

Guillain-Barré Syndrome After Receiving the First Dose of Oxford–AstraZeneca SARS-CoV-2 Vaccine

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Received: 02 Dec. 2021; Accepted: 24 May 2022

Abstract- Safety monitoring of COVID-19 vaccination is paramount of importance. There are limited reports of Guillain-Barré syndrome (GBS) associated with the COVID-19 vaccination. The present study reported a case of GBS following the first dose of the Oxford-AstraZeneca SARS-CoV-2 vaccine. A 32-year-old man presented a history of progressive descending weakness and autonomic features within a month after receiving the Oxford-AstraZeneca SARS-CoV-2 vaccine. The neurological examination was consistent with acute polyneuropathy. The para-clinical investigations were in favor of acute demyelinating polyneuropathy. The patient was diagnosed with GBS, and IVIG was initiated as an acute treatment, which led to significant clinical recovery. We reported a case of GBS after receiving the Oxford-AstraZeneca vaccine. However, our findings dose not conclude a causal association between GBS and COVID-19 vaccination.

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Acta Med Iran 2022;60(6):384-386.

Keywords: Coronavirus disease 2019 (COVID-19); Vaccine; Oxford-AstraZeneca vaccine; Guillain-barré syndrome (GBS)

Introduction

Since the emergence of COVID-19 disease, a number of neurological manifestations have been reported with COVID-19 ranging from a mild headache to severe involvement of the peripheral and central nervous system. However, the neurological complications of COVID-19 vaccine immunization are just beginning to disclose (1,2).

Safety monitoring of vaccination is always an essential priority. On the other hand, temporal bias might confound the correct interpretation of the causal association between adverse events and vaccination. As regards the reports of COVID-19 vaccine adverse events in the Centers for Disease Control (CDC) 's Vaccine Adverse Event Reporting System (VAERS), few reports of Guillain-Barre Syndrome (GBS) in association with mRNA COVID-19 vaccine are in the literature.

Herein, we reported a case of GBS in association with the first dose of the Oxford-AstraZeneca SARS-CoV-2 vaccine.

Case Report

Clinical presentation

The patient was a 32-year-old man who was admitted to our emergency department due to a two-day history of symmetric distal paresis of the upper limbs. His medical history was negative except for self-limited diarrhea just a day before arrival which was related to food poisoning, and the history of his first dose of Oxford-AstraZeneca vaccination 28 days ago.

On examination, the patient was alert and oriented. He was hemodynamically stable, and no significant finding was detected in his systemic examinations. The neurological examination was notable for distal upper limb paresis (4/5) regarding the medical research council (MRC) score accompanied by normal deep tendon reflexes (DTR). No sensory abnormality was found. The patient was ambulatory, and no limb ataxia was observed.

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Investigation

The routine para-clinical investigations, including electrocardiogram, blood chemistry, C-reactive protein, erythrocyte sedimentation rate, chest computed tomography, and brain and cervical magnetic resonance imaging (MRI), were all unremarkable.

On day two, the patient developed progressive descending quadri-paresis and quadri-paresthesia. His neurologic reevaluation revealed flaccid quadri-paresis (3/5 according to the MRC scale) in both proximal and distal extremities, as he was unable to walk without aid. The sensory examination was notable for decreased pinprick and vibration sensations in distal extremities.

With a clinical impression of acute demyelinating polyneuropathy, the patient was admitted to the intensive care unit (ICU). He underwent a lumbar puncture showing an opening cerebrospinal fluid (CSF) pressure of 12 CmH₂O, normal glucose and cell, and elevated protein (220 mg/dL). No evidence of infection was found in the CSF study.

Subsequently, a nerve conduction study (NCS) was performed, which revealed a significant reduction in the compound motor action potentials (CMAP) with prolonged distal latency and reduced conduction velocity of tibial nerves in a range of demyelinating process. The peroneal CMAP was absent bilaterally. The tibial F and H waves were also absent. Moreover, the sensory nerves were all spare. Electromyography (EMG) showed reduced recruitment in lower limbs without evidence of spontaneous activity, which all confirmed the diagnosis of acute, predominantly demyelinating polyradiculoneuropathy.

Treatment

Treatment started with 20 g IVIG intravenously daily for 7 d. On day 4, the patient developed autonomic features such as tachycardia and blood pressure instability, which was well controlled by labetalol infusion and losartan 25 mg twice a day. Good clinical recovery was achieved. The patient was then referred to a rehabilitation facility to complete treatment. His last examination after three months was notable for a muscle force of 4/5 regarding the MRC score.

Discussion

GBS is an acute inflammatory polyradiculoneuropathy that mainly occurs following recent infection, particularly upper respiratory tract infection, trauma, surgery, or vaccination. It accounts for

an annual incidence of 0.4-4.0 cases per 100 000 populations of all ages. While the exact pathophysiology of GBS is poorly understood, it seems to be related to an underlying immune-mediated process (3).

Several studies have highlighted a slightly increased risk of GBS following seasonal and 2009 H1N1 monovalent influenza vaccines, which was significantly lower than the possibility of developing influenza infection (4). During the period of the COVID-19 pandemic, reports of GBS in the context of COVID-19 (either in the course of the disease or as a para-infectious immune disorder) have brought to mind a causal association between COVID-19 and GBS with a maximum rate of 4.7 cases per 100 000 COVID-19 infection. However, there is currently no conclusive evidence favoring a significant increase in the risk of developing GBS after COVID-19 (5). Notably, we recently described four cases who developed GBS following a mild to moderate COVID-19 infection (6).

It is noteworthy that as global immunization is the crucial approach to controlling the current COVID-19 pandemic, attempts to determine the epidemiological extent of infrequent complications of the COVID-19 vaccines are of particular importance.

Sadia Waheed et al., reported the first case of GBS following recent exposure to the COVID-19 vaccine. They reported an 82-year-old female with a history of acute progressive flaccid quadriplegia following recent exposure to the first dose of the Pfizer COVID-19 vaccine. Her neurological examination was in favor of acute symmetric polyneuropathy. The CSF analysis revealed an albuminocytologic dissociation (protein of 88 and WBC of 4). She was diagnosed with GBS. Acute treatment with a 5-day course of IVIG led to complete recovery (7).

Later, *Osakpolor Ogbebor et al.*, reported an 85 year-old woman presenting with a 6-day history of progressive lower limb weakness with muscle strength of 4/5. She reported the onset of symptoms a day after receiving the Pfizer COVID-19 vaccine. The CSF demonstrated albuminocytologic dissociation. With a diagnosis of GBS, a 5-day course of IVIG was administered resulted in significant recovery (8).

In our patient, GBS symptoms developed within a month after receiving the first Oxford-AstraZeneca vaccine. The patient also mentioned diarrhea the day before admission. However, a short interval between diarrhea and the onset of symptoms did not explain the role of diarrhea in the development of GBS.

While monitoring for GBS following COVID-19 vaccination is important, consideration of the possibility

of temporal bias before establishing a contributory relationship between the vaccine and GBS is also fundamental. Interestingly, there is a trial that reported an occurrence of GBS in two patients who were classified in both the vaccine arm and placebo arm. Such findings support the argument for a coincidental rather than a causal association (9,10).

Taking all considerations into account, although our data did not confirm a causal relationship between the COVID-19 vaccine and GBS development, it highlights the need to monitor the rare adverse events of the COVID vaccine.

Herein, we reported the first case of GBS associated with the Oxford-AstraZeneca vaccine. However, our findings do not conclude a causal association between GBS and vaccination.

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