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Rheumatology: Current Research

Short Communication

# Neurological Complications of COVID-19: Insights from a Case Study and Literature Review

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## DESCRIPTION

With the global COVID-19 pandemic, a substantial number of patients have developed neurological lesions, including CLOCCs (Cytotoxic Lesion of the Corpus Callosum). CLOCCs is a rare, self-limited central nervous system disease that is characterized by a decrease in the Apparent Diffusion Coefficient (ADC) of the corpus callosum. It is currently considered to be caused by cytotoxic edema [1]. There are many causes of CLOCCs, among which infection and trauma are the most common. The neurological symptoms of CLOCCs, including changes in behavior and consciousness, neck stiffness, delirium, irritability, hallucinations, dizziness, transient disturbance of consciousness, epilepsy, cognitive impairment, dyskinesia, dysarthria, slurred speech, ataxia, visual impairment, and acute urinary retention, are known to be common neurological symptoms [2]. However, there are still many aspects of diagnosing, treating, and understanding the pathogenesis of these patients that require further in-depth study. This article aims to provide fresh insights for future clinical diagnosis and research into the novel coronavirus's impact on the nervous system by examining and dissecting the diagnosis and treatment journey of a 40-year-old female patient who holds a doctorate in the arts and is afflicted with COVID-19-related CLOCCs.

Shortly after testing positive for COVID-19, the patient exhibited symptoms indicative of rapidly progressive nervous system damage. The swift decline in this patient's symptoms stands out compared to other autoimmune-related neurological conditions. Unfortunately, direct evidence of inflammation and autoimmune reactions such as IL-6 was not evident in the early stages, making it challenging to ascertain whether the response was an acute autoimmune reaction or a lesion caused by direct viral infection [3].

Throughout the treatment course, despite undergoing two gamma globulin treatments and one hormone shock treatment, her symptoms persisted and even worsened, manifesting as nystagmus, blurred vision, and diplopia. Although serum IL-6 tested negative at the time and no acute inflammatory response or common autoimmune antibodies were detected in cerebrospinal fluid, we hypothesise that the escalating neurological symptoms could be linked to molecular or organelle-level influences.

During the course of treatment, extrapyramidal symptoms, particularly gait instability and tremors, were notably pronounced. It is worth noting that the administration of benzhexol and gabapentin significantly exacerbated involuntary tremors. Upon discontinuation of these medications, donepezil was prescribed. The exacerbation of tremors by banks is believed to be due to their blocking of calcium or sodium channels.

Gabapentin regulates excitatory neurotransmitter release, particularly glutamic acid, norepinephrine, and substance P [4]. Therefore, it is suspected that changes in ion channels and neurotransmitters may be implicated. Donepezil primarily works by inhibiting acetylcholine, suggesting a potential association with increased acetylcholine levels or heightened sensitivity.

From an imaging perspective, initial involvement was focused on the splenium of the corpus callosum and the left cerebellar arm. Although initially treated for acute ischemic cerebrovascular disease, the treatment's efficacy was suboptimal, and symptoms continued to worsen. Furthermore, the relatively minor infarction of the corpus callosum and low likelihood of cerebrovascular disease attributable to COVID-19 further complicate the diagnostic picture [3].

An 8-month follow-up revealed mild cerebellar atrophy primarily affecting the cerebellar hemisphere and vermis, with no recurrence of abnormalities in the splenium of the corpus callosum. These changes may be linked to cerebral hypoxia induced by COVID-19 as well as persistent damage from long-COVID syndrome and post-COVID syndrome, along with the lingering presence of SARS-CoV-2 viral remnants, although further clarification is warranted [3,5,6].

Rehabilitation adjunct therapy has shown favourable results during treatment, highlighting the importance of long-term treatment and rehabilitation training in achieving optimal clinical outcomes. Further research across all aspects of human physiology is crucial to understanding whether COVID-19 causes lasting nerve damage and its progression.

The patient, an accomplished artist and doctor, displayed numerous mental symptoms after onset, including irritability,

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**Received:** 14-Apr-2024, Manuscript No. RCR-24-30774; **Editor assigned**: 17-Apr-2024, PreQC No. RCR-24-30774 (PQ); **Reviewed:** 02-May-2024, QC No. RCR-24-30774; **Revised:** 09-May-2024, Manuscript No. RCR-24-30774 (R); **Published:** 16-May-2024, DOI: 10.35841/2161-1149.24.14.398

**Citation:** Li J, Jia XD, Ge J, Yao G (2024) Neurological Complications of COVID-19: Insights from a Case Study and Literature Review. Rheumatology (Sunnyvale). 14:398.

delusions of victimization, and memory loss. Some events from the onset were difficult to recall. While mood stability improved over time, paranoia, irritability, and diminished intelligence persisted. Three months after onset, the patient's MMSE (Mini-Mental State Examination) score was 28 with a score of 0 in graphic drawing. This may be linked to ataxia symptoms; however, even with disease remission, artistic and cognitive abilities failed to fully recover. Given current findings on COVID-19-related damage, parallels with cognitive impairment similar to Alzheimer's disease emerge; further observation is necessary for confirmation [7].

## CONCLUSION

This case highlights the pronounced and severe disease course observed in CLOCC patients associated with COVID-19 compared to other causes. It emphasizes the importance of early differentiation of these complications from ischemic cerebrovascular diseases. We advocate for a deeper understanding of COVID-19's impact on the nervous system and collaborative efforts to develop more effective treatment modalities to mitigate long-term consequences.

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