

Is Covid-19 severe in patients with Alzheimer's disease?

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Abstract

Alzheimer's disease (AD), as a degenerative neurodegenerative disease that has a growing trend causes the exacerbation of the infectious disease of COVID-19, or on the contrary, the infection of Covid-19 can aggravate the symptoms of AD and the severe form of COVID-19 appears. There are several mechanisms for the worsening of AD condition after COVID-19. It is reasonable to assume that COVID-19 has a more severe impact on patients with comorbidities, including those with AD. As such, individuals with AD should take additional preventive measures to avoid contracting COVID-19, as they are at a heightened risk of developing severe symptoms of the disease, which could result in increased mortality rates.

Keywords: Alzheimer's disease, COVID-19, SARS-CoV-2, Infection

Introduction

Alzheimer's disease (AD) is a neurodegenerative condition that gradually affects the brain, resulting in cognitive impairment, memory loss, and alterations in behavior^[1]. While the exact cause of AD is not completely clear, it is thought to result from a combination of genetic, environmental, and lifestyle factors. A key feature of the disease is the buildup of atypical proteins in the brain, such as beta-amyloid plaques and tau protein tangles, which can obstruct normal brain cell activity and lead to neuron degeneration and death overtime^[2]. The symptoms of AD generally begin with mild memory impairment and challenges in acquiring and retaining new information. As the illness advances, symptoms can intensify, causing issues with language, decision-making, and judgment. In the later stages, individuals with AD may encounter difficulties with fundamental daily activities, such as self-care, such as eating, grooming, and dressing ^[3, 4]. Alzheimer's disease is a pressing global health concern, with approximately 55 million individuals worldwide currently afflicted by the condition ^[5]. As the population ages, the incidence of AD is expected to rise, underscoring the importance of ongoing research efforts aimed at elucidating the disease's causes and developing more efficacious treatments ^[6,7]. SARS-CoV-2, the virus responsible for COVID-19, can attack all organs even the nervous system and cause neurological symptoms in patients [8-12]. There is concern that this may lead to long-term neuronal damage and the development of neurodegenerative diseases such as AD. There appears to be a reciprocal relationship between AD and COVID-19, with patients with AD possibly being more susceptible to severe COVID-19, and COVID-19 patients being more likely to develop AD^[8]. There is a hypothesis that SARS-CoV-2 could result in CNS damage either through direct neurotoxicity or indirectly by triggering the host immune response, which may lead to demyelination, neurodegeneration and cellular senescence. Consequently, it could accelerate brain aging and increase the risk of developing neurodegenerative conditions such as dementia ^[9]. Due to factors such as advanced age, the presence of multiple medical conditions, and challenges in adhering to physical distancing guidelines, individuals with dementia are at heightened risk of contracting COVID-19^[10]. Individuals with AD may be significantly affected by COVID-19 infection. This is because the infection can worsen cognitive decline and memory loss in patients with AD, which can result in heightened functional impairment and reduced quality of life [11]

SARS-CoV-2 may exacerbate AD symptoms by directly entering the central nervous system and damaging critical memory storage areas, or as a consequence of chronic hypoxia, oxidative stress, or increased production of peripheral pro-inflammatory cytokines ^[12]. AD may develop after SARS-CoV-2 infection due to various mechanisms, such as mitochondrial dysfunction and heightened oxidative stress, both of which contribute significantly to the pathophysiology of AD. One of the effects of oxidative stress is the reduction of α -Secretase activity, which leads to an increase in beta-amyloid production ^[12]. The induction of a cytokine storm by the coronavirus can have unforeseeable ramifications in the nervous system. One important concern is how this process may influence the onset and progression of neurodegenerative disorders like Alzheimer's and Parkinson's disease. The coronavirus has the potential to trigger microglial activation, leading to the production of inflammatory cytokines, prostaglandin E2, nitric oxide, and free radicals, ultimately resulting in chronic neuroinflammation and cell death [13]. Bianchetti et al. revealed a significantly higher mortality rate of 62.2% among COVID-19 patients with dementia compared to 26.2% in those without dementia. Individuals with dementia were found to be at a 1.84 times higher risk of dying from COVID-19 compared to those who did not have dementia. Among those with dementia, the most common initial symptoms were delirium and a decline in functional status ^[11]. However, within nursing homes in the United States, out of 10,576 confirmed COVID-19 cases, individuals with dementia comprised 52% of the cases, but accounted for 72% of all COVID-19 related deaths, indicating a 1.7 times higher risk of mortality compared to those without dementia [14]. Generally, the most vulnerable to COVID-19 encompasses older adults and individuals with pre-existing medical conditions. Those who reside in long-term care facilities are typically elderly and afflicted with multiple comorbidities, making them particularly susceptible to the virus ^[15]. So, it is reasonable to assume that COVID-19 has a more severe impact on patients with comorbidities, including those with AD. As such, individuals with AD should take additional preventive measures to avoid contracting COVID-19, as they are at a heightened risk of developing severe symptoms of the disease, which could result in increased mortality rates.

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