Turk J Phys Med Rehab 2022;68(2):295-299
DOI: 10.5606/tftrd.2022.9984
Available online at www.turkishjournalpmr.com

Case Report

A case of Guillain-Barre syndrome after the second dose of AstraZeneca COVID-19 vaccination

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Received: October 14, 2021 **Accepted:** January 29, 2022 **Published online:** June 01, 2022

ABSTRACT

Coronavirus disease 2019 (COVID-19) is a novel virus that primarily involves the respiratory system. Due to the COVID-19 pandemic, an extensive vaccination program is underway worldwide. Herein, we present a 68-year-old woman with paresthesia of both hands associated with gait instability, which started three to four days after receiving the second dose of Oxford/AstraZeneca vaccine against the COVID-19 infection. The acute inflammatory demyelinating polyradiculoneuropathy subtype of the Guillain-Barre syndrome, which is the most common subtype, was diagnosed. Regardless of the beneficial effects of the vaccines, this case report aimed to evaluate their severe complications, such as Guillain-Barre syndrome, to reduce their occurrence in the future.

Keywords: AstraZeneca vaccine, COVID-19, Guillain-Barre syndrome.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) primarily involves the human respiratory system and is the cause of the recent coronavirus disease 2019 (COVID-19) pandemic. The usual presentation of COVID-19 is mild/asymptomatic to life-threatening pneumonia.[1,2] In response to the pandemic, vaccination against COVID-19 has been underway worldwide for several months. Its neurological complications and side effects, such as cerebral vein thrombosis and Guillain-Barre syndrome (GBS), have been reported recently.[3,4]

Guillain-Barre syndrome is an immune-mediated polyradiculoneuropathy that is usually caused by various infections and less frequently by vaccination. Clinical features of GBS are progressive symmetric muscle weakness and paresthesia accompanied by absent deep tendon reflexes two to four weeks after the triggering event.[5,6] In this report, we describe a case of acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the most common subtype of GBS, that developed after the second dose of Oxford/AstraZeneca COVID-19 vaccine with distal paresthesia and gait instability.

CASE REPORT

A 68-year-old Iranian female with complaints of paresthesia and numbness of both hands and feet associated with an abnormal gait that persisted for 10 days before was referred to our clinic in 2021. The patient's history did not reveal any significant previous

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Cite this article as:
Bazrafshan N, Mohamadi Jahromi LS, Parvin R, Ashraf A. A case of Guillain-Barre syndrome after the second dose of AstraZeneca COVID-19 vaccination. Turk J Phys Med Rehab 2022;68(2):295-299.

illness. The patient described being relatively well until they developed gradual onset paresthesia and numbness beginning from their hands and progressing to the feet accompanied by the inability to walk independently three to four days after receiving the second dose of AstraZeneca COVID-19 vaccination. The patient claimed they had no history of recent exposures to infected patients with COVID-19 or had no respiratory tract and gastrointestinal problems over the last few weeks. The patient did not exhibit dysphagia, dysarthria, urinary and fecal incontinence, constitutional signs and symptoms (for example, fever), and respiratory manifestations. The patient had received the first dose of AstraZeneca COVID-19 vaccination almost three months ago, with no significant complications after the injection. In physical examination, the patient was a little pale in general appearance, but the vital signs were stable. Muscle strength of the proximal lower limbs and the distal upper and lower limbs were 4/5, 4/5, and 3/5, respectively. All deep tendon reflexes were absent. Light touch and temperature sensations were decreased in both legs and hands. Sensory function was preserved, and Hoffman and Babinski signs were negative in our patient. Cranial nerves were intact. Nerve conduction studies of lower limbs showed prolonged bilateral tibial and peroneal compound muscle action potentials (CMAPs) of more than 6 ms while recording from the abductor hallucis and extensor digitorum brevis muscles with evidence of temporal dispersion in left tibial nerve while stimulating from the popliteal fossa. Nerve conduction velocities were decreased to about 32 ms in tibial pathways. The amplitude of CMAP recording from the left extensor digitorum brevis was decreased to 0.8 mV. The sural and superficial peroneal sensory nerve action potentials (SNAPs) were relatively spared except for a mild decrease in amplitudes. In upper limbs, median, ulnar, and radial SNAPs were absent, and CMAPs recorded from the bilateral abductor pollicis brevis and abductor digiti

TABLE 1
Nerve conduction studies

Nerve/site	Rec/site	Latency(ms)	Amplitude (mv)	Velocity
Sensory				
Left median-wrist	Digit3	Absent	0.0	
Left ulnar-wrist	Digit5	Absent	0.0	
Right ulnar-wrist	Digit5	Absent	0.0	
Right median-wrist	Digit3	Absent	0.0	
Left sural-calf	Ankle	3.45	10.1	
Right sural-calf	Ankle	3.55	10.2	
Motor				
Right median	APB			
Wrist		6.03	8.0	
Elbow		10.80	5.1	34.5+ conduction block
Left median	APB			
Wrist		5.80	6.7	
Elbow		9.90	4.3	44.0+ conduction block
Right ulnar	ADM			
Wrist		5.30	5.50	
Elbow		9.80	4.90	45.7
Left common peroneal	EDB			
Ankle		6.2	0.80	
Fib head		13.90	0.70	39.30
Left tibial	AH			
Ankle		6.35	2.60	
Popliteal fossa		16.45	1.50	32.7+temporal dispersion
F-Waves				
Right tibial-AH		Absent		
Right median-APB		Absent		
Left ulnar-ADM		Absent		
H-Reflex				
Left tibial-soleus		Absent		
Right tibial-soleus		Absent		

APB: Abductor pollicis brevis; ADM: Abductor digiti minimi; EDB: Extensor digitorum brevis; AH: Abductor hallucis.

TABLE 2
Needle electromyography

Muscles	IA	PSW	Fibrillation	Amp	Duration	PP	Recruitment
Left FDI	↑	+1	-	Normal	Normal	+1	↓
Right FDI	↑	+1	-	Normal	Normal	+1	↓
Right TA	↑	-	-	Normal	Normal	+1	↓
Left quadriceps	Normal	-	-	Normal	Normal	Normal	Normal
Left EDB	↑	-	-	+1	+1	+1	↓

IA: Insertional activity; PSW: Positive sharp wave; PP: Polyphasia; FDI: First dorsal interosseous; TA: Tibialis; EDB: Extensor digitorum brevis.

minimi muscles were prolonged to more than 5.5-6 ms while preserving their amplitudes. Partial conduction blocks in bilateral median nerves pathways in the forearms were obvious. Nerve conduction velocities were mildly decreased to about 44 ms except for the right median nerve, which was decreased to 34 ms. F-waves were absent (Table 1). In needle electromyography, the insertional activities in distal muscles of both upper and lower limbs were increased, and evidence of positive sharp waves was detected in bilateral first dorsal interossei muscles. Furthermore, the recruitment of distal muscles was decreased (Table 2). Based on the patient's history, physical examination, and electrophysiological findings, which favored a sensorimotor peripheral polyneuropathy with mostly demyelinating features involving both upper and lower limbs, the patient was diagnosed with AIDP. Thus, the patient was admitted to the neurology department for further evaluation and scheduled for receiving intravenous immunoglobulin (IVIG). During the patient's hospital course, to rule out any structural causes, a non-contrast magnetic resonance imaging of the whole spine was performed, which did not have evidence of transverse myelitis, spinal cord compression, or spinal stenosis. Moreover, the polymerase chain reaction test for SARS-CoV-2 was negative. A lumbar puncture was performed, and the evaluation of the cerebrospinal fluid showed clear fluid, elevated proteins (0.8 g/L; reference: 0.15-0.45 gm/L), white blood cells in the normal range (2/mm^3), and a glucose level of 75 mg/dL. Both the culture and the Gram staining of the fluid were negative.

The patient received IVIG (2 g/kg over five days) without any complications. Since the patient remained stable and could tolerate the IVIG, physical and occupational therapy was started after the last course of medication. Distal muscle strength of the lower limbs improved to 4/5, and the patient could walk independently two to three days after receiving the last session of the rehabilitation at the hospital.

DISCUSSION

Guillain-Barre syndrome is a rare immune-mediated and inflammatory disease involving the peripheral nerves and spinal roots.[7-9] Patients with GBS usually present with acute symmetrical weakness and sensory symptoms beginning from the distal parts to achieve the maximal severity of the disease within two to four weeks.[5,6,8] Acute inflammatory demyelinating polyradiculoneuropathy is the most pathophysiologically prevalent subtype of GBS.[8,9]

Infections caused by some pathogens such as Campylobacter jejuni, Mycoplasma pneumonia, Cytomegalovirus, and Haemophilus influenzae have been reported immunological triggers for GBS.[9] Coronavirus disease 2019 is a novel infection that most commonly manifests in the lower respiratory tract. Moreover, some different neurological complications involving central and peripheral nervous systems have been reported in infected patients with the virus.[10] Guillain-Barre syndrome is an example of neurological manifestations which occur during or after severe acute respiratory symptoms of COVID-19. Recently, the incidence of this complication seems to have increased due to the COVID-19 pandemic. A few patients who developed GBS shortly after receiving vaccinations were also reported in the literature.[5,7] The estimated rate of vaccine-related GBS against Influenza A virus (H1N1) was 1.6 cases per 1,000,000 vaccine recipients in the study by Salmon et al.[11] Today, since the COVID-19 vaccination program advances worldwide, vaccine-related GBS cases are also increasingly reported. This applies to both types of vaccinations, including messenger ribonucleic

acid and adenovirus-vectored COVID-19 vaccines.[5] The first case of GBS after the initial dose of the Pfizer-BioNTech vaccine against COVID-19 infection was reported by Waheed et al.[12] We reported the case of a 68-year-old woman who developed GBS a few days after getting the second dose of the AstraZeneca vaccine. The Oxford/AstraZeneca ChAdOx1 SARS-CoV-2 vaccine comprises a recombinant carrier of the chimpanzee adenovirus (ChAdOx1), including deoxyribonucleic acid, which encodes a kind of spike protein of SARS-CoV-2.[13] This protein induces an immune response in humans. Studies showed that this kind of vaccine could prevent 70% of COVID-19 cases.[7] The most commonly reported complications from clinical studies of this vaccine were tenderness at the injection site, chills, fever, fatigue, joint and muscle pain, nausea, and headache.[7] Reported cases of anaphylaxis, lymphadenopathies, anorexia, and abdominal pain were significantly rare.[7] Recent concerns about the pulmonary, abdominal, and cranial venous sinus thrombosis concomitant with thrombocytopenia after the first exposure to this vaccine have increased. Thus far, a few cases of GBS due to the first dose of AstraZeneca have been reported.[5,7,13] Although most of the local and systemic complications of this type of vaccine were reported after receiving the first dose, our patient developed GBS just a few days after receiving the second dose. Not only had she received the first dose of the same type of vaccine almost three months ago without any complications, but she also denied any signs or symptoms of recent respiratory or gastrointestinal illnesses or exposure to infected patients. The pathogenesis of vaccine-related GBS is not clear. However, it has been suggested that contaminating components of the vaccine, particularly proteins, may increase anti-ganglioside antibody production and induce this immune-mediated process.[14] No vaccine is completely effective or without any side effects.[7] Since vaccines prevent severe diseases and death in most individuals, we can claim that vaccination has more benefits than side effects. Nevertheless, the safety of vaccines against the COVID-19 infection needs to be meticulously evaluated, and essential complications should be immediately reported to reduce the occurrence of these complications in the future, if possible.

In conclusion, we reported a case of Guillain-Barre syndrome as a severe complication of COVID-19 vaccine in order to reduce its occurrence in the future.

Patient Consent for Publication: A written informed consent was obtained from patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed to the conception and design, acquisition and analysis of the data, and preparation of the manuscript and drafting the article.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

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