Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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New-onset neuromyelitis optica spectrum disorder in a patient with COVID-19 and chronic Hepatitis B co-infection

Dmitri Kovalev*, Neeharika Thottempudi, Adil Ahmed, Elena Shanina

The University of Texas Medical Branch at Galveston, United States

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ABSTRACT

Background: Neurological autoimmune disorders are often triggered by bacterial and viral infections, with growing evidence supporting coronavirus disease 2019 (COVID-19) infection precipitation of these disorders. COVID-19 is already implicated in causing discrete para-infectious neurological syndromes: acute disseminated encephalomyelitis (ADEM), transverse myelitis, neuromyelitis optica spectrum disorders (NMOSD), Guillain-Barré syndrome (GBS), and is also associated with encephalopathy, acute cerebrovascular disease, neuromuscular disorders, and seizures.

Case Presentation: We describe a case of a 43-year-old Asian woman with chronic Hepatitis B (HBV) co-infected acutely with COVID-19, presenting with urinary retention, bilateral blindness, thoracic sensory level, and quadriparesis. Extensive workup narrowed down her diagnosis as seronegative NMOSD. She had complete resolution of symptoms after treatment with concurrent plasma exchange (PLEX), high dose corticosteroids, and emtricitabine-tenofovir. Follow-up visit showed no seroconversion at 6 months and no relapses.

Conclusions: Our literature review highlights the likely link between COVID-19 infection and the development of neurologic autoimmune diseases. Our literature review supports a virus-triggered immune-mediated process rather than neuro-invasion. Many viral illnesses have been linked to the development of NMOSD and anti-AQP4 antibody-related myelitis. Additionally, there is limited literature linking chronic HBV infection with the development of optic neuritis and speculation thatcross-reactivity between HBsAg and myelin antigens may lead to the development of demyelinating diseases in the CNS and PNS. We observed remarkable clinical improvement after treatment with alternating days of IV methylprednisolone and therapeutic PLEX.

Introduction

The pathogen causing the outbreak of coronavirus disease 2019 (COVID-19) is termed “severe acute respiratory syndrome coronavirus 2” (SARS-CoV-2) and has been implicated in causing many para-infectious neurological syndromes: acute disseminated encephalomyelitis (ADEM), transverse myelitis, neuromyelitis optica spectrum disorders (NMOSD) and Guillain-Barré syndrome (GBS) (Hassett et al., 23). In a recent systematic review, patients with COVID-19 were found to have various patterns of brain and spinal cord demyelination resembling autoimmune neurologic disorders (Ismail and Salama, 2021).

The pathophysiology of COVID-19 neurological involvement is not clearly established. There is abundant skepticism of the view that COVID-19 directly enters the CNS by retrograde axonal transport mechanisms of the olfactory nerve or other cranial nerves (Baig, 2020 Oct 21; Butowt et al., 2021). Substantial evidence suggests COVID-19 causes a “cytokine storm” with proinflammatory cytokines, including IL-1β, IL-2, IL-4, IL-6, IL-8, IL-10, TNF-α, and IFN-γ that may cross the blood-brain barrier. (Ismail and Salama, 2021) In this manner, the innate immunity mediators of the CNS such as macrophages, microglia, and astrocytes can be affected. Outside the CNS, hyperactivation of proinflammatory T cells, lymphopenia, decreased activity of regulatory T cells may also lead to antibody-mediated damage to the CNS and PNS.

Currently, there is insufficient evidence to demonstrate a strong association between chronic Hepatitis B (HBV) infection and the development of NMOSD. Based on our literature review, COVID-19 is highly immunogenic and is the most likely trigger for demyelination seen in our patient.

Case presentation

A previously healthy 43-year-old Vietnamese woman presented to the hospital with one week of urinary retention and progressive severe bilateral vision loss. Review of systems was otherwise unremarkable. She denied recent infections, travel, or trauma. On admission, she required catheterization for urinary retention, and her visual acuity was

* Corresponding author.
E-mail address: dmitrikovalev@gmail.com (D. Kovalev).

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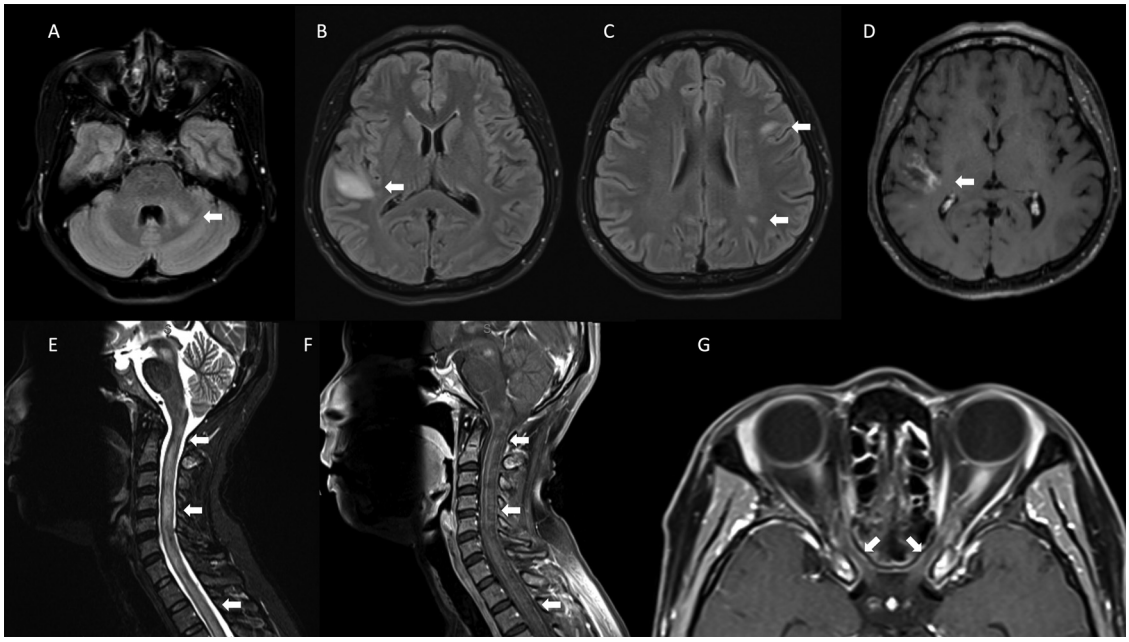

Fig. 1. Magnetic resonance imaging (MRI) findings. A-C: axial T2 FLAIR images show multifocal supra- and infratentorial hyperintense white matter lesions including a large hyperintense lesion in the right temporal lobe. D. Post-contrast axial T1 study demonstrates contrast enhancement. E. Sagittal TIRM (turbo inversion recovery magnitude) image shows longitudinally extensive hyperintense lesion involving cervical and thoracic spinal cord. F. Post-contrast sagittal T1 study shows enhancement of the spinal cord lesion most pronounced above C6 level. G: Post-contrast axial T1 image of the orbit shows enhancement of the optic nerve sheath complexes.

reduced to finger counting, with bilateral loss of color vision. The fundoscopic exam showed mild bilateral disk edema. Otherwise, her cognitive, cranial nerve, motor, cerebellar and sensory examinations were initially normal. She tested positive for nasopharyngeal RAPID PCR for COVID as part of our hospital protocol and expressed positive serum IgM and IgG SARS-CoV-2 antibodies. However, she continued to be afebrile, free of respiratory symptoms or signs of infection. Her neurological deficits rapidly progressed in the following 2 days. Her cognition remained intact with no encephalopathy, cranial nerves were normal except for complete vision loss in both eyes, including loss of light perception, pain with extraocular eye movements. She developed bilateral lower extremity and truncal sensory loss moving up to T4–5 to pinprick and temperature as well as quadriplegia with 0/5 strength in lower extremities on the MRC scale, 1/5 strength in upper extremities, areflexia throughout.

Initial cervical spine MRI was unremarkable. Brain MRI showed a large tumefactive, contrast-enhancing lesion in the right temporal lobe and multiple small T2 hyperintensities with enhancement in the frontal, parietal, cerebellar regions, all suggestive of demyelination. Considering rapid neurological decline, MRI cervical spine was repeated 2 days later demonstrating cord edema and longitudinally extensive T2 hyperintensities throughout the cervical and thoracic cord with patchy enhancement. Additionally, MRI of the orbits showed enhancement of the bilateral optic nerve sheath complexes indicative of optic perineuritis. (Fig. 1).

Diagnostic lumbar puncture showed mild CSF pleocytosis with 27 WBC's, protein of 49 mg/dL, glucose of 46 mg/dL. We did not check for SARS-CoV-2 in the CSF. Oligoclonal bands were absent. Aquaporin-4 receptor (AQP4) and Myelin Oligodendrocyte Glycoprotein (MOG) antibodies were both negative, tested by indirect fluorescent antibody assay utilizing full-length transfected cell lines. Additional tests for viral meningitis/encephalitis panel, West Nile virus, Brucellosis, Tuberculosis, Neurosyphilis, Lyme disease were negative. Laboratory results revealed a normal blood count and liver enzymes, with unremarkable serologies, including ANA, histone antibodies, SSA, SSB, RF. She was also negative for HIV, Hepatitis A and C. Nerve conduction studies (NCS) of the upper and lower extremities were normal, including F-waves.

Our patient was an asymptomatic chronic Hepatitis B carrier with the following serology: Hepatitis B surface (HBs) antigen positive, HBs antibody negative, Hepatitis B core (HBc) antibody positive (unspecified if IgM or IgG), Hepatitis B envelope (HBe) antigen negative and HBe antibody positive. She denied recent travel, history of intravenous drug use, tattoos, unprotected sexual intercourse. There was no transaminitis and no laboratory evidence of liver dysfunction throughout the admission. Initially, we found a low detectable, non-quantifiable level of HBV on quantitative assay by NAAT. Due to our plan to treat her with high-dose steroids, she was at increased risk for HBV reactivation, therefore we started antiviral therapy with tenofovir & emtricitabine (Truvada) to prevent an HBV flare. She continued to have normal liver enzymes and repeat testing for HBe antigen was negative. On 4-month follow-up, quantitative HBV assay by NAAT increased to 1311 IU/ml with no other lab abnormalities.

Based on clinical and neuroimaging findings of bilateral optic neuritis, longitudinally extensive transverse myelitis, atypical supratentorial and infratentorial demyelinating lesions, she was diagnosed with double seronegative NMOSD. She was treated with concurrent therapeutic plasma exchange (PLEX) and intravenous 1 g methylprednisolone on alternating days for a total of 5 sessions each. At the same time, antiviral therapy with tenofovir & emtricitabine was initiated to prevent Hepatitis B reactivation. Upon completion of treatment, her strength in the upper extremities normalized to 5/5 and in the lower extremities improved to 4/5. Color vision returned and visual acuity improved to 20/30. She still had mild residual sensory changes in the distal legs and required self-catheterization for remaining urinary retention. She was discharged home on an oral prednisone taper.

After one month, her urinary retention resolved, her strength improved, and she returned to full-time work without limitations. At the 6-month follow-up, her exam showed intermittent bilateral lower extremity paresthesias and generally brisk reflexes. She did not show seroconversion on repeat testing of serum AQP4 and MOG antibodies via cell assays. Repeat MRI showed resolution of the spinal cord and optic nerves lesions, having only a small residual T2 hyperintensity in the right temporal lobe without enhancement.

Discussion

Our patient fulfilled MRI requirements for NMO on repeat imaging with posterior optic nerve T1-weighted gadolinium enhancing lesions extending over more than one-half the optic nerve length and involving the optic chiasm, intramedullary lesion extending over ≥3 contiguous segments in the spinal cord, (Wingerchuk et al., 2015) demyelinating white matter lesions, although negative for AQP4 antibodies on initial and follow-up testing.

It is known that Hepatitis B vaccinations are not causally associated with the development of MS or optic neuritis. (Mormile, 2015) Despite one study of 10 patients with chronic HBV and seropositive NMOSD hypothesizing a cross-reactivity between HBsAg and myelin proteins, we believe there is currently insufficient evidence to suggest that chronic HBV increases the risk for developing a demyelinating disorder. (Liu et al., 20) COVID-19 is far more likely to be an immunological trigger to developing autoimmune neurological disorders.

A few case reports have suggested the potential for COVID-19 to reactivate HHV-6, HHV-7, and EBV. (Jumah et al., 2021a; Drago et al., 2021; Ciccarese et al., 2020) The risk of HBV reactivation in the setting of COVID-19 co-infection is low; however, this risk is increased with rituximab, high-dose corticosteroids, anthracyclines, and potent TNF-alfa inhibitors. (Liu et al., 2021) In one study, 3 patients with chronic HBV have seen a re-activation after infection with COVID-19. (Liu et al., 2020) Two of these reactivations were thought to be secondary to high dose methylprednisolone, with one unexplained. It is recommended to trend HBV-DNA quantitatively and to use antivirals such as Entecavir or Tenofovir before immunosuppressive treatment in COVID-19 patients with chronic hepatitis B.

We performed a PubMed and Google scholar database search using keywords COVID-19, NMOSD, ADEM, myelitis, optic neuritis, from December 2020 to May 2021. We found 23 case reports of COVID-19 associated optic neuritis and myelitis (see "Table 1" in appendix). Most patients had no significant past medical history, with ages ranging 15 - 70 years old: 12 male (52.2%), 11 female (47.8%). Myelitis was the predominant finding present in 86.9% (20/23) of cases. The average time from COVID-19 infection to neurological symptoms was between 2 and 14 days. One patient had only clinical evidence of myelitis, MRI spine was negative. (de Ruijter et al., 2020) Various treatment regimens were tried: IV steroids, PLEX, IV steroids alternating with PLEX, or intravenous immunoglobulin (IVIG) followed by steroids. IV steroids alone were the most effective treatment.

Out of 16 patients tested, 3 were positive for AQP4 antibody and had longitudinally extensive transverse myelitis (LETM). MOG antibody was positive in 2 patients, both with bilateral optic neuritis. There were 2 reported cases of myelitis with coexistent acute motor axonal neuropathy (axonal GBS variant); one had positive anti-GD1B antibody in serum. (Masuccio et al., 2021; Maideniuc and Memon, 2021) Although the results of nerve conduction studies in our patient were normal, it is important to rule out co-existing acute peripheral nerve involvement which would alter management.

From our literature review, CSF PCR for COVID-19 was obtained in 13 patients out of 23 and all of them tested negative. This supports the theory of immune-mediated mechanisms rather than direct neuroinvasion of the virus. Although long-term outcomes in COVID-19 associated demyelinating disorders are unknown, early recognition of these disorders and prompt treatment is critical for reducing morbidity.

Declaration of Competing Interest

None.

Appendix

Table 1
Reported COVID-19 associated optic neuritis/myelitis.

Case Report	Neurological Findings	AQP4 Antibody	Oligoclonal bands	MOG Antibody	Treatment	Recovery
Rodriguez et al. (Rodríguez de Antonio et al., 2021)	Myelitis	–	–	–	IV steroids	Significant
Masuccio et al. (Masuccio et al., 2021)	Myelitis	Not tested	+	Not tested	PLEX followed by IVIG	Mild to moderate
Batum et al. (Batum et al., 2020)	Myelitis	+	-	-	IV steroids and PLEX on alternate days	Significant
de Ruijter et al. (de Ruijter et al., 2020)	B/l Optic Neuritis	–	–	+	IV steroids	Significant
Ghosh et al. (Ghosh et al., 2020)	Myelitis, hiccups, nausea	+	–	–	IV steroids and rituximab	Significant
Sawalha et al. (Sawalha et al., 2020)	B/l Optic Neuritis	–	–	+	IV steroids	Significant
Zhou et al. (Zhou et al., 2020)	Optic Neuritis + Myelitis (LETM)	–	+	+	IV steroids, steroid taper	Significant
Palao et al. (Palao et al., 2020)	Optic Neuritis	–	+	–	IV steroids	Significant
Munz et al. (Munz et al., 2020)	Myelitis	–	–	–	IV steroids	Significant
Maideniuc et al. (Maideniuc and Memon, 2021)	Myelitis	–	Not tested	Not tested	IV steroids followed by PLEX	Mild to moderate
Chow et al. (Chow et al., 2020)	Myelitis (LETM)	–	Not tested	–	IV steroids	Significant
AlKetbi et al. (AlKetbi et al., 2020)	Myelitis (LETM)	Not tested	Not tested	Not tested	IV steroids	Significant
Chakraborthy et al. (Chakraborty et al., 2020)	Myelitis	Not tested	Not tested	Not tested	IV steroids	Died of COVID-19
Sotoca et al. (Sotoca and Rodríguez-Álvarez, 2020)	Myelitis (LETM)	Not tested	–	Not tested	IV steroids followed by PLEX, followed by IV steroids and taper	Mild to moderate
Zachariadis et al. (Zachariadis et al., 2020)	Clinical Myelitis (MRI negative)	Not tested	–	Not tested	IVIG followed by IV steroids	Mild to moderate
Fumary et al. (Fumery et al., 2021)	Myelitis	–	–	–	IV steroids	Significant
Zoghi et al. (Zoghi et al., 2020)	Myelitis	–	–	–	PLEX	Mild to moderate
Baghbanian et al. (Baghbanian and Namazi, 2020)	Myelitis (LETM)	–	–	–	PLEX	Mild to moderate
Valiuddin et al. (Valiuddin et al., 2020)	Myelitis (LETM)	Not tested	Not tested	Not tested	IV steroids and PLEX	Mild to moderate
Sarma et al. (Sarma and Bilello, 2020)	Myelitis	Not tested	–	Not tested	Oral prednisolone, 2 PLEX sessions	Significant
Shaw VC et al. (Shaw et al., 2020)	Optic Neuritis + Myelitis	+	Not tested	Not tested	(Not treated)	Died of COVID-19
Durrani et al. (Durrani et al., 2020)	Myelitis	–	–	Not tested	IV steroids	Significant
Jumah et al. (M. Jumah et al., 2021)	Myelitis	–	–	+	PLEX followed by oral steroids	Significant

Table 1 abbreviations: "BL" bilateral, "LETM" longitudinally extensive transverse myelitis, "PLEX" plasma exchange, "+" positive, "-" negative.

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