Case report

COVID-19 associated unilateral sciatic neuropathy

S C Weerasinghe[1], S P Mohotti[2], N Harishchandra[3], K G Dayaratne[4], M C Caldera[1]

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Abstract

A 36-year-old woman developed right leg weakness with foot drop while being treated for COVID-19 pneumonia. Her nerve conduction test and electromyography confirmed right sciatic nerve lesion and MRI scan of the lumbar spine, sciatic nerve, and examination of cerebrospinal fluid were unremarkable. Since there was no other possible explanation, the cause was attributed to a neurological complication of COVID-19 infection.

Introduction

In the latter part of 2019, Corona Virus Disease 2019 (COVID-19) epidemic emerged and spread rapidly across the globe becoming a pandemic by March 2020. The causative virus is known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The most common manifestation of the disease is respiratory system related symptoms. Although the neurological manifestations are not the commonest, the scale of the current pandemic could result in large number of cases with neurological complications[1].

The neurological manifestations could be para-infectious or post infectious. The reported neurological manifestations of the disease include anosmia, meningitis, encephalitis, myelitis, central nervous system (CNS) vasculitis, acute disseminated encephalomyelitis (ADEM), Bell's Palsy, Guillain-Barré syndrome (GBS), other acute neuropathies and stroke. These manifestations may be related to non-specific complications of the systemic disease, direct invasion by the virus or the inflammation of the nervous system or the vasculature[2,6].

In this case report we present a case of COVID-19-related unilateral sciatic mono-neuropathy.

History

A 36-year-old woman with no previous co-morbidities, who was unvaccinated for COVID-19, presented with fever and shortness of breath. She had a positive SARS-CoV-2 polymerase chain reaction (PCR) test on nasopharyngeal swab on 13th of June 2021. With the worsening of respiratory symptoms, she was diagnosed as COVID-19 pneumonia and admitted to the High Dependence Unit. She received oxygen therapy, subcutaneous enoxaparin, and dexamethasone, empirical antibiotics, but no biologics for her pneumonia. On 25th of June 2021 (after 12 days from presentation) during her HDU stay, she developed weakness of right leg with foot drop and paraesthesia.

The neurological examination revealed, a high stepping gait due to right foot drop, weakness in knee flexion (MRC 3/5), ankle plantar flexion (MRC 2/5) and ankle dorsiflexion (MRC 2/5). Normal power was observed in hip flexion, extension, abduction, and adduction as well as knee extension.

The right ankle reflex was absent with preserved knee reflex. Sensory impairment was noted below knee except for a narrow area down the medial side of the lower leg and along the medial boarder of the foot. Examination of left lower limb and both upper limbs were neurologically normal. She did not have cranial nerve impairment.

The electromyogram showed, severe proximal right sciatic nerve neuropathy with preferential involvement of the peroneal fibres. MRI lumbosacral spine showed normal vertebral heights, disc spaces and roots. The MRI scan of right sciatic nerve showed normal signal intensity and anatomy, and no contrast enhancement was seen. Cerebrospinal fluid (CSF) analysis showed normal protein, cells, and sugar. CSF was negative for PCR and antibodies for SARS-CoV-2 while her serum showed positive SARS-CoV-2 antibody response. Her inflammatory markers were high during the acute phase of her pneumonia, but antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA) were negative.

Discussion

Although SARS-COV-2 has main manifestations related to the respiratory system, neurologic complications are not uncommon. A recent study found 36.4% of patients with COVID-19 develop neurological symptoms. Anosmia, myalgia, headache, encephalopathy, and dizziness are most common neurological manifestations. Severely affected patients are more likely to develop neurological symptoms than patients who have mild or moderate disease[3].

[1]*Neurologist,* [2]*Neurophysiologist,* [3]*Respiratory Physician,* [4]*Radiologist, Teaching Hospital, Anuradhapura, Sri Lanka.*
Correspondence: M C Caldera E-mail: calderamanjula@yahoo.com

The exact mechanisms of nervous system involvement by the SARS-CoV2 virus are still unknown. However, the possible routes of transmission could be retrograde neuronal transport across infected neurons, entry via the olfactory nerve, infection of the vascular endothelium, leucocytes migration across the blood-brain barrier, or via angiotensin-converting enzyme-2 receptors found on neurons and glial cells[8].

Peripheral nerve involvement in COVID-19 is not commonly reported and the available literature is limited. The mechanism of peripheral nerve involvement is not well understood and probably a result of inflammatory or immunological response of neurons to the virus.

Guillain-Barré syndrome (GBS) or its variants are one of the common manifestations of peripheral neuropathy related to SARS-CoV-2. These neurological manifestations began at a median of 7 days (7 to 24) after respiratory or systemic features. A review on neurological complications of COVID-19 stated, out of 12 GBS patient's 8 had demyelinating disease and axonal disease in remainder[2].

A case of peripheral neuropathy with electrophysiological evidence of mixed sensorimotor neuropathy had been reported. There is a case of painful neuropathy associated with the COVID-19, which is an uncommon type of a neuropathy[4]. A case of sciatic neuropathy was reported by Acharya et al with some similarities to the case we have reported[5].

In our case of right leg weakness following COVID-19 infection, the neurophysiological studies confirmed a right sided severe proximal sciatic nerve lesion. The MRI of the lumbosacral spine was normal with no enhancement to indicate radiculitis. Her CSF protein and cells were normal and SARS-COV-2 antibodies were negative.

The normal imaging has excluded a structural cause for the nerve lesion. The normal CSF makes an inflammatory radiculopathy less likely. Absence of CSF SARS-COV-2 antibodies makes an intrathecal production of immunoglobin G or disruption to the blood brain barrier less likely. She was not intubated, and her consciousness was intact throughout the hospital stay which makes a neuropraxic sciatic nerve lesion due to compression improbable. Unilateral involvement negates the possibility of critical illness neuropathy. Involvement of the sensory findings exclude the possibility of the anterior horn cell lesion.

Our patient was managed with oral dexamethasone short course, physiotherapy and rehabilitation with ankle foot orthosis and made a moderate improvement.

It is possible that the nerve involvement may be immune mediated or a direct viral effect on the peripheral nerve. We did not consider a nerve biopsy since the neurological deficits as a complication could be devastating and the patient made a moderate improvement with treatment.

Conclusion

Although neurological manifestations are now being reported in relation to COVID-19, mono-neuropathies are rare. It is important to consider COVID-19 as a differential diagnosis for the large nerve neuropathies in the appropriate context. Studies of more cases are necessary to define this entity, pathophysiology, and therapeutics since they can result in long term disability.

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