CASE REPORT

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Sensory Predominant Guillain-Barré Syndrome Following Pfizer-BioNTech COVID-19 Vaccine: A Rare Variant

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ABSTRACT The sensory pre-dominant sub-type of Guillain-Barré syndrome (GBS) is a scarce condition that is difficult to diagnose because of the heterogeneity of the patient clinical characteristics so it can be easily missed. A 56-year-old male patient was admitted with numbness in both lower and upper limbs for 5 days following the Pfizer-BioNTech coronavirus disease-2019 (COVID-19) vaccine. His symptoms began with numbness in the lower limbs and spread up all limbs. He also described the burning dysesthesia on both feet and hands. Neurophysiological studies and cerebrospinal analysis findings were consistent with the diagnosis of sensory pre-dominant sub-type of GBS. Significant improvement was observed following the intravenous immunoglobulin treatment. Other types of GBS following the Pfizer-BioNTech COVID-19 vaccine have been reported previously. However, this is the first case in the literature presented with sensory dominance in terms of clinical and neurophysiological studies following the second dose of the Pfizer-BioNTech COVID-19 vaccine.

Keywords: Guillain-Barré syndrome; sensory pre-dominant; Pfizer-BioNTech; coronavirus disease-2019; vaccine

Guillain-Barré syndrome (GBS) is a rare immune-mediated disorder with an incidence of 0.8-1.9/100,000, and one of the most common causes of acute non-trauma-related paralysis in developing countries.[1] Typical GBS presents with progressive ascending paralysis, loss of deep tendon reflexes (DTR), and autonomic symptoms. Furthermore, the sensory deficit presents in 80% of cases but is not usually prominent.[2] Although GBS is often preceded by gastrointestinal or respiratory infections, it may develop following vaccination.

Two mRNA vaccines for coronavirus disease-2019 (COVID-19) were approved by the U.S. Food and Drug Administration in December 2020.[3] The most commonly reported side effects are injection site reactions, myalgia, and fever. Rarely, some GBS cases following the COVID-19 vaccine have been reported. However, they were mostly the classic forms of GBS.[4] We report here a rare case of sensory-predominant GBS that occurred 5 days following the Pfizer-BioNTech 2nd dose vaccine. To the best of our knowledge, this is the 1st reported case in the literature of sensory-predominant GBS after receiving the COVID-19 vaccine.

CASE REPORT

A 56-year-old male patient was admitted with complaints of numbness in both the lower and upper extremities 5 days after the Pfizer-BioNTech COVID-19 vaccine. His symptoms began with numbness in the distal lower limbs 5 days following the vaccine and spread up all limbs within 2 days. He also described the burning dysesthesia associated with a “pins and needles” sensation on both feet and

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TABLE 1: Motor nerve conduction studies at the time of initial diagnosis-Motor nerve conduction studies the 6-month follow-up

Site	Latency (ms)	Amplitude (mV)	Segment	NCV (m/s)	F wave minimal latency (ms)	Latency (ms)	Amplitude (mV)	Segment	NCV (m/s)	F wave minimal latency (ms)
Median left										
Wrist	7.11	10.8	Wrist		-	3.74	10.9	Wrist		31.8
Elbow	10.4	10.1	Wrist-elbow	51.7		8.48	10.8	Wrist-elbow	57	
Median right										
Wrist	4.77	9.1	Wrist		44.75	3.7	11.2	Wrist		33.4
Elbow	10.1	8.6	Wrist-elbow	56.5		8.14	10.7	Wrist-elbow	58.6	
Ulnar left										
Wrist	3.6	8.9	Wrist		37.2	2.52	7.1	Wrist		37
Elbow	9.1	8.5	Wrist-elbow	50.9		8.04	7.2	Wrist-elbow	54.3	
Ulnar right										
Wrist	2.4	7.9	Wrist		-	2.7	9.5	Wrist		-
Elbow	8.8	7.3	Wrist-elbow	51.9		8.3	9.1	Wrist-elbow	53.8	
Peroneal left										
Ankle	8.5	4	Ankle		57	4.75	4.1	Ankle		56
Popliteal	16.2	3.7	Ankle-popliteal	42.9		13.45	3.8	Ankle-popliteal	43.7	
Peroneal right										
Ankle	8.5	4.8	Ankle		56	4.8	4.8	Ankle		56
Head of fibula	14.6	4.4	Ankle-head of fibula	47.1		13.6	4.4	Ankle-head of fibula	47	
Tibial left										
Ankle	5.4	7.7	Ankle		66.1	4.2	7.6	Ankle		55.8
Head of fibula	15.2	7.1	Ankle-head of fibula	40.8		13	7.1	Ankle-head of fibula	48	
Tibial right										
Ankle	5.5	8.2	Ankle		60	4.3	8.3	Ankle		55.7
Popliteal	14.6	7.9	Ankle-popliteal	44		14	7.9	Ankle-popliteal	46.4	

NCV: Nerve conduction velocity.

TABLE 2: Sensory nerve conduction studies at the time of initial diagnosis- Sensory nerve conduction studies at 6-month follow-up.

Site	Latency (ms)	Amplitude (mV)	NCV (m/s)	Latency (ms)	Amplitude (mV)	NCV (m/s)
Median left						
Antidromic-Dig II	-	-	-	2.66	10.1	50.8
Median right						
Antidromic-Dig II	-	-	-	2.94	8.5	44.2
Ulnar left						
Antidromic-Dig V	-	-	-	2.74	6.7	38.3
Ulnar right						
Antidromic-Dig V	-	-	-	4.06	10.6	27.1
Sural left						
Ankle	3.2	6.1	48.6	2.6	14.5	55
Sural right						
Ankle	3.4	8.4	49.2	2.7	13.4	53.2

NCV: Nerve conduction velocity.

hands. Although he used to go for a walk daily, recently he had difficulty walking due to numbness on the soles of both feet. He denied any chronic disease but hypertension. There was no history of viral infection or any neurological diseases. Blood cell count, C-reactive protein, renal and liver function tests, coagulation profile, hemoglobinA1c level, electrolytes, vitamin D, vitamin B_{12}, and folate were all in the normal range. In addition, biomarkers for vasculitis and rheumatic diseases were negative. Cranial and whole spine Magnetic resonance imaging (MRI)s were normal. He denied previous flu-like or gastrointestinal episodes and tested negative for severe acute respiratory syndrome-coronavirus-2 by reverse transcription-polymerase chain reaction (PCR).

On neurological examination, cognitive function and cranial nerve examination were normal. However, he had an antalgic gait secondary to pain. In the sensory examination, hyperesthesia for pain and temperature was detected as a "glove and stocking" distribution at the wrist level on both upper extremities and up to the mid-calf on the lower extremities. Vibration sensation was absent at the toes and reduced in the distal fingertips. Proprioception was intact throughout. Although he denied any weakness besides fatigue, motor strength was 4/5 in both lower limbs, proximally and distally (in knee flexion/extension, hip flexion/extension) according to the Medical Research Council grade. Other muscles' strength was normal. DTRs were absent in both the upper and lower extremities. No pathological reflexes were detected. Cerebrospinal fluid (CSF) analysis revealed elevated protein at 114.1 mg/dl (reference range 15-45 mg/dl) with a normal leukocyte count of 3 cells/mm^3, consistent with albuminocytological dissociation. CSF cultures and Real-time viral PCRs were negative for Herpes simplex virus, *Cytomegalovirus*, Varicella-zoster, *Mycobacterium tuberculosis*, Epstein-Barr Virus, and Enterovirus.

Neurophysiological studies showed prolonged median, peroneal, and tibial distal motor latencies with prolonged peroneal and tibial F waves (Table 1). Also, F waves were absent for the left median and right ulnar nerves. Sensory studies revealed normal sural sensory amplitude and conduction velocity (CV) with absent median and ulnar sensory response (Table 2), which is a characteristic finding of GBS ("sural sparing pattern") (Figure 1, Table 2).[5] The patient was treated with intravenous immunoglobulin (IVIG) at a total dose of 2g/kg over 5 days. Also, pregabalin was initiated for his neuropathic pain. Meanwhile, improvement was observed in both motor and sensory examinations. At a 3 month follow-up examination, neurological symptoms improved significantly. At 6 months, the neurological examination returned to normal, except for minimal tingling sensation in his fingers, and neurophysiological studies were markedly improved (Figure 2) (Table 1 ve Table 2). Written informed consent of the patient was obtained.

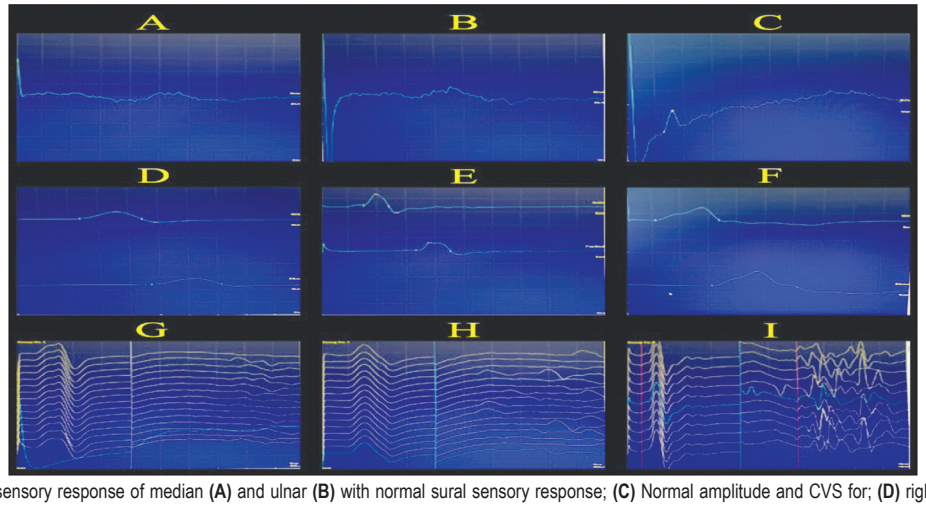

FIGURE 1: Absent sensory response of median **(A)** and ulnar **(B)** with normal sural sensory response; **(C)** Normal amplitude and CVS for; **(D)** right tibial motor nerve; **(E)** right ulnar motor nerve; **(F)** left median motor nerve; Absent F waves on **(G)** left median and **(H)** right ulnar motor nerves; **(I)** prolonged F waves on left tibial motor nerve (at the time of initial diagnosis).

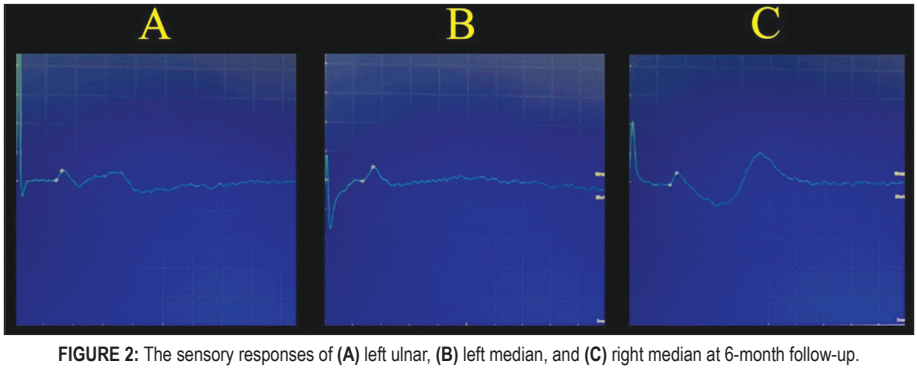

FIGURE 2: The sensory responses of **(A)** left ulnar, **(B)** left median, and **(C)** right median at 6-month follow-up.

DISCUSSION

The underlying etiology of GBS is usually reported as a post-infection in up to 70% of patients.[6] Also, cases of GBS have been reported following vaccinations, such as influenza, meningococcal, polio, tetanus, and rabies.[7] Molecular mimicry theory has a critical role in the pathogenesis of GBS.[8] Some lipopolysaccharides in infectious agents, such as Campylobacter Jejuni, show structural similarities with the gangliosides of peripheral nerve membranes.[9] Also, it has been shown that ganglioside antibodies have different peripheral nerve targets. Anti-GD1a antibodies attach to paranodal myelin, nodes of Ranvier, and neuromuscular junction, whereas GM1 and GQ1B antibodies attach to a peripheral nerve or neuromuscular junction.[10] Therefore, it has been suggested that these different types of peripheral nerve targets may play a critical role in the heterogeneity of the clinical presentation of GBS.[8]

The COVID-19 pandemic has affected the whole world earth-shakingly. In this pandemic, where the death rate was very high, these numbers began to decrease as the vaccination rate increased. Most side effects of vaccination include pain, erythema at the administration site, myalgia, sore throat, and febrile reaction. Furthermore, post-vaccine GBS variants started to be encountered. Recently, Vaccine Safety Datalink reported 36 cases of GBS after mRNA vaccines.[11] Their symptom onset ranged from 0 to 84 days after the vaccine, and acute inflammatory demyelinating polyradiculoneuropathy (AIDP),

Acute Motor Axonal Neuropathy (AMAN), and Acute Motor and Sensory Axonal Neuropathy (AMSAN) subtypes have been reported as the most common subtypes.[11] Our patient is the first case report of sensory predominant of GBS following Pfizer-BioNTech mRNA COVID-19 vaccine. Sensory predominant of GBS has been rarely reported in the literature, but the scarcity and heterogeneity of those cases make their recognition difficult in the first place. Therefore, several diagnostic criteria have been recommended, and the most validated one includes (i) rapid onset, (ii) widespread and symmetrical symptoms, (iii) albuminocytological dissociation, (iv) demyelinating features in neurophysiological studies, and (v) good prognosis.[12] Moreover, as most cases may present some weakness in motor strength, like our case, it makes the diagnosis even more difficult.[13] The most challenging differential diagnosis of sensory GBS is sensory neuronopathy (ganglionopathy). There are several characteristics that can help us make a differential diagnosis.[13,14] First, the progression of symptoms in sensory neuropathy is subacute or chronic, whereas classic GBS is acute. Sensory neuronopathy usually involves the face and scalp, and pain might be the primary symptom. In addition, the neurophysiological impairment of sural sparing is a characteristic finding for classic GBS.[5] And the presence of albuminocytological dissociation supported our diagnosis for the current case. Finally, sensory GBS generally responds well to IVIG treatment with a good prognosis, like in our case.[14]

We were unable to investigate for anti-ganglioside antibodies that would help us identify the sub-variant. As it is already reported as positive in only 36% of patients with GBS, this marker is not necessary for differential diagnosis.[15] Also, we couldn't perform the nerve biopsy that would support our diagnosis.

This case may have several contributions to the current literature. First, neurologists should be aware of such adverse effects following the COVID-19 vaccination. Second, this rare sub-variant of GBS should be considered, as patients benefit from immunotherapy, especially in the early stages. Finally, since the benefits of COVID-19 vaccination far outweigh the risks of severe adverse events, including neurological involvement, such adverse effects should not be discouraging, given the profit and loss ratio.[4]

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

This study is entirely author's own work and no other author contribution.

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