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CASE REPORT

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Novel Coronavirus (COVID-19)-Associated Guillain–Barre' Syndrome: Case Report

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Keywords: COVID-19, Guillain-Barré syndrome, neurology, Ethiopia

Introduction

The global pandemic of extreme acute respiratory coronavirus 2 (SARS-COV-2) and its related diseases, known as coronavirus disease, is currently affecting the healthcare systems. The virus is a novel human pathogen for which there are currently no clear treatment options.¹

COVID-19 affects the respiratory tract and the lungs. However, involvement of other systems such as cardiovascular, renal system and neurological system has also been reported.² Majority of patients with COVID-19 induce respiratory symptoms with mild clinical characteristics, such as Headache, stroke, epilepsy, encephalitis, hypogeusia, and neuralgia are some of the neurological signs of COVID-19.³

Guillain-Barr syndromes (GBSs) are a category of polyneuropathies marked by ascending motor impairment, mild to severe sensory disturbances, cranial nerve involvement, and muscle or radicular pain.⁴ The most common subtypes are acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and Miller Fisher syndrome (MFS), which are characterized by acute ophthalmoplegia, gait ataxia, and areflexia and are identified by the degree of involvement of the motor or sensory nerves, myelin sheath, axon, or cranial nerve predominance.⁵ GBS is a pre-existing viral autoimmune neuropathy, and around 70% of patients develop a flu-like illness, 1–3 weeks before the diagnosis.⁶

Cross-immunity against epitopes of peripheral nerve components, which it shares with epitopes on the cell surface of bacteria that cause an antecedent infection, is thought to be the cause of polyneuropathy in GBS.⁷

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Case Presentation

A 17 years old female patient was referred to emergency department of this Hospital with lower extremity weakness of one week duration in 2020. The weakness was progressive and reached at nadir after seven days of the onset of the illness. She had also right flank pain of the same duration, shortness of breath of 3 days duration but she had no fever and cough. She had decreased urine amount and vomiting for 3 days, there is no bowel and bladder dysfunction. On neurologic examination she is conscious, language and cranial nerves are normal. On motor examination: upper extremity was normal, Lower 0/5 bilaterally, tones: lower extremity flaccid and upper normal and deep tendon reflexes and plantar response are absent; Sensory examination-intact. On laboratory test: Nasopharyngeal swab tested positive for SARSCoV-2 and Lumbar puncture was done and showed.

The findings of CSF analysis are No cell, Protein was 3.5g/dl and Glucose was 97mg/dl.

Gangliosides antibodies test was not performed and EMG was not done. Critical COVID-19 Disease explained respiratory failure secondary to GBS can be entertained.

The patient was started with ceftriaxone and azithromycin but not hydroxychloroquine and dexamethasone.

She was also on intranasal oxygen with nasal cannula later change to facemask.

IVIG was not given b/c it is not available and also costly.

The patient developed respiratory failure after 4 days of the onset of weakness and the patient died of respiratory failure secondary to GBS.

Discussion/Conclusion

The primary presentation of COVID-19 is respiratory but neurological manifestations and complications are increasingly being reported in different literatures. GBS is one of the frequent neurological complications associated with COVID-19. Neurological manifestations of COVID-19 is often associated with severe acute respiratory distress syndrome; this patient develops respiratory failure 4 days after the onset of the weakness.

At this time, there is no clear causal relationship between GBS and COVID-19, and more data is needed to determine the casualty. Cross-immunity against epitopes of peripheral nerve components, which it shares with epitopes on the cell surface of bacteria that cause an antecedent infection, is thought to be the cause of polyneuropathy in GBS. With the Campylobacter jejuni-related GBS, the mechanism of "molecular mimicry" is best understood.⁶

To explain the potential pathophysiological correlation, and to also describe the clinical/electrophysiological trend of new cases of GBS reported in the context of the COVID-19 pandemic, further study and data is needed.⁸

GBS has serious implications, and early detection is important for tracking ambulation failure and initiating immunoglobulin care. Several studies have shown that a 5-day intravenous immunoglobulin 400 mg/kg daily regimen for a suspected diagnosis of GBS can save lives.

Patients who present with paresthesia and trouble moving after experiencing COVID-19 symptoms should be considered for viral-associated myositis.⁹

Weeks after the initial COVID-19 infection, GBS should be treated as rare but a major complication.¹⁰

Consent for Publication

Informed consent was obtained from the patient's relative to publish their case details and any accompanying images.

Informed Consent

A verbal informed consent for the patient information is provided by the relative of the patient.

Disclosure

The authors declared no potential conflicts of interests with respect to publication of the article.

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