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Case Report

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The Development of Guillain Barre Syndrome Subsequent to Administration of Ad26.COV2.S Vaccine

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ABSTRACT

Guillain Barre Syndrome (GBS) is a rare neurologic disorder in which the immune system mistakenly attacks the peripheral nervous system due to molecular mimicry. GBS symptoms can range from a mild episode of weakness to devastating paralysis and respiratory failure. The exact cause of GBS is unknown, but it is often thought to be due to a preceding viral infection or rarely due to vaccination. To date, there has only been one reported case of Guillain Barre Syndrome associated with the administration of Ad26.COV2.S vaccine (Janssen/Johnson & Johnson COVID-19 vaccine) [3]. Here, we describe the case of a 59-year-old woman who received the Johnson & Johnson COVID-19 vaccine and subsequently developed symptoms consistent with GBS. Unfortunately, due to failure to obtain lumbar puncture (LP) and electromyography (EMG), it is only possible to diagnose Guillain Barre syndrome with Level 3 diagnostic certainty using the Brighton criteria [6]. We are of the opinion that our patient developed GBS subsequent to the vaccination, but not necessarily consequent to the vaccination, as it remains possible that she may have contracted an asymptomatic infection prior to inoculation.

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Introduction

The Ad26.COV2.S (Janssen/Johnson & Johnson COVID-19 vaccine) has been reauthorized for use in the United States following concerns of thrombosis with thrombocytopenia syndrome [1, 2]. To the present date, there is only one reported case of Guillain Barre Syndrome (GBS) associated with administration of the Ad26.COV2.S vaccine [3]. This case was in the treatment cohort of the Ad26.COV2.S clinical trial, and another parallel case of GBS occured in the control group of that same trial. With a relative risk of 1 for this adverse event during the trial, and population wide vaccination efforts, it is estimated that for every billion vaccinated patients, roughly 708 incidental cases of GBS syndrome may occur within two weeks of vaccination [4].

Here, we report the case of a middle-aged woman whose complex clinical course and illness trajectory reflects a case of GBS that is subsequent to vaccination with the Ad26.COV2.S vaccine.

Case

A 59-year-old Spanish speaking woman with a history of hypertension, gastroesophageal reflux disease, bipolar disorder, hyperlipidemia, and non-insulin dependent diabetes mellitus was in her usual state of health when she received the Johnson & Johnson COVID-19 vaccine on April 12th, 2021. At baseline, she was independent in all activities of daily living and ambulatory [5]. Two weeks later, she presented to a neighboring emergency department due to difficulty walking, right facial droop, and slurred speech. She was diagnosed with Bell's palsy and discharged, without further intervention or imaging.

The patient continued to experience difficulty ambulating due to worsening lower extremity weakness, and experienced new and increasing weakness to her proximal bilateral upper extremity. At its worst, she endorsed being unable to brush her hair.

She stayed in bed for several days. Two days prior to her arrival at our hospital, she noticed painless left lower extremity swelling. The night of admission, she awoke at approximately 2 am with diffuse low back pain. After being assisted to the restroom, she sustained an episode of dark, red, and bloody loose stool. After five repeat episodes, the patient experienced fatigue, dyspnea, and diaphoresis. EMS brought her to our hospital.

On admission, vital signs were: oral temperature of 34.9°C, heart rate 75 beats per minute, respiratory rate 19 cycles per minute, blood pressure 87/47 mmHg, and oxygen saturation 100% on room air. Her body mass index (BMI) was 26.79 kg/m2. Physical exam demonstrated right sided facial droop with no sparing of the forehead, decreased left forehead wrinkles, slurred speech, weakness and reduced sensation to the bilateral lower extremities, diminished deep tendon reflexes to the lower extremity, distended bladder, and decreased rectal tone.

Initial hematology studies demonstrated a total white blood cell count of 11,500/uL, red blood cell count of 2.12 M/uL, hemoglobin of 6.2 g/dL, hematocrit of 19.1%, platelet count of 118,000/uL.

Arterial blood gas and initial chemistries demonstrated non-anion gap metabolic acidosis with a pH of 7.1, potassium of 8.0, blood urea nitrogen (BUN) of 204 mg/dL, and serum creatinine of 5.81 mg/dL.

Bladder scan demonstrated > 2L of retained urine.

She was admitted to our intensive care unit on May 13th, 2021 for gastrointestinal hemorrhage secondary to uremic gastropathy, due to obstructive uropathy.

She received urgent blood transfusion and was placed on a Protonix drip. A permacatheter was placed for urgent hemodialysis, as well as a foley catheter.

A non-contrast MRI of the brain failed to demonstrate any acute intracranial abnormality. However, an MRI of the thoracic and lumbar spine showed an abnormal T2 bright signal within the left psoas muscle and bilateral erector spinae of the lumbar spine (Figure 1). This finding was interpreted as reflecting a possible myopathic condition such as hyperCKemia or myositis.

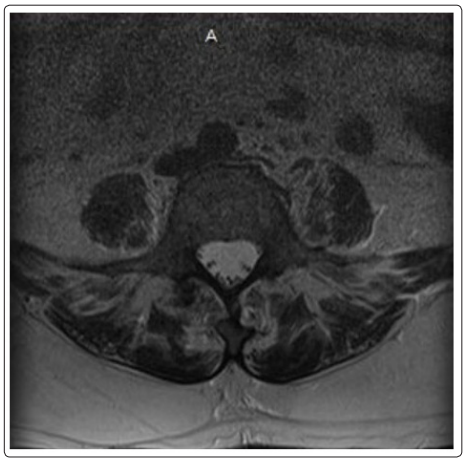

Figure 1: MRI Lumbar Spine demonstrating abnormal T2 bright signal within the left psoas muscle and bilateral erector spinae

Nonetheless, her initial creatine kinase was 653 iU/L on admission. The remainder of her rheumatologic panel remained noncontributory as shown in Table 1.

Table 1: Rheumatological Panel

Lab	Result	Reference Range
Rheumatoid Factor	<14 IU/mL	<14 IU/mL
Antinuclear Antibody	Negative	Negative
Antineutrophil Cytoplasmic Antibody	Myeloperoxidase Ab <1.0 AI Negative, Proteinase-3 Ab <1.0 AI Negative	<1.0 AI
Anti-Jo 1	<1.0 AI	<1.0 AI
Complement Component C3	97 mg/dL	83-193 mg/dL
Complement Component C4	25 mg/dL,	15-57 mg/dL
Anti- DNAse B Antibody	153 U/mL,	<301 U/mL
Glomerular Basement Membrane Antibody	<1.0 AI	<1.0 AI
Anti-Mitochondrial M2 antibody	<20.0 AI	<20.0 AI
Erythrocyte Sedimentation Rate	19 mm/h,	0-30 mm/hr

An ultrasound of the left lower leg showed acute femoral and popliteal deep vein thrombosis (DVT). She received an inferior vena cava (IVC) filter as the risk of heparinization exceeded the possible benefit, given her recent gastrointestinal hemorrhage.

On May 14th, the patient's laboratory values had stabilized and she was downgraded from the ICU. Her dialysis access was removed on May 18th as she no longer required dialysis. She continued to experience urinary retention during voiding trials. Her muscular strength mildly improved throughout her hospital course, without any targeted intervention such as corticosteroids, IVIg, or plasmapheresis.

She was ultimately discharged, with Foley in place, to sub-acute rehabilitation on May 21st with plans for outpatient rheumatologic, neurologic, and urologic follow up.

Electromyography (EMG) and lumbar puncture (LP) were not obtained given the patient's clinical improvement, edematous lower extremity, and concerns of artifact from subclinical diabetic neuropathy.

Discussion

Unfortunately, due to failure to obtain LP and EMG, it is only possible to diagnose Guillain Barre syndrome with Level 3 diagnostic certainty using the Brighton criteria [6]. Evidence in support of our largely clinical diagnosis are laboratory results which exclude a myopathic or rheumatological etiology, and a history highly concerning for GBS [7]. We are of the opinion that our patient developed GBS subsequent to the vaccination, but not necessarily consequent to the vaccination, as it remains possible that she may have contracted an asymptomatic infection prior to inoculation. The composite of her physical exam, laboratory studies and imaging point away from an active infection as a cause of her GBS, as well as an alternative diagnosis, such as a myopathic illness.

The only remarkable piece of her history was the administration of the Ad26.COV2.S vaccine two weeks prior to symptom onset. Anecdotally, GBS can occur a few weeks post vaccination, though this risk is likely overstated [8]. In addition, adenovirus vector based vaccines have a robust safety and efficacy profile. Prior to the introduction of Ad26.COV2.S, and as of September 2020, 114,000 individuals have received adenovirus vector based vaccines, mostly for tropical viral illnesses such as ebola, filovirus, and zika. Importantly, every received Ad26-based vaccine was well tolerated, and there were no significant safety issues which could be found from the data that was available in the AdVac safety database a little over a year ago, prior to the introduction of the Ad26.COV2.S vaccine [9].

As the Ad26.COV2.S vaccine is reintroduced for widespread inoculation efforts, incidental cases of GBS are expected to occur [4]. Only with ongoing vigilant reporting, can a meaningful association between Ad26.COV2.S vaccine and Guillain Barre syndrome be established. This case represents the first of such a report for receiving the Ad26.COV2.S vaccine and the development of Guillain Barre syndrome.

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