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Cognitive Impairment in Patients After COVID-19 Infection

Patteera Thanasetsothorn
Praharuthai Convent School, Prakanong, Wattana, Bangkok Thailand 10110

***Corresponding Author:**
Patteera Thanasetsothorn
Praharuthai Convent School, Prakanong, Wattana, Bangkok Thailand 10110

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Abstract

COVID-19 infection has been linked to several neuropsychiatric complications, including cognitive impairment and dementia. Cognitive impairments are common in patients with severe COVID-19 infections and those with other risk factors. In people with a mild infection, post-COVID-19 cognitive symptoms have been observed that are distinct from those caused by other viruses. COVID-19 cognitive symptoms may last longer than those caused by other infectious diseases and are more prevalent in children and adolescents. Brief screening tests for cognitive impairment have suboptimal diagnostic performance, and accurate diagnosis requires the use of standardised criteria. Cognitive impairment caused by post–COVID-19 can significantly affect a patient's quality of life and functional independence, regardless of the presence of other post–COVID-19 symptoms. There is no approved treatment for post–COVID-19 cognitive impairment, though cognitive stimulation may benefit some patients. Cognitive symptoms are prevalent and widely co-occur with other systemic symptoms following COVID-19. Neuropsychological evaluations may be beneficial in diagnosing and quantifying the severity of neurological disorders and their long-term prognosis. Comprehensive and individual assessments of cognitive impairment may allow for the development of therapeutic interventions.

Keywords: Cognitive Impairment, COVID-19 Infection, SARS-CoV-2

Introduction

SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) is a highly contagious and severe coronavirus that initially surfaced in China and then spread over the world, causing a pandemic of acute respiratory illness known as coronavirus disease 2019 (COVID-19) [1,2]. COVID-19 was described as a disease of the lower respiratory tract [3]. The virus causes mild to moderate respiratory infection, such as pneumonia, as well as other common symptoms such as fever, cough, and dyspnea in most people who are infected [4,5]. However, some people may have severe symptoms and acute respiratory failure insufficiency (5). There may be long-term neurologic consequences in those who survive COVID-19 infection [6-8]. These folks frequently complain of cognitive impairment, sometimes known as brain fog [9,0]. The prevalence of post–COVID-19 cognitive dysfunction as well as its relationship to the severity of the disease are not well understood [11,12]. SARS-CoV-2 has a complicated etiology, but the specific pathophysiological mechanisms driving COVID-19's neurological and mental outcomes are still undetermined [13,14]. Therefore, the goal of this literature review is to look into objective cognitive impairment in COVID-19 patients.

Mechanisms of Covid-19 etiology that lead to neurological and psychological effects

COVID-19, a malady caused by the SARS Coronavirus 2 (SARS-CoV-2), has spread over the world, posing a risk to human health and public safety [15]. Acute Respiratory Distress Syndrome (ARDS) is a condition that causes heart, kidneys, hematology, digestive system, and nervous system issues [16, 17]. The virus's direct influence on the

central Nervous System (CNS) via the olfactory bulb, neuroinflammation, systemic infection, and persistent hypoxia have all been linked to COVID-19's cognitive symptoms [13,18]. The following neurotoxic pathways have been identified in earlier coronavirus infection as being involved in neurotropism and direct capacity to penetrate neurons and glial cells, resulting in neuronal malfunction and destruction (neuroinvasion), and subsequently encephalitis [6,19]. The virus could enter the CNS either indirectly through the blood-brain barrier or directly through olfactory neuron axonal transmission [5,20]. Affection of the cerebral blood vessels, as well as coagulopathies that cause ischemic or hemorrhagic strokes, have previously been mentioned [21]. Furthermore, global ischemia due to respiratory insufficiency, respirator treatment, and the so-called acute respiratory distress syndrome (ARDS) were also reported, as well as secondary negative effects of excessive systemic inflammatory responses, "cytokine storm," and peripheral organ dysfunctions affecting the brain [17, 22, 23].

Relationship between Covid-19 and cognitive impairment

Patients with severe COVID-19 who are admitted to the ICU are at risk for developing post-intensive care syndrome (PICS), which includes physical, cognitive, and psychosocial changes [24, 25]. In around 6–51% of patients after being hospitalized in ICU, PICS raises the likelihood of long-term cognitive damage [25, 26]. Furthermore, delirium is one of the leading causes of cognitive impairment, and it was one of the most common COVID-19 behavioral symptoms in up to 11% of all hospitalizations [27, 28]. The symptoms in patients with severe SARS-CoV-2 have shown to have varied degrees of the impairment of cognition in studies thus far [29]. The most impaired functions were sustained attention, speed of processing, verbal memory, language, and executive function [14, 30]. The amount of time spent in the intensive care unit (ICU), the existence of delirium, systemic inflammation at baseline, neurological symptoms (such as headache, anosmia, or dysgeusia), or psychiatric symptoms have all been identified as predisposing factors in patients with coronavirus disease 2019 (COVID-19) [31, 32]. The factors that increase the risk of cognitive impairment in patients that have recovered from COVID-19 have been the subject of previous research [33-35]. In this cohort, however, the influence of protective factors on cognition is understudied [35]. Cognitive Reserve (CR), a term that is linked to lifestyle, educational, and intellectual levels and refers to the brain's adaptability, is a risk factor for dementia, as well as a predictor of better cognitive functioning and a decreased risk of cognitive impairment following ICU discharge in people with psychological illnesses [36, 37]. Another study found that patients with COVID-19 had a significant rate of cognitive impairment months after contracting the virus. Executive functioning, processing speed, category fluency, memory encoding, and recall were all found to be impaired within hospitalized patients [11, 38]. The sparseness of memory recognition reflects an executive pattern in the context of poor encoding and recall [39]. This trend matches early reports of a COVID-19-induced dysexecutive syndrome, and it has significant consequences for occupational, psychological, and functional outcomes [21, 40, 41]. Although it is generally recognized that particular groups of people are more vulnerable to cognitive impairment following critical illness; yet, a significant proportion of the relatively younger generations who have recovered from COVID-19 for a few months showed some cognitive dysfunction in the current study [11, 34]). The results of this study are broadly in line with those of other virus studies (such as influenza) [42, 43].

Executive Functioning and Attention

Several studies investigated executive function and attention, either through subtest scores from global cognitive tests (MMSE, MoCA) or through the use of more sophisticated and certain neuropsychological assessments (i.e. computerized attentive tasks) [44, 45]. Notably, every research discovered some form of executive or attentional impairment, and every study that examined executive function also discovered some form of attentional deficit [46, 47]. A study discovered that just one patient (2.9%) demonstrated problems with processing speed (Trail Making Test A) and inhibition (Stroop Interference), whereas two patients (5.7%) got abnormal results on a test evaluating split attention and visual scanning (Symbol Digit Modalities Test, SDMT) [48, 49]. Three (8.6%) people had trouble with cognitive flexibility (Trail Making B) and attention span (Inverse Digits) [50].

Volume 5, Issue 2; March-April 2022; Page No 1255-1263

Three trials evaluated patients using the Frontal Assessment Battery (FAB), which measures many elements of executive functioning such as fluency, inhibition, conceptualization, and others [51, 52]. All these studies discovered, to varying degrees, impaired executive scores. Negrini and team discovered one patient (11.1%) with abnormal scores, but Beaud et al. discovered aberrant scores in 8 of the 13 cases (61%) [53]. Recent investigations discovered difficulties with fluency and linguistic activities [53]. Beaud and colleague discovered lexical fluency deficiencies, but Negrini et al. and Almeria et al. discovered abnormal phonemic fluency scores in 11% of patients [48, 53, 54]. Semantic fluency, on the other hand, was affected in 5.7 % of the subjects [48, 53, 54].

Memory deficits

The studies included cognitive memory tests, with three revealing deficits. Almeria et al. discovered a pathological score (2.9%) on a verbal memory test (Tavec Total) in only one patient, whereas two other studies discovered short-term memory problems [48, 50]. The questionnaire-based assessments of fatigue, forgetfulness, motivation, sleep abnormalities, depression, and anxiety levels were comparable to those of age-matched controls in the study [34, 39]. Participants initially demonstrated typical behavioural performance, but this was followed by a progressive reduction in performance relative to age-matched controls, implying a "lower ability to track and keep information attentively over time," according to the authors [55].

Linguistic skill

A study examined domain-specific linguistic activities then discovered that one patient had an inferior naming score (Boston naming test) [48, 56]. A study on linguistic skill showed that five patients (16.7%) exhibited a subjective decline in word discoveries, which was confirmed objectively using TICS language tasks to test the linguistic domain [57]. When compared to healthy controls, the authors discovered statistically notable reductions in concentration and language skills, but specific numbers are not disclosed [57].

Visuospatial Ability

A study conducted by Beaud et al discovered abnormalities in visuospatial functions while using MoCA, whereas Raman et al. discovered that 40% of patients with COVID-19, in comparison to 16% of the control group, had impairments in the visuospatial domain when using MoCA [54]. When tested using Trail Making B, Zhou et al. discovered no significant difference between COVID-19 patients and the control group [58]. Almeria et al. found no problems when visual reproduction was assessed using the Wechsler Memory Scale, and visuospatial organization was assessed using the copy of the Rey-Osterrieth Complex test [50].

Discussion

According to documented research, people with COVID-19 infection appear to experience varying degrees of short-term cognitive impairment [59]. All of the research included in this review revealed that a more significant proportion of patients exhibited global cognitive decline when compared to healthy controls [58]. Specific cognitive domains, most notably attentional and executive skills, appear to be more susceptible to deficits. On the other hand, data on memory, language, and visuospatial functions are less dependable [58]. The latter may be connected to observed methodological variation, including the type of instrument or test utilized, the period of assessment (early and late in the disease phase), the inclusion and exclusion criteria used, and the extent to which precise data were recorded. There was a few test data were available for the related SARS-CoV or Mers-CoV infections with cognitive issues during the early stage, only a fifth of patients developed cognitive impairments later in the early phase of the infection [60, 61]. At the same time, this would be consistent with De Lorenzo, and team findings that a quarter of COVID-19 patients demonstrated cognitive impairment upon discharge [62]. An additional study using comparable approaches would be required to assess time gradients for cognitive damage following COVID-19 infections [63].

According to the research, cognitive sequelae are widespread, with 19% of the group performing below the screening test criteria for mild cognitive impairment [64]. Apart from verbal memory, the most affected domains were speed processing and executive function, correlating with findings from similar studies [38]. As previously proven, the length of time spent in the ICU did not predict cognitive impairment, but mechanical ventilation did [65]. This

Volume 5, Issue 2; March-April 2022; Page No 1255-1263

conclusion is consistent with previous research suggesting that supplemental oxygen could be explained by the chronic hypoxia produced by COVID-19 infection associated with pulmonary illness [14]. Hypoxia may increase the risk of brain injury, particularly in limbic regions such as the hippocampus [7]. CA3 neurons in the hippocampal region are particularly vulnerable to hypoxia, and this region is associated with episodic memory [66]. The encoding process is carried out by CA2, CA3, and the dentate gyrus, while the retrieval process is carried out by CA1 and the subiculum [66]. Given that hypoxia and ARDS are common symptoms associated with COVID-19 infection, it is reasonable to consider memory impairment as a risk factor in these patients [66]. Although sedation has been linked to cognitive impairment following ICU discharge, delirium in the ICU appears to be the most important predictor of long-term cognitive impairment and is associated with aberrant brain anatomy [67]. Other characteristics in the ICU, such as delirium or the number of days spent sedated, also predicted poor memory function [68]. In our investigation, similar to earlier findings, delirium in the intensive care unit was associated with poor verbal memory performance six months later [68]. Thus, delirium appears to be a risk factor for long-term memory impairment in COVID-19 patients hospitalized in the intensive care unit [7]. As a result, it is necessary to observe and evaluate these patients' cognitive performance to facilitate the early detection of probable long-term cognitive impairments [34]. Another risk factor for cognitive impairment, according to Mazza et al. (2021), could be inflammatory mechanisms associated with early phases of COVID-19 infection [38]. They discovered that systemic inflammation at the start of the study predicted cognitive functioning and the severity of depression at three months follow-up, implying a possible shared brain change [38]. This study discovered that days with elevated PCR predicted higher interference scores and interacted with CR to predict poor phonemic fluency, implying a link between inflammation and cognitive function in COVID-19 patients hospitalized in the intensive care unit [46]. Although nearly 50% of our group reported subjective cognitive impairment (SCI), cognitive complaints did not correlate with neuropsychological performance [69]. There was, however, a correlation between SCI and anxiety scores, with patients with high anxiety having more substantial SCI [70]. These findings corroborate previous research emphasizing the importance of examining the affective and anxiety states of patients with cognitive complaints following COVID-19 infection in order to develop a comprehensive treatment strategy that addresses neuropsychological sequelae and psychological symptoms associated with functional impairments associated with high disease burden. Sociocultural status, premorbid intellect (IQ), and other proxy measures are connected with the cognitive reserve construct (CR) [46, 70]. As stated previously, this construct refers to individual variability in vulnerability to age-related cognitive and changes in the brain that are pathologic [46]. CR may operate as a moderator between pathology and clinical outcome, as it enables the brain to actively cope with brain damage through pre-existing cognitive processing strategies or the enlistment of compensatory strategies [70]. This suggests that those with a high CR are likely to be more resilient to brain damage than individuals with a low CR [69]. In this scenario, we expected that CR would be involved in the relationship between medical risk factors for brain injury and neurocognition [70].

Future perspectives

Further research is needed to determine whether severe SARS-CoV-2 infection is a risk factor for cognitive impairment or if the deficiencies are caused mostly by other medical diseases linked to it (such as ICU admission and delirium) [27]. It is required to compare severe COVID-19 patients admitted to the intensive care unit with control groups admitted for other reasons [71]. Additionally, a larger sample size would allow for the capture of CR variation in the population and a better understanding of how CR may operate as a protective factor against the SARS-CoV-2 effect on cognition [71]. In addition, psychological variables such as sadness and anxiety may be associated with cognitive complaints in these individuals and may account for some functional issues observed in some instances [69]. Thus, it is as critical to investigate these patients' psychological well-being as it is to investigate their cognitive or physical well-being [47]. Additionally, long-term follow-up of these patients is essential to ascertain the extent of the deficiencies produced by the COVID-19, as well as whether this population is at a

Volume 5, Issue 2; March-April 2022; Page No 1255-1263

heightened risk of neurodegenerative disorders, as some researchers have hypothesized [6].

Finally, more research into cognitive protective factors in severe COVID-19 patients and other critically sick patients requiring ICU admission could be pursued in the future [6]. Thus, treatments aimed at promoting cognitive reserve in the community and cognitive stimulation as primary, as well as secondary preventative measures for severe COVID-19 patients, respectively, should help minimize the incidence of cognitive deficits in this subgroup and the long-term health and economic health repercussions of this condition [6, 72].

Conclusion

The connection between COVID-19 and executive function raises serious issues about patients' long-term treatment. COVID-19-infected ICU patients may have a long-term cognitive impairment, notably about processing speed, verbal memory and executive function when applied to this group of people, medical risk factors such as agitation, drowsiness, inflammation, hypoxia, and mechanical ventilation while in the hospital can all contribute to long-term cognitive functioning reductions in this population. Further research is needed to explain cognitive impairment's risk factors and mechanisms, and potential treatment options.

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Volume 5, Issue 2; March-April 2022; Page No 1255-1263

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Volume 5, Issue 2; March-April 2022; Page No 1255-1263

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Volume 5, Issue 2; March-April 2022; Page No 1255-1263

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Volume 5, Issue 2; March-April 2022; Page No 1255-1263

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Volume 5, Issue 2; March-April 2022; Page No 1255-1263