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Guest Editorial

Guillain-Barré syndrome: An ALARM in the Management of COVID-19 with Respiratory Insufficiency

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Corona virus disease 2019 (COVID-19) has infected a great number of patients with a pandemic behavior in the world and therefore even rare manifestations of the virus infection have been reported during a short period of time. Corona viruses have special neurotropic potential with neurological complications occurring both in central nervous and peripheral nervous systems [1]. Neurological manifestations occur in up to 36.5% of COVID-19 infected patients [2]; Guillain-Barré syndrome (GBS) accounts for 1.4% of the reported cases [1]. Para-infectious immune mediated mechanism is thought to be the underlying cause of GBS in COVID-19 disease [3,4]. It is however shown that neurological complications occur in severe cases of COVID-19 infection [2]. GBS in the setting of COVID-19 can contribute to respiratory failure. High level of suspicion and in particular when the patient has lost the consciousness is therefore critical. On the other hand proper diagnosis and treatment of GBS can help better controlling respiratory insufficiency.

We have been consulted for a 47 year old woman with several days of cough and generalized body pain and low grade fever infected with COVID-19 and admitted to internal medicine. She had past medical histories of diabetes mellitus, hypothyroidism, hyperlipidemia, ischemic heart disease and hypertension. Along with typical chest CT for COVID-19, treatment with anti COVID-19 medications (Table 1) was started for her (Figure 1). After one week of treatment, her respiratory condition deteriorated and lead to intubation and mechanical ventilation. During treatment with mechanical ventilation and on third week of therapy for COVID-19, the clinical picture manifested with quadriparesis and loss of tendon reflexes with preserved bulbar muscle function.

Electrodiagnostic studies were performed without patient cooperation, which demonstrated absent motor action potentials in tested nerves (Median, ulnar, tibial and deep peroneal nerves) with normal sensory potentials in (Median, ulnar and radial nerves). Median and tibial F waves and lower extremity H reflex were absent. In electromyography study no spontaneous potentials were detected from tested

Dexamethasone Intra Venous	4 mg three times daily
Enoxaparin Subcutaneous	60 mcgr twice daily
Aspirin Per Oral	80 mg at evening
Famotidine Per oral	40 mg at night
Atorvastatin Per oral	40 mg at night
Metoprolol Per Oral	25 mg daily
Spironolactone Per Oral	25 mg at evening

Table 1: *Medications used for the patient with relevant therapeutic effects on COVID-19.*

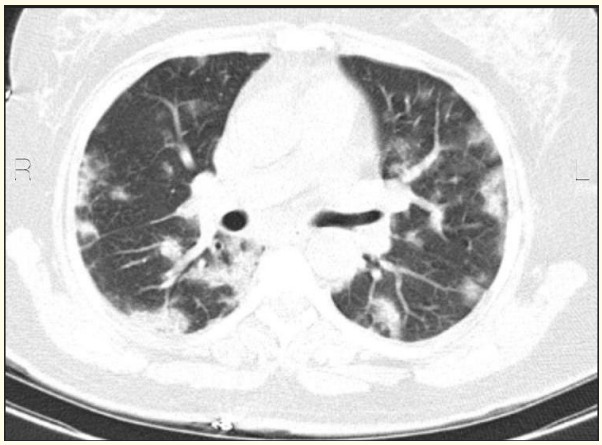

Figure 1: *Chest computed tomography done during first week of hospital admission.*

muscles (Biceps brachii, pronator teres, flexor carpi radialis, first dorsal interossei, vastus medialis, tibialis anterior, peroneus longus, gastrocnemius), volitional potentials could not be evaluated due to lack of patient cooperation.

The patient was diagnosed as a case of acute motor axonal neuropathy (AMAN type GBS) and was treated with Intravenous Immunoglobulin (IVIG) 0.4 gr/kg body weight for 5 consecutive days. The patient respiratory symptoms were more stable on starting IVIG. Respiratory condition along with general condition of the patient deteriorated in the following 2 weeks and the muscle weakness did not respond to IVIG therapy. The patient expired in the course of treatment with the differential diagnoses of pulmonary thrombo-emboli vs. COVID induced systemic inflammatory response.

White Blood Cell count	6700 /mL
Hemoglobin	11.9 gr/dL
Platelet count	141000 /mL
BUN	24 mg%
Creatinine	0.8 mg%
Total Bilirubin	0.95 mg%
Direct Bilirubin	0.29 mg%
AST	42 IU/L
ALT	24 IU/L
ALP	241 IU/L
Magnesium	1.8 mg/L

Table 2: *Laboratory results during hospital admission.*

Discussion

COVID-19 presents with several neurological manifestations, however peripheral neuropathy is less reported between various presentations of COVID-19 [3]. In a recent review of the literature recruiting 37 GBS cases from several case report and case series related to

COVID-19 infection, it is shown that GBS associated with COVID-19 infection occurs predominantly in the age of fifties with a tendency to affect more male patients (64.9%) [5]. The most common non specific manifestations before the onset of GBS symptoms are cough and fever (91.9%) and anosmia (18.9%). Among neurological manifestations of GBS, hyporeflexia or areflexia (100%), limb weakness (67.6% increasing to 78.3% during hospital course) and paresthesia (67.6%) are predominant signs and symptoms. Interestingly cranial nerve involvement including unilateral or bilateral facial palsies, ophthalmoplegias and bulbar weakness have been reported in up to 41% of cases [5].

In our patient, severe COVID-19 course was associated with the symptoms of GBS. The patient had respiratory failure requiring mechanical ventilation and is not clear whether GBS associated muscle weakness was an exacerbating factor. The patient was not awake during neurological examination and the EMG/NCV study was done without cooperation. Plasmapheresis was not available in the center and the patient received intravenous immune globulin (IVIG) 0.4gr/kg/day for 5 consecutive days. The severe condition and its fatal course did not allow further therapeutic assessment and gauge of therapy.

The herein proposed therapeutic idea is that GBS can be a contributing factor to COVID-19 respiratory failure that needs its proper treatment. The patients may be unconscious and high level of clinical suspicion is needed for its timely diagnosis. Reports of GBS in the setting of COVID-19 infection make an alarm for involving neurologists in case of respiratory failure particularly when the lung involvement cannot fully explain severe and lasting respiratory conditions especially failure to wean off ventilator.

Limitations and Recommendations

The EMG study has to be repeated after a duration of 2 to 3 weeks that was not possible in our case because of critical condition. Protein content of cerebrospinal fluid can be helpful in better diagnosing GBS that was not performed in the patient. It is demonstrated that acute axonal GBS, not well responding to first dose of IVIG, benefit from second dose of IVIG [6]. With proper treatment strategy the therapeutic response rate of GBS in the context of COVID-19 infection is reported to be 89% with varying improvement degrees of 73% in the following days [5]. The end clinical comment is that, in general, GBS is a major cause of respiratory failure with up to 30% of cases requiring mechanical ventilation [7,8] and it can complicate COVID-19 infection and especially in severe cases of COVID-19, in which neurological manifestations are more common [2]. High clinical suspicion and appropriate treatment on the other hand helps better managing respiratory insufficiency in COVID-19. Finally the immune suppressive effect of plasmaphresis and immune modulatory and hypercoagulable effects of IVIG, two well recognized treatment modalities of GBS, have to be considered in therapeutic context of COVID-19 marked with hyperinflammatory state and predisposition to secondry infection and coagulable abnormalities.

Conflict of Interests Declaration

No conflicts of interests.

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