

The First Ethiopian Case Report of Post-Covid-19 Vaccine Associated Guillain-Barre Syndrome

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Received Date: 06 August 2022; **Accepted Date:** 20 August 2022; **Published date:** 21 September 2022

Citation: Z. Demissie MD, E. Tesfaye MD, A. Bitew MD, A. Worku MD, T. Haile MD (2022). The First Ethiopian Case Report of Post-COVID-19 Vaccine Associated Guillain-Barre Syndrome. *Neurons and Neurological Disorders*. 1(1); DOI: 10.58489/2836-8851/002

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Abstract

We report the first Ethiopian case of a rare neurologic complication, Guillain-Barre Syndrome (GBS), following Ad26.COV2. S (Janssen/Johnson & Johnson) COVID-19 vaccination. Although a temporal relationship is identified, a causal relationship between the Janssen/Johnson & Johnson COVID-19 vaccine and GBS is not established. Clinician awareness of this association is essential to early GBS diagnosis and treatment. Further, global data collection is important to ascertain the true magnitude and possible causality of post-COVID-19 vaccine associated GBS.

Keywords: covid-19 vaccine, neurologic

Introduction

Nearly two years have passed since the onset of the COVID-19 pandemic, first identified as the cause of a cluster of pneumonia cases in Wuhan, a city in the Hubei Province of China [1]. Since then, several preventive and therapeutic interventions have been developed including highly effective vaccines. Even though the vaccines have brought significant control of COVID-19 community spread and reduced disease severity [2,3,4,5], some rare adverse effects have been identified including thrombosis with thrombocytopenia, myocarditis, and Guillain-Barre Syndrome (GBS). [6,7,8].

GBS, a rare cause of acute onset polyneuropathy, has been reported as a safety concern following COVID-19 infection and COVID-19 vaccines. Post-release data suggested a possible increased risk of GBS during the first 6 weeks after Ad26.COV2. S (Janssen/Johnson & Johnson) vaccination, and led the U.S. Food and Drug Administration (FDA) to place a specific warning on the vaccine.

We report the first Ethiopian case of a previously healthy middle-aged adult man who developed GBS 4 weeks after receiving the Janssen/Johnson & Johnson COVID-19 vaccine.

Case Presentation

A 46-year-old man presented to the Emergency Department (ED) of Tikur Anbessa Specialized Hospital in Addis Ababa, Ethiopia after he developed bilateral lower extremities weakness of two hours, which progressed, within 24 hours, to include bilateral upper extremity, associated with paresthesia. He denied urinary or fecal incontinence, fever, cough, upper respiratory tract symptoms, diarrhea or a prior history of trauma or neurologic disease.

On arrival to the ED, the patient was conscious and not in distress. His blood pressure was 150/100mmHg, pulse rate was 100/min, respiratory rate was 18/min, and oxygen saturation was 94% on room air. His physical examination was normal except for respiratory and neurologic systems. His vital capacity was 2.8 liters, which was 64% of the predicted value. His motor strength was 1/5 in both upper and lower limbs, proximally and distally. All extremities were hypotonic. Deep tendon reflexes were absent in the ankles and 1/4 in both knees. Cranial nerves and sensory response in both upper and lower extremities were intact; he had negative meningeal signs. His Glasgow Coma Scale was 15/15 (E4V5M6).

Neurons and Neurological Disorders

The patient was transferred to the intensive care unit (ICU) for further evaluation of possible GBS. Lumbar puncture was performed, and cerebrospinal fluid analysis revealed albuminocytologic dissociation with no cells, protein of 238mg/dl, and glucose 90mg/dl. Gram stain and culture of the fluid were negative. Viral markers including HIV, Hepatitis B, and C were negative. His complete cell count, renal and liver function, electrolytes, and coagulation tests were all normal. Electrocardiogram study and chest radiography were unremarkable along with a negative SARS-Cov-2 RT-PCR test.

Nerve conduction study showed diffuse severe motor axonal polyneuropathy. Specifically, absent F-waves on the left median and bilateral ulnar nerves, nonpersistent F-waves on his right tibial, and prolonged F-waves on both peroneal nerves.

His clinical and laboratory evaluation confirmed the diagnosis of GBS. He completed 5 doses of intravenous immunoglobulin (0.4gm/kg/day). Daily physiotherapy was initiated. His blood pressure was controlled by Amlodipine (10mg oral daily) and the pain was relieved by Gabapentin (200mg three times per day). Respiratory failure never ensued but motor weakness persisted until his discharge from hospital, after he stayed 4weeks. Upon discharge, his muscle strength was 1/5 and hypotonia in all extremities.

Discussion

GBS is a disease of peripheral nerves that causes acute onset of ascending type, flaccid paralysis, usually starts from lower extremities. But in about 10% of patients, it begins on the face or upper extremities. [9] The estimated worldwide annual incidence is 1–2 cases per 100,000 population. Several variants of GBS have been identified, including acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy, acute motor-sensory axonal neuropathy, and Miller Fisher syndrome.10 GBS is an autoantibody-mediated process that is triggered by molecular mimicry.[10] GBS can follow infections or vaccinations. The risk of GBS after infection is substantially greater than after vaccines.[11] Antecedent infections with respiratory pathogens or *Campylobacter jejuni* occur in more than two-thirds of GBS patients. [12,13] GBS has been documented following vaccinations for rabies, hepatitis A and B, polio, and influenza.[14]

GBS in individuals receiving the Jansen/Johnson & Johnson COVID-19 vaccines have also been reported. [8,15,16] As in our patient, a temporal relationship has been identified between the timing of

vaccination and onset of symptoms of GBS, but a causal relationship has not been clearly identified. In the largest study by Woo et al, 130 individuals developed GBS after nearly 13.2 million Janssen/Johnson & Johnson vaccinations. In their study, the median age was 56 years old and the median time to the onset of GBS following vaccination was 13 days.[8] Our patient was younger and had a later onset of symptoms after vaccination.

Conclusions

GBS is an uncommon adverse effect of the Janssen/Johnson & Johnson COVID-19 vaccine. Clinicians need to be aware of this association to allow early GBS diagnosis and treatment. Further, global data collection is important to ascertain the true magnitude and possible causality of post-COVID-19 vaccine associated GBS.

Availability of data and materials

The datasets used in this case report are available from the corresponding author on responsible requests.

Funding Statement

This case report didn't receive any grant from funding governmental or non-governmental organizations.

Author contribution

ET did a literature review. ZD wrote the original draft of the manuscript. TH, AW, and AB revised the original manuscript and supervised the overall writing process.

Research registration

Non-applicable.

Declaration of competing interest

None to be declared.

Acknowledgments

We thank the Neurology Department of Tikur Anbessa Specialized Hospital for performing the nerve conduction studies.

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