Para Infectious Guillain–Barre Syndrome (GBS) in Covid-19 – A Case Report

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INTRODUCTION

Covid-19 is a disease caused by severe acute respiratory syndrome corona virus 2 (SARS-CoV-2). SARS-CoV-2 which affects respiratory, gastrointestinal and neurological systems. It not only causes atypical pneumonia with acute respiratory distress syndrome (ARDS), but also, acute cardiac damage, acute renal failure and gastrointestinal complications.[1] It is a disorder that not only presents with fever and respiratory symptoms but can involve the nervous system with varied presentations in form of cerebrovascular accident, loss of taste, loss of smell, myelopathy, neuropathy, meningitis and encephalitis.[2] Some cases of Guillain–Barre syndrome (GBS) associated with SARS-CoV-2 have been reported in the literature. GBS is acute immune mediated inflammatory polyradiculopathy.[3] GBS presents as limb weakness or cranial nerve weakness, loss of deep tendon reflex, autonomic dysfunction due to peripheral nerve demyelination and sensory root demyelination.[4]

PRESENTATION OF CASE

A 22-year-old male patient came to the casualty with fever for 7 days followed by ascending weakness of both lower limbs and both upper limbs for 2 days. There was no history of back pain, headache, diarrhoea, trauma, sore throat, breathlessness and cough.

On physical examination, he was afebrile, pulse - 110 / min, regular, blood pressure 120 / 80 mmHg, respiratory rate was 16 / minute, and oxygen saturation of 98 % on room air.

On neurologic examination - Higher mental function was normal, all the cranial nerves were intact, motor system examination revealed weakness in four limbs with a Medical Research Council (MRC) scale of 1 / 5 in proximal, 1 / 5 in distal of the upper extremities and 1 / 5 in proximal, 1 / 5 in distal of the lower extremities. Deep tendon reflexes were absent. Sensory examination was normal. The patient was admitted to the isolation ward. Oropharyngeal swab was sent for RTPCR of Covid-19 which came out to be positive.

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DISCUSSION OF MANAGEMENT

Investigations revealed; Haemoglobin (Hb)- 12.1 gm %; Total leukocyte count (TLC) - 5800 cells / mm^3, platelets - 2.67 lakh / litre, mean corpuscular volume (MCV) - 85.8 fl, Urea - 32 mg / dL, creatinine - 0.70 mg / dL, sodium – 144 mmol / L, potassium - 5.5 mmol / L, liver and renal function tests were normal. His C-reactive protein - 0.5 mg / L (normal range < 10 mg / L), D dimer - 1.3 mcg / mL (normal range < 0.40 mcg / ml), serum ferritin - 67.3 ng / mL (normal < 300 ng / mL), interleukin-6 (IL) = 5 pg / mL (normal < 7 pg / mL). CSF analysis revealed; TLC - 3 cells / mm^3 all were lymphocytes, protein = 380 mg / dL, glucose = 59 mg / dL, lactate dehydrogenase (LDH) = 64 mg / dL. High-resolution computed tomography (HRCT) thorax revealed CT severity index of 0 / 25.

Nerve conduction study was suggestive of prolonged proximal latency and low amplitude compound motor action potential (CMAP) in left ulnar nerve (Figure 1), right tibial nerve (Figure 2) and left tibial nerve.

Non-recordable CMAP in median and perineal nerves absent F waves in left ulnar and tibial nerves. Normal sensory nerve action potential (SNAP) amplitude in both sural, median and ulnar, all above findings suggestive of demyelinating motor polyneuropathy. Diagnosis of GBS was made and the patient was started on tablet doxycycline 100 mg bid, vitamin C, vitamin D and intravenous immunoglobulins at a dose of 0.4 g / Kg for 5 days. There was a progressive recovery of muscle power council (MRC) scale of 1 / 5 in proximal, 1 / 5 in distal of the upper extremities and 1 / 5 in proximal, 1 / 5 in distal of the lower extremities and deep tendon reflexes over next 2 weeks. The patient became afebrile and was discharged after 2 weeks.

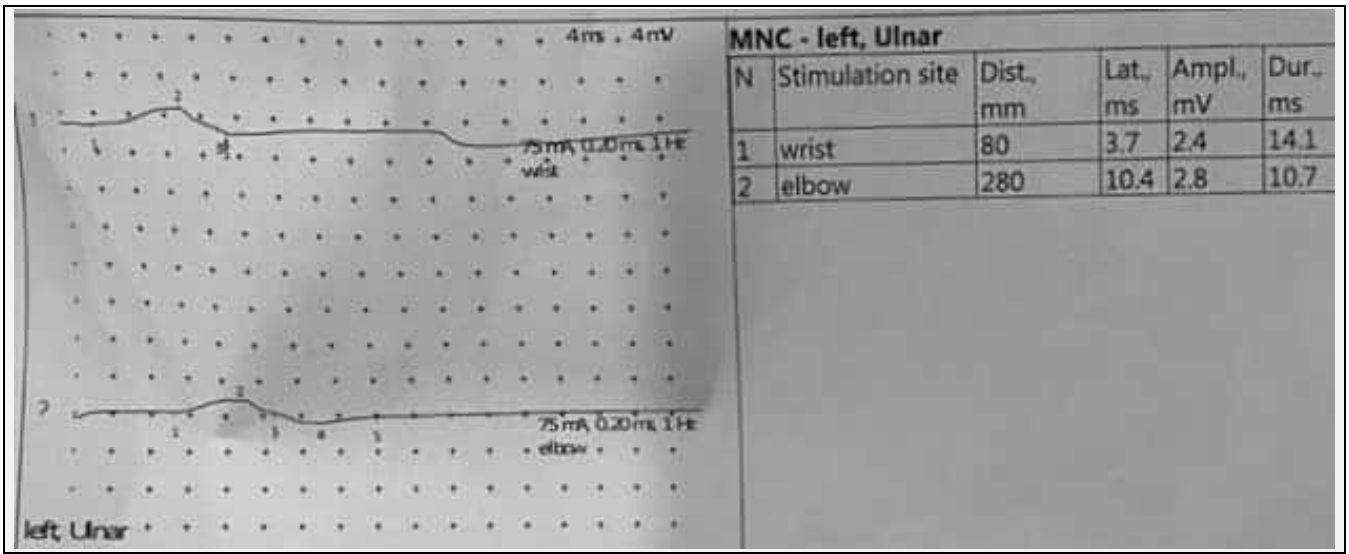

Figure 1. Showing Prolonged Proximal Latency and Low Amplitude CMAP and Absent 'f' Waves in Left Ulnar Nerve

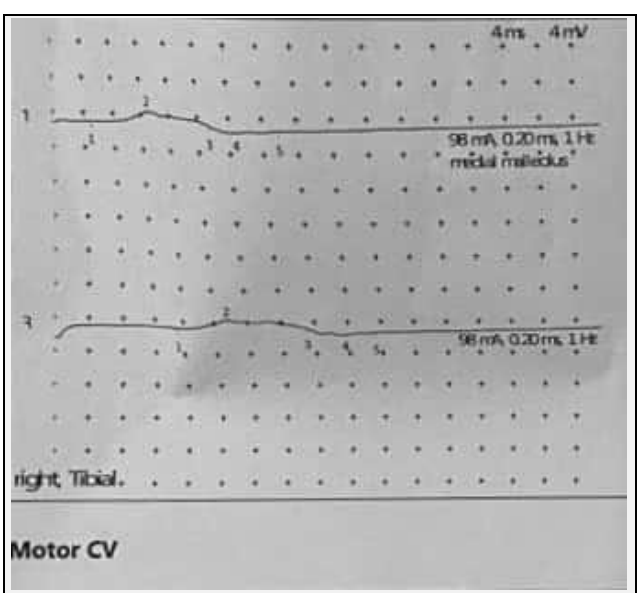

Figure 2. Nerve Conduction Study Showing Prolonged Proximal Latency, Low Amplitude CMAP and Absent 'f' Waves in Right Tibial Nerve

DISCUSSION

SARS-CoV-2 is a beta coronavirus that mainly affects the respiratory system causing atypical pneumonia and ARDS but affects other systems also. Covid-19 infects cells via angiotensin-converting enzyme 2 receptor which is present on the lung, kidney, liver, neurons and skeletal muscles.[5,6] In the brain SARS-CoV-2 enters via olfactory epithelium, cribriform bone and through interruption of blood brain barrier.[7,8] Proposed and established mechanisms of neurological involvement includes hypoxic brain injury and an immune mediated damage to the CNS.[9,10] Neurological complications in SARS-CoV-2 are acute cerebrovascular disease, encephalopathy, transverse myelitis, acute haemorrhagic necrotising encephalopathy, encephalitis, epilepsy, ataxia, GBS and myelopathy.

Covid-19 infection induces cytokine storm which leads to immune-mediated damage of neurons.[11] GBS is an autoimmune disorder. Covid-19 causes GBS by immune activation precipitated by cytokine storm and also through molecular mimicry.[12,13]

According to some case reports, there is an association between GBS in Covid-19 patients.[14] Para-infectious GBS

occurs during Covid-19 infection and post-infectious GBS arises after SARS-CoV-2.[15]

Interaction between SARS-CoV-2 spike protein with the N-acetylgalactosamine (GalNAc) residue of GM1 and ganglioside dimers for anchoring to surface of cell and cross-reaction of sugar residues of surface peripheral nerve glycolipids with an epitope of spike bearing gangliosides of peripheral nerves is the possible mechanism proposed for the nerve damage.[16] Molecular mimicry between SARS-CoV-2 and organs and tissues is a known mechanism of autoimmunity in Covid-19.[17,18] A study showed that SARS-CoV-2 cross reacts with human heat shock proteins (HSPs). Heat shock proteins are involved in development of multiple immune mediated disorders. Immunologic cross reaction through molecular mimicry causes GBS.[19] Our case did not have an overt cytokine storm so molecular mimicry could be the cause for GBS.

CONCLUSIONS

In certain forms of GBS, appearance of antibodies against specific gangliosides due to Covid-19 is still not clear. At present, number of patients of Covid-19 with neurological features and complications are less but in future number of patients with neurological features in Covid-19 will increase.[20]

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Disclosure forms provided by the authors are available with the full text of this article at jemds.com.
Informed consent was taken from the patient before writing this case report.

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