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CLINICAL MANIFESTATIONS OF POST-COVID SYNDROME CAUSED BY DIFFERENT SARS-CoV-2 STRAINS IN NEUROLOGICAL PRACTICE

Introduction

According to the World Health Organization (WHO), to date, more than 774 million confirmed cases of COVID-19 infection and over 7 million deaths have been reported globally.

The WHO describes the term PCS as a post-COVID-19 condition occurring in individuals with a history of probable or confirmed SARS CoV-2 infection, usually after 3 months from the onset of COVID-19 disease, with symptoms lasting at least 2 months and cannot be explained by an alternative diagnosis. Post-COVID syndrome appears to be a multisystem disease, occurring even after a relatively mild acute illness. The incidence of post-COVID syndrome is estimated at 10–35%, while for hospitalized patients it may reach 85%. Fatigue is the most common symptom reported in 17.5–72% of post-COVID cases, followed by residual dyspnea with an incidence ranging from 10–40%. Mental problems, chest pain, and olfactory and gustatory dysfunction may affect up to 26, 22 and 11% of patients, respectively. More than one third of patients with post-COVID syndrome have pre-existing comorbidities, hypertension and diabetes mellitus being the most common. Post-infectious olfactory function which may affect over 60% of those with SARS-CoV-2 infection including asymptomatic infections, represents also an important frequent symptom of post-COVID syndrome [1]. Involvement of the central or peripheral nervous system is noted in more than one-third of patients with antecedent severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. Moreover, as post-COVID is conceptualized as a multi-organ disease, central nervous system and/or peripheral nervous system involvement may present alone or in combination with pulmonary, cardiovascular, psychiatric, endocrine, renal, gastrointestinal, or immunological symptoms. Similar to WHO, the National Institutes of Health has linked post-COVID to symptoms such as fatigue, shortness of breath, brain fog, sleep disorders, fever, gastrointestinal symptoms, anxiety, and depression, thereby acknowledging neurological symptoms as core aspects of post-COVID. Moreover, recent reports indicate an extremely high prevalence of long-term neurological manifestations among COVID-19 survivors, with nearly one-third of patients being diagnosed with neurological or psychiatric illnesses in the first 6 months following acute COVID-19. In accordance with the spatial distribution of ACE2 receptors in the CNS, which are predominantly expressed in the olfactory bulb, amygdala, hippocampus, middle temporal gyrus, posterior cingulate cortex, and the brainstem, a multitude of neurological symptoms encountered in post-COVID patients

including hyposmia, mood disorders, cognitive impairment, sleep disorders, and dysautonomia, have been linked to dysfunction of ACE2-rich brain areas [2].

Since the beginning of the COVID-19 epidemic, SARS-CoV-2 has evolved, mutated, and produced variants with difference in transmissibility and virulence. The SARS-CoV-2 variants emerged from the original wild-type strain, which includes: Alpha (B.1.1.7), Beta (B.1.351), Gamma (P.1), Delta (B.1.617.2), and Omicron (B.1.1.529). Due to their increased transmissibility, virulence and resistance to vaccine these variants quickly became the main virus variants worldwide. The common symptoms of long COVID-19 caused by the wild-type strain were fatigue or muscle weakness, as well as mild dyspnea [3]. Delta is more transmissible and increases the hospitalization and mortality rate when compared to Alpha, whereas Omicron is highly transmissible but is thought primarily to infect the upper respiratory tract and less the lungs, thus resulting in a milder disease in most patients. Anosmia is uncommon when infected by Omicron compared to infection by wild type or Alpha variants, the cell-entry of Omicron differs from that of the other SARS-CoV-2 variants; its binding capacity to ACE2 is lower and Omicron also uses an endocytic pathway.

Goal

To determine the clinical manifestations of post covid syndrome and to characterize the neurological manifestations in regard to what caused unique SARS-CoV-2 strains caused it.

Material and methods of research

The analysis and generalization of modern medical scientific literature on neurological manifestations, post covid syndrome and different variants of SARS-CoV2 strains.

The results of the research and their discussion

PCS is found mainly in adults, less frequently in children and adolescents. It can develop both in patients who initially had only mild symptoms or none at all and in those who had a severe course of COVID-19. SARS-CoV-2 infection can cause vascular inflammation that leads to impaired microcirculation and endothelial dysfunction (ED). A third of patients with PCS exhibit ED in endothelial dysfunction testing as well as elevated levels of the potent vasoconstrictor endothelin-1 by 6 months following mild COVID-19. ED can also cause changes to the retina and affect reproductive health, for example, via new-onset erectile dysfunction.

Emerging SARS-CoV-2 variants show multiple mutations in the S protein which can impair the attachment of virus to host cell receptors and membrane fusion crucial for virus entry into the host cell. For example, S protein mutations that are seen in the Alpha variant increases its replication in human ACE2-deficient cells compared to the D614G variant. However, mutations in the S protein of Omicron BA.1 variant results in inadequate S1/S2 cleavage associated with a shift in cellular tropism away from TMPRSS2 expressing cells favoring the endosomal entry route compared to the D614G a Delta variant. Activation and fusion of Spike (S) proteins is essential for the entrance of the virus into the host cells. These proteins bind to the cellular ACE2 receptor, which is also present in neurons. Protein S is the surface glycoprotein of the virus responsible for its crown shape. This protein is composed by two subunits, S1 and S2. The S1 subunit consists of the N-terminal domain (NTD) and the C-terminal domain (CTD). The receptor-binding domain (RBD) in the CTD is responsible for binding to the host cell. The S2 subunit allows the fusion with membranes. Full-length protein S, RBD domain, S1 subunit, and NTD are used as antigens to develop SARS-CoV-2 vaccines, including adenoviral, RNA-based, DNA-based, and protein subunit vaccines.

With regard to COVID-19 variants and neurological disorders we found that Patients infected with the Delta variant had an increased risk of vascular disorders including ischemic stroke and hemorrhagic stroke, cognitive deficits, insomnia, anxiety disorders, central venous

thrombosis and epilepsy while patients infected with the Omicron variant had an increased risk of encephalitis, encephalopathy and Bell palsy. Moreover, even though the Omicron variant is associated with lower mortality rates, the risk of psychiatric or neurological problems remains similar to that of Delta. Anosmia and post-COVID are less frequently associated with Omicron infections compared to infections caused by Alpha, or Delta variants, but no differences are observed in the risk of neurological and psychiatric outcomes between the Delta and Omicron variant.

Conclusions

It is now clear that this infection does not only include the respiratory system but has consequences that affects the cardiovascular, neurological, and musculoskeletal system in addition to other organs. Recognizing the post COVID-19 of different strains is a crucial in scheming an appropriate health management strategy. It was found that Delta is more transmissible and has high mortality rate with increased risk of vascular disorders and anosmia and post-COVID is less common when seen with omicron variant compared to Alpha and Delta variants, but no differences between them in terms of risks of neurological and psychiatric outcomes. At present there is lack of studies that can be used to evaluate the CT and PFT results of post COVID-19 caused by unique variants. Thus, more research studies are needed. Also, patients who stay longer in the intensive care unit may advance to post-intensive care syndrome. Therefore, besides paying attention to the respiratory syndrome, it is necessary to evaluate and treat anxiety, depression, or other neurological disorders that derived from a condition with prolonged bed rest and invasive mechanical ventilation.

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AN OVERVIEW REVIEW OF PARKINSON'S DISEASE IN INDIAN POPULATION

Introduction

Parkinsons disease, one of the second most neurodegenerative disease in the ongoing population in India as well as worldwide after the Alzheimer's disease. It is known as one of the chronic, progressive, degenerative disorder of nervous system which majorly effects the population among the elders. According to the recent times of research it has been noted that approximately 1.5% of Indian population over the age of 60 suffers from Parkinson's disease and its associated further increase in the cases. Age has been depicted as of the major risk factors for the Parkinson's disease in the recent times. Hence AGE – RELATED Parkinson's disease has are of increasing concern in present days. We also see that there is almost 5–10% of the patients have been noticed as age groups below 20 years, which is 2% of 2 million people. Although