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Pulmonary Post-Acute-Covid-19-Illness Sequelae

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Pulmonary function abnormalities in post-acute-COVID-19-illness survivors could be due to a decrease in both diffusing capacity of the lung for carbon dioxide (DLCO) and transfer coefficient of the lung for carbon monoxide (KCO) [1]. Alveolar-capillary damage, microvascular pathological lesions, or anemia can contribute to reduction of the KCO [1]. Post-acute-COVID-19-illness pulmonary fibrosis and post-acute-MERS-associated pulmonary fibrosis were highly associated with old aging ranging from 60- to 70-years age group (13 out of 30 (43.3 %)), demonstrated by Wong et al and Das et al [2, 3]. Post-acute-COVID-19-illness pulmonary fibrosis was 1.3 times more predominant in males, compared to females, possibly explained by the effect of androgen that promotes the transcription of transmembrane protease serine 2 gene, and was much higher incidence in cigarette smoker (18 of 30 patients (60 %)), compared to non-smoking patients [4, 5]. This gene impairs hose's antibody response and facilitates the fusion of the virus-hose cells. Dyspnea, the most common persistent symptom of the pulmonary sequelae ranged from 42 % to 66 % prevalence at 60-100 days of following-up, supported by the result of lower median 6-minutes walking distance comparing to the normal reference values in one-fourth of the patients at 6 months in the post-acute-COVID-19-illlness Chinese study that was similar to the prevalence in SARS and MERS survivors [6-11].

Lungs of the post-acute-COVID-19-illness survivors demonstrated endothelial- cell-dysfunction vasculitis [12]. Detected incident-clinical sequelae that are commonly identified in other serious viral infections, such as kidney injury, stroke, and hypertension was nearly twice, compared to a normal-year-incidence in general population, suggesting more urgently needed planning for healthcare resources for management of the COVID-19 survivors' health complications [13]. Post-acute-COVID-19-illness pulmonary aspergillosis that frequently are associated with acute respiratory distress syndrome (ARDS) could be due to host factors, such as poorly controlled diabetes, anti-interleukin 6 (tocilizumab) treatment in hospitalized post-acute-COVID-19-illness patients, and long-term use of corticosteroids in severe COVID-19-associated pneumonia, in addition to damage to the respiratory epithelium directly caused by SARS-CoV-2 (COVID-19) resulting in mucociliary dysfunction and fungal invasion of the respiratory epithelium. Serum galactomannan levels, chest roentgenographic and chest computerized tomographic imaging, and fungal isolation in the bronchoalveolar lavage or bronchial washing or tracheal aspirates are the diagnostics. The proposed first-line treatment for this pulmonary sequelae is isavuconazole or voriconazole [14]. Additionally, pulmonary and rhino-orbital-cerebral mucormycosis are the other two mold-related manifestations found in the post-acute-COVID-19 survivors. Biopsy with fungal isolation is the gold standard of diagnosis, whereas the initial treatment includes surgical debridement with intravenous amphotericin-B switching by oral posaconazole for avoiding nephrotoxicity [15]. Blood-stream-candida infection (invasive candidiasis), a late complication of COVID-19-related pneumonia in 15 post-acute-COVID-19-ilness patients in New Delhi, India was also reported [16].

In conclusion, prediction of pulmonary fibrosis in potential-high-risk-post-acute-COVID-19-illness patients could assist in decreasing the morbidity and mortality by introduction of early medical therapeutics, such as anti-fibrotic agents.

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