

Impact of COVID-19 on Cognitive and Way to Resolve

Mohammad Azizur Rahman^{1,*}, Nabidur Rahman¹

¹Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka 1342, Bangladesh.

Corresponding author:

Mohammad Azizur Rahman, Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka 1342, Bangladesh.

Keywords:

Alzheimer's disease; Cognition; COVID-19; Dementia; SARS-CoV-2.

Received: Oct 30, 2021

Accepted: Nov 10, 2021

Published: Nov 20, 2021

Editor:

Raul Isea, Fundación Instituto de Estudios Avanzados -IDEA.

DOI:

10.14302/issn.2692-1537.ijcv-21-4007

Abstract

Cognitive abilities are of immense importance for the normal life sustenance of a human being. As cognitive impairment ensues, the living caliber declines. Among multiple factors, the current epidemic coronavirus diseases 2019 (COVID-19) has been implicated in worsening cognitive perfor-

mance of the COVID-19 sufferers. Present article pinpoints the etiology and patho physiology as well as recommendations to overcome the COVID-19 led cognitive decline.

Introduction

Among the grave concerns of COVID-19, the cognitive issue is foremost. Most of the COVID-19 survivors are either sufferers of cognitive impairment or remain vulnerable to the same as their short- or long-term consequences. Thus, the reasons behind this impediment should be delved out and appropriate recommendations should be formulated to save the global innumerable populace. In this context, the present study reports the cognitive impairments of the COVID-19 sufferers, the etiopathology, pathomechanism and putative recommendations.

Cognitive Performance

Cognitive performance comprises a number of abilities, including but not limited to, attention and thinking, listening and understanding, viewing and judging, learning and memory, reasoning and problem solving, justifying and decision making, as a whole, mental well being [1]. Among different organs of the body, the central nervous system (CNS) is intricately linked with cognitive performance of a person [2]. Any perturbation

of the CNS affects the organismal cognitive performance [2]. Though all of the cognitive attributes might not be affected equally, impairment of one type influences other, albeit modifies the normalcy [3]. Recently, this type of induced cognitive impairment have been noticed in the COVID-19 sufferers [3].

COVID-19

Epidemic coronavirus diseases 2019 (COVID-19) has taken a heavy toll worldwide. Caused by the severe acute respiratory distress syndrome coronavirus serotype 2 (SARS CoV-2), COVID-19 affects mainly the respiratory system [4]. Unfortunately, the entry route of SARS CoV-2, the angiotensin converting enzyme 2 receptor (ACE2R), abounds in the CNS and is a haven for this virus [4]. Consequently, the COVID-19 patients co-manifest CNS abnormalities along with respiratory anomalies [4]. As the CNS becomes affected, associated cognitive functions become disrupted [4]. In this way, the secondary complications of the COVID-19 patients has been the deranged cognitive attributes that warrant adequate withstanding and management strategy.

COVID-19 and Deranged Cognition – the Achilles Heel

The “brain fog”, manifested through cognitive deficit are the etiological and patho-physiological resultants COVID-19. Following etiological and patho-physiological concerns could be attributed to this muddle.

ACE2 Overexpression

COVID-19 patients exhibit hyper expression of the ACE2 receptor that welcome the invading SARS-CoV-2 exceedingly [4-5]. Later on, pro-inflammatory cytokine led “cytokine storm” cripples both the respiratory system and CNS. Deranged CNS can hardly afford cognitive performance [4-5]. Shockingly, hippocampus and temporal lobes, the brain regions involved in memory and cognition, express ACE2 receptors that only worsen the cognitive level of the COVID-19 sufferers [4-5].

Neurotoxicity

SARS-CoV-2 led direct neurotoxicity vandalize the neurogenesis, synaptic plasticity, neurotransmission that are the neuropsychiatric hallmarks of cognitive decline [4-6].

Hypoxic-Ischemic Brain Injury

Depleted supply of oxygen to the CNS due to pulmonary debility attenuate CNS performance [7-8]. Besides, cerebrovascular injury and bursting of pro-inflammatory cytokines scars the CNS. Also, ischemic brain damage and stroke are seminal hallmarks of dementia and cognitive impairment [7-8].

Neurovascular Dysfunction

Blood brain barrier (BBB) and endothelial dysfunction leading to cerebral microvascular damage propensities SARS-CoV-2 malediction, otherwise accentuates cognitive malfunction [9-10].

Co-Morbidity

Cognitive decline in COVID-19 patients are co-morbid expression of multiple pathophysiological symptoms [11]. Besides, pre-existing cognitive decline associated with dementia and Alzheimer’s disease (AD) had been found to be overly diminished in COVID-19 sufferers [12]. Diabetes and hypertension are among the modifiable co-morbidities of COVID-19 and cognitive decline. Alzheimer’s disease (AD), the most common form of dementia, has been reported to be interlinked with COVID-19 [14-18].

Recommendations

People suffering from cognitive impairments during COVID-19 crises,

1. should be checked through COVID-19 tests.
2. should have adequate behavioral support.
3. should be supplied with medication that minimizes the consequences of neurovascular injury.

National and international health care

professionals should formulate state of the art guidelines to lower the burden of cognitively impaired persons.

Conclusion

COVID-19 has plagued the global humanity, especially those who are already cognitively impaired. Even, *de novo* cognitive impairments have been detected among the COVID-19 sufferers. Immediate measures against COVID-19 led cognitive impairment could reduce global economic burden. Further studies are called for withstanding this global crisis.

References

1. Stenfors CUD, Van Hedger SC, Schertz KE, et al. Positive Effects of Nature on Cognitive Performance Across Multiple Experiments: Test Order but Not Affect Modulates the Cognitive Effects [published correction appears in *Front Psychol*. 2019 Oct 17;10:2242]. *Front Psychol*. 2019;10:1413. Published 2019 Jul 3. doi:10.3389/fpsyg.2019.01413.
2. Nichols, M., Newsome, W. The neurobiology of cognition. *Nature* 402, C35–C38 (1999). <https://doi.org/10.1038/35011531>.
3. Alzheimer's Association International Conference® (AAIC®) 2021. alz.org/aaic/pressroom.asp
4. Rahman MA, Islam K, Rahman S, Alamin M (2020). Neurobiochemical Cross-talk Between COVID-19 and Alzheimer's Disease. *Molecular Neurobiology*. 19:1–7. doi: 10.1007/s12035-020-02177-w. PMID: 33078369; PMCID: PMC7571527.
5. Lukiw WJ, Pogue A, Hill JM. SARS-CoV-2 Infectivity and Neurological Targets in the Brain. *Cell Mol Neurobiol*. 2020:1–8. <https://doi.org/10.1007/s10571-020-00947-7>.
6. Solomon IH, Normandin E, Bhattacharyya S, Mukerji SS, Keller K, Ali AS, Adams G, Hornick JL, Padera RF Jr, Sabeti P. Neuropathological Features of Covid-19. *N Engl J Med*. 2020;383(10):98–92. doi:10.1056/NEJMc2019373.
7. Pendlebury ST, Rothwell PM, Oxford Vascular S. Incidence and prevalence of dementia associated with transient ischaemic attack and stroke: analysis of the population-based Oxford Vascular study. *Lancet Neurol*. 2019;18(3):248–258. doi: 10.1016/S1474-4422(18)30442-3.
8. Mijajlovic MD, et al. Post-stroke dementia - a comprehensive review. *BMC Med*. 2017;15(1):11. doi: 10.1186/s12916-017-0779-7.
9. Teuwen LA, et al. COVID-19: the vasculature unleashed. *Nat Rev Immunol*. 2020;20(7):389–391. doi: 10.1038/s41577-020-0343-0.
10. Varga Z, et al. Endothelial cell infection and endotheliitis in COVID-19. *Lancet*. 2020;395(10234):1417–1418. doi: 10.1016/S0140-6736(20)30937-5.
11. Williamson EJ, et al. Factors associated with COVID-19-related death using OpenSAFELY. *Nature*. 2020;584(7821):430–436. doi: 10.1038/s41586-020-2521-4.
12. Mok VCT, Pendlebury S, Wong A, Alladi S, Scheltens P. Tackling challenges in care of Alzheimer's disease and other dementias amid the COVID-19 pandemic, now and in the future. *Alzheimers Dement*. 2020;16(11):1571–81. doi:10.1002/alz.12143.
13. Ostergaard L, et al. Cerebral small vessel disease: capillary pathways to stroke and cognitive decline. *J Cereb Blood Flow Metab*. 2016;36(2):302–325. doi: 10.1177/0271678X15606723.
14. Rahman MA, Rahman N, Habiba U, Rahman J, Shakil S (2021) Psychological Problems of COVID-19 Sufferers. *Psychol J Res Open*, 3(3): 1-2.
15. Rahman MA, Habiba, U (2021). COVID-19 and neuropsychiatric disorders: Common links and extended networks. *J Neurol Neurol Sci Disord* 7(1): 024-026. doi.org/10.17352/jnnsd.000044.
16. Rahman MA, Rahman N, Shakil S, Habiba U. Therapeutic approaches towards COVID-19: A critical

insight. *J Clin Images Med Case Rep.* 2021; 2(4): 1231.

17. Rahman MA, Rahman MS, Alam N (2020). Heightened Vulnerability of Alzheimer's disease in COVID-19 Cataclysm and Putative Management Strategies. *Annals of Alzheimer's disease and Care.* 4(1):027-029. DOI:10.17352/aadc000015.
18. Rahman MA, Hossan T, Hsan K, Alam N, Shafique MR (2020). Alternative Medicine-Based COVID-19 Therapy: Lesson from a Bangladeshi Patient. *Medical Research and Clinical Case Reports.* 4.2:15-27.