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CASE REPORT

WILEY

A case of chronic inflammatory demyelinating polyneuropathy following COVID-19 vaccine

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Abstract

We report a 44-year-old woman who presented with bilateral weakness of the hands and distal paresthesia of the arms on the next day of the second COVID-19 vaccine, and gradually progressed ascending weakness of the arms and legs, and sensory ataxia beyond 2 months. She was diagnosed as a chronic inflammatory demyelinating polyneuropathy (CIDP) following COVID-19 vaccine on the basis of clinical and electrophysiological findings. This is a first case diagnosed as a CIDP following COVID-19 vaccine alone.

KEYWORDS
chronic inflammatory demyelinating polyneuropathy, COVID-19 vaccine, Guillain–Barrė syndrome

1 | INTRODUCTION

Recently, the number of case reports regarding to Guillain–Barrė syndrome (GBS) after Coronavirus disease 2019 (COVID-19) infection or following COVID-19 vaccine has been increasing.[1] Here, we described the first case of chronic inflammatory demyelinating polyneuropathy (CIDP) following COVID-19 vaccine alone. Differentiating CIDP from GBS is crucial at the initial clinical presentation, because there have been different treatment and outcomes.

2 | CASE PRESENTATION

A 44-year-old woman, with a medical history of dysmenorrhea, insomnia, palmoplantar pustulosis, operation to umbilical hernia, and allergy to some food (Pacific mackerel) had a local swelling and pain on left upper arm of the intramuscular injection area with a high fever on the next day of the second Pfizer/BioNTech BNT162b2 COVID-19 vaccination. At the same time, she felt a difficulty in turning the faucet with ascending bilateral (right-side dominantly) weakness of the arms and distal paresthesia. Her neurological symptoms gradually progressed for 4 weeks, she dropped somethings in grasping them by her right hand and could not wash her hair by herself. She visited home doctor 10th week later from the onset of her neurological symptoms and the doctor recognized that her symptoms progressed over 8 weeks, and consulted our hospital considering of neuropathy. At the 15th weeks later from the onset, her neurological examination showed weakness of the upper [Medical Research Council (MRC), 4/5] and lower extremities (MRC, 4/5), numbness in the distal extremities, the absence of deep tendon reflexes of four extremities. She had some distal paresthesia of four extremities, decreased light touch sensation and proprioception in the lower limbs from the groin distally. There was the absence of cerebellar signs and no evidence of cranial nerve abnormality. She could walk without support, but tandem gait was impossible by the sensory ataxia (Figure 1A). Because her activities of daily living, Hughes grade was II and (INCAT) Overall Disability Sum Scale was 5 [4 (0–5) on the upper limbs and 1(0–5) on the lower limbs],[2] we progressed minute investigation. Nerve conduction study (NCS) was performed thereafter which showed marked demyelinating sensorimotor polyneuropathy of median, ulnar, and sural nerve and prolonged F wave latencies of median nerve and absent of ulnar nerve (Table 1).

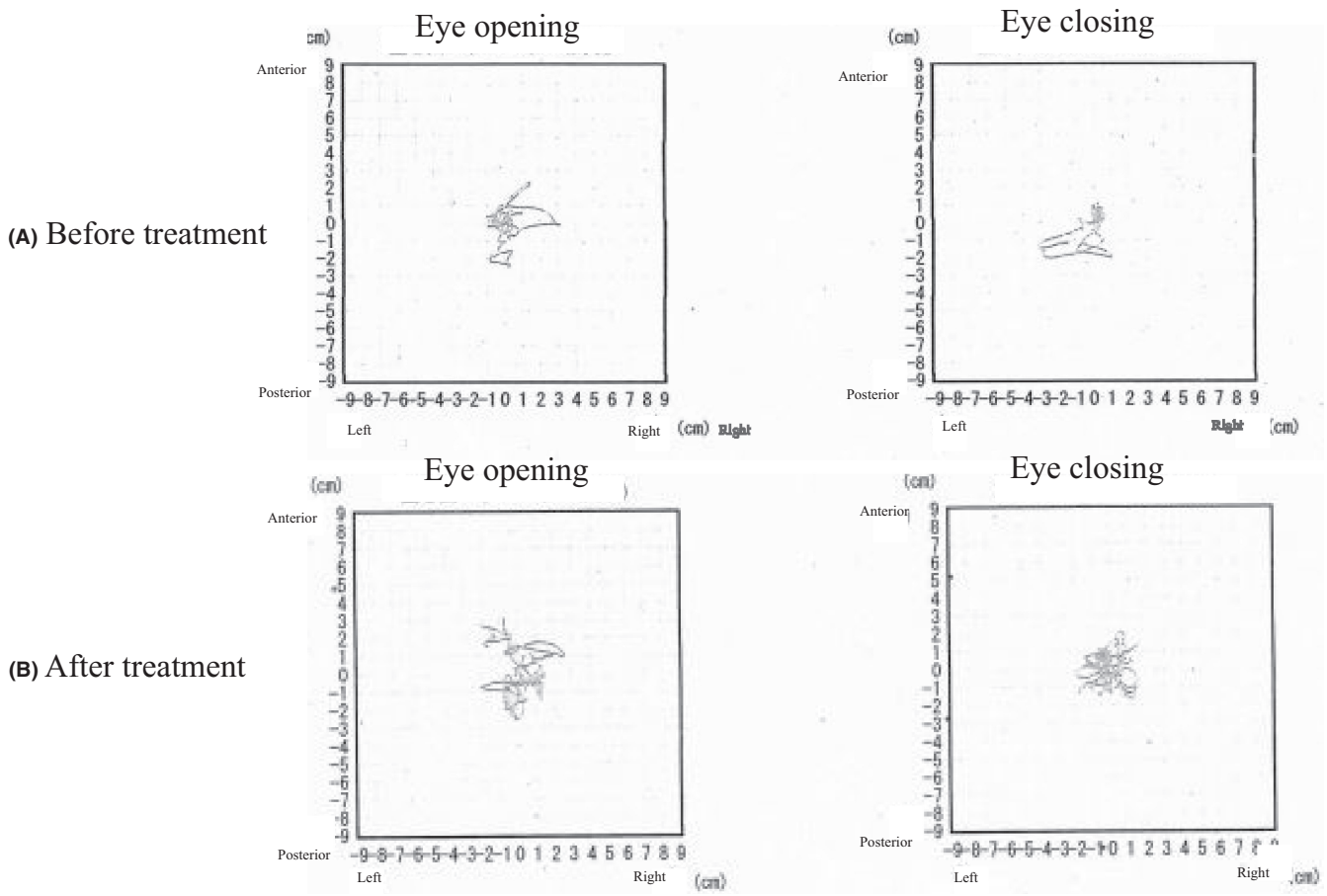

FIGURE 1 The stabilographs showed the unstable standing posture center position of the subject calculated for 1 min during both the opening and closing eyes before (A) and after (B) treatment

Magnetic resonance imaging of whole spinal cord did not show abnormality. Basic peripheral neuropathy screen including HbA1c, glucose level, vitamin B_{12} level, folate level, copper level, thyroid function tests, protein electrophoresis, paraprotein level, hepatitis screen, syphilis, and HIV were performed, and these results were negative. Antinuclear antibody and SS-A/SS-B antibody were negative. Antineutrophil cytoplasmic antibody as well as C3 and C4 levels were done as a part of vasculitis screening and results were within normal limit. Anti-ganglioside antibodies were negative. As she denied a recent history of infection, deterioration of palmoplantar pustulosis, and recent change of medication, we diagnosed her case as a CIDP following COVID-19 vaccination to fulfill the diagnostic criteria for CIDP.[3] At the 20th weeks from the onset, her neurological symptoms gradually deteriorated to the (INCAT) Overall Disability Sum Scale 6 [4 (0–5) on the upper limbs and 2 (0–5) on the lower limbs], she commenced on intravenous immunoglobulin (IVIG) at a dose of 0.4 g/kg body weight/day for 5 days. After 10 days, muscle weakness began to improve, but a 1-month follow-up NCS showed demyelinating pathology and sensory ataxia (Figure 1B) and albumin-cytologic dissociation (total protein count 129 mg/dL and one mono-nuclear cell) with examination of cerebrospinal fluid. The second IVIG was performed at the 28th weeks from the onset, and maintaining therapy of IVIG was needed because of the fluctuation of her neurological symptoms.

3 | DISCUSSION

The large population-based study revealed that the risk of neurological complications from SARS-CoV-2 infection was substantially higher than the risk of adverse events from vaccinations and that there was an increased risk of GBS and Bell`s palsy with ChAdOx1nCoV-19 and an increased risk of hemorrhagic stroke with BNT162b2.[4] CIDP is characterized by an insidious onset showing slow and progressive course, but may present acutely in up to 13% of patients, who rapidly progress within 4 weeks and initially may be diagnosed with GBS.[3] The first case of acute-onset CIDP in association with COVID-19 disease and its vaccination was reported.[5] To our knowledge, our case diagnosed as CIDP following COVID-19 vaccine alone is first described. This case might indicate the possible causal relationship between COVID-19 vaccination and CIDP. However, because CIDP may develop by chance, the causal relationship between two events should be considered with caution in the same manner as GBS.[6]

TABLE 1 The results of motor and sensory nerve conduction studies and F waves

Motor nerve conduction study						
Site	**Latency (ms)**	**Normal range (ms)**	**CMAP amplitude at distal site (mV)**	**Normal range (mV)**	**MCV (m/s)**	**Normal range (m/s)**
Left median nerve	6.5	3.5 ±0.3	7.3	6.9 ±3.2	33.7	57.7 ±4.9
Left ulnar nerve	5.3	2.6 ±0.4	4.9	5.7 ±2.0	34.4	58.7 ±5.1
Left peroneal nerve	15.9	3.8 ±0.9	2.6	5.1 ±2.3	43.8	48.3 ±3.6
Left tibial nerve	18.3	3.9 ±1.0	7.3	5.8 ±1.9	43.0	48.5 ±3.6
Sensory nerve conduction study						
Site	**Latency (ms)**	**Normal range (ms)**	**SNAP amplitude at distal site (μV)**	**Normal range (μV)**	**SCV (m/s)**	**Normal range (m/s)**
Right median nerve	3.7	2.8 ±0.3	6.0	38.5 ±15.6	38.8	56.2 ±5.8
Left ulnar nerve	4.0	2.5 ±0.3	7.0	35.0 ±14.7	34.3	54.8 ±5.3
Left sural nerve	2.7	2.8 ±0.3	17.7	17.2 ±6.7	55.6	51.1 ±5.9
F waves						
Site	**Latency (ms)**	**Normal range (ms)**	**Ocurrence (%)**	**Normal range (%)**	**Conduction velocity (m/s)**	**Normal range (m/s)**
Left median nerve	42.1	26.6 ±2.2	44	>40	41.3	65.3 ±4.7
Left ulnar nerve	Not evoked	27.6 ±2.2	0	>40	Not evoked	65.3 ±4.8
Left peroneal nerve	30.0	48.4 ±4.0	84	>40	43.8	49.8 ±3.6
Left tibial nerve	42.8	47.7 ±5.0	13	>40	55.5	52.6 ±4.3

Abbreviations: CMAP, compound muscle action potential; MCV, motor conduction velocity; SCV, sensory conduction velocity; SNAP, sensory nerve action potential.

CONFLICT OF INTEREST

The authors declare no conflict of interest for this article.

ETHICAL APPROVAL

This case report was conducted in accordance with the Declaration of Helsinki, and the identity of the patient has been protected.

CONSENT STATEMENT

Informed consent was obtained from the patient to publish these features of her case.

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