ejpmr, 2022, 9(6), 345-347

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Study

ISSN 2394-3211

EJPMR

POST- COVID-19 NEUROLOGICAL COMPLICATION: BULBAR GBS, A MERE COINCIDENCE OR SOMETHING MORE: A CASE REPORT

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Article Received on 27/03/2022 **Article Revised on 17/04/2022** **Article Accepted on 07/05/2022**

ABSTRACT

Covid-19 was first discovered in Wuhan, China. Covid is caused by SARS-CoV-2 virus. World Health Organization has declared Covid-19 as a pandemic. Symptoms include pyrexia, cough, malaise, and myalgia. Very few post-neurological complications of Covid-19 infection are recorded including GBS. Guillain-Barré syndrome or GBS is a rare auto-immune condition affecting the peripheral nerves leading to demyelination and finally paralysis. The aetiology of the disease is still not known, but people of all age are affected. It is believed to occur post-surgery, infection or even vaccine administration. Many variants of GBS are reported globally including AMAN, Pharyngeal-cervical-brachial, Bickerstaff Encephalitis, and Miller Fisher syndrome. It can be diagnosed using CSF examination, neurological examination, and electromyography. The possible treatment includes IVIG and plasma therapy. The most common side-effect being respiratory compromise and bulbar palsies. The exact cause and treatment of the deadly disease is not known yet. Just like during the Zika virus outbreak, many cases of GBS were reported, similarly various cases are being reported post-Covid infection. We present a case report of GBS with bulbar palsy, post- Covid-19 infection.

KEYWORDS: Covid-19, Guillain-Barré syndrome, GBS, Miller Fisher syndrome.

BACKGROUND

The novel virus break out in Wuhan city of the people's republic of China in December 2019 led to a deadly disease known to mankind. The virus is ranked similar to SARS coronavirus and MERS coronavirus and was named COVID-19. The virus is named as 'Severe Acute Respiratory Syndrome Coronavirus 2' (SARS-CoV-2), and the disease is named as Coronavirus disease (COVID-19).[1]

The novel coronavirus first case was reported in India from Kerala in January.[2] As of 12 April, 6.15 pm (CEST) 497,960,492 confirmed cases have been reported, with 6,181,850 deaths globally.[3] World Health Organisation declared COVID-19 as a pandemic on 11th March 2020.[1] It is a member of family Coronaviridae whose member have been implicated in the causation of disease in wide range of host.[4] The symptoms vary from mild to severe. The most common symptoms include pyrexia, cough, myalgia, and malaise. While dyspnoea, haemoptysis and diarrhoea were less commonly seen.[5]

Though rarely encountered acute and post-acute neurological complications have been reported. This could affect both central and peripheral nervous system, like Guillain-Barré syndrome, critical illness neuromyopathy (CIM), encephalitis and demyelinating disease (CNS inflammatory diseases).[6]

Guillain-Barré syndrome (GBS) is an acute auto-immune condition affecting the peripheral nerves leading to their demyelination resulting in paralysis. It is acute in onset and causes ascending and symmetrical paralysis. Typically, both sides of the body are involved with muscle weakness (initially in the hands and feet, following up to upper body and arms), pain or sensation in the back. People of all ages are affected, but adult males are more affected. The aetiology is unknown, but it's believed to occur after an infection of the respiratory or gastrointestinal tract, surgery or even vaccine administration. In case of Zika virus infection, in the affected countries an unexpected rise in the cases of GBS was seen. The explanation for the same was given by WHO and stated that Zika virus infection acted as a trigger for Guillain-Barré syndrome.[7]

Many variants of GBS have been recorded. The most common variant in Asian countries is AMAN (Acute Motor Axonal Neuropathy).[8] The other variant being Pharyngeal-cervical-brachial, primarily affects pharyngeal, neck and upper extremity muscles.[9] Other variant termed as Bickerstaff Encephalitis, involves CNS

(Central Nervous System).[10] The most common variant being Miller Fisher syndrome, a triad of areflexia, ataxia, and ophthalmoplegia. Along with oculomotor nerves, other cranial nerves are also affected.[11]

Diagnosis of GBS is done by Electromyography, neurological, and cerebro spinal fluid (CSF) examination, including retardation or complete loss of reflexes by deep tendons. Nerve conduction studies (NCS) helps distinguish axonal and demyelinating neuropathy. Acuity of patient's symptoms can be determined by needle electromyography.[12] Cerebrospinal fluid shows albumin-cytological dissociation, that is, normal WBC (white blood cell) count and elevated CSF protein.[13] Ganglioside antibodies namely anti-GM1, anti-GD1A, anti-GT1A, and anti-GQ1B are associated with GBS.[14] MRI (Magnetic Resonance Imaging) findings in GBS cases show enhancement of nerve roots, indicating inflammation leading to breakdown of blood-nerve barrier.[15]

A negative inspiratory force (NIF) test is conducted on patients with suspected GBS, patients who are unable to perform NIF of -20 to -30 H_2O are considered as high-risk patients.[16]

There are two possible treatments for GBS, one being intravenous immunoglobulin (IVIG) and other being plasma exchange. IVIG is divided over a period of 5 days dose being set at 2 gram/kilogram. IVIG is thought to work by immune-modulating action,[17] while plasma is thought to act by removing humoral mediators, pathogenic bodies, and complement proteins involved in the GBS pathogenesis. Yet the exact mechanism of both the method of treatment is not known.[18]

The most severe complication of GBS includes respiratory compromise and bulbar palsies.[16]

GBS despite being life-threatening disorder, there is no known cause and treatment for the disease.

CASE PRESENTATION

A 41-year-old obese male of Indian origin who is a known case of diabetes presented to the emergency department with complains of immobility of the right lower and right upper limb. He had a history of upper respiratory tract infection for which he got a RT-PCR done in wake of ongoing pandemic which was positive for SARS-Cov2, three weeks prior to presentation. The symptoms began suddenly starting with loss of grip of upper hand. One day after admission the weakness involved all the four limbs, and the patient was unable to walk. He then developed difficulty in breathing and swallowing for which he was put on a ventilator support.

INVESTIGATION

MRI brain revealed few non-specific T2W and TIRM bifrontal hyperintensities and evidence of significant mucosal disease in paranasal sinus was found. Nerve conduction studies showed distal, symmetrical, large fibre motor axonal polyneuropathy/polyradiculoneuropathy involving both upper and lower limb.

Neurological investigation showed absent deep tendon reflexes. Muscle Strength Scale had a score of 0 out of 5 for all four limbs. The patient also had facial weakness.

COVID-19 total antibody test was positive (Total Antibody - 262). CSF sample was incubated for 72 hours, and no organisms were isolated. It revealed a normal protein level and showed a cell count of 2 per cubic millimetre. The ADA (Adenosine deaminase) was 0.07 U/L. The CSF tested negative for anti-SARS-CoV-2 IgG. It tested negative for *Mycobacterium tuberculosis*, *Cryptococcus neoformans*, *Cytomegalovirus*, *Enterovirus*, *Herpes simplex* 1 and 2, *Varicella-zoster* virus and bacteria commonly associated with meningitis. Blood investigations showed a rise in level of total leukocyte count (TLC) (Counts- 17.65 thousand per microliter). A raised AMC (Absolute monocyte count) was also raised (1236/microliter). RBCs showed mild anisocytosis showing normocytic normochromic cells. Neutrophilic leucocytosis with lymphopenia and absolute monocystosis and no hemoparasite was seen in the smear.

BacT/Alert blood culture was negative. IgM dengue MAC ELISA test and Scrub typhus ELISA test was negative. Hepatitis C and B virus antibody was also negative.

Endotracheal secretion showed few polymorphonuclear leukocytes, few squamous epithelial cells, mixed upper respiratory tract flora.

Urine culture from catheter showed no pathogenic organism after 24 hours of incubation. Urine investigation showed glucosuria of 1000 mg/dL (+++) and was negative for nitrite, ketone, and albumin.

TREATMENT

The patient was managed with IVIG (intravenous immunoglobulin) therapy according to weight (2 gram/kg) for 5 days and was put on mechanical ventilation due to breathing difficulties and intubation. The patient was put under ICU induced sedation. The patient was also catheterised. The patient did not respond well to the treatment and succumbed to it after a month.

DISCUSSION

This case study showed Guillain-Barré syndrome occurring post Covid-19 infection. This case revealed a rare form of GBS known as Acute Bulbar Palsy Plus syndrome. Dysphagia and facial weakness were noted which interfered with eating and swallowing.[19] There are different variants of GBS, acute inflammatory demyelinating polyradiculopathy is most commonly

encountered. The other variants like acute motor axonal neuropathy, acute motor sensory axonal neuropathy and Miller Fisher syndrome are also documented. There is an immunological response generated in GBS against the myelin sheath of neurons which results in its loss.

This syndrome can involve nerve which are involved in the transmission of sensory signal, the nerves which are involved in the maintenance of muscle contraction and their basal resting tone. Besides these the nerves associated with the autonomic nervous system can also be affected. Nearly 220 patients around the world have reported GBS as post infectious sequelae of Covid-19.[20] The mechanism by which Covid-19 induce generation of autoimmune response against neuronal myelin sheath is still not clear yet.

CONCLUSION

Based on this case study, we cannot for sure determine Covid-19 to be the etiological agent. But many cases of GBS were reported post-Covid infection. Further research is required.

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