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Case Report
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Two Case Reports of Chronic Inflammatory Demyelinating Polyneuropathy After COVID-19 Vaccination

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ABSTRACT

The occurrence of chronic inflammatory demyelinating polyneuropathy (CIDP) related to coronavirus disease 2019 (COVID-19) has rarely been reported. We describe two patients who were diagnosed with CIDP after COVID-19 vaccination. A 72-year-old man presented with a progressive tingling sensation and weakness below both knees for two weeks. He had been vaccinated against COVID-19 (mRNA-1273 vaccine) a month before the appearance of symptoms. Demyelinating polyneuropathy was observed in the nerve conduction studies (NCS). Intravenous immunoglobulin (IVIg) was administered under the diagnosis of Guillain-Barré syndrome (GBS), and his symptoms were improved. However, his symptoms relapsed at 10 weeks from the onset. Oral prednisolone, azathioprine, and IVIg were administered as treatment. The second case was a 50-year-old man who complained of a bilateral leg tingling sensation and gait disturbance lasting four weeks. He had received the Ad26.COV2.S vaccine against COVID-19 five weeks prior. Demyelinating polyneuropathy was observed in the NCS. He was treated with oral prednisolone, azathioprine, and IVIg for CIDP because his symptoms had lasted for more than 12 weeks from the onset. A causal relationship has not been established between COVID-19 vaccination and CIDP; however, CIDP may follow COVID-19 vaccination. As CIDP treatment is different from that for GBS, clinicians should closely monitor patients diagnosed with GBS associated with COVID-19 whether they deteriorate after initial treatment.

Keywords: COVID-19 Vaccines; Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP); Peripheral Nervous System Diseases

INTRODUCTION

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a relapsing-remitting or progressive immune-mediated acquired neuropathy classified as an autoimmune disorder. CIDP is characterized by a slow and progressive course, with relapsing symptoms and gradual worsening over a period of more than eight weeks.[1] CIDP is diagnosed based on clinical features and neurological examination with the help of electrophysiological studies and other supportive findings (**Table 1**).[2] On the other hand, acute inflammatory demyelinating

Table 1. European Academy of Neurology/Peripheral Nerve Society guideline on diagnosis of CIDP

Clinical inclusion criteria for typical CIDP require both of the following:
Chronically progressive, stepwise, or recurrent symmetric proximal and distal weakness and sensory dysfunction of at least two limbs, developing over two months or longer; cranial nerves may be affected
Absent or reduced tendon reflexes in all extremities
Clinical inclusion criteria for CIDP variants require one of the following, but otherwise as in typical CIDP. However, tendon reflexes may be normal in unaffected limbs:
Predominantly distal (distal acquired demyelinating symmetric neuropathy) or
Asymmetric (multifocal acquired demyelinating sensory and motor neuropathy) or
Focal
Pure motor
Pure sensory
Clinical exclusion criteria:
Neuropathy probably caused by *Borrelia burgdorferi* infection (Lyme disease), diphtheria, drug or toxin exposure
Hereditary demyelinating neuropathy
Prominent sphincter disturbance
Diagnosis of multifocal motor neuropathy
IgM monoclonal gammopathy with high titer antibodies to myelin-associated glycoprotein
Other causes of demyelinating neuropathy include POEMS syndrome,[a] osteosclerotic myeloma, diabetic and non-diabetic lumbosacral radiculoplexus neuropathy, peripheral nervous system lymphoma and amyloidosis
Electrodiagnostic criteria for CIDP include:
Parameters to identify motor and sensory conduction abnormalities that suggest demyelination
Supportive criteria for CIDP:
Elevated CSF protein with leukocyte count < 10/mm^3 (albuminocytological dissociation)
MRI showing gadolinium enhancement and/or hypertrophy of the cauda equina, lumbosacral or cervical nerve roots, or the brachial or lumbosacral plexuses
Abnormal sensory electrophysiology in at least one nerve
Objective clinical improvement following immunomodulatory treatment
Nerve biopsy showing unequivocal evidence of demyelination and/or remyelination by electron microscopy or teased fiber analysis

CIDP = chronic inflammatory demyelinating polyneuropathy, CSF = cerebrospinal fluid, MRI = magnetic resonance imaging.
[a]POEMS syndrome, a paraneoplastic syndrome characterized by polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin abnormalities.

polyneuropathy, also called Guillain-Barré syndrome (GBS), is characterized by a monophasic course, with a clinical peak within four weeks of disease onset.[3]

Several immune-mediated neurological diseases have been reported in close temporal relationship with coronavirus disease 2019 (COVID-19) infection or following vaccination against COVID-19.[4] The immune-mediated neuropathies related to COVID-19 include GBS, recurrence of GBS, and exacerbation of pre-existing CIDP.[5] Although GBS was officially recognized as a complication of COVID-19 vaccines, the causal relationship between new occurrence of CIDP and vaccination against COVID-19 is not yet clear. However, as the pandemic continues, several cases related to these have been reported. In this report, we describe two patients diagnosed with CIDP after COVID-19 vaccination.

CASE DESCRIPTION

Case 1

A 72-year-old man presented with a progressive tingling sensation and weakness below both knees for two weeks. He was vaccinated against COVID-19 with the mRNA-1273 vaccine a month before. As the symptoms progressed, the patient could not walk independently and had difficulty using chopsticks. He had no underlying diseases or recent infection history. He had received the first and the second doses of the BNT162b2 vaccine, which caused no adverse effects. Neurological examinations identified distal dominant limb weakness, deficits of light touch and vibration sensation in the bilateral fingertips and below both knees, and areflexia in all four limbs. Blood tests for polyneuropathy evaluation including complete blood

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cell counts, liver function tests, renal function tests, serum electrophoresis, thyroid function tests, vitamin B12/folate levels, vitamin D levels, hepatitis marker, anti-Ro/La antibodies, anti-neutrophilic cytoplasmic antibodies, anti-myelin-associated glycoprotein antibody, anti-ganglioside antibodies, and urine tests including urinalysis and electrophoresis revealed no abnormalities. Cerebrospinal fluid (CSF) examination revealed albuminocytological dissociation (white blood cell [WBC] < 5 mm^3 [normal range < 5 mm^3]; total protein [TP] 72 mg/dL [normal range < 45 mg/dL]). Nerve conduction studies (NCS) revealed demyelinating polyneuropathy (**Tables 2** and **3**,[6] **Fig. 1A-D**). Limb weakness and sensory deficits improved after intravenous immunoglobulin (IVIg) treatment, and subsequent NCS showed improved demyelinating polyneuropathy (**Tables 2** and **3**,[6] **Fig. 1E-H**). Approximately ten weeks after the onset, he experienced worsened tingling sensation on the feet with gait disturbance, and the following NCS results were aggravated compared with the previous result (**Tables 2** and **3**,[6] **Fig. 1I-L**). Under the tentative diagnosis of acute onset chronic inflammatory demyelinating polyneuropathy (A-CIDP), oral prednisolone (30 mg/day) was started, and he felt improvement in the tingling sensation and gait disturbance. However, he complained of aggravated gait disturbance and sensory changes in both feet with the tapering of oral steroids (15 mg/day). Subsequent NCS showed aggravated demyelinating polyneuropathy (**Tables 2** and **3**,[6] **Fig. 1M-P**). IVIg and azathioprine were administered with oral prednisolone, which improved his symptoms, except for the mild gait disturbance. The clinical features of this case were recurrent symmetric distal limb weakness, sensory dysfunction, and areflexia at all limbs lasting more than eight weeks. Although GBS was considered as initial diagnosis, we finally considered chronic demyelinating polyneuropathy rather than treatment related fluctuation of GBS because the symptoms progressed for more than 8 weeks and recurred more than 3 times. In addition, other etiologies for chronic polyneuropathy including infection, inflammation, paraneoplastic, metabolic, and toxic causes were excluded, and the symptoms were improved by immune-modulating therapy. The case met the clinical criteria for typical CIDP according to the European Academy of Neurology/Peripheral Nerve Society (EAN/PNS) guideline. The NCS met the demyelinating criteria of motor and sensory nerves, and therefore we diagnosed him as having typical CIDP according to EAN/PNS guideline.

Table 2. Serial nerve conduction studies for case 1: motor nerve (height: 164 cm)

Motor nerve	2022.01.25			2022.03.02 (improved)			2022.03.31 (worsened)			2022.06.20 (worsened)			Normal values		
	Latency, ms	Amp, mV	NCV, m/s	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV
Median, right															
APB-Wr	4.7[a]	10.5		6.2[a]	12.8		5.1[a]	14.5		5.6[a]	9.7		3.6	> 5.0	
Wr-Eb		9.9	39[a]		11.8	39[a]		13.5	37[a]		8.7	36[a]		> 5.0	49.9
Eb-Ax		8.0	55[a]		10.1	50[a]		12.9	50[a]		7.9	55[a]		> 5.0	55.9
F-wave	NR[a]			39.2[a]			38.5[a]			29.8[a]			26.7		
Ulnar, right															
ADM-Wr	5.5[a]	2.1[a]		6.0[a]	9.4		5.7[a]	8.8		5.4[a]	6.8		2.5	> 5.0	
Wr-Eb		2.7[a]	28[a]		5.2	22[a]		3.9[a]	22[a]		2.5[a]	21[a]		> 5.0	50.6
Eb-Ax		3.7[a]	40[a]		5.2	38[a]		4.1[a]	30[a]		2.5[a]	47[a]		> 5.0	52.6
F-wave	NR[a]			NR[a]			54.9[*]			NR[a]			26.6		
Tibial, right/left															
AH-Ak	10.4[a]/8.4[a]	4.8[a]/3.8[a]		8.9[a]/9.1[a]	7.2/7.1		9.3[a]/10.9[a]	6.8/3.0[a]		9.9[a]/9.9[a]	2.2[a]/1.2[a]		5.1	> 5.0	
Ak-P		3.1[a]/4.7[a]	26[a]/29[a]		7.0/5.4	30[a]/30[a]		5.4/3.0[a]	25[a]/28[a]		2.6[a]/2.0[a]	22[a]/25[a]		> 5.0	40.6
F-wave	NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			46.8		
Fibular, right/left															
EDB-Ak	5.2[a]/5.1[a]	7.3/2.3[a]		6.6[a]/5.9[a]	8.5/7.2		7.0[a]/6.5[a]	8.5/6.4		7.2[a]/6.5[a]	1.0[a]/1.3[a]		4.78	> 3.0	
Ak-P		5.8/3.0[a]	23[a]/20[a]		6.0/4.4	24[a]/25[a]		4.3/3.4	21[a]/22[a]		0.3[a]/0.9[a]	17[a]/19[a]		> 3.0	41.8
F-wave	NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			46.2		

ADM = abductor digiti minimi, AH = abductor halluces, Ak = ankle, Amp = amplitude, APB = abductor pollicis brevis, Ax = axillar, Eb = elbow, EDB = extensor digitorum brevis, NCV = nerve conduction velocity, P = popliteal fossa, Wr = wrist, NR = not recorded.
[a]Indicates abnormal values.[6]

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Table 3. Serial nerve conduction studies for case 1: sensory nerve (height: 164 cm)

Sensory nerve	2022.01.25			2022.03.02 (improved)			2022.03.31 (worsened)			2022.06.20 (worsened)			Normal values		
	Latency, ms	Amp, μV	NCV, m/s	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV
Median, right															
Wr		16	36[a]		20	32[a]		15	33[a]		NR[a]			> 10.0	41.2
Eb		18	45[a]		23	47[a]		24	44[a]		NR[a]			> 10.0	49.3
Ax		53	65		20	59		85	55		72	50[a]		> 10.0	53.9
Ulnar, right															
Wr		7[a]	36[a]		11	31[a]		NR[a]			NR			> 8.0	39.2
Eb		7[a]	43[a]		12	33[a]		NR[a]			NR			> 10.0	47.4
Ax		19	48[a]		16	47[a]		15	46[a]		25	43[a]		> 10.0	48.1
Sural, right/left		16/13	38/39		16/19	39/39		18/18	34[a]/34[a]		9[a]/8[a]	33[a]/34[a]		> 6.0	34.6
H reflex, right/left	NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			NR[a]/NR[a]			28.7		

Ax = axillar, Eb = elbow, NCV = nerve conduction velocity, Wr = wrist, NR = not recorded.
[a]Indicates abnormal values.[6]

Case 2

A 50-year-old man received the first dose of the Ad26.COV2.S vaccine against COVID-19 five weeks prior and experienced a tingling sensation in both legs after a week and gait disturbance three weeks after that. He had hypertension and no recent history of infection. Neurological examinations identified vibration sensation deficits below the ankles, bilateral distal dominant motor weakness on the lower extremities, areflexia, and positive results in the Romberg test. Blood tests including complete blood cell counts, liver function tests, renal function tests, HbA1C, thyroid function tests, hepatitis marker, anti-human immunodeficiency virus antibodies, anti-ganglioside antibodies, antibodies for rheumatologic diseases, vitamin B12/folate levels, serum electrophoresis, tumor marker, paraneoplastic antibodies, and urine tests including urinalysis and electrophoresis revealed no abnormalities. Magnetic resonance imaging (MRI) of brain and whole spinal cord, and positron emission tomography-computed tomography showed no specific findings. Albuminocytological dissociation (WBC < 5 mm^3; TP 158 mg/dL) was observed in the CSF analysis, and motor demyelinating polyneuropathy was confirmed in the initial NCS (**Tables 4** and **5**,[6] **Fig. 2A-D**). We suspected a case of CIDP because of the progressive symptoms lasting more than four weeks. High-dose oral prednisolone (60 mg/day) was started according to Korean medical insurance guidelines. However, this led to severe tremors and palpitations without improvement. We reduced the dose of oral prednisolone to 15 mg/day and added azathioprine, which partially improved the patient's symptoms. He complained of persistent gait disturbance and worsened bilateral hand weakness, and serial NCS revealed aggravated demyelinating polyneuropathy 12 weeks after the onset (**Tables 4** and **5**,[6] **Fig. 2E-H**). IVIg was administered for two days along with oral prednisolone and azathioprine. His symptoms improved, the motor power and Romberg test results were normalized; and the last NCS showed improvement (**Tables 4** and **5**,[6] **Fig. 2I-L**). In this case, the patient had chronic progressive symmetric distal limb weakness, sensory deficits and areflexia in all limbs lasting more than eight weeks, and the results of motor NCS were compatible with demyelinating neuropathy according to EAN/PNS criteria. Although he complained of tingling sensation and sensory ataxia, the NCS of sensory nerves were within normal range. However, the results of the CSF analysis and the response to treatment (especially to corticosteroids) supported a diagnosis of possible typical CIDP. In addition, because no specific findings were observed on the lumbosacral spinal MRI, multiple lumbosacral radiculopathies were excluded as a diagnosis. And the demyelinating features were more clearly identified in NCS performed at the time of recurrence than in initial NCS, which led to the consideration of possible typical CIDP as a diagnosis.

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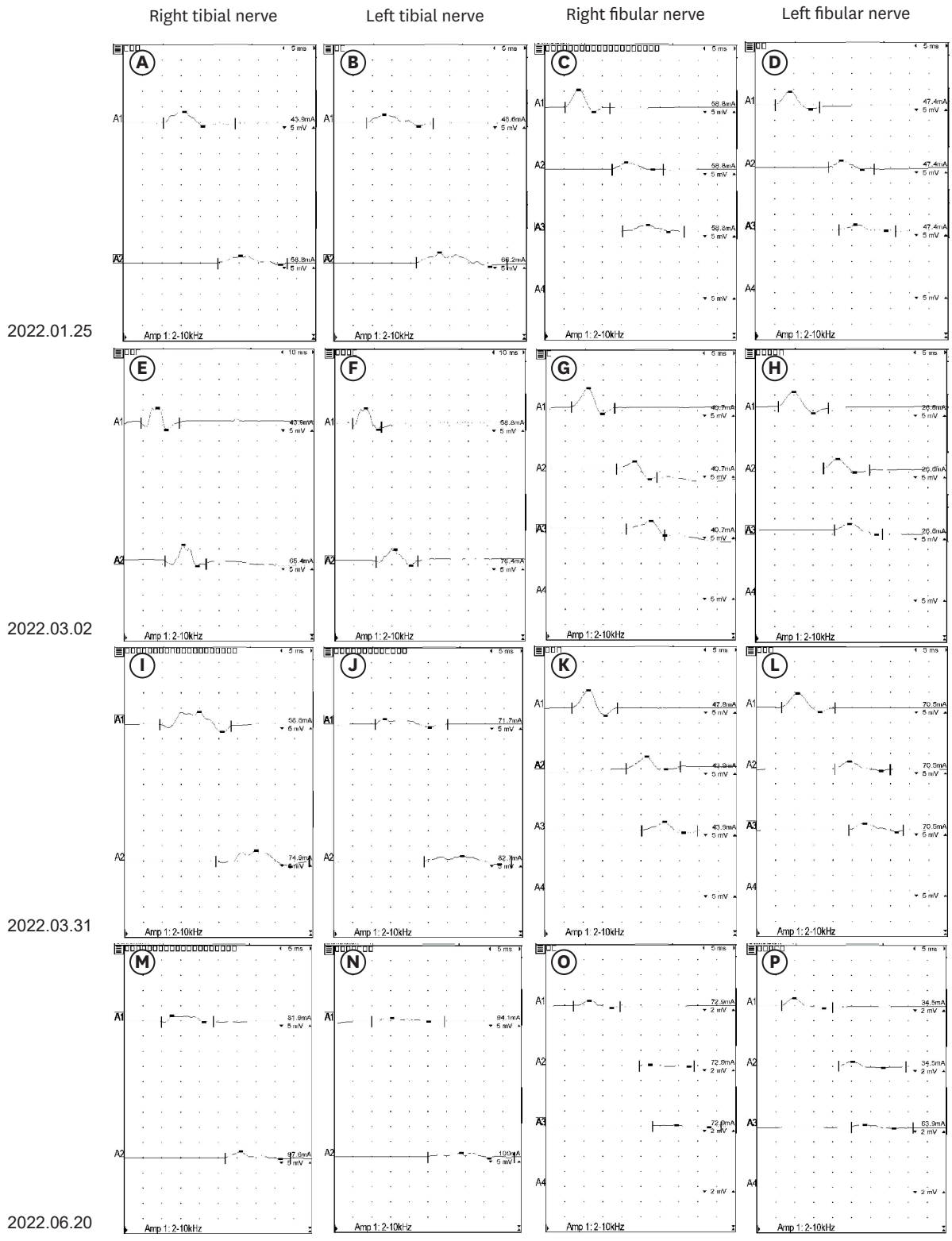

Fig. 1. Waveform of NCS of case 1. (**A**-**D**) Initial NCS of bilateral lower extremities: (**A**) right tibial nerve; (**B**) left tibial nerve; (**C**) right fibular nerve; (**D**) left fibular nerve. (**E**-**H**) Second NCS. The demyelinating polyneuropathy were improved after intravenous immunoglobulin administration: (**E**) right tibial nerve; (**F**) left tibial nerve; (**G**) right fibular nerve; (**H**) left fibular nerve. (**I**-**L**) Third NCS. The recurrence occurred: (**I**) right tibial nerve; (**J**) left tibial nerve; (**K**) right fibular nerve; (**L**) left fibular nerve. (**M**-**P**) Fourth NCS. The demyelinating polyneuropathy aggravated after tapering of oral prednisolone (30 → 15 mg): (**M**) right tibial nerve; (**N**) left tibial nerve; (**O**) right fibular nerve; (**P**) left fibular nerve.
NCS = nerve conduction studies.

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Table 4. Serial nerve conduction studies for case 2: motor nerve (height: 167 cm)

Motor nerve	2021.07.05			2021.08.25 (worsened)			2021.11.05 (improved)			Normal values		
	Latency, ms	Amp, mV	NCV, m/s	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV
Median, right												
APB-Wr	3.5	7.9		3.8[a]	5.2		3.7[a]	8.8		3.6	> 5.0	
Wr-Eb		7.5	51		4.9[a]	47[a]		7.5	52		> 5.0	49.9
Eb-Ax		7.3	59		4.2[a]	65		7.7	65		> 5.0	55.9
F-wave	26.7			32.5[a]			34.8[a]			26.7		
Ulnar, right												
ADM-Wr	2.5	9.1		3.0[a]	11.7		2.7[a]	12.6		2.5	> 5.0	
Wr-Eb		7.2	52		9.0	45[a]		11.1	53		> 5.0	50.6
Eb-Ax		6.4	77		7.5	62		9.2	73		> 5.0	52.6
F-wave	25.6			29.4[a]			29.4[a]			26.6		
Tibial, right/left												
AH-Ak	6.3[a]/6.3[a]	7.0/4.3[a]		6.4[a]/7.0[a]	4.9[a]/4.9[a]		5.8[a]/6.6[a]	10.9/8.6		5.1	> 5.0	
Ak-P		5.7/3.7[a]	43/43		4.4[a]/4.6[a]	40[a]/40[a]		8.6/7.9	43/45		> 5.0	40.6
F-wave	NR[a]/NR[a]			NR[a]/NR[a]			57.2[a]/58.5[a]			46.8		
Fibular, right/left												
EDB-Ak	9.2[a]/5.8[a]	3.0[a]/5.6		9.7[a]/7.1[a]	2.8[a]/4.4		8.0[a]/5.6[a]	4.4/6.6		4.78	> 3.0	
Ak-P		2.7[a]/4.9	46/45		2.2[a]/4.3	35[a]/35[a]		3.7/5.5	42/42		> 3.0	41.8
F-wave	NR[a]/NR[a]			NR[a]/NR[a]			57.5[a]/56.5[a]			46.2		

ADM = abductor digiti minimi, AH = abductor halluces, Ak = ankle, Amp = amplitude, APB = abductor pollicis brevis, Ax = axillar, Eb= elbow, EDB = extensor digitorum brevis, NCV = nerve conduction velocity, P = popliteal fossa, Wr = wrist, NR = not recorded.
[a]Indicates abnormal values.[6]

Table 5. Serial nerve conduction studies for case 2: sensory nerve (height: 167 cm)

Sensory nerve	2021.07.05			2021.08.25 (worsened)			2021.11.05 (improved)			Normal values		
	Latency, ms	Amp, μV	NCV, m/s	Latency	Amp	NCV	Latency	Amp	NCV	Latency	Amp	NCV
Median, right												
Wr		15	50		16	48		19	46		> 10.0	41.2
Eb		29	53		12	52		37	56		> 10.0	49.3
Ax		33	59		50	55		59	79		> 10.0	53.9
Ulnar, right												
Wr		9	42		10	46		13	48		> 8.0	39.2
Eb		35	50		21	50		49	56		> 10.0	47.4
Ax		51	65		46	61		87	72		> 10.0	48.1
Sural, right/left		38/32	45/45		29/31	41/40		35/38	44/40		> 6.0	34.6
H reflex, right/left	35.8[a]/35.0[a]			NR[a]/NR[a]			NR[a]/NR[a]			28.7		

Ax = axillar, Eb = elbow, NCV = nerve conduction velocity, Wr = wrist, NR = not recorded.
[a]Indicates abnormal values.[6]

Ethics statement

Informed consent for publication of clinical data was obtained from the case patients.

DISCUSSION

As the pandemic persists, cases of inflammatory neuropathies associated with COVID-19 vaccination are increasingly being reported, and large-scale studies are underway.[7] GBS cases have occurred after COVID-19 vaccinations regardless of the type of vaccine (ChAdOx1 nCoV-19 and BNT162b2) in South Korea,[8] and GBS was officially recognized as a complication of COVID-19 vaccines in July 2021. The pathomechanism of GBS is associated with the immune response to a preceding infection, which leads to a cross-reaction and causes the production of auto-antibodies directed to epitopes of the myelin sheath or to peripheral nerves and roots.[9] The molecular mimicry between microbial proteins or proteins produced by vaccination and the nerve cell surface has been one of the suggested pathophysiologies of GBS associated with COVID-19.

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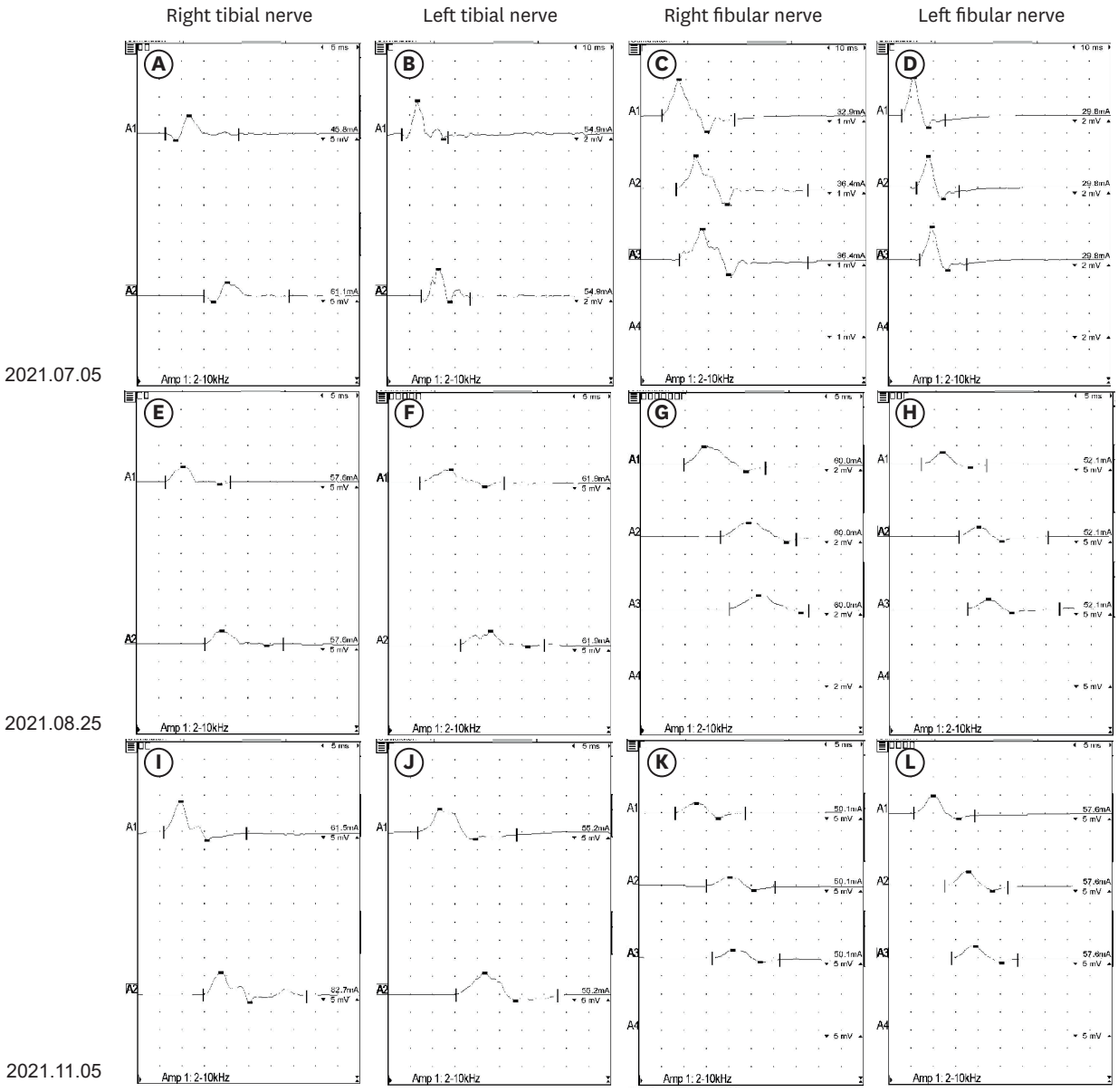

Fig. 2. Waveform of NCS of case 2. (**A**-**D**) Initial NCS of bilateral lower extremities: (**A**) right tibial nerve; (**B**) left tibial nerve; (**C**) right fibular nerve; (**D**) left fibular nerve. (**E**-**H**) Second NCS. The demyelinating polyneuropathy aggravated despite of high-dose oral prednisolone: (**E**) right tibial nerve; (**F**) left tibial nerve; (**G**) right fibular nerve; (**H**) left fibular nerve. (**I**-**L**) Third NCS. The demyelinating polyneuropathy were improved after oral prednisolone (15 mg/day), azathioprine, and intravenous immunoglobulin administration: (**I**) right tibial nerve; (**J**) left tibial nerve; (**K**) right fibular nerve; (**L**) left fibular nerve.
NCS = nerve conduction studies

CIDP, which is a type of immune-mediated demyelinating polyneuropathy, has potential to be induced by COVID-19 vaccination, just like GBS. Several cases of CIDP after COVID-19 vaccination have been reported recently. Suri et al.[10] reported the case of a 47-year-old male patient diagnosed with CIDP who presented with bilateral limb weakness and facial diplegia after ChAdOx1 nCoV-19 vaccination. Abo-zed et al.[11] reported a case of GBS that developed shortly after administration of the mRNA-1273 vaccine and subsequently evolved into CIDP. In Australia, a case series of four patients diagnosed with CIDP was reported by de Souza et al.[12] All four patients had received the ChAdOx1 nCoV-19 vaccine before the onset of symptoms. However, there has not yet been multicenter research on the incidence of CIDP after vaccination

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against COVID-19, or on the association between types of vaccines and CIDP. Also, there have been no reports of the occurrence of CIDP after COVID-19 vaccination in South Korea.

CIDP is an idiopathic condition in which a primarily T cell-mediated immune response is directed against myelin components of peripheral nerves.[13] CIDP may be triggered by viruses and vaccines, through mechanisms triggering self-reactive T cell and cytokine upregulation that may induce MHC class II expression.[14] The cases reported here had been vaccinated with two types of vaccine with different mechanisms. mRNA-1273 is an mRNA vaccine containing instructions for expressing a SARS-CoV-2 protein, but it does not contain the virus and prepares the body to defend itself against COVID-19.[15] On the other hand, vaccines such as Ad26.COV2.S and ChAdOx1 nCoV-19 are modified adenovirus vector vaccines. Virus vector vaccines have a higher transmission rate of genes within the cell than mRNA vaccines, and they cause natural infection and require a stronger immune response compared to an mRNA vaccine. Many neurological diseases such as GBS have been reported after administration of adenovirus vector vaccines.[10,12,16,17] However, the mRNA vaccine may also cause various neurological diseases, including immune-mediated neuropathies. There have been 383 reports of GBS in the UK following vaccination with ChAdOx1 nCoV-19, and 42 reports following the BNT162b2 vaccine.[15] In South Korea, a series of 13 GBS cases following COVID-19 vaccination was reported. Of the 13 patients, eight had received ChAdOx1 nCoV-19, and five had received the BNT162b2 vaccine.[8] In addition to GBS, a case of CIDP after vaccination with mRNA-1273 vaccine was reported.[18] The pathomechanisms underlying the association between CIDP and specific vaccines against COVID-19 are not well known, but CIDP seems to occur in response to various types of COVID-19 vaccines.

The treatments for GBS and CIDP are quite different. Therefore, it is important to distinguishing CIDP from other demyelinating neuropathies, including GBS. In GBS, IVIg is administered for a short period, without maintenance treatment. On the other hand, oral corticosteroids or steroid-sparing immunosuppressants have been used as treatment for CIDP. However, A-CIDP is difficult to distinguish from GBS. If symptoms improve after IVIg administration and then worsen for more than eight weeks, the possibility of A-CIDP should be considered. Maintenance treatment for CIDP should be initiated without delay in A-CIDP. The first of the cases was considered A-CIDP, because the patient had been experiencing recurrences three times, with more than eight weeks from the onset of symptoms.

Herein, we summarize the first reported cases of a new occurrence of CIDP following vaccination against COVID-19 in South Korea. These cases indicate that COVID-19 vaccination may be a possible trigger factor for CIDP as well as for GBS. Although a causal relationship has not been established between COVID-19 vaccination and CIDP, CIDP may follow COVID-19 vaccination, regardless of different mechanisms of action. As CIDP treatment is different from that of GBS, clinicians should closely monitor patients with GBS associated with COVID-19 whenever they deteriorate after initial treatment.

REFERENCES

1. Van den Bergh PY, Hadden RD, Bouche P, Cornblath DR, Hahn A, Illa I, et al. European Federation of Neurological Societies/Peripheral Nerve Society guideline on management of chronic inflammatory demyelinating polyradiculoneuropathy: report of a joint task force of the European Federation of Neurological Societies and the Peripheral Nerve Society - first revision. *Eur J Neurol* 2010;17(3):356-63. PUBMED | CROSSREF

2. Van den Bergh PY, van Doorn PA, Hadden RD, Avau B, Vankrunkelsven P, Allen JA, et al. European Academy of Neurology/Peripheral Nerve Society guideline on diagnosis and treatment of chronic inflammatory demyelinating polyradiculoneuropathy: report of a joint task force-second revision. *J Peripher Nerv Syst* 2021;26(3):242-68.
PUBMED | CROSSREF
3. Van der Meché FG, Van Doorn PA, Meulstee J, Jennekens FG; GBS-consensus group of the Dutch Neuromuscular Research Support Centre. Diagnostic and classification criteria for the Guillain-Barré syndrome. *Eur Neurol* 2001;45(3):133-9.
PUBMED | CROSSREF
4. Sriwastava S, Sharma K, Khalid SH, Bhansali S, Shrestha AK, Elkhooly M, et al. COVID-19 vaccination and neurological manifestations: a review of case reports and case series. *Brain Sci* 2022;12(3):407.
PUBMED | CROSSREF
5. Abu-Rumeileh S, Abdelhak A, Foschi M, Tumani H, Otto M. Guillain-Barré syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. *J Neurol* 2021;268(4):1133-70.
PUBMED | CROSSREF
6. Oh SJ. *Clinical Electromyography: Nerve Conduction Studies*. Philadelphia, PA, USA: Lippincott Williams & Wilkins; 1993.
7. Hanson KE, Goddard K, Lewis N, Fireman B, Myers TR, Bakshi N, et al. Incidence of Guillain-Barré syndrome after COVID-19 vaccination in the Vaccine Safety Datalink. *JAMA Netw Open* 2022;5(4):e228879.
PUBMED | CROSSREF
8. Kim JE, Min YG, Shin JY, Kwon YN, Bae JS, Sung JJ, et al. Guillain-Barré syndrome and variants following COVID-19 vaccination: report of 13 cases. *Front Neurol* 2022;12:820723.
PUBMED | CROSSREF
9. Leonhard SE, Mandarakas MR, Gondim FA, Bateman K, Ferreira ML, Cornblath DR, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. *Nat Rev Neurol* 2019;15(11):671-83.
PUBMED | CROSSREF
10. Suri V, Pandey S, Singh J, Jena A. Acute-onset chronic inflammatory demyelinating polyneuropathy after COVID-19 infection and subsequent ChAdOx1 nCoV-19 vaccination. *BMJ Case Rep* 2021;14(10):e245816.
PUBMED | CROSSREF
11. Abo-Zed A, Pinevich A. Guillain-Barré syndrome, or acute on chronic inflammatory demyelinating polyneuropathy, following Moderna COVID-19 vaccine. *Chest* 2021;160(4):A898.
CROSSREF
12. de Souza A, Oo WM, Giri P. Inflammatory demyelinating polyneuropathy after the ChAdOx1 nCoV-19 vaccine may follow a chronic course. *J Neurol Sci* 2022;436:120231.
PUBMED | CROSSREF
13. Pascual-Goñi E, Martín-Aguilar L, Querol L. Autoantibodies in chronic inflammatory demyelinating polyradiculoneuropathy. *Curr Opin Neurol* 2019;32(5):651-7.
PUBMED | CROSSREF
14. Albert LJ, Inman RD. Molecular mimicry and autoimmunity. *N Engl J Med* 1999;341(27):2068-74.
PUBMED | CROSSREF
15. Kim JW, Kim YG, Park YC, Choi S, Lee S, Min HJ, et al. Guillain-Barre syndrome after two COVID-19 vaccinations: two case reports with follow-up electrodiagnostic study. *J Korean Med Sci* 2022;37(7):e58.
PUBMED | CROSSREF
16. Maramattom BV, Krishnan P, Paul R, Padmanabhan S, Cherukudal Vishnu Nampoothiri S, Syed AA, et al. Guillain-Barré syndrome following ChAdOx1-S/nCoV-19 vaccine. *Ann Neurol* 2021;90(2):312-4.
PUBMED | CROSSREF
17. Patel SU, Khurram R, Lakhani A, Quirk B. Guillain-Barre syndrome following the first dose of the chimpanzee adenovirus-vectored COVID-19 vaccine, ChAdOx1. *BMJ Case Rep* 2021;14(4):e242956.
PUBMED | CROSSREF
18. Singh S, Sanna F, Adhikari R, Akella R, Gangu K. chronic inflammatory demyelinating polyneuropathy post-mRNA-1273 vaccination. *Cureus* 2022;14(4):e24528.
PUBMED | CROSSREF