CASE REPORT

Encephaloradiculoneuropathy: A Rare Manifestation of COVID-19 Infection

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Abstract

Neurological complications are being recognized as important outcomes of the coronavirus disease-2019 (COVID-19) pandemic. We report a rare case of both the central nervous system (CNS) and peripheral nervous system (PNS) involvement, encephalitis with polyradiculoneuropathy in a single patient of COVID infection.

Keywords: Acute encephalitis, COVID-19 infection, Critical illness polyneuromyopathy, Guillain-Barré syndrome.

Indian Journal of Critical Care Medicine (2022): 10.5005/jp-journals-10071-24222

Highlights

This case is unique with respect to both the central and peripheral nervous system manifestations of coronavirus disease-2019 (COVID-19) infection in the same patient.

Also, this prolonged encephalitis as a manifestation of COVID-19 is unique in its presentation and has not been reported to date.

Introduction

An outbreak of the novel coronavirus NCoV-19 (SARS-CoV-2) responsible for the coronavirus disease has rapidly spread globally with approximately 100 million confirmed infections and 2.3 million deaths to date. Neurological complications are being recognized as important outcomes of this pandemic. Central nervous system (CNS) manifestations like stroke, seizures, venous sinus thrombosis, meningitis, encephalitis, myelitis, CNS vasculitis, and PNS manifestations like GBS, myopathy have been reported with COVID-19 infection.[1,2]

We report a case of both CNS with PNS involvement and encephalitis with polyradiculoneuropathy in a single patient.

Case Report

A 56-year-old man, presented with fever, cough, headache, and myalgia for 5 days followed by an altered sensorium and three episodes of generalized tonic-clonic seizures. He was a diabetic and hypertensive and had past history of one cerebrovascular accident 12 years back. On examination he was drowsy arousable to deep pain stimulus, febrile with pulse rate 120/minute, blood pressure 100/70 mm Hg, and tachypneic with RR—20/minute. His COVID RT-PCR from the nasopharyngeal swab was positive.

He was admitted to the COVID critical care unit. His Chest CT scan showed bilateral ground-glass opacities, interstitial and septal thickening, traction bronchiectasis suggestive of CO-RADS 5 grade III COVID pneumonia with moderate 50–60% parenchymal lung involvement.

All his metabolic parameters like- serum electrolytes, serum glucose, serum ammonia, renal function tests, liver function tests, serum procalcitonin, and CO_2 levels were normal. CRP and LDH were raised. His MRI brain was normal. His EEG was showing intermittent generalized slowing. Autoimmune encephalitis antibodies (NMDA, AMPA, GABA) were negative. The patient was started on the treatment for COVID infection. He was given injection methylprednisolone, injection remdesivir, and low molecular weight heparin.

Two weeks later he developed acute onset progressive flaccid areflexic quadriparesis, without bowel bladder involvement.

A neurological examination showed weakness in all four limbs and truncal and neck flexor weakness. Medical Research Council (MRC) score is 1/5. Deep tendon reflexes were absent in all limbs. Babinski's sign was positive. The cranial nerves were spared.

His nerve conduction study (attached) showed diffuse axonal pure motor polyradiculoneuropathy and EMG showed cervical and lumbosacral radiculopathy. MRI brain with the spine was repeated which was normal, there was no root enhancement. CSF showed albuminocytological dissociation WBC (0.001 × 109/L), protein (0.8 g/L). CSF COVID RT-PCR was negative. He was started on IV immunoglobulin 2 gm/kg for 5 days. There was an improvement in his weakness. However, the patient had one episode of generalized tonic-clonic seizure in the hospital and his sensorium further deteriorated he was immediately intubated and put on ventilatory support. He developed post-COVID lung fibrosis and eventually succumbed (Fig. 1).

Discussion

SARS-CoV-2 is the largest and the most severe pandemic since the 1918 influenza pandemic.

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How to cite this article: Chakor RT, Barvalia PP, Nadaf S, Manjunath V. Encephaloradiculoneuropathy: A Rare Manifestation of COVID-19 Infection. Indian J Crit Care Med 2022;26(5):646–648.

Source of support: Nil

Conflict of interest: None

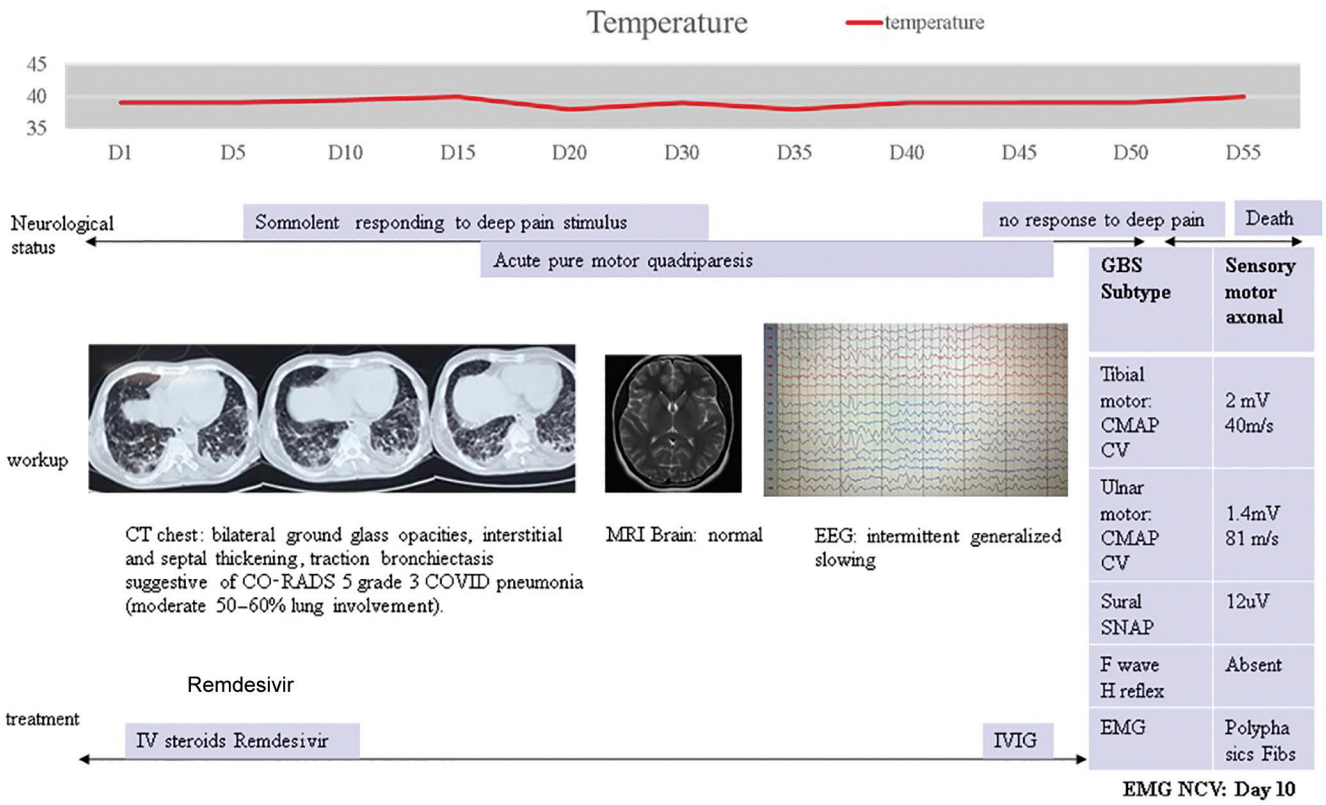

Fig. 1: The image depicts time line of the patient from day 1 to day 55 of hospital stay. CMAP, compound motor action potential; CO-RADS, coronavirus disease-2019 (COVID-19) reporting and data system; CT, computer tomography; EEG, electroencephalogram; GBS, Guillain-Barré syndrome; MRI, magnetic resonance imaging; NCV, nerve conduction velocity

Both CNS and PNS symptoms have been described with COVID-19 infection.[2] These clinical manifestations can be considered as direct effects of the virus on the nervous system, parainfectious, postinfectious immune-mediated disease, or neurological complications of the systemic effects of COVID-19.

Encephalitis is the inflammation of the brain parenchyma, usually caused by an infection or the body's immune defenses. Clinical evidence of brain inflammation, such as a CSF pleocytosis, neuro-imaging changes, or focal abnormalities on EEG, and exclusion of all causes of encephalopathy, indicates a diagnosis of encephalitis.[3]

Encephalopathy has been reported for 93 patients in total, including 16 (7%) of 214 hospitalized patients with COVID-19 in Wuhan, China, and 40 (69%) of 58 patients in intensive care with COVID-19 in France. Encephalitis has been described in eight patients, and Guillain-Barré syndrome in 19 patients. SARS-CoV-2 has been detected in the CSF of some patients.[3]

In a Lancet Review of neurological manifestations of COVID-19 virus, three encephalitis cases were described, all three cases had a viral prodrome like our patient with fever, cough, headache followed by altered sensorium and seizures in two cases and rhombencephalitis with ataxia, diplopia and bifacial weakness in one case. All three cases had positive nasopharyngeal swab RT-PCR like our patient and only one case had positive CSF RT-PCR. All three cases had associated lung involvement. Our patient's CSF showed albuminocytological dissociation, other published cases had CSF pleocytosis. In our case, the patient had to slow on EEG, although neuroimaging was normal. MRI was abnormal in two published cases showing hyperintense signal in the right medial temporal lobe and hippocampus, right inferior cerebellar peduncle on T2-weighted images and EEG showed epileptiform discharges in one case. One patient expired and one was discharged.[3]

Normal MRI brain has often been reported in COVID encephalitis.[4]

In a case series of five patients with severe COVID-19-related encephalitis, treated with therapeutic plasma exchange and corticosteroids, three cases showed dramatic improvement.[5] Our patient was given immunotherapy with IV immunoglobulins and steroids.

In a study published in NEJM, five GBS cases were observed in 1,200 COVID patients. Their three GBS cases were similar to our case showing axonal polyradiculoneuropathy. On electromyography, fibrillation potentials were present in four patients.[6]

In the Lancet Review of Neurological manifestations of COVID-19, seven cases of GBS were reviewed. Five out of seven had lung involvement and albuminocytological dissociation like our patient. The nerve conduction study showed axonal polyradiculoneuropathy in three cases and demyelinating polyradiculoneuropathy in four cases. All cases were treated with IVIG and one case needed additional plasma exchange.[3]

For critically ill patients, determining whether the neurological features are nonspecific manifestations of critical illness or are specific to the virus itself is challenging. No reliable markers exist for neurological disease caused by critical illness, although it tends to occur after several weeks. Up to 70% of patients with sepsis might develop encephalopathy or polyneuropathy.[2] Hence the exclusion

of alternate etiology like sepsis, metabolic or critical illness-related neuro-myopathy is crucial in the diagnosis of COVID-19 related neurological manifestations.

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References

1. Montalvan V, Lee J, Bueso T, De Toledo J, Rivas K. Neurological manifestations of COVID-19 and other coronavirus infections: a systematic review. Clin Neurol Neurosurg 2020;194:105921. DOI: 10.1016/j.clineuro.2020.105921.
2. Kumar A, Kumar P, Ganapathy U, Goel K, Diwan S, Kohli S, et al. Neurological manifestations of COVID-19: a series of seven cases. Indian J Crit Care Med 2021;25(2):219–223. DOI: 10.5005/jp-journals-10071-23723.
3. Ellul MA, Benjamin L, Singh B, Lant S, Michael BD, Easton A, et al. Neurological associations of COVID-19. Lancet Neurol 2020;19(9):767–783. DOI: 10.1016/S1474-4422(20)30221-0.
4. Delorme C, Paccoud O, Kas A, Hesters A, Bombois S, Shambrook P, et al. COVID-19-related encephalopathy: a case series with brain FDG-positron-emission tomography/computed tomography findings. Ecol Manag Restor 2020:ene.14478. DOI: 10.1111/ene.14478 [Accessed November 9, 2020].
5. Cao A, Rohaut B, Le Guennec L, Saheb S, Marois C, Altmayer V, et al. Severe COVID-19-related encephalitis can respond to immunotherapy. Brain 2020;143(12):e102. DOI: 10.1093/brain/awaa337.
6. Toscano G, Palmerini F, Ravaglia S, Ruiz L, Invernizzi P, Cuzzoni MG, et al. Guillain–Barré syndrome associated with SARS-CoV-2. N Engl J Med 2020;382(26):2574–2576. DOI: 10.1056/NEJMc2009191.