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UNDERSTANDING INTERSTITIAL LUNG DISEASE IN POST COVID-19 PATIENTS AS A NEW FIBROINFLAMMATORY DISEASE

Radio-Diagnosis

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ABSTRACT

Introduction: The COVID-19 pandemic has revealed a spectrum of pulmonary complications, ranging from acute respiratory distress syndrome (ARDS) to long-term sequelae such as interstitial lung disease (ILD). Post-COVID ILD presents a novel challenge, characterized by a fibroinflammatory process that differs from traditional ILDs. This article aims to provide a comprehensive understanding of post-COVID ILD, exploring its pathogenesis, clinical manifestations, diagnostic approaches, and therapeutic considerations. We delve into the interplay between viral infection, dysregulated immune responses, and fibrotic pathways, shedding light on the mechanisms underlying disease progression. Clinical phenotypes and radiological patterns of post-COVID ILD are elucidated, emphasizing the importance of multidisciplinary collaboration for accurate diagnosis and management. By synthesizing current evidence and clinical insights, this article aims to enhance clinicians' understanding of post-COVID ILD, facilitating optimized care for affected individuals in the post-pandemic era. **Material and Methods:** The study was a retrospective, observational cross-sectional study conducted in JNUIMSRC, Jaipur, Rajasthan. A total of 15 patient cases who presented to the Outpatient Department of JNUIMSRC, Jaipur from August 2022 to March 2023, with symptoms of cough, shortness of breath, chest pain and dyspnea with symptom history of more than 3 months were evaluated using High resolution computed tomography. We assessed the clinical symptoms and radiological patterns of the cases to determine the trajectory of the disease. This study included patients of all ages and genders who presented with symptoms of dyspnea, cough and chest pain with symptom duration > 3 months with a clinic-radiological diagnosis of COVID-19 infection who consented to be a part of this study. **Results-** The proposition that post-COVID-19 syndrome contributes to Interstitial Lung Disease suggests that tissue damage occurs directly, potentially inciting autoimmunity, and is compounded by exposure to neoantigens, as well as the generation of antibodies whose identities remain elusive. This phenomenon arises from a complex interplay involving immunological responses, genetic predispositions, tissue damage resulting from COVID-19 infection, the impact of acute respiratory distress syndrome, and the infiltration of macrophages during the acute phase of the disease. These factors collectively instigate a fibroproliferative cascade, culminating in the recruitment of fibroblasts and myofibroblasts, alongside excessive extracellular matrix deposition within the pulmonary interstitium. This process ultimately leads to fibrosis and architectural damage within the lung tissue. **Conclusion-** After experiencing SARS-CoV-2 pneumonitis, a subset of patients develop interstitial lung disease, resulting in enduring functional impairment of the lungs. Even a minor proportion of individuals affected by this condition is poised to impose a considerable burden of disease, underscoring the critical importance of promptly identifying this specific medical condition.

KEYWORDS

INTRODUCTION

COVID-19 is a viral infectious disease that emerged in late 2019 in Wuhan, China. It is caused by novel coronavirus leading to Severe Acute Respiratory Syndrome (SARS). It was declared a pandemic in March 2020 affecting over 100 million people. The clinical course of the disease was very variable ranging from asymptomatic to severe respiratory distress and multiorgan failure and death. The presenting symptoms mostly include cough, fever, breathlessness, anosmia, and fatigue. Morbidities associated with COVID-19 affect a large percentage of the surviving patients, including ARDS, thromboembolic disease, myocarditis, and pericarditis.

SARS-CoV-2 virus causes the attachment of its virion spike protein- S protein to angiotensin-converting enzyme ACE2 receptor present in the alveolar cells of the lung epithelium leading to infection, inflammation and injury of the lungs.

This causes inflammation of the lungs leading to the development of acute respiratory distress syndrome in a majority of patients. As the disease settles, there is the development of persistent or even progressive fibrosis. Long term implications of COVID-19 can lead to interstitial lung disease.

Initially, COVID is profoundly inflammatory manifesting as organizing pneumonia with ARDS. The fibrogenic stage can develop post-COVID interstitial lung disease.[1]

By using thin sections in High-resolution computed tomography, one can diagnose Interstitial Lung Disease in its early stage even when the chest radiograph appears normal. The use of High-resolution computed tomography is not just for the diagnosis of Interstitial Lung Disease but also plays an important role in the non-invasive evaluation of disease activity in Interstitial Lung Disease.

We describe our understanding of post-COVID interstitial lung disease which may facilitate the adequate intervention or arrest of progression of lung damage. We also evaluated the relationship between Interstitial Lung Disease and the long-term effects of COVID-19 infection.

MATERIALS AND METHODS

The study was a retrospective, observational cross-sectional study conducted in JNUIMSRC, Jaipur, Rajasthan after ethical clearance from University's ethics committee.

A total of 15 patient cases who presented to the Outpatient Department of JNUIMSRC, Jaipur from August 2022 to March 2023, with symptoms of cough, shortness of breath, chest pain and dyspnea with symptom history of more than 3 months were evaluated using High resolution computed tomography and the scans were reviewed. The patient cases were scanned using Toshiba Alexion 16-slice helical scanner working on compact mechanism with CT protocol using 120 kV, 5o mA in Scannogram and 120 kV, 150 mA on HRCT with rotation time of 0.75 sec per slice and 360-degree range. FOV- 350.0(M) with effective mAs 78. We assessed the clinical symptoms and radiological patterns of the cases to determine the trajectory of the disease. This study included patients of all ages and genders who presented with symptoms of dyspnea, cough and chest pain with symptom duration > 3 months with a clinic-radiological diagnosis of COVID-19 infection who consented to be a part of this study. These patient cases were admitted for COVID-19 infection and the CT severity score was more than 10 at the time of presentation. Oxygen therapy was required by 88% of the patients and only 42% required intensive care therapy at the time of admission for COVID-19 infection.

Patients who had co-morbidities like Tuberculosis, COPD, Pleural diseases and pregnant women were excluded from the study.

HRCT detected parenchymal abnormalities with great accuracy and

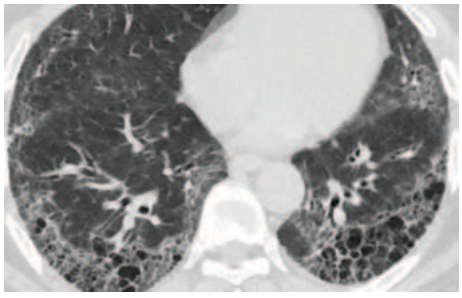
the radiological patterns observed included reticulation, ground-glass opacification, honeycombing, tractional bronchiectasis, thickening of interlobular septa and fibrosis.

RESULTS

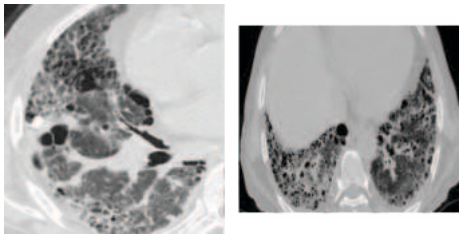
Between August 2022 and March 2023 patients were diagnosed with Interstitial Lung Disease with a clinico-radiological diagnosis of COVID-19 infection and cases included in this study were predominantly female with an average age of 64.2 years. Most of the patients had at least one co-morbidity, with the commonest being hypertension. The diagnosis was made on the basis of analysis of HRCT scans by studying the radiological patterns in the observed cases.

A spectrum of findings observed included patients who had near-normal chest radiographs but with abnormal HRCT. Other cases had persistent changes on both chest radiographs and HRCT. All the cases had diffuse interlobular septal thickening. In the majority of cases, diffuse ground glass opacification, fibrosis and honeycombing in subpleural location and basal segments were observed. In 13% of the cases, crazy paving pattern was observed and only in 6% of the cases, mosaic attenuation and traction bronchiectasis was seen.

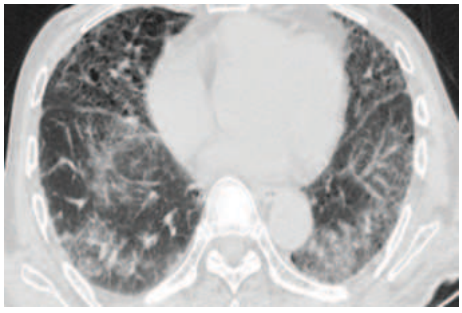
RADIOLOGICAL PATTERN ON HRCT	PERCENTAGE OBSERVED
Interlobular Septal Thickening	100%
Diffuse ground glass opacification	93%
Honeycombing pattern	78%
Fibrosis	60%
Crazy paving pattern	13%
Mosaic attenuation pattern	13%
Traction bronchiectasis	6%



Honeycombing With Interlobular Septal Thickening In Subpleural Location



Honeycombing In The Lower Lobes Of The Lung With Diffuse Interlobular Septal Thickening And Ground Glass Opacification



Diffuse Ground Glass Opacification With Interlobular Septal Thickening

DISCUSSION

The hypothesis of post-COVID-19 syndrome causing Interstitial Lung Disease includes damage directly to the tissue causing autoimmunity and injury due to exposure to neoantigens and the generation of currently unidentified antibodies. This results due to an interaction between immunology, genetic factors, tissue injury from COVID-19 infection, the effect of acute respiratory distress syndrome, and infiltration of macrophages in the acute disease process which triggers a fibroproliferative cascade causing the recruitment of fibroblasts, myofibroblasts and excessive extracellular matrix in the pulmonary interstitium leading to fibrosis and destruction of lung architecture.[3]

There is also a hypothesis that COVID-19 infection unmasked or accelerated pre-existing undiagnosed or asymptomatic Interstitial Lung Disease or even accelerated a known or classifiable ILD. Other hypotheses could be viral persistence and microthrombi triggering PC-ILD.[4]

In post COVID-19 era when the study was conducted, it was observed that most of the cases presented radiologically with ground glass opacities, interlobular septal thickening, and honeycombing, predominantly in basal segments and areas of patchy fibrosis.

It was also observed that patients can develop Post COVID-19 interstitial Lung Disease whether they had a history of having ARDS or mechanical ventilation in the acute phase of the infection.

Whether PC-ILD can be categorized into fibrotic or inflammatory type based on different radiological patterns is controversial as the radiological features are ambiguous and there is an absence of sufficient histologic evidence to support this. Features like ground-glass opacities may show areas of fibrosis with an overlap of inflammation.[4]

The precedence, severity and impact of PC-ILD on health system is currently unknown as it can be influenced by new variants of the virus, future possibility of re-infection, vaccination, ventilation, systemic hyperinflammation and immunomodulation by use of corticosteroids. It is imperative that an adequate clinical, physiological, laboratory, radiological and histological co-relation is made for the appropriate treatment of PC-ILD. Understanding this disease entity has better global health implications and it also presents a opportunity to understand cellular and molecular mechanisms of starting, progression and resorption of lung fibrosis due to the long-term effects of COVID-19 infection. [4][2]

CONCLUSION

Following SARS-CoV-2 pneumonitis, a population of patients are left with interstitial lung disease with persistent functional lung deficit. Even a small percentage of such patients with such morbidity is likely to represent a significant disease burden and so prompt identification of this disease entity is essential.

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