

The Effects of COVID -19 on the Neurological System: A Review Study

Parichehr Heydarian*, Kiana Mostaan

¹General Practitioner, School of Medicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran

Corresponding Author*

Parichehr heydarian
General Practitioner, School of Medicine, Ahvaz Jundishapur
University of Medical Sciences, Ahvaz, Iran
E-mail: pheydarian1991@gmail.com

Copyright: ©2022 Heydarian, P. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received: 07-Nov-2022, Manuscript No. jnn-22-77721; **Editor assigned:** 09- Nov -2022, PreQC No. jnn-22-77721 (PQ); **Reviewed:** 23- Nov -2022, QC No. jnn-22-77721 (Q); **Revised:** 28-Nov -2022, Manuscript No. jnn-22-77721 (R); **Published:** 29-Nov-2022, DOI: 10.35248/2332-2594.22.13(11).606

Abstract

Background: In December 2019, a pandemic termed COVID-19 broke out in Wuhan, China, attracting worldwide attention. The virus prompted widespread dread and terror among people around the world, and the World Health Organization (WHO) declared the epidemic a public health emergency. Fever, weariness, and dry cough are the most prevalent symptoms, but neurological and cognitive symptoms such as headache, cognitive impairment, and paresthesia have also been demonstrated.

Method: This review looked at all English-language works of literature published until May 4, 2020, on the topic of COVID-19 and its neurological and cognitive impacts. This search was conducted using the keywords coronavirus infection, COVID-19, and Nervous system, and the relevant material was extracted using the recognized scientific databases Bio Rxiv, Google Scholar, Scopus, and PubMed.

Conclusion: This study highlighted reports of viral attacks on the central nervous system and the development of viral inflammation of the brain, as well as the immune system's mediating role in defense against infection. The loss of smell and taste in infected people, as well as its association with the neurological system, are other major symptoms of the disease and one of the earliest signs of nervous system involvement. Acute cerebrovascular disease was also discussed, as well as the method through which it develops as a result of hypoxia and other infection-related consequences. Thus, it is important to consider the occurrence of neurological symptoms during initial assessments, as only some patients report these symptoms.

Keywords: Coronavirus infection • COVID-19

• Nervous system

Introduction

The outbreak of the new coronavirus in 2019 has become a health challenge in the world which was accompanied by extensive studies of the clinical status of infected patients and recognition of the infectivity of the disease [1-3]. Immune system over-activity has been found to contribute to the severity of signs, symptoms, and multi-organ failure in COVID-19 patients [4,5]. The Novel Coronavirus (COVID-19), formerly known as SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus) is a new virus that first appeared in December 2019 that causes COVID-19. The virus generates a syndrome that can progress to a serious respiratory condition in some patients, necessitating specific disease management in the Intensive Care

Unit (ICU). It is the seventh coronavirus that has ever infected people. Other beta-coronaviruses have caused epidemics in Asia in the last two decades, such as SARS-COV in China between 2002-2003 and MERS-COV3 in Saudi Arabia between 2012-2013 [6].

Literature Review

COVID-19 is a new disease with unclear clinical, biochemical, and radiographic features as well as numerous discrepancies in research. In this disease, the most common route of transmission is human-to-human contact via respiratory droplets. Symptoms of acute respiratory infection are seen in the early stages of the disease, and some patients develop ARDS and other serious manifestations which eventually lead to organ damage. In the 1960s, coronaviruses were recognized as a separate viral family following the discovery of many new human respiratory diseases. In late 2002 and early 2003, about 40 years after the discovery of this group of viruses, a coronavirus known as SARS (COAR) or Acute Respiratory Syndrome (SARS) developed and caused serious respiratory difficulties in humans [7,8].

The unexpected development of SARS prompted fresh research on the key processes of replication and toxicity of members of this virus family to control them globally. The SARS-COV outbreak infected 8096 persons and killed 794, with a 9.8% mortality rate. Then, in 2012, a new virus from this family, MERS (MERS-COV), emerged in the Middle East, particularly in Saudi Arabia, infecting 2.260 persons and killing 35.5 percent. The emergence of COVID-19 disease by RNA - SARS-CoV-2 virus in December 2019 in Wuhan City, Hui Province, China, was accompanied by signs of severe respiratory syndrome followed by a widespread and rapid epidemic, seven years after the onset of Morse sickness [9].

Although this virus is called an acute respiratory syndrome, it has recently been confirmed that in addition to systemic respiratory symptoms, 4 cases out of 91 patients suffering from COVID-19 had neurological symptoms including headache, loss of consciousness, and paresthesia. These neurological symptoms were more observed in severe cases than in mild or moderate infections [10].

Also, autopsy reports of this group of patients reported brain tissue inflammation and degeneration. There was also a nervous component reported in deceased patients [11]. Moreover, one of the above-mentioned lesions is a case of viral brain inflammation (Encephalitis). Due to COVID-19, the effect of the virus attack on the Central Nervous System (CNS) is reported, as well as reviews of the laboratory data of cerebrospinal fluid have shown that this virus has the potential to cause serious damage to the nervous system [12]. Due to the unique characteristics of the coronavirus as well as the mechanism of their multiplication, the emergence of coronavirus was unexpected. These special features can be noted in the high level of recombination of the virus leading to the emergence of new and unknown strains. The reasons for this diversity in genetic combinations can be vast. Compared to other viruses, complex multiplication, and error of the virus replicator and ultimately to the extent of widespread hosting of this virus in humans and attributed to different animals. In the hard war between genetic matter and viral intelligence with intellect, mankind is on the move to win. In addition to adequate knowledge of this invasive and contagious virus, we need to make the right decisions in a timely manner and all people should try to take action against this disease by personal and public health care manners [13]. The fact is that at the time of the epidemic disease such as coronary artery disease, fear of disease and death in addition to the impairment of daily activities, affects people with an anxiety disorder [14].

Fear of the unknown reduces perception in humans and always has been terrifying for them. This lack of scientific information also exacerbated this anxiety [15]. At this time people seek more information to eliminate their anxiety. Due to the high prevalence of the virus in Iran, researchers and scholars need to gather accurate and scientific information in order to publish and detect as many unknown aspects of the virus as possible and to reduce this ambiguity and confusion. Physicians and specialists can also work on the treatment of various effects related to the virus. The main purpose of this review article is to introduce the neurological and cognitive effects of COVID-19 from several articles published from 2019 to 2020 in authentic BioRxiv databases, Google Scholar, Scopus, and PubMed collected, and for this purpose, the keywords are Coronavirus Nervous system, COVID-19 and infection. This article is divided into three sections: an introduction, the effect of the virus on the Central Nervous System (CNS), and an overall assessment. The virus is able to infect the Central Nervous System (CNS) and may cause serious damage to the structure and function of nerves, mainly, acute inflammation and encephalitis. Viral infections that cause severe systemic and acute lesions involve the myelin of neurons [16].

This field is generally divided into 4 categories:

- Symptoms in the Central Nervous System (CNS) include dizziness, headache, disturbed consciousness, acute illness cerebral palsy, ataxia, and seizures.
- Symptoms in the Peripheral Nervous System (PNS) includes olfactory disorder, taste disturbance, visual disturbance, and diffuse neural pain.
- Symptoms of musculoskeletal injuries in the patient demonstrate as muscle pain and creatinine levels are higher than usual.
- Awareness impairment or changing its level include drowsiness, dizziness, and coma, also changes in the content of consciousness include delirium and delusion [14].

Discussion

Viral inflammation of the brain (viral encephalitis)

Viral infection of the brain parenchyma has an acute onset and symptoms include headache, fever, vomiting, seizures, and impairment of consciousness and need to be followed closely for proper treatment - early diagnosis is very important. researchers were able to detect the virus genome sequences in cerebral inflamed tissue caused by this virus, while genetic materials and even viral proteins were different in samples prepared from systemic neural tissues such as cerebrospinal fluid and brain. The study indicates a direct attack of the virus on the nerves and causes harm in it [17].

Mao et al. also believed due to intense hypoxia, 40% of patients suffer from encephalopathy along with headache, impaired consciousness, and other disorders in the function of the brain. In general, we can say that it is likely that Covid-19 causes viral inflammation of the gose, although more detailed studies have been performed in this area [14].

Severe brain diseases

Respiratory infections are an important risk factor for serious brain diseases [18, 19]. The SARS virus may cause acute cerebrovascular disease by increasing blood-brain barrier permeability with Cytokine storm syndromes [20, 21] This reduces the number of blood platelets. In patients who are at risk of acute cerebrovascular events a warning should be given to the patients. When the virus infects lung tissue, it disrupts gas exchange in the lungs, leading to hypoxia and lack of oxygen in the central nervous system. Due to increased metabolism and anaerobicity in the mitochondria of brain cells, acid will accumulate in these areas, which causes dilation of cerebral arteries, cell inflammation, intermediate obstruction of cerebral blood flow, and even headaches. If hypoxia is not treated may result in brain swelling and disorder in circulation which, in turn, may result in increased blood pressure in the head, decreased brain function, conjunctivitis, or even coma

[22]. If the patient is seriously ill, it makes rapid progress in their condition and leads to serious brain injury and strokes - one of the causes of the high mortality rate [23].

Conclusion

The severity of COVID-19 depends on the cellular immune response of the host. Those with mild COVID-19 infection usually have an immune response capable of eliminating the virus. As a result, managing the immune response may help to mitigate the viral burden. In addition to affecting the nervous system and cognitive system, the Coronavirus has now been demonstrated to affect a person's immune system and result in a persistent infection and prolonged disease. It can also cause nervousness. During the spread of this virus, it is vital to observe and evaluate patients who exhibit related symptoms, and take the necessary steps to isolate and quarantine them. In the future, it will also be important to examine the effects of long-term effects on neural activity and cognitive behavior.

Conflict of Interest

The authors declare there is no conflict of interest.

References

1. Abedini, A., et al. Can Hesperidin be the Key to the Treatment of Severe Acute Respiratory Syndrome COV-2? *Biomed. Biotechnol. Res. J. (BBRJ)*. 4.5(2020):108.
2. Guo, YR., et al. The origin, transmission and clinical therapies on coronavirus disease 2019 (COVID-19) outbreak—an update on the status. *Mil. med. res.*7.1(2020):1-0.
3. Fallahzadeh, M., et al. Efficacy of Interferon β -1a in Treatment of Hospitalized COVID-19 Patients; SBMU Taskforce on the COVIFERON Study.
4. Robinson DJ and Christenson RH. Creatine kinase and its CK-MB isoenzyme: the conventional marker for the diagnosis of acute myocardial infarction. *J. emerg. med.*, 17.1(1999):95-104.
5. Feldmann, M., et al. Trials of anti-tumour necrosis factor therapy for COVID-19 are urgently needed. *Lancet*. 39.10234(2020):1407-9.
6. Xu, P., et al. Profiles of COVID-19 clinical trials in the Chinese Clinical Trial Registry. *Emerg. Microbes Infect.* 9.1(2020):1695-701.
7. Mair, J. Cardiac troponin I and troponin T: are enzymes still relevant as cardiac markers? *Clin. Chim. Acta*. 257.1(1997):99-115.
8. Martins, JT., Et al. Comparison of cardiac troponin I and lactate dehydrogenase isoenzymes for the late diagnosis of myocardial injury. *Am. j. clin. pathol.* 106.6(1996):705-8
9. Ghormade, PS., et al. Distribution & diagnostic efficacy of cardiac markers CK-MB & LDH in pericardial fluid for postmortem diagnosis of ischemic heart disease. *J. forensic leg. med.*28(2014):42-6.
10. Jaffe, AS., et al. Comparative sensitivity of cardiac troponin I and lactate dehydrogenase isoenzymes for diagnosing acute myocardial infarction. *Clin. chem.* 42.11(1996):1770-6.
11. Lai, CC., et al. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and coronavirus disease-2019 (COVID-19): The epidemic and the challenges. *Int. j. antimicrob. Agents.*55.3(2020):105924.
12. Lu CW, Liu XF and Jia ZF. 2019-nCoV transmission through the ocular surface must not be ignored. *Lancet*. 395.10224(2020):e39.
13. Mao, L., et al. Neurological manifestations of hospitalized patients with COVID-19 in Wuhan, China: a retrospective case series study. *MedRxiv*. 2020.
14. Mao, L., et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China *JAMA neurol.* 77.6(2020):683-90.

15. Mehta, P., et al. COVID-19: consider cytokine storm syndromes and immunosuppression. *Lancet*. 395.10229(2020):1033-4.
16. Michalicova, A., et al. How viruses infiltrate the central nervous system. *Acta Virol*. 61.4(2017):393-400.
17. Schoeman D and Fielding BC. Coronavirus envelope protein: current knowledge. *Virology*, 16.1(2019):1-22.
18. Van, Doremalen N., et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. *N. Engl. j. med*. 382.16(2020):1564-7.
19. Ren, LL., et al. Identification of a novel coronavirus causing severe pneumonia in human: a descriptive study. *Chin. med. j.*, 133.9(2020):1015-24.
20. Wang, C., et al. A novel coronavirus outbreak of global health concern. *Lancet*. 395.10223(2020):470-3.
21. Sharifi-Razavi A, Karimi N, et al. COVID-19 and intracerebral haemorrhage: causative or coincidental? *New microbes new infect*. 35(2020):100669.
22. Warren-Gash, C., et al. Laboratory-confirmed respiratory infections as triggers for acute myocardial infarction and stroke: a self-controlled case series analysis of national linked datasets from Scotland. *Eur. Respir. J.*, 51.3(2018).
23. Wang W, et al. Updated understanding of the outbreak of 2019 novel coronavirus (2019-nCoV) in Wuhan, China. *J. med. virol*, 92.4(2020):441-7.