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SARS-COV-2, A NEW ETIOLOGICAL AGENT OF GUILLAIN-BARRE SYNDROME: A CASE REPORT

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ABSTRACT:

Guillain-Barre syndrome (GBS) is an acute progressive polyradiculoneuropathy where body's immune system mistakenly attacks the peripheral nerves and their spinal roots.Covid 19 is a highly contagious disease declared as a global pandemic in 2020. We report a case of GBS in asymptomatic non vaccinated (against novel corona virus) covid 19 patient. A 18 years old male patient with no co morbidities admitted with 2 days history of sudden onset weakness of all four limbs. He was tested covid 19 RTPCR positive with no previous history of any symptoms of covid 19. His anti SARS COV2 antibody was positive. Nerve conduction velocity study showed severe acute motor neuropathy. He was electively intubated and ventilated, successfully treated with intravenous immunoglobulins. We conclude SARS COV2 can induce immune system without any prodomal symptoms, so we should aware about SARS COV 2 as an etiological agent of GBS.

INTRODUCTION

The symptoms ofcovid19 can vary from asymptomatic illness to life threatening respiratory failure, acute respiratory distress syndrome. It could be associated with certain neurological symptoms like headache, dizziness,ageusia, anosmia and complications like febrile seizure, encephalitis, stroke and acute peripheral nerve diseases.[1,2] Guillain-Barre syndrome is an acute, progressive, paralytic polyneuropathy resulting from an autoimmuno response following an infectious disease, affecting the peripheral nerves and their spinal roots.[3] There are few reported cases about the incidence of GB syndrome in SARS COV2 positive patient[4-8]. Here we present a case of 18 years old male patient developed Guillain- Barre syndrome who was diagnosed Covid19 RTPCR positive (asymptomatic and non- vaccinated) and also positive for anti SARS COV2 antibody.

CASE REPORT

A 18 years old male patient with no co-morbidities admitted in emergency with H/O sudden onset inability to get up from bed and generalized weakness for last 2 days.He was diagnosed as covid 19 RTPCR positive. He had no previous H/O fever, cough, shortness of breath, diarrhea or any symptoms related to covid 19. He was non vaccinated against SARS COV2.On admission, he had weakness of proximal and distal muscle of both upper and lower extremities, neck muscle weakness, difficulty in getting up from supine position. He had difficulty in passing urine, so urinary catheterization was done. Physical examination showed normal temperature of 97°F, blood pressure 110/70 mm Hg, heart rate 61 beats/minute, respiratory rate 23/minute, and oxygen saturation of 97% on room air. On neurological examination, the patient was alert, conscious, and oriented with normal speech and higher mental functions. Cranial nerve examination showed suppressed gag and cough reflexes. Motor examination showed decreased tone, muscle weakness in 4 limbs with a Medical Research Council scale of grade 2/5 in proximal muscles, grade 3/5 in distal muscles of the upper extremities, grade 3/5 in proximal and distal muscles of the both lower extremities. Deep tendon reflexes were absent in all four limbs, planter responses were absent bilaterally. Sensory assessment was normal allover. Patient was put on elective invasive mechanical ventilation in view of rapid progression of weakness, depressed cough reflex and single breath count which was 15. Initial laboratory investigations were within normal limit. Anti SARS- COV- 2 antibody was positive, value 10 u/ml. Echocardiography study was normal. MRI brain, MRI whole spine study were absolutely normal. Motor nerve conduction study showed tibial (Figure 1), peroneal, median and ulnar nerves were in- exitable. Bilateral tibial (Figure 2), median and ulnar nerves F latencies were non recordable. Sensory nerve conduction showed onset latencies, conduction velocities and SNAPs amplitude were normal in all these nerves along with sural nerve (Figure 3). This report suggessed the patient had severe motor neuropathy. As NCV stydy confirmed the diagnosis of GBS and duration of illness was less than 7 days, we didn't do CSF study.There was fluctuation of heart rate from 60 to 110, systolic blood pressure was ranging fron 110 to 170 mm of Hg. cardiology opinion taken in view of cardiac dysautonomia. The patient received intravenous immunoglobulin (IVIG) at the dose of 2 gram per day for a duration of 5 days and he had marked improvement of his symptoms, he was extubated 5 days after installation of mechanical ventilation. Muscle function was improved by 2 grades. He was discharged 15 days after admission.

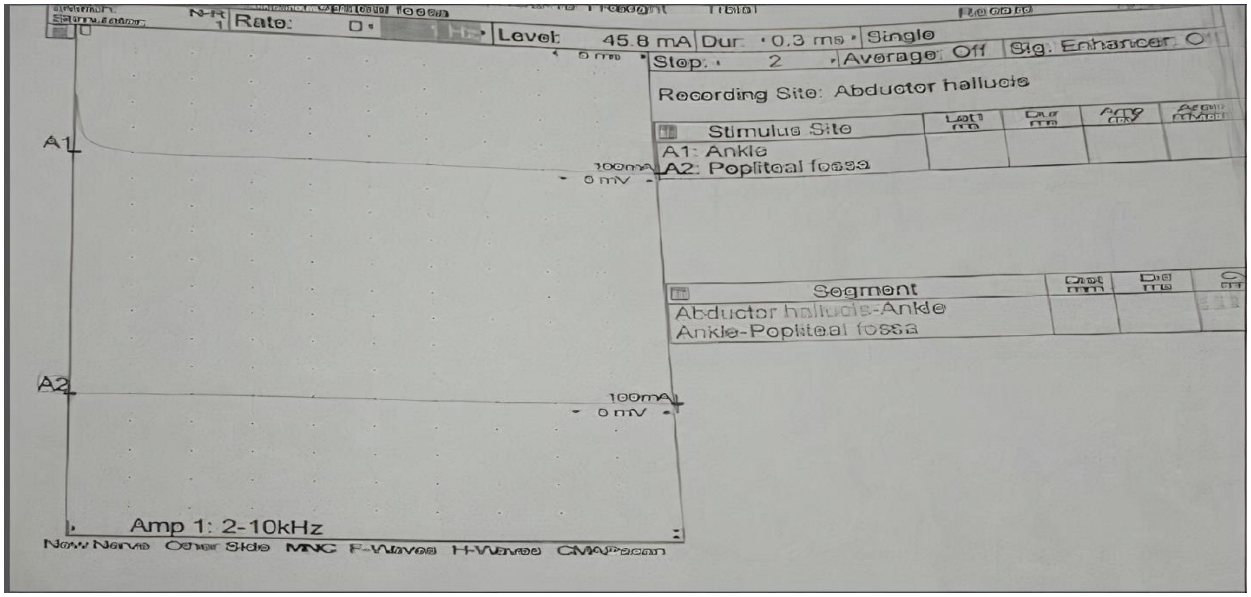

Figure1: Motor nerve conduction study of Right Tibial nerve showing in-exitable nerve

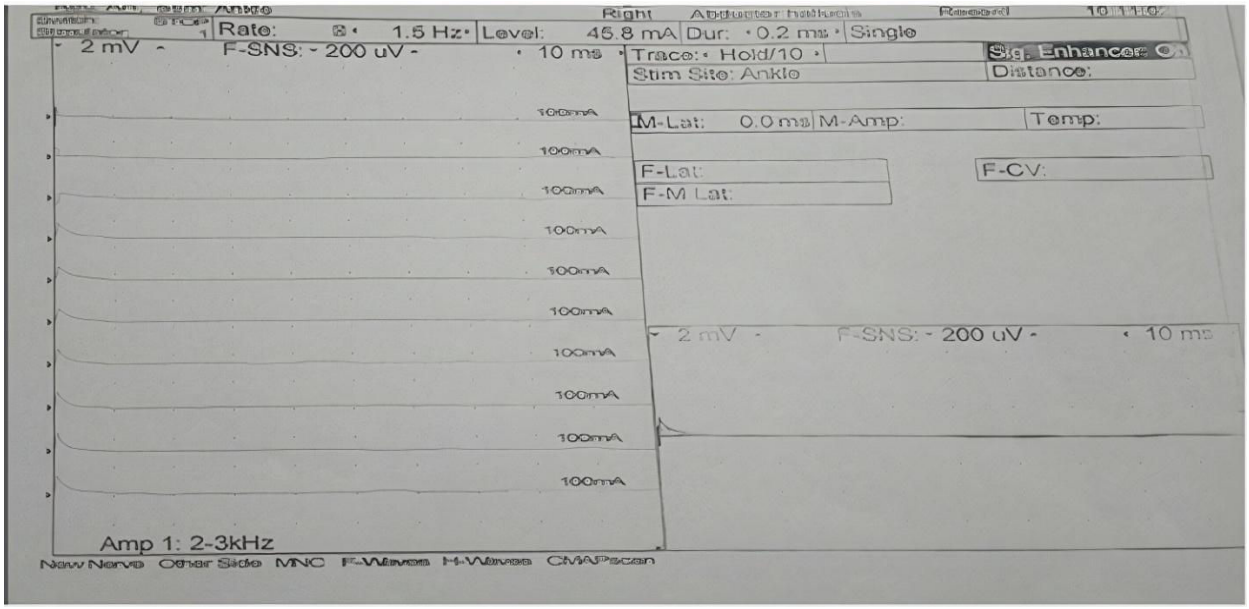

Figure 2: Non recordable F wave response of Right tibial nerve

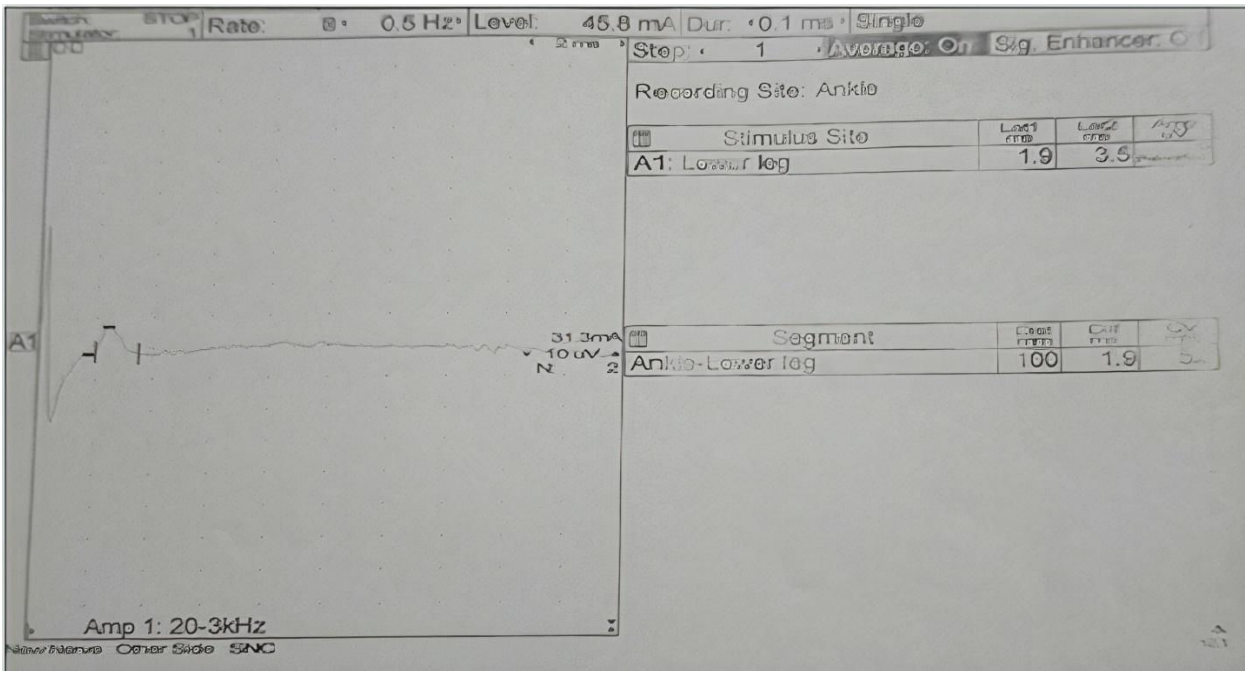

Figure 3: Normal Sensory Nerve Conduction of Right Sural nerve

DISCUSSION

Since December 2019, coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, has rapidly swept the world. It primarily targets the angiotensin converting enzyme 2 (ACE 2) receptors of respiratory tract epithelium. There is aslo neurological manifestations which can worsen disease outcome. This damage can occur by direct viral cytopathy mainly through afferent branches of 1st (olfactory) and 5th (trigeminal) cranial nerves, or para infectious through cytokine mediated damage (mainly IL6) or post infectious immune mediated phenomenon[9]. Both IgG and IgM antibodies can be detected within 1-3 weeks after SARS-COV-2 infection[10]. Time interval between covid 19 illness and Guillain-Barre syndrome may ranges from 5 days to 3 weeks.[11]. Our patient had history of weakness for 2 days duration for which he was admitted, incidentally he was found to be covid 19 RTPCR positive with no symptoms related to covid 19 and also positive for anti SARS COV2 antibody. He was non vaccinated against SARS COV2, it suggests he might get infected earlier followed by antibody production which triggered the immune system followed by GBS.We believe this patient had severe motor neuropathy due to covid19 as the patient was non vaccinated, covid 19 RTPCR was positive along with positive anti SARS COV2 antibody titre. NCV study clearly defined about severe motor neuropathy variants of Guillain-

Barre syndrome. Dysregulated immune response against peripheral nerves or spinal nerve root related to covid 19 may trigger the development of GBS due to molecular mimicry.To date no literature reported about clear predilection for axonal or demyelinating variants of GBS in SARS COV2 infection[12].Our patient received 2gm Immunoglobulin daily for 5 days. Autonomic dysfunction has also subsided and muscle power improved by 2 grades. There is a case report where SARS-COV-2 virus found in CSF of a patient with GBS during the phase of viremia suggesting its neuroinvasive nature[13]. Further studies are needed to establish more severe form of GBS in covid 19 and which mechanism do responsible for more seve form, para-infectious or post-infectious?

CONCLUSION

We conclude that GB syndrome can occur in covid 19 even in asymptomatic patient. It is neurological emergency, quick symptoms recognition and diagnosis is important for management of the patient. Further studies are needed to established the severity of GBS due to SARS-COV-2. We should consider testing all patient with GBS for SARS-COV-2 infection.

HIGHLIGHTS

This patient presented with limb weakness which is not familier with covid 19 symptoms. Clinical manifestations and NCV study confirmed this as Guillain-Barre syndrome. Here we report SARS-COV-2 as a new etiological agent of GBS. To the best of our knowledge no study report SARS-COV-2 as GB syndrome etiological agent.

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