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CASE REPORT

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Post COVID-19 vaccination Guillain-Barre syndrome: three cases

Seyedehnarges Tabatabaee[a], Fatemeh Rezania[b], Sayed Mohammed Jawad Alwedaie[c], Ensieh Malekdar[d], Zahra Badi[e], Seyed Mohammad Tabatabaei[f], and Zahra Mirzaasgari[a,g]

[a]Department of Neurology, Firoozgar Hospital, School of Medicine, Iran university of medical sciences, Tehran, Iran; [b]Neurosciences Department, St Vincents Hospital, Melbourne, Australia; [c]Royal College of Surgeons in Ireland, Medical university of Bahrain, Manama, Bahrain; [d]Department of Neurology, Motamedi Hospital, Garmsar, Iran; [e]Department of Neurology, Erfan Hospital, Tehran, Iran; [f]Department of Electrical and Computer Engineering, University of Tehran, Tehran, Iran; [g]Shafa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran

ABSTRACT

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy. In two-thirds of patients, it is preceded by an upper respiratory or gastrointestinal tract infection. Temporally associated cases of GBS following COVID-19 vaccination have been described with different COVID-19 vaccines. In this study, we report three cases of GBS patients following COVID-19 vaccine. Two of the studied patients received the Sinopharm vaccine and one patient received the AstraZeneca vaccine. All patients were diagnosed with acute motor axonal neuropathy (AMAN) type of GBS, on nerve conduction studies. All three patients responded well to treatment with intravenous immunoglobulin (IVIg). The association between COVID-19 vaccination and GBS is not well understood and more studies are needed to establish whether it is merely an association or a causal relationship.

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Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy. In two-thirds of patients, it is preceded by an upper respiratory or gastrointestinal tract infection.[1,2] GBS has also been repeatedly reported during or after severe COVID-19 infection.[3] A number of vaccines have been shown to be associated with an increased risk of GBS.[1] There have been increasing reports of GBS following vaccination.[4–8] Temporally associated cases of GBS following COVID-19 vaccination have been described with messenger-RNA vaccine,[4] adenovirus-vectored COVID-19 vaccine,[5–7] and inactivated COVID-19 vaccines.[8] Here, we describe three patients who developed GBS shortly after being vaccinated against COVID-19 with the AstraZeneca second dose and Sinopharm first dose in the absence of any other known precipitating factors.

Case 1

A 46-year-old man who was previously well presented with one-week history of lower limbs weakness and 3 days of upper limbs weakness. His symptoms started three days after receiving the second dose of AstraZeneca vaccine. He described an ascending weakness together with pain in his lower limbs. He did not report back pain or bowel/bladder dysfunction. He denied fever, upper respiratory tract symptoms, or gastrointestinal illness leading up to his presentation. On examination, he was afebrile and hemodynamically stable. An evaluation of muscle power revealed Medical Research Council (MRC) grade +4/5 in upper limbs muscles, and MRC 4/5 in lower limbs. His deep tendon reflexes were absent in the ankles, 1+ in the knees and 2+ in biceps, triceps, and brachioradialis. All sensory modalities were intact in upper and lower limbs. His cranial nerve examinations were unremarkable.

Laboratory tests, including HIV viral serology, syphilis serology, and vasculitic screening showed negative results; however, his cerebrospinal fluid (CSF) examination revealed a high protein reading (184 mg/dL; reference range 15–45 mg/dL) with no cells. A nasopharyngeal (NP) swab and CSF for SARS-CoV-2 real-time polymerase-chain reaction (RT-PCR) test were negative. He underwent electromyography and nerve conduction studies (EMG-NCS) that showed features consistent with an acute motor axonal neuropathy (AMAN) subtype of GBS (Table 1). As a result, a diagnosis of GBS was considered. The patient was treated with 170-g intravenous immunoglobulin (IVIG) (2 g/kg) which resulted in partial improvement in his symptoms. (Table 2)

Case 2

A previously healthy 36-year-old man, presented to the Emergency Department with progressive generalized weakness, difficulty walking and clumsiness of his hands. He received his first dose of the Sinopharm vaccine five days prior to his presentation. He denied any history of prodromal infection prior to the onset of his symptoms.

He experienced a generalized weakness within the first 24 h after receiving his vaccination. However, his symptoms only progressed three days post vaccination with deterioration in his mobility requiring assistance for ambulation.

CONTACT Zahra Mirzaasgari mirzaasgari@gmail.cominstitution Department of Neurology, Firoozgar Hospital, School of Medicine, Iran university of medical sciences, Tehran, Iran

Table 1. Nerve conduction study parameters in patient 1.

			Amplitude*			Latency (ms)			Conduction velocity (m/s)			F wave latency (ms)		
Nerve stimulated	Stimulation site	Recoding site	Rt	Lt	Ref	Rt	Lt	Ref	Rt	Lt	Ref	Rt	Lt	Ref
Median (M)	Wrist	APB	1.1	1.2	≥4	4.6	4.1	≤4.4				NM	NM	≤31
	AF	APB	1.1	1.3		9.25	8.9		55.9	54.2	≥49			
Ulnar (M)	Wrist	ADM	1.7	1.0	≥6	3	3.65	≤3.3				NM	NM	≤32
	BE	ADM	1.5	0.9					58	59	≥49			
	AE	ADM	1.2	0.8					56	60	≥49			
Tibial (M)	Ankle	AHB	1.0	0.3	≥4	4.95	8.0	≤5.8				62	62	≤56
	PF	AHB	0.8	0.3					40	45	≥41			
Peroneal (M)	Ankle	EDB	0.9	0.3	≥2	6.5	7.7	≤6.5	48.1			54	55	≤56
	BF	EDB	0.8	0.3						48.4	≥44			
	LPF	EDB	0.7	0.2						47.1	≥44			
Median (S)	Wrist	Digit 2	28	26	≥20	3.75	3.85	≤3.5	49	47.5	≥50			
Ulnar (S)	Wrist	Digit 5	19.8	18.7	≥17	3.4	3.4	≤3.1	45	45	≥50			
Sural (S)	Calf	Post. ankle	16.6	14.5	≥6	4.6	4.4	≤4.4	53	52	≥40			

There was significant drop in compound muscle action potential (CMAP) amplitude in 4 limbs with preserved conduction velocity and F-wave latency (were tested). Sensory responses were normal in 4 limbs. These findings were in keeping with acute motor axonal neuropathy (AMAN) subtype of GBS.

M = motor responses, S = antidromic sensory responses; ms = milli second, m/s = meter per second; Rt = right; Lt = left; Ref = Reference range; NM = not measured; AF = anterior fossa; BE = below elbow; AE = above elbow; BF = below fibula; LPF = lateral posterior fossa; APB = abductor pollicis brevis; ADM = abductor digiti minimi; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis.

*Motor studies' amplitudes measured in millivolts (mV) and sensory studies' amplitudes measured in microvolts (μV).

The patient did not experience any sensory symptoms and gave no history of sphincteric dysfunction. He had a normal cranial nerve examination.

Motor examination demonstrated normal bulk and tone in bilateral upper and lower extremities. Strength in bilateral upper extremities was noted to be MRC 4/5. The examination of muscle strength in bilateral lower extremities showed MRC grade 3/5 in all muscle groups. Deep tendon reflexes were diminished in all four limbs. He had a normal sensory examination. There was no evidence of appendicular ataxia. MRI of the brain was normal. A NP swab for SARS-CoV-2 PCR test was negative. Routine lab tests were unrevealing. CSF examination showed a slight elevation of protein (54 mg/dL) with white cell count of 0. The patient underwent EMG-NCS that showed features consistent with an AMAN form of GBS (Table 3). This patient received an induction dose of IVIG (2 g/kg), which led to relative clinical improvement. (Table 2)

Case 3

A 32-year-old healthy male with no previous medical history, presented 14 days after the first dose of the Sinopharm COVID-19 vaccination, with an acute ascending weakness. The patient had an afebrile diarrhea a week prior to the onset of his symptoms. Four days before his presentation, he developed distal lower limbs weakness that progressed gradually to involve his upper limbs. He had no sensory complaints. He had normal cranial nerves function. Motor examination showed loss of dexterity in hands and MRC grade 4/5 in both proximal and distal muscles of lower limbs. Deep tendon reflexes were diminished in all four limbs while having an intact sensory function. No abnormality was noted on other examinations. MRI of the brain was normal. SARS-CoV-2 PCR test was negative in NP swab. His CSF study showed a normal protein level of 30 mg/dL with no white cells. He underwent EMG-NCS that showed features consistent with an AMAN variant of GBS (Table 4). His symptoms improved partially after IVIG therapy (2 g/kg) and a period of rehabilitation. (Table 2)

Discussion

GBS is an inflammatory polyradiculoneuropathy that is usually precipitated by various infections and is one of the most common causes of acute flaccid paralysis,[9] presenting with varying degrees of weakness, sensory abnormalities, and autonomic dysfunction.[10] While the exact etiopathogenesis of GBS seems to be unclear, molecular mimicry and immune-mediated mechanisms like antiganglioside antibody production and complement activation are the most widely accepted theories.[9,11] Approximately two-thirds of GBS cases happen to occur after an episode of infectious disease with 1–3 weeks interval.[12] Various reports of GBS following COVID-19 infection have been published so far.[3] Although a temporal relationship between GBS and some vaccines has been reported,[10,13,14] the establishment of a causal link is proved to be controversial. Nevertheless, a small increase in the incidence of GBS after influenza vaccination is relatively well established.[13,15] It is prudent to closely monitor the potential adverse events of COVID-19 vaccines such as COVID-19 vaccination-associated GBS. Recently, the co-occurrence of GBS with various COVID-19 vaccines has been reported.[4,5,7,16] Likewise, we described a temporal link between receiving Sinopharm and AstraZeneca COVID-19 vaccines and GBS onset within 34–14 days after injection. To date, the most common reported side effects of COVID-19 vaccines are injection site reactions, flu-like complaint, headache, and asthenia that mostly are mild and self-limiting conditions and do not require any intervention.[17,18]

We report the first cases of GBS post COVID-19 vaccination in Iran that initially presented with the typical GBS symptoms of ascending weakness. This is in contrast to the previous three published case reports, which have described 12 patients in

Table 2. Characteristics of three patients with GBS after the COVID-19 vaccination.

Patient No.	Characteristics	symptoms Onset of Neurologic	COVID-19 Vaccine and Timeline of Symptoms	Neurologic Signs and Symptoms	CSF analysis	EMG-NCS subtype	Treatment	Outcome
1	46-year-old male	7 days prior to admission	AstraZeneca, 3 days after the second dose	Progressive ascending quadriparesis, Decreased or absent DTR in weak limbs	elevated protein level (184 mg/dl) no cells	AMAN	170 g IVIG physiotherapy	Relative improvement
2	36-year-old male	5 days prior to presentation	Sinopharm, 5 days after the firstdose	Progressive generalized weakness, diminished DTR in all limbs	Elevated protein level (54 mg/dL)WBC <10 (normal)	AMAN	160 g IVIG physiotherapy	Relative improvement
3	32-year-old male	4 days prior to presentation	Sinopharm, 14 days after the first dose	Progressive ascending quadriparesis, Decreased or absent DTR in weak limbs diarrhea	protein level (30 mg/dL) no cells	AMAN	185 g IVIG physiotherapy	Relative improvement

EMG-NCS: electromyography and nerve conduction studies; CSF: cerebrospinal fluid; IVIG: intravenous immunoglobulin; AMAN: acute motor axonal neuropathy; DTR: Deep tendon reflex.

Table 3. Nerve conduction study parameters in patient 2.

			Amplitude*			Latency (ms)			Conduction velocity (m/s)			F wave latency (ms)		
Nerve stimulated	Stimulation site	Recoding site	Rt	Lt	Nl	Rt	Lt	Nl	Rt	Lt	Nl	Rt	Lt	Nl
Median (m)	Wrist	APB	3.1	2.9	≥4	4.10	3.80	≤4.4						≤31
	AF	APB	2.6	2.5	≥4	8.02	8.35		44.0	42.2	≥49			
Ulnar (M)	Wrist	ADM	3.5	2.6	≥6	2.75	2.63	≤3.3	54.2	51.3				≤32
	BE	ADM	2.8	1.7	≥6	7.4	7.35				≥49			
	AE	ADM	1.8	1.0	≥6						≥49			
Tibial (M)	Ankle	AHB	1.1	0.8	≥4	5.20	5.35	≤5.8	43.5	40.7		62	67	≤56
	PF	AHB	0.7	0.6	≥4						≥41			
Peroneal (M)	Ankle	EDB	1.3	1.5	≥2	7.20	8.10	≤6.5	51.7	54.2		56	57	≤56
	BF	EDB	0.7	1.1							≥44			
	LPF	EDB	0.7	0.9							≥44			
Median (S)	Wrist	Digit 2	28	32	≥20	3.65	3.40	≤3.5	38.5	42.5	≥50			
Ulnar (S)	Wrist	Digit 5	35.4	32.3	≥17	2.8	2.90	≤3.1	45.9	44.3	≥50			
Sural (S)	Calf	Post. ankle	13.4	14.5	≥6	4.15	4.35	≤4.4	31.7	34.8	≥40			

There was significant drop in compound muscle action potential (CMAP) amplitude in 4 limbs with preserved conduction velocity and F-wave latency (were tested). Sensory responses were normal in 4 limbs. These findings were in keeping with acute motor axonal neuropathy (AMAN) subtype of GBS.

M= motor responses, *, S=antidromic sensory responses; ms = milli second, m/s = meter per second; Rt = right; Lt = left; Ref = Reference range; NM = not measured; AF = anterior fossa; BE = below elbow; AE = above elbow; BF = below fibula; LPF = lateral posterior fossa; APB = abductor pollicis brevis; ADM = abductor digiti minimi; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis.

*Motor studies' amplitudes measured in millivolts (mV) and sensory studies' amplitudes measured in microvolts (μV).

total with post AsteraZeneca vaccine GBS within all of them having uncommon presentation of bilateral facial weakness as presenting symptom.[6,7,19] Various subtypes of GBS differ clinically and electrophysiologically. However, GBS typically presents with a symmetric weakness of both upper and lower limbs with depressed or complete lack of reflexes (areflexia).[11] To the best of our knowledge, there have been only three cases of GBS after the COVID-19 Sinopharm vaccine reported so far in the literature.[8] Here, we described two further cases of GBS associated with COVID-19 Sinopharm vaccine. Post vaccine GBS has been mostly shown to be an acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype electrophysiologically,[20–22] in contrast to our cases of AMAN subtype of GBS. Despite reports on higher morbidity and mortality rates in AMAN patients, especially in those with lower CMAPs amplitudes,[22,23] our patients showed a considerable short-term improvement after receiving IVIG therapy.

Given the current situation of the COVID-19 pandemic, vaccination against this virus has become one of the most imperative strategic plans in many countries. Several mechanisms may be involved in the association of autoimmune diseases after vaccination such as the cross reaction of contaminating proteins or other vaccine components that share similarities with myelin or axon epitopes with peripheral nerves.[24]

An extensive epidemiological study has not been performed yet to investigate the relationship between GBS after COVID-19 vaccination. However, since GBS is a debilitating disease, a diligent reporting of these rare adverse events on the safety of the COVID-19 vaccine not only allows physicians to recognize and treat this rare condition in a timely fashion but also enables scientists with an opportunity to further investigate a potential link between GBS and COVID-19 vaccination.

Table 4. Nerve conduction study parameters in patient 3.

			Amplitude*			Latency (ms)			Conduction velocity (m/s)			F wave latency(ms)		
Nerve stimulated	Stimulation site	Recoding site	Rt	Lt	Nl	Rt	Lt	Nl	Rt	Lt	Nl	Rt	Lt	Nl
Median (M)	Wrist	APB	3.5	3.6	≥4	4.15	3.75	≤4.4						≤31
	AF	APB	2.9	3.3	≥4	9.05	8.3		49.0	52.7	≥49			
Ulnar (M)	Wrist	ADM	3.9	2.3	≥6	2.65	2.75	≤3.3	57.1	54.2				≤32
	BE	ADM	2.9	1.6	≥6	7.2	7.55				≥49			
	AE	ADM	1.8	0.9	≥6						≥49			
Tibial (M)	Ankle	AHB	1.3	0.9	≥4	5.45	5.7	≤5.8	47.3	41.7		65	68	≤56
	PF	AHB	0.8	0.7	≥4						≥41			
Peroneal (M)	Ankle	EDB	1.5	1.8	≥2	7.4	8.65	≤6.5	54.4	59.3		58	55	≤56
	BF	EDB	0.8	1							≥44			
	LPF	EDB	0.7	0.9							≥44			
Median (S)	Wrist	Digit 2	35	42	≥20	3.85	3.50	≤3.5	37.5	41.5	≥50			
Ulnar (S)	Wrist	Digit 5	37.7	40.1	≥17	2.9	3.00	≤3.1	46.8	47.9	≥50			
Sural (S)	Calf	Post. calf	12.1	15.5	≥6	3.85	4.40	≤4.4	32.4	36.7	≥40			

There was significant drop in compound muscle action potential (CMAP) amplitude in 4 limbs with preserved conduction velocity and F-wave latency (were tested). Sensory responses were normal in 4 limbs. These findings were in keeping with acute motor axonal neuropathy (AMAN) subtype of GBS.

M= motor responses, S= antidromic sensory responses; ms = milli second, m/s = meter per second; Rt = right; Lt = left; Ref = Reference range; NM = not measured; AF = anterior fossa; BE = below elbow; AE = above elbow; BF = below fibula; LPF = lateral posterior fossa; APB = abductor pollicis brevis; ADM = abductor digiti minimi; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis.

*Motor studies' amplitudes measured in millivolts (mV) and sensory studies' amplitudes measured in microvolts (μV).

Abbreviations

GBS	Guillain-Barré syndrome
MRC	Medical Research Council
CSF	cerebrospinal fluid
NP	nasopharyngeal
RT-PCR	real time polymerase chain reaction
EMG-NCS	electromyography and nerve conduction studies
AMAN	acute motor axonal neuropathy
IVIG	intravenous immunoglobulin
AIDP	acute inflammatory demyelinating polyradiculoneuropathy

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ORCID

Sayed Mohammed Jawad Alwedaie http://orcid.org/0000-0003-0101-2137
Zahra Mirzaasgari http://orcid.org/0000-0002-4046-8471

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