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RESEARCH ARTICLE

REVERSIBLE LUNG FIBROSIS FOLLOWING SEVERE COVID-19 INFECTIONA CASE REPORT

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Abstract

After wide spread of COVID-19 pneumonia with thousands of cases complicated with acute respiratory distress syndrome (ARDS) of which high percentage fully recovered and resumed their normal life without any disability. It was known that those patients will have residual fibrosis which may affect their daily activity on the long run.There was always paramount question about the reversibility of such fibrosis especially with well-known concept about absence of any radical treatment for such etiology.We present a case of 54 years old male patient, non-smoker with irrelevant medical history apart from well controlled hypertension. He was referred to our facility with fever, productive cough, and shortness of breath. An initial polymerase chain reaction (PCR) screening for SARS-COV2 was positive. A high-resolution CT (HRCT) chest revealed Multi-focal patchy air-space ground glass density with peripheral and basal predominance more affecting the left side, suggestive of viral pneumonia. The patient was commenced on low flow oxygen therapy and symptomatic treatment. Four days later the symptoms aggravated and oxygen requirement increased to non-rebreathing oxygen mask (NRM). We treated him according to COVID protocol withtherapeutic anticoagulation, corticosteroids and oxygen therapy which was adjusted to maintain saturation 92%, but his condition deteriorated and was shifted to intensive care unit. Later patient improved andwas shifted back to the medical ward. We were able to wean him off oxygen and he was discharged home oncorticosteroidsand scheduled for a follow up visit in the pulmonology clinic. Repeated HRCT chest showed regressive course concerning both the airway space disease and the fibrotic changes. The patient returned to his normal life activity with no residual limitation of his physical activity or development of shortness of breath with exertion. Latest HRCT was completely normal.

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Introduction:-

Coronavirus disease 2019 (COVID19), has been hitting the world brutally since December 2019, started from Wuhan province in china, with worldwide spread within few months, WHO estimated the number of confirmed cases 170 million and death of 3.5 million globally until the date.

Various studies have reported that around 70–80% of patients who recovered from COVID-19 presents with persistence of at least one or more symptoms, even after being declared COVID-free.[1, 2]

It is a challenging task to manage COVID-19 Sequelae which may vary from mild in terms of fatigue and body pain to severe forms due to lung fibrosis, significant cardiac abnormalities and stroke leading to impairment in quality of health. While the majority of those infected will not have long-term pulmonary sequelae, 5%-10% will develop severe COVID-19 pneumonia and acute respiratory distress syndrome (ARDS).

Wu et al. described that 40% of patients who recovered from COVID-19 might develop ARDS consequently, and 20% of the ARDS patients may progress to pulmonary fibrosis. [3]

Abdel-Hamid et al. conducted a prospective observational study on 85 moderately and severely affected COVID-19 patients. They found that 38.5% of them have pulmonary residuals after three weeks. Male gender, high body mass index (BMI), high serum ferritin and C-reactive protein levels, low lymphocyte count, consolidation, and mixed consolidation/ground-glass opacities on initial CT scans are the independent predictors of post COVID- 19 pulmonary residuals. [4]25% to 85% of patients can have remnant images compatible with pulmonary fibrosis on the chest images. [5]

Considering millions of COVID19 cases worldwide, even small proportion of post COVID lung fibrosis is worrisome. Many active clinical trials and studies are underway to know more about the entity post COVID pulmonary fibrosis.

Case presentation:

A 54-year-old male patient non-smoker with irrelevant medical history apart from well controlled hypertension. He was admitted to the medical wardof another health care facility with fever started two days before admission complicated with productive cough and shortness of breathing. An initial polymerase chain reaction (PCR) screening for SARS-COV2 was negative. A high resolution CT chest was done revealing Multi-focal patchy air-space ground glass density with peripheral and basal predominance more affecting the left side, suggestive of viral pneumonia. (Fig. 1)

The patient was commenced on low flow oxygen therapy and symptomatic treatment. Four days later the symptoms aggravated and oxygen requirement increased to non-rebreathing oxygen mask (NRM). Another SARS-COV2 screening was done with a positive result. So, therapeutic anticoagulation and corticosteroids therapy were added and oxygen therapy was adjusted to maintain saturation above 92%.

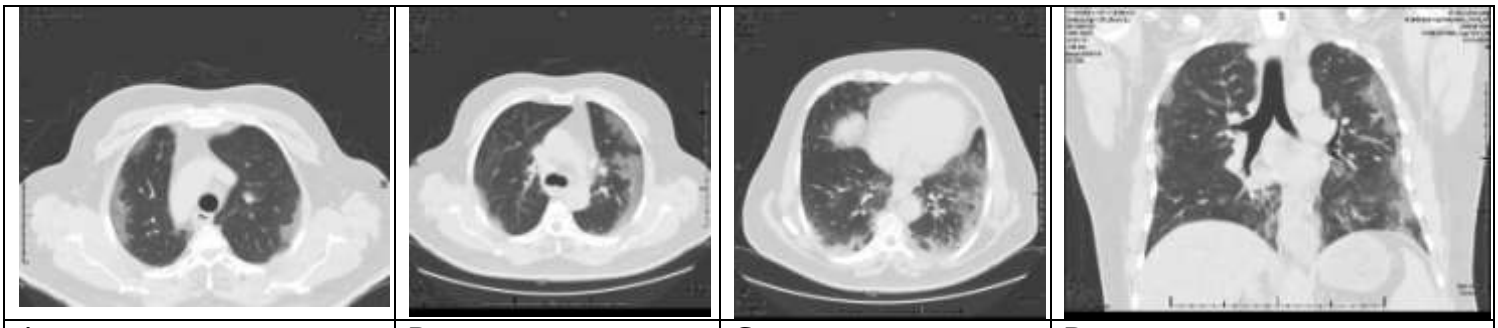
After four days, the patient was transferred to the intensive care unit (ICU) of our hospital with sever hypoxia and symptoms of respiratory distress. Upon admission, oxygen therapy was advanced to high flow nasal cannula(HFNC). The symptoms were dramatically improved and oxygen saturation was maintained above 92% on FiO2 of 0.8. SARS-COV2 PCR screening was done andthe initial laboratory investigation showed elevated sepsis markers and serum Ferritin levelwith normal interluken-6 (IL-6). The infectious disease management team advisedto startHydroxychloroquine, Favipiravir, Meropenemand convalescent plasma concomitantly with the currenttherapeutic anticoagulation and corticosteroids therapy. Moreover, the awake prone positioning protocol was implemented. The pending SARS-COV2 PCR screening showed negative result, it was repeated after 3 days and was also negative.

The patient showed stationary course for one week. However, his condition worsened and non-invasive ventilation (NIV) was commenced alternating with HFNC to relief the symptoms and FiO2 was adjusted to maintain accepted oxygen saturation.

Three days later, the patient showed progressive improvement, NIV was no more required, he maintained accepted oxygen saturation on HFNC which was gradually weaned to NRM then venturi mask then low flow nasal cannula

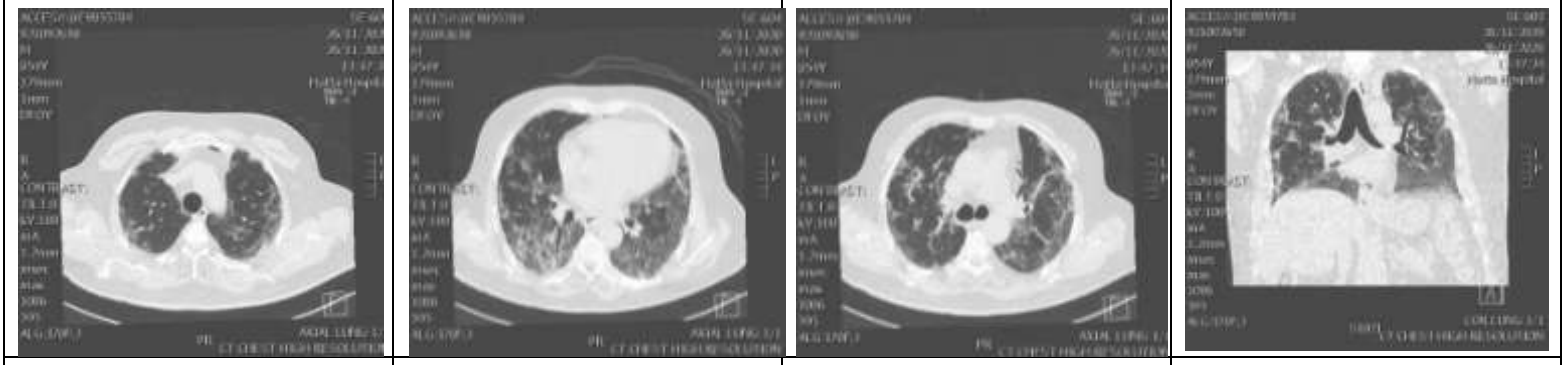
over the next five days.The patient was transferred out from the ICU to the medical ward. Five days later, a follow up HRCT chest was done, it showed findings compatible with post COVID-19 pulmonary fibrosis and bilateral basal traction bronchiectasis. (Fig. 2)The corticosteroids therapy was tapered while the patient continued improvement then he was discharged to home after one week.

The patient was scheduled for follow up visit in the pulmonology clinic after one month and follow up HRCT chest was arranged which showed regressive course concerning both the airway space disease and the fibrotic changes. (Fig. 3)The patient returned to his normal life activity with no residual limitation of his physical activity or development of shortness of breath with excretion.



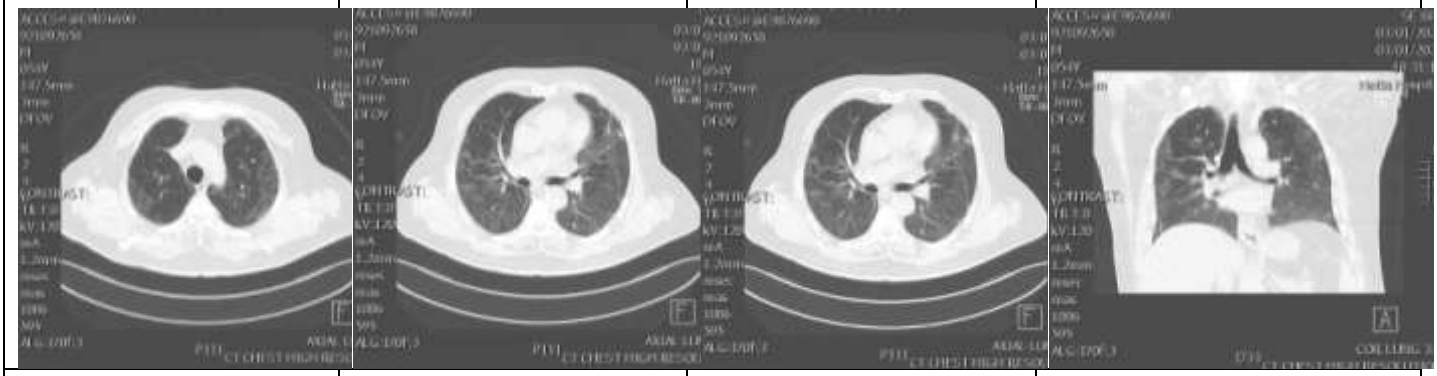
A B C D

Fig 1,HRCT chest done on 31/10/2020 selected axial images lung widow aupper,middle and lower lung zones A,B,C ,coronal d revealed multifocal areas of ground glass veiling more affecting the left lower lobe showing peripheral subpleural distribution ,more along the lung base,normal lung volume.



A B C D

Fig2 ,follow up HRCT chest done on 26/11/2020 selected axial images lung widow aupper,middle and lower lung zones A,B,C ,coronal D showing regressive course as regarding the extent and density of the prviously seen areas of ground glass veiling however extensive reticular formations involving mainly the lung periphery and basal lung zones as well as diffuse parenchymal coarsening.



A	B	C	D

Fig3 ,follow up HRCT chest done on 3/1/2021 selected axial images lung widow aupper,middle and lower lung zones A,B,C,coronal D showing regrsive course as rgrding the parenchymal coarsning hower multifocal regressing residual ground glass veiling of subpleural distribution still noted,and thin peripheral linear bands are seen reaching the pleural srfaces and showing minor surface tractions pointing to interstitial process ,lung volume is within the normal limits

Discussion:-

Pulmonary fibrosis happening after viral pneumonia was explained by increased deposition of extracellular matrix proteins, stimulation of fibroblast chemotactic migration, and fibroblast to myofibroblast transition. It was known as accepted consequences after recovery from ARDS.[6]

Nowadays after spread of COVID-19 pneumonia it was found that 40% of COVID-19 patient will develop ARDS which is very high rate for fibrosis outcome.[3]

As many studies has concluded that the duration of the etiology played vital role in determining the rate of fibrosis post ARDS. A recent paper published concluded that 4% of cases with disease course less than 1 week, 24% of cases with disease course between 1-3weeks and 61% of cases with disease course more than 3 weeks developed pulmonary fibrosis.[7]

Our case course was nearly 3 weeks in the critical care unit followed by 1 week in the step down unit followed by medical team.

In one studyCT chest was performed one day before discharge, two weeks and four weeks after discharge. While comparing with CT scan before discharge, the CT abnormalities (including focal/multiple GGO, consolidation, interlobular septal thickening, subpleural lines and irregular lines) in lungs were graduallyabsorbed in the first and second follow-ups after discharge.The lung lesions of 64.7% discharged patients were fullyabsorbed after 4-week follow-up. It indicated that the damageto lung tissue by COVID-19 could be reversible for the common COVID-19 patients. It also suggested that the prognosis ofnon-severe patients is favorable, and the clinical interventionshould be conducted in time to prevent common COVID-19 patients from worsening to severe patients.

As it was shown in the HRCT images the patient developed findings suggestive of airway space disease in the form of multifocal areas of ground glass veiling,yet lung volumes were preserved.

In the follow up HRCT chestafter 1 month there was regression in the course of the airwayspace diseae but early fibrotic changes started to be evident in the form of extensive reticular formations which were expected to be permenant .

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Many studies have shown that most common abnormality of lung function in discharged survivors with COVID-19 is impairment of diffusion capacity, followed by restrictive ventilatory defects, both associated with the severity of the disease.[9,10]

In the last HRCT the was even regression in the fibrotic process with thin peripheral linear bands are seen reaching the pleural surfaces and showing minor surface tractions pointing to interstitial process which proves reversible course of the fibrotic changes which affected the patient lung.It was also proved by patient full recovery and complete weaning from O2 and returning to his ordinary life activities without any limitations.

Those patients were given prednisolone for 3 months after their first covid positive swab, which might be the cause of this marked imporvment.

Conclusion:-

After the wide spread of COVID infection world wide with severly elevated numbers of complicated cases with ARDS which eventually ends with different degree of lung fibrosis according to the degree of lung invasion and duration of the ARDS course.This case review give us some hope that prognosis of COVID peumonia complicated with ARDS is mainly dependant on the duration of the illness and that the fastest the recovery from the ARDS the fully reversible the fibrotic lung changes .

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