



CASE REPORT

New Onset of Multiple Sclerosis Following Recent COVID-19 Infection

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Abstract

Multiple sclerosis (MS) is a chronic autoimmune disease that affects the central nervous system. There is evidence to support a link between herpesvirus infections and an associated increased risk of developing MS. Over the past two years, neurological complications associated with COVID-19 infection have been reported and are speculated to be caused by direct viral invasion. A relatively low connection between COVID-19 infection and MS development has been identified in the scientific literature. Herein, we describe a unique case of newly-diagnosed MS following a recent COVID-19 infection.

Introduction

Multiple sclerosis (MS) is a chronic autoimmune disease that affects the central nervous system (CNS) and is defined by the presence of inflammation, demyelination, gliosis, and neuronal loss [1]. Women are three times more susceptible than men at contracting MS [2]. Epidemiologic observations suggest a polygenic etiology for MS along with a higher disease incidence in people who live in northern Europe and North America [3].

There is evidence to support the association between herpesvirus infections (EBV and VZV) increasing the risk of developing MS [4]. Over the past two years, neurological complications associated with SARS-CoV-2 infection have been reported and are speculated to be caused by direct viral invasion [5]. A relatively low connection between SARS-CoV-2 (COVID-19) infection

and MS development has been identified in the scientific literature [6].

Herein, we describe a unique case of newly-diagnosed MS following a recent COVID-19 infection.

Case Description

A 43-year-old male with a past medical history of hyperlipidemia and right-sided ulnar neuropathy, who was vaccinated for COVID-19, and contracted a moderate case of COVID-19 infection. He initially presented with a temperature of 104 °F and a cough. He did not require hospitalization or oxygen supplementation. He presented to the emergency department approximately 8 weeks later with progressive weakness and worsening paresthesia. His symptoms of paresthesia initially began in the bilateral fingertips and toes and eventually progressed proximally to the thighs and abdomen. He further reported feeling a band-like sensation in the lower chest/upper abdomen along with generalized fatigue, fasciculations, and lower extremity weakness upon ambulation.

On neurological examination, allodynia was noted in the proximal lower extremities and lower abdomen, as well as a decreased sensation of vibration, light touch, and temperature in the lower extremities, bilaterally. No evidence of nystagmus, motor weakness, clonus, or hyperreflexia was noted. Babinski's sign was negative. He was admitted for further workup and management of his progressive weakness and paresthesia.



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MRI of the brain showed foci of signal abnormality involving the posterior body and splenium of the corpus callosum, along the undersurface of the corpus callosum and in the upper cervical spinal cord. CT spine imaging showed cervical and thoracic spinal cord T2 hyperintense lesions, suggestive of active demyelination.

Laboratory results showed the presence of oligoclonal bands in the CSF that were not present in the patient's corresponding serum sample. Serum EBV VCA IgG and nuclear antigen were positive. CSF and serum studies ruled out autoimmune, infectious, or nutritional causes. His vitamin D level was low and he was started on supplementation. During his hospitalization, the patient reported complaints of increased bilateral eye pressure with mild light sensitivity. Ophthalmology was consulted, and upon fundoscopic exam, optic neuritis was excluded. He was treated with 5 days of IV Methylprednisolone but had minimal response. He was discharged with baclofen for lower extremity muscle spasms and gabapentin for neuropathic pain in his fingertips, respectively. At his one-month follow-up with an MS specialist, the patient noted improvement in his symptoms one week following his discharge. Neurologic exam was unremarkable and the decision was made to start B-cell depleting therapy with ofatumumab (Kesimpta).

Discussion

It is known that certain viruses such as EBV can act as potential triggers for developing multiple sclerosis. While the exact pathophysiology behind this phenomenon is still unclear, the currently accepted theory is that viral antigens that are structurally similar to myelin are presented by antigen-presenting cells to auto-reactive CD4+ helper T cells, in turn inducing inflammatory destruction of nerve tissue [7]. Other viruses such as VZV, influenza, or adenovirus have been associated with more frequent and severe relapses in patients with MS [8].

While, COVID-19 has been shown to cause certain neurological manifestations such as anosmia, dysgeusia, and Guillain-Barre Syndrome, there is little evidence demonstrating the implication of COVID-19 as a possible trigger for developing a CNS demyelinating disease like MS. There have only been a few cases where COVID-19 infection has been linked to the development of CNS demyelinating diseases [9,10]. One of which describes a patient who presented with decreased visual acuity, retro-ocular pain, lower extremity hyperreflexia, and ankle clonus following COVID-19 infection. The patient was also shown to have periventricular lesions on brain MRI and the presence of oligoclonal bands in the CSF confirming the diagnosis of MS [9]. Previous animal studies have demonstrated that intracranial inoculation of mice with Coronavirus mouse hepatitis virus (MHV) induced acute severe encephalomyelitis infecting oligodendrocytes, astrocytes, and microglia, with persistent viral expression infiltrating T-cell and

macrophage demyelination within the CNS [11].

We report a unique case of a patient being diagnosed with MS following COVID-19 infection a month prior to admission. Based on the temporal relationship of physical symptoms, it is possible that the infection may have precipitated the disease. It is also noteworthy that the patient tested positive for EBV VCA IgG and nuclear antigen, which would suggest EBV reactivation. However, the patient did not report any previous diagnosis of EBV or exhibited symptoms of prior infection. It is unclear whether the patient's COVID-19 infection induced the development of MS or whether COVID-19 induced the reactivation of EBV, which in turn induced the development of MS. The etiopathology of the aforementioned phenomena needs to be investigated further. It has been shown that patients infected with COVID-19 who have a history of EBV infection tend to test positive for reactivated EBV during the acute phase. Indicating COVID-19's ability to reactivate EBV [12]. Therefore, it may be conceivable that this patient's COVID-19 infection reactivated his prior EBV triggering the development of MS. It is also possible that co-infection of COVID-19 and EBV may have expedited the development of MS symptoms.

Conclusion

MS is an inflammatory disease that results in CNS demyelination. It has been associated with viral triggers such as EBV, VZV, Influenza, or Adenovirus. However, only a few cases have shown COVID-19 as a potential trigger for MS. This case not only illustrates COVID-19 as a possible trigger for the development of MS but also presents a question of whether co-infection with or reactivation of EBV might have contributed to the development of MS.

Author Contributions

Conceptualization: AO, LD, JG & AP. Data curation: AO & LD. Project administration: AO. Original draft preparation: AO, LD, JG & AP. Writing, review and editing, AO, LD, JG & AP. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

Ethical Standard

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1975 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was obtained from the patient included in the study.

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