Long Term Effect of COVID-19 on the Brain: Review

Shreerag Devkumar¹, Rakesh Kumar Jha²* and Dhruba Hari Chandi³

¹Datta Meghe Medical College, Nagpur, India.
²Datta Meghe Medical College, Shalinitai Meghe Hospital and Research Centre Nagpur, India.
³Department of Microbiology Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences Sawangi (Meghe), Wardha, India.

Authors’ contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2021/v33i39B32173
Editors:
(1) Prof. Mohamed Fawzy Ramadan Hassanien, Zagazig University, Egypt.
(2) Dr. S. Prabhu, Sri Venkateswara College of Engineering, India.
Reviewers:
(1) Ildefonso Rodríguez-Leyva, Zona Universitaria, México.
(2) Bayisa Bereka Negussie, Jimma University, Ethiopia.
(3) Pallavi Mahajan, Jammu University, India.
Complete Peer review History: https://www.sdiarticle4.com/review-history/71005

Received 29 May 2021
Accepted 29 July 2021
Published 31 July 2021

ABSTRACT

Corona viruses (CoVs) are well-covered RNA viruses that cause enteric and respiratory infections in humans and animals. Many human CoVs have recently gained global interest because of their lethal power and high contagious power. SARS-CoV-2, or COVID-19, is a pathogenic coV that first appeared in Wuhan, China.

Corona virus Disease 2019 (COVID-19) created by SARS-CoV-2, has been declared as pandemic by the World Health Organization since March 11, 2020. The epidemic started in Wuhan and spread rapidly around the world. Corona virus is facing a major epidemic: Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome (MERS). SARS-CoV-2 is a virus closely related to SARS.

Corona virus (COVID-19) is caused by SARS-CoV-2 VIRUS, a complex clinical disorder characterized by severe pneumonia and acute respiratory stress syndrome. Serious and neurological disorders, such as encephalitis, coma, fever, epilepsy, and Guillain-Barré syndrome, are more common in cases of COVID-19. In addition, chronic autoimmune and neurodegenerative diseases may occur in SARS-CoV-2 immunopathology and colonization of intestinal and central nervous system, as well as systemic inflammatory response during COVID-19. Parkinson’s disease...
INTRODUCTION

COVID-19 is worse than a welfare emergency. It has the ability to inflict severe social and economic issues with long-term consequences [1]. To curb the spread of the virus, countries rush to treat patients, block transportation, evacuate residents, postpone major sporting events and concerts, and close schools. Most people experience respiratory symptoms from COVID-19, although there is growing evidence that some people with severe infections often experience emotional symptoms such as confusion, stroke, epilepsy, or loss of smell and taste [2,3]. Another risk factor is the vulnerability of patients with neurological conditions to COVID-19 and emerging community-based treatment [4]. First, people with dementia may have difficulty remembering isolation and control strategies (such as wearing masks), leaving them at risk of infection [5].

Older people have a much higher mortality rate. Changes in aging, as well as age-related comorbidities such as heart and lung disease, diabetes, and dementia, are contributing factors [6]. Many disorders in older patients can be considered a sign of weakness because they increase a person’s susceptibility to depression and inhibit the many systemic compensation efforts to maintain homeostasis [7]. Immune senescence refers to a series of age-related changes in the immune system of the elderly [8].

According to the facts, coronavirus not only affects the respiratory system, but can also infect the central nervous system. Human-coronavirus RNA found in human brain samples clearly suggests that these respiratory viruses usually infect humans and can cause long-term infections in the central nervous system [9].

About 36% of patients have neurological complications during the acute period of COVID-19 infection, and 25% of those suffering from immediate central nervous system involvement [10]. Dizziness, headache, diminished consciousness, and seizures are among the most common symptoms. Cases with or without pre-existing neurological conditions is included in the neurological symptoms group [11]. Patients on intensive care units displayed anxiety, confusion, and symptoms of the corticospinal tract, such as increased tendon reflexes and clonus [12].

Convulsions, confusion, and symptoms of corticospinal tract, such as the development of tendon reflexes and clonus, are seen in patients in intensive care units. Olfactory (85.6%) and gustatory (88.0%) malfunctions were recorded by patients with symptoms of low to severe disease. Most importantly, anosmia occurs before all other health symptoms in about 11% of cases [13].

MATERIALS AND METHODS

This review study was conducted at Datta Meghe Medical College, Nagpur. The data for this article was collected from Google Scholar and other available resources at my reputed university (Datta Meghe Institute of Medical Sciences, Sawangi).

DISCUSSION

Neurological complications of para including Guillain-Barré syndrome, transverse myelitis, and widespread encephalomyelitis, identified in the Zika virus epidemic of 2015-2016, but to a greater extent given the number of people affected, can be dangerous greater than an excessive CNS virus attack. With regard to the fact that parasitic conditions often peak within four weeks, there was no clear trace from the countries affected at the start of the epidemic studies, which is reassuring. On the other hand, such interactions can develop over time and have significant clinical consequences. Patients with neurological problems may continue to be referred to intensive care for a long time, adding to the workload of the program.

The adverse effect of COVID-19 on the CNS can be caused by at least four pathogenic mechanisms: (1) direct viral encephalitis, (2) systemic inflammation, (3) immune dysfunction (liver, kidney, lung), and (4) cerebrovascular changes. On the other hand, the emotional expression of COVID-19, is usually the result of a combination of the factors described above.

COVID-19 survivors are at risk for long-term neurological symptoms due to one or more of
these mechanisms that exacerbate or exacerbate chronic neurological disease or trigger a new disease. According to the findings, one in three patients had symptoms of mental retardation and a lack of motor skills when exiting, confirming the question. With a direct walk from face-to-face visits where possible, a particular study can continue, at least in part. By promoting testing and compliance, as well as accessing tests and tracking results, these technologies have helped change that can have positive long-term results.

4. COVID-19 SEVERELY AFFECTS MOSTLY THE ELDERLY PEOPLE

4.1 Parkinson's Disease (PD)

Due to the depletion of dopaminergic cells in substantia nigra pars compacta and Lewy-synuclein (-syn) -positive antibodies, PD is the second most common neurodegenerative disorder, characterized by progressive motor and motor impairment. The link between HIV infection and Parkinson's disease can be traced back to the early 20th century, when a series of cases of post-encephalitic Parkinsonism were discovered during the outbreak [14].

The mechanisms underlying this connection could point to direct neuronal damage as a result of virus infection of the central nervous system (CNS) and subsequent loss of dopaminergic cells in the substantia nigra pars compacta. H1N1 Influenza-A virus infection has been shown to suppress protein degradation at the autophagosome–lysosome system level and precipitate -syn accumulation in Rag knockout mice. Additional studies showed that influenza A virus impairs dopaminergic transmission and compromises the integrity of the blood-brain barrier (BBB), whereas hepatitis C impairs dopaminergic transmission and jeopardizes BBB integrity. As a result, the infection can disrupt the cellular pathways involved in Parkinson's pathogenesis, which can lead to the onset of the disease [15-17].

Pull leaflets can allow SARS-CoV2 to enter the brain. Levy's body accumulation begins in the enrichment process and spreads to other brain structures through olfactory system integration, which has led to neuronal degeneration in Parkinson's disease. Finally, no matter what the cause, emotional disorders are related to neuroinfigueation and often have negative effects on the CNS, leading to emotional development. As a result, COVID-19 directly represents a stressful event that may have contributed to the onset of Parkinson's disease [18.19].

5. CONCLUSION

Patients surviving COVID-19 are at increased risk of developing future neurological disorders, including Alzheimer's disease, according to evidence. Neurologists, psychiatrists, and caregivers should be informed of the possible increase in such cases among COVID-19 survivors. As a result, we can say that COVID-19 has an effect on the brain.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

It is not applicable.

ETHICAL APPROVAL

Ethical clearance: Taken from institutional ethics committee

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Efrat Shadmi, Yingyao Chen, et al. Health equity and COVID-19: global perspectives. Int J Equity Health. 2020;19:104.
2. Fried TR, Vaz Fragoso CA, Rabow MW. Caring for the older person with chronic obstructive pulmonary disease. JAMA. 2012;308(12):1254-1263.
3. Buran T, Sanem Gökçe Merve Kılıç, Elmas Kasap. Prevalence of Extraintestinal Manifestations of Ulcerative Colitis Patients in Turkey: Community-Based Monocentric Observational Study. Clinical
4. Alomari SO, Abou-Mrad Z, Bydon A. COVID-19 and the central nervous system. Clin Neurol Neurosurg. 2020;198:106-116.

5. Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. JAMA Neurol. 2020:e201127.

6. Daniel V, Daniel K. Perception of Nurses’ Work in Psychiatric Clinic. Clinical Medicine Insights. 2020;1(1):27-33. https://doi.org/10.52845/CMI/2020v1i1a5

7. Lechien JR, Chiesa-Estomba CM, De Siati DR, Horoi M, Le Bon SD, Rodriguez A, et al. Olfactory and gustatory dysfunctions as a clinical presentation of mild-to-moderate forms of the coronavirus disease (COVID-19): a multicenter European study. Eur Arch Otorhinolaryngol. 2020; 6:1–11.

8. Daniel V, Daniel K. Diabetic neuropathy: new perspectives on early diagnosis and treatments. Journal of Current Diabetes Reports, 2020;1(1):12–14. https://doi.org/10.52845/JCDR/2020v1i1a3

9. Kalia LV, Lang AE. Parkinson’s disease. Lancet. 2015;386:896–912.

10. Imbriani P, D’Angelo V, Platania P, et al. Ischemic injury precipitates neuronal vulnerability in Parkinson’s disease: insights from PINK1 mouse model study and clinical retrospective data. Park Relat Disord. 2020;74:57–63.

11. Daniel V, Daniel K. Exercises training program: It’s Effect on Muscle strength and Activity of daily living among elderly people. Nursing and Midwifery. 2020; 1(01):19-23. https://doi.org/10.52845/NM/2020v1i1a5

12. Marreiros R, Müller-Schiffmann A, Trossbach SV et al Disruption of cellular proteostasis by H1N1 influenza A virus causes α-synuclein aggregation. Proc Natl Acad Sci USA. 2020; 117:6741–6751.

13. Wang R, Zhu Y, Lin X et al Influenza M2 protein regulates MAVS-mediated signaling pathway through interacting with MAVS and increasing ROS production. Autophagy. 2019;15:1163–1181.

14. Vishwa S et al. Effect of COVID 19 Affecting Geriatric Patients. Int J Cur Res Rev. 2020;12(17):182-187.

15. Meng L, Shen L, Ji HF. Impact of infection on risk of Parkinson’s disease: a quantitative assessment of case-control and cohort studies. J Neurovirol. 2019; 25:221–228.

16. Schirinzi T, Martella G, Imbriani P et al. Dietary Vitamin E as a protective factor for Parkinson’s disease: clinical and experimental evidence. Front Neurol. 2019;10:148.

17. Pisani V, Stefani A, Pierantozzi M et al. Increased blood-cerebrospinal fluid transfer of albumin in advanced Parkinson’s disease. J Neuro-inflammation. 2012;9:670.

18. Schirinzi T, Madeo G, Martella G et al. Early synaptic dysfunction in Parkinson’s disease: insights from animal models. Mov Disord. 2016;31:802–813.

19. Vlajinac H, Sipetic S, Marinkovic J et al. The stressful life events and Parkinson’s disease: a case-control study. Stress Heal. 2013; 29:50–55.

© 2021 Devkumar et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.