

Letter to the Editor

[The Editor is not responsible for the views expressed by the correspondents]

Guillain-Barre Syndrome Following Covishield Vaccine : A Case Report

SIR, — Guillain-Barre syndrome (GBS), formerly known as Landry’s paralysis, is a condition in which the body’s immune system affects a portion of the peripheral nervous system. The precise causes of GBS are largely unresolved¹. The disorder can be seen days or weeks after respiratory or digestive infection and can occur rarely after recent vaccination or surgery. The condition may result in acute flaccid paralysis of limbs and areflexia^{2,3}. Complete recovery has been observed in most patients, and the mortality rate is about 5%⁴. The frequency of occurrence in men is one and a half times more than in women, and adults are more susceptible to GBS than children⁵. This study reports a 63-year-old female patient with Acute Motor Sensory Axonal Neuropathy (AMSAN) subtype of GBS, 10 days following the preliminary dosage of the ChAdOx1nCoV-19 Coronavirus vaccine (COVISHIELD™).

A 63-year-old female with a medical history of hypertension and type 2 diabetes mellitus on regular medication presented to the general medicine department with chief complaints of abrupt onset of aggravating generalized weakness in both upper and lower limbs, body pains, and back pain, difficulty to stand or walk without support, difficulty in rolling over the bed, chest pain, breathlessness, and loss of appetite, over the previous 10 days following the first dose of ChAdOx1nCoV-19 Coronavirus vaccine (COVISHIELD™) (Fig 1).

The patient had no previous medical history of trauma, weight loss, fever, or respiratory or gastrointestinal diseases. On physical examination, the patient was found to be afebrile, with blood pressure – of 130/90 mmHg, SpO₂ – 96% on RA, Pulse Rate – 92 bpm, GRBS –149 mg/dl. Neurological examination revealed acute areflexic quadriparesis of both upper and lower limbs. With normal bone density and tone in the upper limbs and decreased tone in the lower limbs, the power of the upper limbs was found to be 4/5, and power in the lower was found to be 3/5. The patient’s serum electrolytes revealed decreased sodium levels of 114 mEq/L, and decreased chloride levels of 81mEq/L respectively, CBP revealed leucocytosis of 15.04[10³/μl], increased blood urea levels of 45.7 mg/dl.

Her nerve conduction investigations revealed sensory-motor axonal neuropathy in both the lower and upper limbs (Tables 1&2). In nerve conduction examinations, both motor and sensory nerves exhibited lower amplitude. On inspection, there was no evidence of cranial nerve involvement. The results of all other systemic exams were within normal ranges.

Electroneuromyography (ENMG) (Table 3) revealed the absence of both the sural nerve sensory nerve action potential (SNAPS) and a reduction in the tibial nerve compound muscle action potential (CMAP), as well as significantly elevated common peroneal CMAP distal latency and significantly lowered CMAP amplitude (with spatial and temporal dispersion). The F wave latency was within standard ranges.

Her treatment procedure was started with intravenous immunoglobulin (IVIg) at a dose of (2 g/kg) five vials 24h



Fig 1 — Patient’s Covishield Vaccine Certificate

Nerve	Dist. Lat	CMAP	CV	F Wave
Left median	15.5/18.6	3.2/0.2	70	NR
Right median	12.8/20.7	1.1/1.0	27	NR
Left ulnar	42/8.8	1.7/1.2	51	NR
Right ulnar	5.7/9.5	3.0/2.1	69	NR
Left CPN	NR	-	-	-
Right CPN	8.3/18.8	1.0/0.9	34	NR
Left PTN	NR	-	-	-
Right PTN	7.3/16.8	1.6/1.4	40	NR

Dist. Lat – Distal latency, CMAP – Compound Muscle Action Potential, CPN – Common Peroneal Nerve, PTN – Posterior Tibial Nerve, NR – No response

Nerve	Latency
Left median	NR
Right median	NR
Left ulnar	NR
Right ulnar	NR
Left sural	NR
Right sural	NR

NR – No Response

Table 3 — ENMG

Distal latency	Prolonged
CMAP amplitude	Reduced
Temporal dispersion	Short
Conduction velocity	Reduced Rt median, left CPN
F wave latency	NR
Conduction block	Absent
SNAP'S	NR
SNAP – Sensory Nerve Action Potential, CMAP – Compound Muscle Action Potential, CPN – Common Peroneal Nerve, NR – No Response	

infusion for five days with a close watch for respiratory failure. She responded well to IVIg therapy and remained stable, hence IVIg therapy was discontinued and physiotherapy has been advised. She is still being monitored at home and receiving regular assessments and follow-ups at the Neurology outpatient department.

Guillain-Barré syndrome is an uncommon autoimmune condition that causes inflammatory demyelination of nerve fibers and usually arises after infection⁵. Although there have been numerous case reports of post-vaccination GBS, the global incidence of GBS in society is estimated to be 17 cases per million people per year. An analysis of earlier post-vaccination periods (Swine flu vaccination campaigns in 1976/1977 and H1N1 vaccination programs in 2008/2009) indicated no increased prevalence of post-vaccination GBS³.

Even though the fact that there is no proven link between the Covishield vaccination and GBS, the Food and Drug Delivery (FDA) has updated GBS warnings for another vaccine [GlaxoSmithKline's SHINGRIX™ (Zoster Vaccine Recombinant, Adjuvanted)] as of 24 March 2021, after observing 3 additional cases of GBS per million within 42 days after administration of the vaccine^{1,2}. The development of GBS was seen within 10-14 days of the covid 19 vaccination administration in every case where GBS development was linked to its administration. Although the pathophysiology of GBS caused by the administration of the COVID-19 vaccine is unknown, it is considered that when a COVID-19 mRNA vaccine is given, the mRNA enters human cells and instructs them to recognize the spike protein located on the surface of SARS-CoV-2, the virus that results in COVID-19. The spike protein is then identified by our bodies as an invader, which causes the development of antibodies to fight it. These antibodies are prepared to detect and eliminate the virus when it is encountered, blocking it from spreading and causing harm. This immune response

may trigger autoimmune processes in some patients, leading to the development of antibodies against myelin causing GBS^{6,7}.

In this case report, the GBS patient has been diagnosed with the condition despite having neither a history of the illness in her family nor a history of an infection that would have contributed to it. Therefore, it is feasible to hypothesize that in this case, the COVID-19 vaccine is what caused the GBS.

We present this case because a global vaccination drive is underway to control the pandemic. A correct analysis of the neurological drug reactions linked to the vaccination and the onset of occurrence can reduce morbidity and mortality through early disease detection. Using immunoglobulin to treat GBS shows quicker results.

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