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Post COVID-19 vaccine Guillain-Barré syndrome

Bikash Khadka,[1] Kishor Khanal[1]

ABSTRACT

Guillain-Barré syndrome is an acute generalized polyneuropathy which usually follows infection with a virus or bacteria, although rarely vaccination may be associated with it. We present a case of a 44-year-man who presented with progressive weakness of both lower limbs since 6 days, neurological examination findings were consistent with flaccid-type paraplegia and investigation findings including lumbar puncture and nerve conduction studies were consistent with the diagnosis of Guillain-Barré syndrome. He had received the Johnson and Johnson corona virus disease-19 vaccine intramuscularly 15 days before his presentation. Only potential triggering factor in this case was positive finding of Jansen Vaccine.

Keywords: Guillain-Barré syndrome; immunoglobulin; jansen vaccine; polyneuropathy.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute generalized polyneuropathy affecting 1 to 2 out of 100,000 people per year affecting male population more than females. It causes paralysis and about one-third of patients may develop respiratory failure needing ventilation support. A mild respiratory or gastrointestinal infection precedes the neuropathic symptoms by 1-3 weeks, in about 60% of the patients.[1]

Since the first reported case of COVID -19 on 23rd January 2020 in Nepal, more than 200 thousand cases had been reported. The vaccination program has been going on from January 2021 with Covishield(AstraZanacea) and Vero cell(Sinopharm) vaccines till July 2021. On 13th July 2021, 1.5 million doses of the Janssen vaccine (Johnson and Johnson) was donated by the United States to extend the vaccination program. As of 19th July 2021, 3,067,118 people have received their first shot and 1,134,317 people have received both shots of the COVID-19 vaccine in Nepal as per the government data.[2]

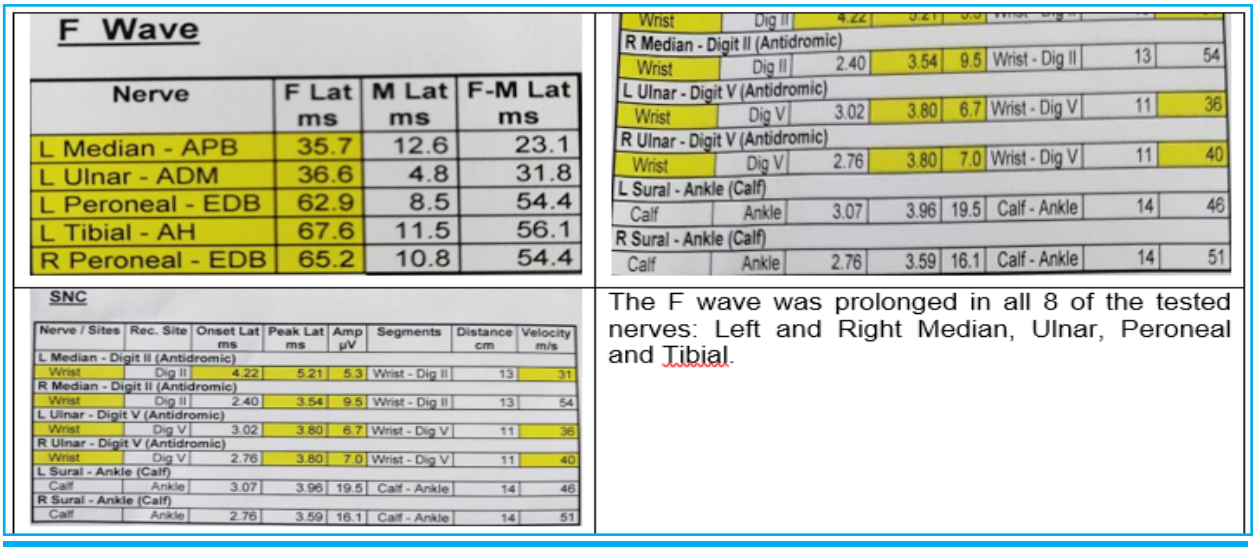
We present a case of GBS with probable association with Janssen vaccination.

CASE PRESENTATION

A 44-year-old male with a history of gradual weakness of bilateral lower limbs, preceded by paraesthesia and numbness, for 6 days was referred to our center. The weakness started from the left lower limb and gradually progressed to the right lower limb on the next day.He had received Janssen vaccine intramuscularly 15 days before his presentation. Neurological examination findings were consistent with flaccid paraplegia of bilateral lower limbs with muscle power of 4/5 in the right lower limb and 3/5 in the left lower limb, whereas normal bilateral upper limbs. Muscle tone and deep tendon reflexes were also reduced in bilateral lower limbs along with paraesthesia.There were no cerebellar signs and cranial nerve examination revealed normal results.His single breath count ranged from 28-36.

Routine lab tests, peripheral neuropathy and vasculitis screening tests were unremarkable.Non-contrast CT head was done to rule out any intracranial pathologies and MRI spine to rule out spinal cord compression,infarction or stenosis and transverse myelitis. Cerebrospinal fluid (CSF) analysis revealed albumin-cytological dissociation with protein 90 mg/dl and WBC 5 cells/cumm with 100% lymphocytes, glucose 70 mg/dl and negative for culture.A nerve conduction study (NCS) showed electrophysiological evidence of sensorimotor axonomyelenic polyneuropathy of severe severity on both arms and legs, prolonged F-waves latencies consistent with Acute Inflammatory Demyelinating Polyneuropathy.

Correspondence: Bikash Khadka1, Nepal Mediciti Hospital, Lalitpur, Nepal. Email: Khadka.vkas@gmail.com, Phone: +9779841411827.



F Wave

Nerve	F Lat ms	M Lat ms	F-M Lat ms
L Median - APB	35.7	12.6	23.1
L Ulnar - ADM	36.6	4.8	31.8
L Peroneal - EDB	62.9	8.5	54.4
L Tibial - AH	67.6	11.5	56.1
R Peroneal - EDB	65.2	10.8	54.4

SNC

Nerve / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	Amp µV	Segments	Distance cm	Velocity m/s
L Median - Digit II (Antidromic)							
Wrist	Dig II	4.22	5.21	5.3	Wrist - Dig II	13	31
R Median - Digit II (Antidromic)							
Wrist	Dig II	2.40	3.54	9.5	Wrist - Dig II	13	54
L Ulnar - Digit V (Antidromic)							
Wrist	Dig V	3.02	3.80	6.7	Wrist - Dig V	11	36
R Ulnar - Digit V (Antidromic)							
Wrist	Dig V	2.76	3.80	7.0	Wrist - Dig V	11	40
L Sural - Ankle (Calf)							
Calf	Ankle	3.07	3.96	19.5	Calf - Ankle	14	46
R Sural - Ankle (Calf)							
Calf	Ankle	2.76	3.59	16.1	Calf - Ankle	14	51

Wrist Dig II 4.22 5.21 5.3 Wrist - Dig II 13 31
R Median - Digit II (Antidromic)
Wrist Dig II 2.40 3.54 9.5 Wrist - Dig II 13 54
L Ulnar - Digit V (Antidromic)
Wrist Dig V 3.02 3.80 6.7 Wrist - Dig V 11 36
R Ulnar - Digit V (Antidromic)
Wrist Dig V 2.76 3.80 7.0 Wrist - Dig V 11 40
L Sural - Ankle (Calf)
Calf Ankle 3.07 3.96 19.5 Calf - Ankle 14 46
R Sural - Ankle (Calf)
Calf Ankle 2.76 3.59 16.1 Calf - Ankle 14 51

The F wave was prolonged in all 8 of the tested nerves: Left and Right Median, Ulnar, Peroneal and Tibial.

Figure 1. Demonstrating Nerve Conduction Test Results.

MANAGEMENT, OUTCOME AND FOLLOWUP

Classic CSF and NCS findings confirmed the diagnosis of GBS. The patient was put on intravenous immunoglobulin(IVIG) at a dose of 2 g/kg body weight divided over 5 consecutive day, 35 gm with premedication of intravenous paracetamol, chlorphenaramine, ranitidine and hydrocortisone.Oral metoprolol 12.5 mg daily was administered for hemodynamic liability. Physiotherapy was started and was put on high calorie, high protein oral diet. After the third day of IVIG treatment, there was a gradual improvement in the power of the lower limbs (4/5 on bilateral lower limbs). Single breath count decreased to 20-24 over the first 2 days treatment but increased from day 3 to 40-48. Following completion of immunoglobulin therapy, patient was shifted to ward and later discharged to home after 21 days of admission.

DISCUSSION

GBS appears to be an autoimmune response where the peripheral nervous system is involved. The diagnostic criteria for GBS include progressive, relatively symmetrical weakness with decreased or absent myotatic reflexes.*Campylobacter jejuni*, one of the most common causes of gastroenteritis worldwide, causes 30% to 35% of GBS cases.[3] A small percentage of patients develop GBS after immunization, surgery, trauma, or bone marrow transplantation.[3] In our case, there was no symptoms suggestive of gastroenteritis but positive history of immunization.CoV infections can itself affect the nervous system that may lead to neurological diseases such as viral encephalitis, acute cerebrovascular disease and infectious toxic encephalopathy[4,5] This may occur due to direct nerve injury or hypoxia in the CNS, increasing anaerobic metabolism, activation of immune cells causing chronic inflammation,damaging BBB and attacking the vascular system.[6]

Patients with GBS classically have elevated protein levels and normal WBC counts, condition known as albuminocytologic dissociation.[4] Intravenous immunoglobulin 2 gm/kg over 5 days and plasma exchange 200-250 mL/kg for five sessions are the accepted therapy.

Janssen COVID-19 Vaccine is manufactured by Janssen Pharmaceuticals Companies of Johnson & Johnson, a single shot viral vector type vaccine. Vector enters a cell, uses cell's machinery to produce a spike protein, found on surface of virus causing COVID-19 which is recognized by immune system and triggers the production of antibodies to protect itself against future infection of COVID-19. As per US Advisory Committee on Immunization Practices (ACIP) until 30th June, 100 cases of GBS had been reported with 12.2 million doses of Janssen vaccine.[5] Incidence of GBS was about two weeks after vaccination and mostly in males, above 50 years and had issued a safety warning. Benefits outweighs the risks of the vaccine[6] and no actual association has been found till date between vaccine and GBS.

CONCLUSIONS

As it is well reported that GBS can occur a few weeks post vaccination and only potential triggering factor in this case was positive finding of Jansen Vaccine, we

believe there is a reasonable possibility that GBS has occurred post-vaccine.

Author Affiliations
[1]Nepal Mediciti Hospital, Lalitpur, Nepal

Competing interests: None declared

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