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Mini Review

Effects of COVID-19 on Alzheimer's Patients: A Brief Overview

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Abstract

Alzheimer's disease is the most common form of dementia. Old age people are mostly effected by AD. Ab oligomers and fibrils are the principal components of the plaque which is formed by the accumulation of beta amyloid. According to US Census Bureau data, between 2000 and 2020, the number will raised by over 200% and the number of people between 90 - 95 years will be double. However, prevalence rates of AD seem to be lower (1 - 3%) in India and sub Saharan.

COVID-19 pandemic has had impact on all age groups but highly effected old age people and AD patients. Studies suggested that AD patients are more likely contract COVID-19 than non-AD people. In thus review we have discussed how COVID-19 effects AD patients.

Keywords: *COVID-19; Alzheimer's Disease; Amyloid Beta; APOE4; Dementia; Neurogenerative Disorder*

Introduction

Alzheimer's disease (AD) is the most common cause of Dementia [1,2]. Currently, approximate 5 - 6 million AD patients are in United States and European Union and the number of AD patients would be expected to double by 2040 [3,4]. AD was discovered 100 years ago but unfortunately there is no effective cure for AD, compounded to it, the diagnosis of AD is a huge challenge. However, scientific research has reached an important achievement by identifying the early molecular changes which indicate certain pathological events occurring in brain. The symptoms of AD show after 10 - 20 years [5,6], with molecular and biological changes [7-11].

Effects of COVID-19 on AD patients

In December 2019, a novel infection caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) emerged on the global scene. The positive strand RNA virus started spreading rapidly to over 100 countries [12,13]. It was declared a pandemic by the World Health Organization (WHO) on March 2020. More than 700,000 deaths caused by the Pandemic globally. In their studies, Mao., *et al.* found that, neurological manifestations including acute cerebrovascular disease and impaired consciousness showed among the severe COVID patients [14]. Although, recent studies showed neurological association with COVID-19 [15-18]. Angiotensin-Converting Enzyme 2 (ACE2) is a transmembrane protease that cuts Angiotensin I and II into smaller peptides Ang(1-9) and Ang(1-7) [19]. Mainly ACE2 is present in airway epithelia, Kidney, small intestine, lung parenchyma, vascular endothelia, and brain [20,21].

It is yet not confirmed that how SARS-CoV-2 may invade the CNS. Severe Acute Respiratory Syndrome coronavirus (SARS-CoV) and the Middle East Respiratory Syndrome coronavirus (MERS-CoV) can be found parallelly with other coronaviruses (CoVs). SARS-CoV-2 entry

receptor ACE2 is highly expressed in nasal goblet and ciliated cells corroborating the hypothesis that SARS-CoVs might enter the human brain by olfactory nerves [22].

Beta-amyloid (Aβ) is a key factor in AD pathogenesis [23-25]. By different *in vivo* experimental models and human clinical trials the beta-amyloid theory of AD has been further confirmed. Currentlythe main target of Pharmacological research is on reducing accumulation. According to Soscia et al. higher antimicrobial activity in whole brain homogenates from AD compared with age-matched non-AD samples, and that Antimicrobial peptide (AMP) corelation with Aβ levels [26]. Consistent with Aβ- mediated activity, the increased antimicrobial action was removed by immune-depletion of AD brain homogenates with anti-Aβ antibodies. According to these findings, virus transient infection may cause accumulation of Aβ in the brain that cause AD.

Conclusion

All age groups are at risk of COVID-19 but old age people and AD patients have a high risk of contracting COVID-19. People with AD may forget to wash their hands or take other precautions (use of face mask) to prevent COVID-19. So it is important for caregivers to consider the risk and take appropriate safety precautions for AD patients.

Conflict of Interest

Author has no conflict of interest.

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