

Case Report

Guillain-Barré Syndrome Associated with COVID-19 Infection: Case Report

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Abstract: Guillain-Barré syndrome (GBS) is an acute demyelinating disease and the most common cause of acute flaccid paralysis (AFP). We report a case of Guillain-Barré syndrome associated with corona virus which is a novel virus. At presentation the patient was hypoxic and was intubated in view of high oxygen requirement. He was weaned off from ventilator but there was extubation failure. He was reintubated. Other causes of extubation failure were ruled out. His motor power was 1/5 in upper limb, 0/5 in lower limb along with autonomic involvement. He was diagnosed with GBS. He was tracheostomised and managed with dual session of IVIG (intravenous immunoglobulin) and 3 sessions of plasmapheresis. Patient's motor power improved. He was weaned off from ventilator and decannulated. Motor power improved to 5/5 in upper limb and 3/5 in lower limb and he was discharged on room air.

Keywords: Guillain-Barré syndrome, Corona virus, IVIG, Plasmapheresis.

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INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute demyelinating disease and the most common cause of acute flaccid paralysis (AFP), with an incidence of 1.2 to 2.3 per 100,000 persons per year [1]. GBS has different variants including acute motor axonal neuropathy (AMAN), acute inflammatory demyelinating polyneuropathy (AIDP), acute motor sensory axonal neuropathy (AMSAN) and Miller Fisher variant [1, 2]. The male-to-female ratio is 3:2 [2]. GBS typically follows an infection, most often an upper respiratory tract infection (58%) and gastrointestinal infection (22%), but can also be caused by different surgeries [3]. Among bacterial infections, *Campylobacter jejuni* is the most common organism associated with the acute motor axonal neuropathy and other GBS variants [3]. Several viral infections are also associated with GBS like cytomegalovirus, Epstein-Barr virus, HIV, varicella-zoster virus, ZIKA virus and hepatitis A and B [4]. The clinical features of this syndrome were described by Guillan, Barre' and Strohl in 1916, which included bilateral symmetrical areflexia,

limb weakness and uncommonly sensory loss proceeding to neuromuscular paralysis including lower cranial nerves, facial and respiratory function with maximum severity of symptoms in 2-4 weeks. Common clinical pattern is a bilateral symmetrical ascending paralysis which evolves over hours to few days with tingling and dysesthesias in the extremities. Lower limbs are more affected than upper limbs and facial diparesis occurs in 50% of affected population [3, 5]. Guillain-Barré syndrome (GBS) is an immune-mediated post-infectious radiculopathy which commonly leads to ascending weakness. It has been reported as possible rare sequelae of COVID-19 infection, though the primary case reported in Wuhan, China suggested a para-infectious presentation [6, 7]. We report a case of GBS associated with severe COVID-19 infection from North India.

CASE PRESENTATION

A 30-year-old man with no co-morbidities, presented with chief complaints of low grade fever for 3-4 days and shortness of breath for 1 day. In an outside

hospital, on 10th April 2021 his oropharyngeal swabs tested positive for SARS-CoV-2 on RT-PCR. HRCT chest was suggestive of covid pneumonia. CTSI was 11/25. He was managed on high flow oxygen but was referred to a higher centre due to increased oxygen requirement. On 12th April he presented with SpO₂ 88% on non-rebreathing mask with 15 litre of oxygen and respiratory rate of 40/min to our hospital. He was afebrile and hemodynamically stable.

In covid ICU, High Flow Nasal Cannula was applied with 60 L flow (Fio₂ - 90%) alternate with Non-invasive ventilator support. The patient was electively intubated due to persistent tachypnea and worsened hypoxemia. Tracheal culture was sent. Adequate fluid resuscitation was done. He was given broad spectrum antibiotics Meropenem, Teicoplanin and antiviral agent Remdesivir for 5 days. On admission complete blood count, renal function test with electrolytes were normal; inflammatory markers (IL-6- 226, CRP- 184, LDH-115, D-DIMER- 1540, PCT-2.7 ng/ml) were on the higher side. The patient was started on methylprednisolone 80 mg intravenous twice a day, enoxaparin 0.4 ml subcutaneous twice a day from day 1.

After 9 days of mechanical ventilation patient's required FiO₂ decreased to 40%. Steroids were tapered. Weaning trial was given. Post-extubation the patient remained tachypneic despite NIV support. Blood gases done 2 hours post-extubation showed type-2 respiratory failure. Patient was electively reintubated. Repeat oropharyngeal swab for SARS-CoV-2 was sent. Chest radiology was done which showed no new findings. Serum electrolytes were normal which ruled out other metabolic causes. There was no deterioration in patient's mental status.

Repeat RT-PCR for SARS-CoV-2 done on 10th day of admission was negative. Patient was shifted to non-covid ICU, where planned tracheostomy was done. Sedation was stopped. Neurology consultation was done to find out the cause of respiratory muscle weakness. Neurology examination showed that the patient had developed facial diplegia along with proximal weakness in upper and lower limbs without any sign of facial deviation. Bilateral pupils were reacting to light. Gag reflex was sluggish and neck holding was inadequate. His motor power according to Medical Research Council (MRC) Muscle Scale was 0/5 in lower limbs, 0/5 in proximal upper limbs and 1/5 in distal upper limbs. He had normal sensation to pinprick and reduced vibration sense in feet. All deep tendon reflexes were absent. His cerebrospinal fluid (CSF) examination was inconclusive. Serum CPK was 428 U/L. Motor NCS (nerve conduction study) was done in all four limbs. It showed severely reduced CMAP (compound muscle action potential) amplitude in bilateral tibial and peroneal nerves, left ulnar nerve. F wave was absent in bilateral lower limbs and right ulnar

nerves. Bilateral sural and upper limb sensory NCV studies were normal. After fulfilment of the Brighton criteria a diagnosis of GBS was confirmed on day 10.

IVIG (intravenous immunoglobulin) (0.4mg/kg/day for 5 days) was started on day 10. The patient was continued on mechanical ventilation support due to poor respiratory function. He had minimal improvement in muscle power after IVIG. On day 30th (15 days after last dose of IVIG) plasmapheresis was started every alternate day for 5 sessions. The same was stopped after 3 sessions due to severe sepsis. There was no significant improvement in motor power so a repeat cycle of IVIG was started on 45th day for 5 days. 4-5 days after the second cycle of IVIG, patient showed improvement in muscle power to 1/ 5 in lower limbs and 3/5 in right upper limb and 2/5 in left upper limb. Antibiotics were de-escalated and steroids were tapered off as per protocol. The patient was shifted to CPAP and protocolised weaning started. The patient was weaned off completely from mechanical ventilation on 65th day.

Serial chest radiographs showed improvement. The MRC muscle scale improved to 4/5 in right upper limb, 3/5 in left upper limb and 2/5 in lower limbs by 69th day. The patient was able to lift his head for 5 seconds. Gag reflex improved and patient was able to accept liquid diet. Patient remained tracheostomised, without any respiratory distress on room air for 7 days and was discharged on 72nd day of admission with double lumen tracheostomy tube in-situ on room air.

On follow up the muscle power had improved to 5/5 in upper limbs and 3/5 in lower limbs. Patient was decannulated and he started vocalising 80th days after admission.

A 30-year-old male with no co-morbidities, presented with chief complaints of low grade fever for 3-4 days and shortness of breath for 1 day. his outside oropharyngeal swabs tested positive for SARS-CoV-2 on RT-PCR. HRCT chest was suggestive of covid pneumonia and CTSI was 11/25. Patient presented to our hospital on 12th April was afebrile, hemodynamically stable, tachypenic and SpO₂ was 88% on non rebreathing mask with 15 liter.

In covid ICU, High Flow Nasal Cannula (HFNC) was applied with 60 L flow and Fio₂ - 90% alternate with Non-invasive ventilator (NIV) support. Patient electively intubated as hypoxemia worsen. Tracheal culture was sent. Adequate fluid resuscitation was done. He was given broad spectrum antibiotics and antiviral agent Remdesivir for 5 days. On admission complete blood count, renal function test with electrolytes were normal; inflammatory markers (IL-6- 226, CRP- 184, LDH-115, D-DIMER- 1540, PCT-2.7 ng/ml) were on the higher side. The patient was started on methylprednisolone 80 mg intravenous twice a day

and enoxaparin 0.4 ml subcutaneous twice a day from day 1.

After 9 days of mechanical ventilation patient's oxygenation improved, steroids were tapered to lower dose and weaning trial was given and patient extubated. Post extubation the patient remained tachypneic despite NIV support. Blood gases done after 2 hour of extubation showed type-2 respiratory failure. Patient was electively reintubated and repeat oropharyngeal swab for SARS-CoV-2 was sent. To rule out the cause of extubation failure, chest radiology was done which showed no new findings. Serum electrolytes were normal which ruled out other metabolic causes. There was no deterioration in patient's mental status.

Repeat RT-PCR for SARS-CoV-2 done on 10th day of admission was negative. Patient was shifted to non-covid ICU and done tracheostomy. Sedation stopped and Neurology consultation was done to find out the cause of respiratory muscle weakness. Neurology examination showed that the patient had developed facial diplegia along with proximal weakness in upper and lower limbs without any sign of facial deviation. Bilateral pupils were reacting to light. Gag reflex was sluggish and neck holding was inadequate. His motor power according to Medical Research Council (MRC) Muscle Scale was 0/5 in lower limbs, 0/5 in proximal upper limbs and 1/5 in distal upper limbs. He had normal sensation to pinprick and reduced vibration sense in feet. Deep tendon reflexes were absent. His cerebrospinal fluid (CSF) examination was inconclusive. Serum CPK was 428 U/L. Motor NCS (nerve conduction study) was done in all limbs. It showed severely reduced CMAP (compound muscle action potential) amplitude in bilateral tibial and peroneal nerves, left ulnar nerve. F wave was absent in lower limbs and right ulnar nerves. Bilateral sural and sensory NCV studies were normal. After fulfilment of the Brighton criteria a diagnosis of GBS was confirmed on day 10.

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On follow up the muscle power had improved to 5/5 in upper limbs and 3/5 in lower limbs. Patient was decannulated and he started vocalising on 80th day.

DISCUSSION

Historically, SARS-CoV-2 has been known to affect body systems other than the respiratory system. COVID-19 has also been reported to affect multiple body systems apart from the respiratory system, one of them being the nervous system. Although a detailed study into the mechanism of neurological manifestations is needed, various theories explaining it have been proposed. Active retrograde transport from a peripheral neuron up to the brain, across synapses is one possibility [8]. Other probabilities are hypoxia-related injury, cytokine storm sequelae or via angiotensin receptor -2 (ACE-2) [9]. There could also be generation of antibodies by the virus against specific glycolipids in certain GBS variants [7].

The neurological symptoms associated with COVID-19 were first reported by Mao *et al.*, [10]. They also reported that the neurological symptoms were more likely in severe covid cases. The first covid associated GBS case was reported from Wuhan, which presented with GBS symptoms followed 7 days later by covid symptoms [11]. The case we have presented was a more typical presentation with respiratory symptomatology followed 2 weeks later by GBS symptoms. Tiet MY *et al.*, also presented a case report where respiratory infection symptoms occurred 3 weeks prior to onset of GBS (6). Korem S *et al.*, also presented a case of covid associated GBS with a viral illness preceding weakness [12]. Other cases have been presented where concurrent respiratory and neurological symptoms occurred [13, 14]. In few others the duration from onset of respiratory symptoms to neurological manifestations varied from 5-24 days [15]. The recovery varied from full recovery to no change in extremity function. But the use of intravenous immunoglobulins and/or plasmapheresis is recommended in all reported cases.

Unique features – severity, difficulty in treatment, associated prolonged paralytic ileus.

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