS SCORE	CTSS SCORE	LEFT BLANK	CTSS CATEGORY	REMDESVIR (antiviral injection)	STERIODS	NIV DURING COVID	intubated	Length of hosp stay	HTN	T2DM	IHD	COPD	BA	OTHERS	SPINE / CHEST WALL ABNORMALITIES	FVC	FEV1	SPIROMETRY VALUES FEV1 TO FVC RATIO	PEF	FEF 25-75	MVV	VC	VC MAX	CTSS SCORE	CTSS CATEGORY	post covid FOLLOW UP HRCT	post covid FOLLOW UP HRCT fibrotic changes	COMPLICATIONS	E/O FIBROSIS	MAIN COMPLAINTS AT FOLLOW UP	FOLLOW UP VITALS SP02
1	40	M	24.2	NO	NO	YES	5	21		SEVERE	YES	YES	YES	NO	12	NO	YES	NO	NO	NO	NIL	NIL	31	36	119	39	61	45	35	35	21	SEVERE	LEFT PNEUMOTHORAX	yes	FT PNEUMOTHORAX / YE	YES	BREATHLESSNESS ON EXERTION	94
2	46	M	24	NO	NO	YES	5	15		MODERATE	YES	YES	YES	NO	13	NO	NO	NO	NO	NO	NIL	NIL	54	51	93	29	43	80	60	60	15	MODERATE	B/L RETICULAR PATTERN IN B/L LL WITH TRACTION BRONCHIECTASIS	yes	NIL	YES	fatigue	90
3	45	M	25.17	NO	NO	YES	5	12		MODERATE	YES	YES	NO	NO	9	NO	YES	NO	NO	NO	NIL	NIL	50	54	109	83	77	82	43	50	12	MODERATE	FIBRROTIC BANDS WITH LLL CONSOLIDATION	yes	NIL	YES	NIL	98
4	23	M	32.5	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	6	NO	NO	NO	NO	NO	NIL	NIL	67	66	97	50	75	44	64	67	10	MODERATE	FEW FIBROTIC BANDS	yes	NIL	YES	NIL	98
5	30	M	27.6	NO	YES	NO	5	16		SEVERE	YES	YES	YES	NO	9	NO	NO	NO	NO	NO	NIL	NIL	73	78	107	80	120	106	68	73	16	SEVERE	E/O FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	90
6	38	M	22.9	NO	NO	YES	5	6		MILD	NO	NO	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	76	81	108	119	133	96	69	76	6	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
9	59	M	38.7	YES	NO	NO	5	15		MODERATE	YES	YES	YES	NO	5	YES	NO	NO	NO	NO	CKD	NIL	80	87	80	110	126	76	87	87	15	MODERATE	NORMAL STUDY	NO	NIL	NO	BREATHLESSNESS ON EXERTION	99
8	62	M	30.4	YES	NO	NO	4	6		MILD	NO	NO	NO	NO	4	YES	YES	YES	NO	NO	NIL	NIL	68	64	96	23	55	80	74	74	6	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
9	59	M	38.7	YES	NO	NO	5	15		MODERATE	YES	YES	YES	NO	7	YES	NO	NO	NO	NO	CKD	NIL	80	87	80	110	126	76	87	87	15	MODERATE	NORMAL STUDY	NO	NIL	YES	NIL	99
10	40	M	22.7	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	11	NO	NO	NO	NO	NO	NIL	NIL	55	55	100	43	59	68	47	58	18	SEVERE	FEATURES OF FIBROSIS	yes	NIL	NO	BREATHLESSNESS ON EXERTION	93
11	45	M	22.7	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	5	YES	YES	NO	NO	NO	NIL	NIL	67	66	92	50	75	44	43	78	10	MODERATE	FIBROTIC BANDS	yes	NIL	YES	NIL	99
12	75	M	16.2	YES	NO	YES	5	15		MODERATE	YES	YES	YES	yes	7	NO	NO	YES	YES	NO	NIL	NIL	46	58	78	60	19	45	35	58	15	MODERATE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
13	74	M	23.07	YES	NO	NO	5	8		MILD	YES	YES	NO	NO	5	YES	YES	NO	NO	NO	NIL	NIL	110	132	83	61	67	82	125	132	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	98
14	40	M	23.8	NO	NO	YES	5	8		MILD	YES	YES	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	77	81	105	89	88	82	64	77	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
15	61	M	30	YES	NO	NO	5	21		SEVERE	YES	YES	YES	yes	16	YES	YES	YES	NO	NO	NIL	NIL	86	94	110	46	104	77	83	86	21	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
16	52	M	35.5	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	4	YES	YES	NO	NO	NO	NIL	NIL	77	73	106	129	102	60	67	73	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	98
17	64	M	25.5	YES	NO	NO	5	15		MODERATE	YES	YES	YES	yes	14	YES	NO	YES	NO	NO	NIL	NIL	43	50	118	64	85	41	44	43	15	MODERATE	LEFT PNEUMOTHORAX	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
18	45	M	22.7	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	7	YES	YES	NO	NO	NO	NIL	NIL	67	66	92	50	75	44	43	78	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	99
19	26	F	32.8	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	6	NO	NO	NO	NO	NO	NIL	NIL	101	109	106	126	164	98	95	100	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	98
20	48	F	25	NO	NO	YES	5	15		MODERATE	YES	YES	YES	yes	16	YES	YES	NO	NO	NO	NIL	NIL	55	50	90	30	43	80	48	60	15	MODERATE	RIGHT PNEUMOTHORAX	yes	GHT PNEUMOTHORAX/Y	YES	BREATHLESSNESS ON EXERTION	92
21	50	F	34.7	NO	YES	NO	5	21		SEVERE	YES	YES	YES	yes	17	YES	YES	NO	NO	NO	NIL	NIL	62	61	47	27	13	44	62	65	21	SEVERE	SUB PLEURAL ATELECTASIS OF L ML AND L LL	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
22	40	F	28.2	NO	YES	NO	5	16		SEVERE	YES	YES	YES	NO	10	NO	NO	NO	NO	NO	NIL	NIL	74	76	102	82	120	104	68	73	16	SEVERE	FEW FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
23	70	M	17.2	YES	NO	NO	5	8		MILD	NO	NO	NO	NO	5	YES	YES	YES	NO	NO	NIL	NIL	46	58	78	60	19	45	46	58	18	SEVERE	B/L LL WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
24	71	F	22.08	NO	YES	NO	5	21		SEVERE	YES	YES	YES	yes	15	NO	NO	YES	NO	NO	NIL	NIL	64	56	87	27	42	48	64	66	21	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
25	62	M	20.5	NO	YES	NO	5	21		SEVERE	YES	YES	YES	yes	14	YES	NO	NO	NO	NO	NIL	NIL	59	58	99	34	50	38	59	60	21	SEVERE	FEATURES OF FIBROSIS	yes	LEFT EMPYEMA/YES	YES	BREATHLESSNESS ON EXERTION	95
26	71	F	22.08	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	10	YES	YES	YES	NO	NO	NIL	NIL	50	56	112	30	50	48	64	66	18	SEVERE	FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
27	42	F	22.9	NO	NO	YES	5	23		SEVERE	YES	YES	YES	yes	14	NO	NO	NO	NO	NO	NIL	NIL	50	47	93	31	35	46	50	52	23	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
28	65	F	20.7	NO	YES	NO	5	23		SEVERE	YES	YES	YES	yes	16	NO	YES	YES	NO	NO	NIL	NIL	59	58	99	33	50	36	59	62	23	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
29	25	M	22.9	NO	NO	YES	5	10		MODERATE	NO	NO	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	80	81	102	57	91	60	82	84	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	99
30	59	F	29.6	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	6	NO	YES	YES	NO	NO	NIL	NIL	66	66	100	88	63	74	66	68	10	MODERATE	B/L FIBROTIC BANDS	yes	NIL	YES	FATIGUE	99
31	67	M	22.08	YES	NO	NO	5	18		SEVERE	YES	YES	YES	NO	8	YES	YES	YES	NO	NO	NIL	NIL	73	79	86	89	88	82	73	77	18	SEVERE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
32	65	F	22.2	NO	NO	YES	5	21		SEVERE	YES	YES	YES	yes	11	NO	YES	YES	NO	NO	NIL	NIL	52	50	96	26	42	44	52	52	21	SEVERE	FIBROTIC BAND WITH TRACTION	NO	NIL	YES	BREATHLESSNESS ON EXERTION	96
33	45	F	23	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	92	79	85	38	47	86	92	94	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
34	32	M	28.7	NO	NO	YES	5	21		SEVERE	YES	YES	YES	NO	10	NO	NO	NO	NO	NO	NIL	NIL	69	75	109	112	86	96	69	72	21	SEVERE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
35	42	M	17.6	YES	NO	YES	5	23		SEVERE	YES	YES	YES	yes	16	NO	YES	NO	NO	NO	NIL	NIL	31	29	93	17	24	28	26	31	23	SEVERE	B/L FIBROSIS WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
36	27	F	22	NO	NO	YES	5	22		SEVERE	YES	YES	YES	NO	12	NO	NO	NO	NO	NO	NIL	NIL	73	73	99	60	69	60	73	87	22	SEVERE	E/O FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
37	64	M	25.5	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	6	YES	YES	YES	NO	NO	NIL	NIL	115	116	100	48	108	55	106	116	8	MILD	FEW FIBROTIC BANDS	yes	NIL	YES	fatigue	99
38	73	F	26.6	NO	NO	YES	5	13		MODERATE	YES	YES	YES	NO	8	NO	YES	YES	NO	NO	NIL	NIL	68	67	97	45	63	53	68	69	13	MODERATE	B/L LL FIBROTIC BANDS	yes	NIL	YES	fatigue	97
39	67	M	26.8	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	11	YES	NO	YES	NO	NO	NIL	NIL	73	79	109	78	117	56	65	75	18	SEVERE	SUB PLEURAL ATELECTASIS OF LLL	yes	NIL	YES	BREATHLESSNESS ON EXERTION	97
40	71	F	22.1	NO	YES	NO	5	21		SEVERE	YES	YES	YES	NO	12	NO	YES	YES	NO	NO	NIL	NIL	63	54	85	27	40	48	63	66	21	SEVERE	B/L LL FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
41	46	M	30.5	YES	NO	NO	5	20		SEVERE	YES	YES	YES	NO	8	YES	NO	NO	NO	NO	NIL	NIL	45	43	96	30	42	53	41	45	20	SEVERE	B/L FIBROSIS	yes	NIL	NO	NIL	96
42	23	F	22.4	NO	NO	NO	5	10		MODERATE	NO	YES	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	74	73	98	59	81	53	65	69	10	MODERATE	NORMAL STUDY	yes	NIL	NO	NIL	98
43	60	M	26	YES	NO	NO	5	15		MODERATE	YES	YES	YES	NO	6	YES	YES	YES	NO	NO	NIL	NIL	66	63	95	47	64	58	52	60	15	MODERATE	B/L LL FIBROTIC BANDS	NO	NIL	YES	BREATHLESSNESS ON EXERTION	95
44	46	M	23.6	YES	NO	NO	4	8		MODERATE	YES	YES	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	96	88	91	66	90	72	90	98	8	MILD	NORMAL STUDY	yes	NIL	YES	NIL	98
45	50	M	26.9	NO	YES	NO	5	8		MODERATE	NO	YES	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	86	86	100	100	87	83	86	77	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
46	66	F	25.6	NO	YES	NO	5	20		SEVERE	YES	YES	YES	NO	9	YES	YES	YES	NO	NO	NIL	NIL	48	32	66	19	30	46	45	48	20	SEVERE	B/L FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
47	58	F	27	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	11	NO	YES	YES	NO	NO	NIL	NIL	58	53	91	95	88	50	54	56	18	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	EFT PNEUMOTHORAX/YE	YES	BREATHLESSNESS ON EXERTION	94
48	52	M	24.16	YES	NO	NO	5	20		SEVERE	YES	YES	YES	NO	10	YES	NO	NO	NO	NO	NIL	NIL	100	65	65	41	31	69	91	100	20	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
49	60	F	26	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	9	YES	YES	YES	NO	NO	NIL	NIL	68	66	97	78	76	65	68	56	18	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
50	65	M	25	YES	NO	NO	5	16		SEVERE	YES	YES	YES	NO	9	YES	YES	NO	NO	NO	NIL	NIL	58	53	91	90	84	56	60	65	16	SEVERE	B/L FIBROSIS WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
51	56	F	26.4	NO	NO	YES	5	12		MODERATE	YES	YES	YES	NO	7	NO	YES	YES	NO	NO	NIL	NIL	65	60	92	87	86	55	63	70	18	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	RIGHT EMPYEMA/YES	YES	BREATHLESSNESS ON EXERTION	94
52	43	M	23.6	NO	NO	YES	5	18		MODERATE	NO	YES	YES	NO	9	YES	NO	NO	NO	NO	NIL	NIL	54	53	98	52	63	56	40	60	18	SEVERE	B/L FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
53	50	F	26.5	NO	NO	YES	5	12		MODERATE	YES	YES	NO	NO	7	NO	YES	NO	NO	NO	NIL	NIL	74	73	98	60	86	60	66	66	12	MODERATE	B/L FIBROTIC BANDS	yes	NIL	YES	NIL	98
54	48	M	24.6	YES	NO	NO	5	15		MODERATE	YES	YES	YES	NO	7	NO	YES	NO	NO	NO	NIL	NIL	60	54	90	56	68	56	50	64	15	SEVERE	B/L RETICULAR BANDS	NO	NIL	YES	BREATHLESSNESS ON EXERTION	95
55	44	F	25	NO	NO	YES	5	18		SEVERE	YES	YES	YES	NO	8	NO	NO	NO	NO	NO	NIL	NIL	100	65	65	41	40	55	88	96	18	SEVERE	SUB PLEURAL ATELECTASIS OF L ML AND L LL	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
56	68	M	24	YES	NO	NO	5	12		MODERATE	YES	YES	YES	NO	12	YES	YES	YES	NO	NO	NIL	NIL	66	63	95	48	70	50	56	60	12	MODERATE	SEPTAL THICKENING	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
57	60	F	25.5	NO	NO	YES	5	18		SEVERE	YES	YES	YES	NO	10	YES	YES	YES	NO	NO	NIL	NIL	70	66	94	50	74	54	60	60	18	SEVERE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
58	46	F	24.8	NO	NO	YES	5	12		MODERATE	YES	YES	NO	NO	6	NO	YES	NO	NO	NO	NIL	NIL	74	73	98	66	90	64	66	68	12	MODERATE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
59	42	M	19.8	NO	NO	YES	5	12		MODERATE	YES	YES	NO	NO	7	NO	NO	NO	NO	NO	NIL	NIL	100	65	65	40	58	55	88	94	12	MODERATE	B/L FIBROTIC BANDS	yes	NIL	YES	NIL	98
60	63	M	31.2	NO	NO	YES	5	15		MODERATE	YES	YES	YES	NO	11	NO	YES	YES	NO	YES	NIL	NIL	60	67	111	69	99	55	57	60	15	MODERATE	LL FIBROTIC BANDS IN LLL	yes	NIL	YES	fatigue	98
61	74	M	23.07	YES	NO	NO	5	8		MILD	NO	NO	NO	NO	8	YES	YES	NO	NO	NO	NIL	NIL	110	132	83	61	67	82	125	132	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
62	61	M	30	YES	NO	NO	5	21		SEVERE	YES	YES	YES	yes	14	YES	YES	NO	NO	NO	NIL	NIL	86	94	110	46	104	77	83	86	21	SEVERE	E/O FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
63	70	M	17.2	YES	NO	NO	5	8		MILD	YES	YES	NO	NO	8	YES	YES	YES	NO	NO	NIL	NIL	46	58	78	60	19	45	46	58	18	SEVERE	B/L LL WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	97
64	59	M	38.7	YES	NO	NO	5	15		MODERATE	YES	YES	YES	NO	5	YES	NO	YES	NO	NO	CKD	NIL	80	87	80	110	126	76	87	87	15	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	98
65	67	M	22.08	YES	NO	NO	5	18		SEVERE	YES	YES	YES	NO	9	YES	YES	YES	NO	NO	NIL	NIL	73	79	86	89	88	82	73	77	18	SEVERE	E/O FIBROSIS	yes	NIL	YES	fatigue	97
66	27	F	22	NO	YES	NO	5	22		SEVERE	YES	YES	YES	NO	12	NO	NO	NO	NO	NO	NIL	NIL	73	73	99	60	69	60	73	87	22	SEVERE	E/O FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
67	62	M	30.4	YES	NO	NO	4	6		MILD	NO	NO	NO	NO	3	YES	YES	YES	NO	NO	NIL	NIL	68	64	96	23	55	80	74	74	6	MILD	NORMAL STUDY	NO	NIL	NO	NIL	95
68	46	M	24	NO	NO	YES	5	15		MODERATE	NO	NO	YES	NO	6	NO	NO	NO	NO	NO	NIL	NIL	54	51	93	29	43	80	60	60	15	MODERATE	B/L RETICULAR PATTERN IN B/L LL	yes	NIL	YES	fatigue	98
69	65	F	22.2	NO	YES	NO	5	21		SEVERE	YES	YES	YES	NO	12	NO	YES	YES	NO	NO	NIL	NIL	52	50	96	26	42	44	52	52	21	SEVERE	FIBROTIC BAND WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
70	52	M	24.16	YES	NO	NO	5	20		SEVERE	YES	YES	YES	NO	9	YES	NO	NO	NO	NO	NIL	NIL	100	65	65	41	31	69	91	100	20	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
71	43	M	23.6	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	9	YES	NO	NO	NO	NO	NIL	NIL	54	53	98	52	63	56	40	60	18	SEVERE	L LL FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
72	44	F	25	NO	NO	YES	5	18		SEVERE	YES	YES	YES	NO	8	NO	NO	NO	NO	NO	NIL	NIL	100	65	65	41	40	55	88	96	18	SEVERE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
73	71	F	22.08	NO	NO	YES	5	21		SEVERE	YES	YES	YES	NO	13	NO	NO	YES	NO	NO	NIL	NIL	64	56	87	27	42	48	64	66	21	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
74	64	M	25.5	YES	NO	NO	5	15		MODERATE	NO	YES	YES	NO	13	YES	NO	YES	NO	NO	NIL	NIL	43	50	118	64	85	41	44	43	15	MODERATE	LEFT PNEUMOTHORAX	NO	NIL	YES	BREATHLESSNESS ON EXERTION	95
75	42	F	22.9	NO	NO	YES	5	23		SEVERE	YES	YES	YES	NO	15	NO	NO	NO	NO	NO	NIL	NIL	50	47	93	31	35	46	50	52	23	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	93
76	71	F	22.1	NO	NO	YES	5	21		SEVERE	YES	YES	YES	NO	16	NO	YES	YES	NO	NO	NIL	NIL	63	54	85	27	40	48	63	66	21	SEVERE	B/L LL FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	95
77	60	F	26	NO	NO	YES	5	18		SEVERE	YES	NO	YES	NO	10	YES	YES	YES	NO	NO	NIL	NIL	68	66	97	78	76	65	68	56	18	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	GHT PNEUMOTHORAX/Y	YES	BREATHLESSNESS ON EXERTION	98
78	43	M	23.6	NO	NO	YES	5	18		SEVERE	YES	YES	YES	NO	8	YES	NO	NO	NO	NO	NIL	NIL	54	53	98	52	63	56	40	60	18	SEVERE	B/L FIBROSIS WITH TRACTION	NO	NIL	YES	BREATHLESSNESS ON EXERTION	98
79	48	F	25	NO	NO	YES	5	15		MODERATE	YES	YES	YES	NO	7	YES	YES	NO	NO	NO	NIL	NIL	55	50	90	30	43	80	48	60	15	MODERATE	RIGHT PNEUMOTHORAX	NO	GHT PNEUMOTHORAX/Y	NO	NIL	99
80	45	F	23	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	92	79	85	38	47	86	92	94	8	MILD	NORMAL STUDY	NO	NIL	YES	NIL	99
81	63	M	31.2	NO	NO	YES	5	15		MODERATE	YES	NO	YES	NO	9	NO	YES	NO	NO	YES	NIL	NIL	60	67	111	69	99	55	57	60	15	MODERATE	R LL FIBROTIC BANDS	yes	NIL	YES	fatigue	97
82	64	M	25.5	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	5	YES	YES	YES	NO	NO	NIL	NIL	115	116	100	48	108	55	106	116	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	98
83	45	M	22.7	NO	NO	YES	5	10		MODERATE	NO	NO	NO	NO	5	YES	YES	NO	NO	NO	NIL	NIL	67	66	92	50	75	44	43	78	10	MODERATE	SUB PLEURAL ATELECTASIS OF R LL	yes	NIL	YES	NIL	99
84	67	M	22.08	YES	NO	NO	5	18		SEVERE	YES	YES	YES	yes	11	YES	YES	NO	NO	NO	NIL	NIL	73	79	86	89	88	82	73	77	18	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	fatigue	96
85	67	M	26.8	NO	NO	YES	5	18		SEVERE	YES	YES	YES	NO	9	YES	NO	NO	NO	NO	NIL	NIL	73	79	109	78	117	56	65	75	18	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	NIL	98
86	40	M	23.8	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	77	81	105	89	88	82	64	77	8	MILD	NORMAL STUDY	NO	NIL	YES	NIL	98
87	46	M	26	NO	NO	YES	5	15		MODERATE	YES	NO	YES	NO	6	NO	NO	NO	NO	NO	NIL	NIL	58	54	93	29	43	80	60	60	15	MODERATE	B/L RETICULAR PATTERN	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
88	67	M	26.8	NO	YES	NO	5	18		SEVERE	YES	YES	YES	NO	10	YES	NO	YES	NO	NO	NIL	NIL	73	79	109	78	117	56	65	75	18	SEVERE	E/O FIBROSIS	yes	NIL	YES	NIL	97
89	52	M	35.5	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	5	YES	YES	NO	NO	NO	NIL	NIL	77	73	106	129	102	60	67	73	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99
90	40	F	28.2	NO	NO	YES	5	16		SEVERE	YES	YES	YES	NO	7	NO	NO	NO	NO	NO	NIL	NIL	74	76	102	82	120	104	68	73	16	SEVERE	FEW FIBROTIC BANDS	yes	NIL	NO	NIL	98
91	74	M	23.07	YES	NO	NO	5	8		MILD	NO	NO	NO	NO	5	YES	YES	YES	NO	NO	NIL	NIL	110	132	83	61	67	82	125	132	8	MILD	FEW BASAL FIBROTIC BANDS	yes	NIL	NO	NIL	97
92	23	F	22.4	NO	NO	YES	5	10		MODERATE	NO	NO	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	74	73	98	59	81	53	65	69	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	98
93	44	M	30.1	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	5	YES	NO	NO	NO	NO	NIL	NIL	77	74	106	129	102	64	68	73	8	MILD	NORMAL STUDY	NO	NIL	NO	fatigue	97
94	58	F	25.6	NO	NO	NO	5	16		SEVERE	YES	YES	YES	NO	7	YES	YES	NO	NO	NO	NIL	NIL	48	32	66	34	50	46	45	48	20	SEVERE	B/L FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	94
95	42	F	22.9	NO	YES	NO	5	20		SEVERE	YES	YES	YES	NO	11	NO	NO	NO	NO	NO	NIL	NIL	55	50	93	40	48	46	50	52	20	SEVERE	FEATURES OF FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	92
96	48	F	25.6	NO	NO	YES	5	12		MODERATE	YES	YES	YES	NO	10	YES	NO	NO	NO	NO	NIL	NIL	58	50	86	36	66	80	48	60	12	MODERATE	LEFT PNEUMOTHORAX	NO	EFT PNEUMOTHORAX/YE	NO	NIL	96
97	46	M	25.6	YES	NO	NO	4	8		MODERATE	NO	YES	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	96	88	91	66	90	72	90	98	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	98
98	26	F	32.8	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	6	NO	NO	NO	NO	NO	NIL	NIL	101	109	106	126	164	98	95	100	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	98
99	38	M	22.9	NO	NO	YES	5	6		MILD	NO	NO	NO	NO	4	NO	NO	NO	NO	NO	NIL	NIL	76	81	108	119	133	96	69	76	6	MILD	NORMAL STUDY	NO	NIL	NO	NIL	98
100	25	M	22.9	NO	NO	YES	5	10		MODERATE	YES	YES	NO	NO	6	NO	NO	NO	NO	NO	NIL	NIL	80	81	102	57	91	60	82	84	10	MODERATE	NORMAL STUDY	NO	NIL	NO	NIL	98
101	56	F	26.4	NO	NO	YES	5	12		MODERATE	YES	YES	YES	NO	7	NO	YES	YES	NO	NO	NO	NIL	65	60	92	87	86	55	63	70	18	SEVERE	B/L LOWER LOBE FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	96
102	64	F	22.2	NO	YES	NO	5	21		SEVERE	YES	YES	YES	yes	14	NO	YES	NO	NO	NO	NIL	NIL	52	50	96	26	42	44	52	52	21	SEVERE	FIBROTIC BAND WITH TRACTION	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
103	36	M	28.7	NO	NO	YES	5	21		SEVERE	YES	YES	YES	NO	9	NO	NO	NO	NO	NO	NIL	NIL	69	75	109	112	86	96	69	72	21	SEVERE	B/L FIBROTIC BANDS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
104	39	M	27.6	NO	YES	NO	5	16		SEVERE	YES	YES	YES	NO	7	NO	NO	NO	NO	NO	NIL	NIL	73	78	107	80	120	106	68	73	16	SEVERE	E/O FIBROSIS	yes	NIL	YES	BREATHLESSNESS ON EXERTION	90
105	48	F	25	NO	NO	YES	5	15		MODERATE	YES	YES	YES	NO	9	YES	YES	NO	NO	NO	NIL	NIL	55	50	90	30	43	80	48	60	15	MODERATE	RIGHT PNEUMOTHORAX	NO	GHT PNEUMOTHORAX/Y	NO	NIL	99
106	55	M	26	NO	NO	YES	5	15		MODERATE	YES	NO	YES	NO	8	NO	NO	NO	NO	NO	NIL	NIL	58	54	93	29	43	80	60	60	15	MODERATE	B/L RETICULAR PATTERN	yes	NIL	YES	BREATHLESSNESS ON EXERTION	98
107	50	F	25	NO	NO	YES	5	15		MODERATE	YES	YES	YES	NO	10	YES	YES	NO	NO	NO	NIL	NIL	55	50	90	30	43	80	48	60	15	MODERATE	RIGHT PNEUMOTHORAX	yes	GHT PNEUMOTHORAX/Y	YES	BREATHLESSNESS ON EXERTION	92
108	50	F	23	NO	NO	YES	5	8		MILD	NO	NO	NO	NO	5	NO	NO	NO	NO	NO	NIL	NIL	92	79	85	38	47	86	92	94	8	MILD	NORMAL STUDY	NO	NIL	NO	NIL	99