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Aims and Scope

Journal of Electrodiagnosis and Neuromuscular Diseases (*J Electrodiagn Neuromuscul Dis*, JEND) is a peer-reviewed journal concerning both normal and abnormal functioning of the muscle, the neuromuscular junction, and the peripheral motor, sensory and autonomic nerves. The journal publishes clinical studies, reviews, and case reports in the fields of electrophysiology, electrodiagnosis, imaging studies including ultrasonography, and management, about neuromuscular diseases. The journal is aimed to provide an open forum for original research in basic science and clinical research that will improve our fundamental understanding and lead to effective treatments of neuromuscular diseases.
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Ultrasonographic Evaluation of Ulnar Neuropathy Around the Elbow in Diabetes Mellitus

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Objective: We evaluated the usefulness of ultrasonography for subclinical ulnar neuropathy at the elbow (UNE) in patients with diabetes.
Methods: Ultrasonography of the ulnar nerve was performed on 140 limbs in 73 patients with diabetes mellitus at 8 standardized sites proximal and distal to the medial epicondyle (1-cm intervals). The ulnar nerve cross-sectional area (CSA) at each site was analysed according to the presence of electrophysiological diabetic polyneuropathy (DPN) or UNE (UNE_e).
Results: Fifty-nine limbs were electrophysiologically normal, 22 limbs had UNE, 39 limbs had DPN, and 20 limbs had DPN and UNE_e. In patients without DPN, the maximal CSA, swelling ratio, and CSAs were greater in the UNE_e group than in the normal group ($p < 0.01$). No significant differences were noted between the DPN and DPN+UNE_e groups regarding the CSAs, maximal CSA, and swelling ratio.
Conclusion: Ultrasonography of ulnar nerve swelling is helpful for diagnosing subclinical UNE in patients with diabetes.

Keywords: Ulnar neuropathies; Diabetes mellitus; Ultrasonography; Electrodiagnosis; Polyneuropathies

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Introduction

Ulnar neuropathy at the elbow (UNE) is the second most common focal entrapment neuropathy in patients with diabetes following carpal tunnel syndrome. Standard 10-cm nerve conduction studies revealing reduced conduction velocity (less than 50 m/s) or conduction block in the elbow segment are commonly used to diagnose UNE [1]. However, it is difficult to diagnose UNE based on the ulnar motor conduction velocity (MCV) in patients with diabetic polyneuropathy (DPN) [2]. In particular, it can be extremely challenging to establish diagnostic criteria for UNE in patients with diabetes who have a forearm segment MCV less than 50 m/s. In these patients, decreased conduction velocity in the elbow segment can result from UNE or DPN. Short-segment nerve conduction studies are also used to diagnose UNE with higher diagnostic accuracy [3–5].

High-resolution ultrasonography is widely applied to investigate peripheral nerve lesions. Ultrasonography has also been recommended as a useful and reliable secondary approach for diagnosing UNE [6]. In many ultrasonography studies [7–9], UNE was diagnosed based on the maximum cross-sectional area of the ulnar nerve around the elbow (CSAmax); the cut-off values of the ulnar nerve cross-sectional areas (CSAs) varied marginally from 8.3 to 11.0 mm^2. In patients with diabetes, however, the cut-off values of the ulnar nerve CSAs for diagnosing UNE are unknown.

Subclinical UNE (UNE_sc) is frequently encountered in patients with diabetes during electrodiagnostic studies. Jang et al.

[10] examined 105 patients with diabetes and diagnosed UNE_sc using the ulnar MCV and inching techniques with a 1-cm interval of stimulation around the elbow for increased detection of ulnar nerve lesion. The authors suggest that diagnosing UNE_sc early is important because it can exacerbate ulnar neuropathy through external compression or continuous elbow flexion. However, the morphologic change of the ulnar nerve at the elbow in patients with diabetes with UNE and/or DPN remains unknown. In the present study, we identify the changes in ulnar nerve CSA that occur around the elbow in patients with diabetes according to the presence of electrophysiological DPN or UNE to evaluate the diagnostic utility of ultrasonography for UNE_sc in patients with diabetes.

Materials and Methods

1) Patients

Patients with type-2 diabetes mellitus diagnosed according to the criteria of the American Diabetes Association with no symptoms of ulnar neuropathy were enrolled in the present study prospectively. Patients were excluded if they had a history of previous elbow surgery or trauma, cervical radiculopathy, any inflammatory disorder, any malignancy, and/or a medical disease associated with polyneuropathy other than diabetes mellitus. Patients who had brachial plexopathy or who chronically consumed alcohol were also excluded. The study protocol was approved by the Institutional Review Board of Korea University Ansan Hospital (AS13033) and all patients provided written informed consent before participating in the study.

2) Electrodiagnostic study

Nerve conduction studies were performed in the bilateral ulnar motor and sensory nerves, unilateral median motor and sensory nerves, peroneal, tibial motor nerves and sural, superficial peroneal sensory nerves using the Viking Select EMG NCS Machine (Nicolet Viasys; Nicolet Viasys Healthcare, Madison, WI, USA). H-reflex and the ulnar, median, peroneal, and tibial F-waves were also obtained. Prior to the nerve conduction studies, the skin temperature was maintained above 32°C. The inching test of the ulnar nerve was performed with bilateral abductor digiti minimi recording to detect abnormal focal slowing of the nerve using the TenElectrodes stimulator [11]. Although there is no gold standard for the electrodiagnosis of DPN, in this study, patients were diagnosed with DPN if the electrophysiological criteria from the Diabetes Control and Complication Trial, with some modifications, were met [12,13]. Specifically, electrophysiologic DPN was diagnosed when the sural sensory amplitude was less than or equal to 5 μV and 2 or more of the median sensory, peroneal motor, peroneal F-wave, H-reflex, and fibrillations in the lower extremity muscles were abnormal.

Electrophysiological UNE (UNE_e) in patients with diabetes was established using the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) guidelines and the results of the inching test [14]. The AANEM guidelines for diagnosing UNE_e were as follows: (1) absolute ulnar MCV of the elbow segment less than 50 m/s or (2) the elbow segment was more than 10 m/s slower than the forearm segment. In the inching test, the ulnar nerve is stimulated at intervals of 1 cm beginning 4 cm distal to the medial epicondyle (ME) and ending 3 cm proximal to the ME. We diagnosed a patient with UNE_e when the latency difference per 1-cm segment was greater than 0.4 ms. If the absolute MCV of the forearm segment was less than 50 m/s, which could result from DPN, then a diagnosis of UNE_e was made when the MCV of the elbow segment was slower than that of the forearm segment by 10 m/s or more or based on the results of the inching test. Patients were classified into the following 4 groups according to the electrophysiological diagnosis: normal, UNE_e, DPN, or DPN with UNE_e (DPN+UNE_e).

3) Ultrasonographic study

Ultrasonography was performed using the ACCUVIX V20 system (Samsung Medison, Seoul, Korea) interfaced with a 6 to 13 MHz linear array transducer by a physiatrist with more than 8 years of experience in ultrasonography of peripheral nerves. Sonographic examinations were conducted with the patient in a supine position and the elbows flexed at 10 degrees with the forearms supinated. Initially, marks were made on the skin overlying the ulnar nerve at the level of the ME; 3, 2, and 1 cm proximal to the ME; and 4, 3, 2, and 1 cm distal to the ME (proximal to dis-

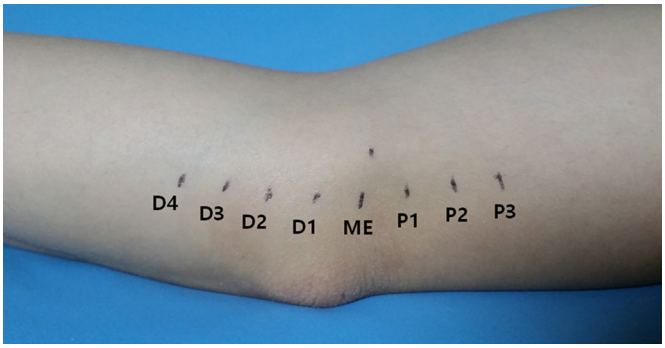

Fig. 1. Sites of nerve stimulation and cross-sectional area measurements of the ulnar nerve. Stimulation was performed at the medial epicondyle (ME); 1, 2, and 3 cm proximal (P1, P2, and P3, respectively) to the ME; and 1, 2, 3, and 4 cm distal (D1, D2, D3, and D4, respectively) to the ME.

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tal: P3, P2, P1, ME, D1, D2, D3, and D4 in Fig. 1). The probe was placed in the transverse plane on the proximal arm to verify the ulnar nerve and then moved distally. The probe was held perpendicular to the skin. Minimal pressure was applied to avoid extrinsic nerve compression. At each of the 8 standardized and marked sites, the CSA of the ulnar nerve was measured within the hyperechoic rim of the nerve using the "AreaTrace" function of the ultrasonography system. The ulnar nerve-swelling ratio was calculated by dividing the CSAmax by the CSA measured from D4.

4) Statistical analyses

Statistical analyses were performed with IBM SPSS ver. 20.0 for Windows (IBM Corp., Armonk, NY, USA). Independent t-tests were used to evaluate differences in the demographic data between patients with and without DPN. Categorical values (gender and presence of DPN symptoms) were analysed using χ^2 tests. To compare the ultrasonographic nerve CSAs among the 4 patient groups, analyses of variance were employed. Fisher Least Significant Difference tests were used for the post hoc analyses. The cut-off values of the CSA at the ME, CSAmax, and swelling ratio for diagnosing UNE_sc were calculated using receiver operating characteristic (ROC) curves and the resultant specificity and sensitivity were obtained. Differences were considered statistically significant at $p < 0.05$.

Results

From the 73 patients with diabetes mellitus in this study, 140 upper limbs (59 normal, 22 UNE_e, 39 DPN, and 20 DPN +UNE_e) were included in the analysis. Demographic data are presented in Table 1. Differences in the age, diabetes duration, DPN symptoms, and glycosylated hemoglobin were noted between patients with and without DPN.

In patients without DPN (normal and UNE_e groups), significant increases in the ulnar nerve CSAs were identified in the UNE_e group (Fig. 2) when compared with the electrophysiologically normal group at all sites except D4 and D3 ($p < 0.01$). The CSAmax and swelling ratio were also significantly greater in the UNE_e group than in the normal group ($p < 0.001$). Conversely, no significant differences in the ulnar nerve CSAs, CSAmax, and swelling ratio were identified between the DPN and

Table 1. Patients' Demographic and Clinical Data

Measurement	Without DPN (n = 38)	With DPN (n = 35)	p-value
Sex (female:male)	12:26	13:22	0.617
Age (y)	52.4 ± 10.3	60.2 ± 11.0	0.003
Height (cm)	165.5 ± 6.7	163.8 ± 9.6	0.384
Weight (kg)	68.0 ± 11.2	65.3 ± 12.4	0.327
Diabetes mellitus duration (mo)	64.9 (1-300)	138.0 (1-480)	0.003
DPN symptoms*	12	28	0.001
Symptom duration (mo)†	11.3 (1-120)	24.5 (1.5-120)	0.081
Glycosylated hemoglobin	7.5 ± 1.5	9.0 ± 2.5	0.003

Values are presented as mean ± standard deviation or mean (range).
DPN, diabetic polyneuropathy.
*DPN symptoms include unsteady walking and numbness, burning pain, and a prickling sensation in the distal legs and feet. †Diabetes mellitus duration refers to the time interval between the diagnosis of diabetes mellitus and recruitment.

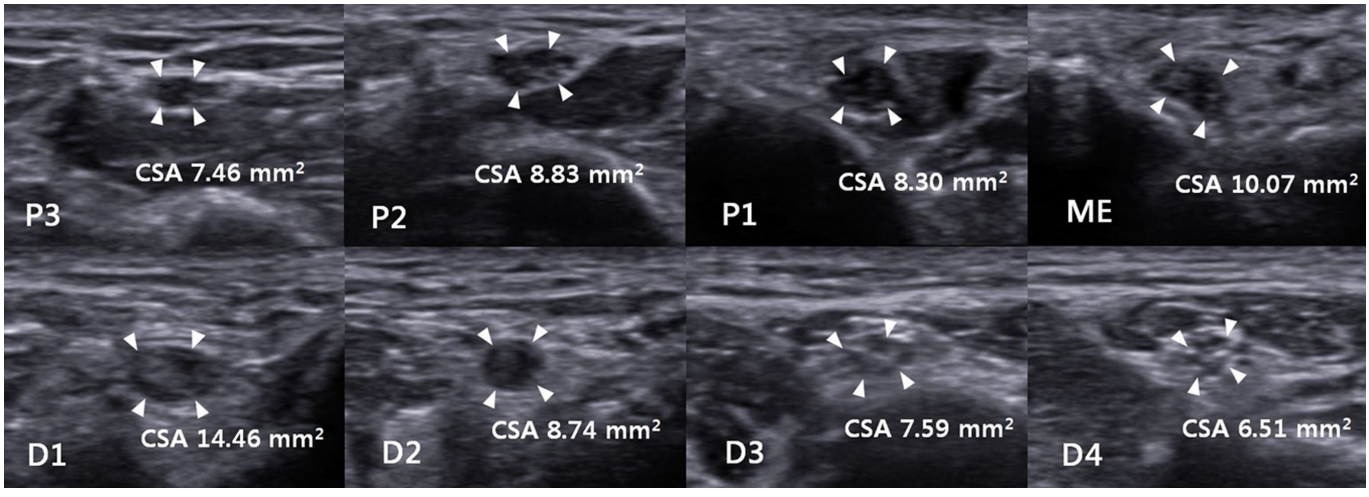

Fig. 2. Ultrasound image of the ulnar nerve in a patient without diabetic polyneuropathy, but with subclinical ulnar neuropathy at the elbow (UNE_e group) at 8 stimulation sites, including 3 sites proximal (P3, P2, and P1) to the medial epicondyle (ME), ME, and 4 sites distal (D1, D2, D3, and D4) to the ME. The maximum cross-sectional area (CSA) was observed 1 cm distal (D1) to the ME.

DPN+UNE_e groups. The ulnar nerve CSA at the ME and CSAmax were greater in the DPN group than in the normal group ($p < 0.01$). Significant increases in the ulnar nerve CSAs near to the ME (D1, ME, and P1) and in the CSAmax were identified in the DPN+UNE_e group compared with the normal group. Finally, the swelling ratio was significantly greater in the UNE_e group than in the DPN group (Table 2).

The areas under the ROC curves were significant for the ulnar nerve at CSAmax, CSA swelling ratio, and CSA at ME. The cut-off values for diagnosing UNE_sc in patients without DPN were 9.85 mm^2 for the CSAmax, 1.43 for the CSA swelling ratio, and 9.85 mm^2 for the CSA at ME (Table 3). However, no meaningful sensitivity and specificity values of the CSAs were identified for diagnosing UNE_sc in patients with DPN.

Discussion

In the present study, ultrasonographic evaluations performed at 1-cm intervals were conducted to investigate the changes in the CSA of the ulnar nerve around the elbow with the aim of identifying UNE_sc in patients with diabetes. In the absence of electrophysiologic DPN, the UNE_e group demonstrated greater swelling than the electrophysiologically normal group without UNE_e, especially near to the ME (P1, ME, and D1). Conversely, in the presence of DPN, no significant swelling differences were identified between the DPN and DPN+UNE_e groups. These results reflect that ulnar nerve swelling can be induced by the UNE_e and DPN simultaneously. They also suggest that DPN-induced ulnar nerve swelling can limit further enlargement of the ulnar nerve from UNE_e, although the exact pathophysiology remains unclear. Although the examined nerve was different, the results of the present study resemble those of a prior study reporting that the distal median nerve CSA did not differ significantly between those with and without superimposed carpal tunnel syndrome in patients with DPN [15]. These findings imply that a nerve swelling at a common entrapment region in a patient with DPN can be a consequence of DPN, not just entrapment neuropathy.

The present study also revealed that, in the absence of UNE_e, patients with DPN (DPN group) had more swelling at the ME level than did patients without DPN (normal group). These findings are similar to those of a previous study on the upper extremity nerves in patients with diabetes, which reported that the CSAs of the ulnar nerve were larger at the ME level in patients

Table 2. Mean CSAs of the Ulnar Nerve around the Elbow

CSA	Normal (n = 59)	UNE_e (n = 22)	DPN (n = 39)	DPN + UNE_e (n = 20)	p-value
CSA_D4	6.9 ± 1.8	7.3 ± 1.5	7.6 ± 1.9	7.6 ± 1.5	0.140
CSA_D3	7.2 ± 1.8	8.1 ± 1.9	7.9 ± 1.8	7.7 ± 1.4	0.089
CSA_D2	6.9 ± 1.8	8.3 ± 1.9*	7.8 ± 2.2	7.8 ± 1.6	0.014
CSA_D1	7.2 ± 2.1	9.8 ± 3.0**	8.4 ± 2.0	9.4 ± 3.6‡	< 0.001
CSA_ME	7.9 ± 2.1	11.3 ± 3.5**	9.6 ± 2.1†	9.8 ± 3.3‡	< 0.001
CSA_P1	8.2 ± 2.4	11.0 ± 3.9**	9.3 ± 2.3	10.4 ± 3.9‡	< 0.001
CSA_P2	7.6 ± 2.3	9.2 ± 2.4*	8.6 ± 2.3	9.0 ± 2.4	0.011
CSA_P3	6.9 ± 2.1	8.4 ± 2.1*	7.8 ± 2.0	7.5 ± 1.7	0.016
CSAmax	9.0 ± 2.4	12.6 ± 3.8**	10.5 ± 2.5†	12.0 ± 4.1‡‡	< 0.001
CSA_SR	1.3 ± 0.3	1.7 ± 0.5**^	1.4 ± 0.3	1.6 ± 0.3	< 0.001

CSA, cross-sectional area; UNE_e, electrophysiological ulnar neuropathy at elbow; DPN, diabetic polyneuropathy; ME, medial epicondyle; D4, D3, D2, and D1, 4, 3, 2, and 1 cm distal to the ME, respectively; P1, P2, and P3, 1, 2, and 3 cm proximal to the ME, respectively; CSAmax, maximum CSA; SR, swelling ratio (CSAmax/CSA_D4).
Post hoc analyses using the Fisher least significant difference tests: *$p < 0.01$ and **$p < 0.001$ for UNE_e vs. normal; †$p < 0.01$ for DPN vs. normal; ‡$p < 0.01$ and ‡‡$p < 0.001$ for DPN + UNE_e vs. normal; ^$p < 0.001$ for UNE_e vs. DPN.

Table 3. Cut-Off Values of the Ulnar Nerve CSA for Diagnosing Subclinical Ulnar Neuropathy at the Elbow in Patients with Diabetes

CSA	Without DPN			With DPN		
	Cut-off value	Sensitivity	Specificity	Cut-off value	Sensitivity	Specificity
CSA_ME	9.85	72.7	83.1	9.45	45.0	51.3
CSAmax	9.85	77.3	74.6	11.60	55.0	66.7
CSA_SR	1.43	72.7	72.9	1.46	50.0	64.1

CSA, cross-sectional area; DPN, diabetic polyneuropathy; ME, medial epicondyle; CSAmax, maximum CSA; SR, swelling ratio (CSAmax/CSA_D4).

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with DPN than in those with diabetes without DPN [16]. This implies that peripheral nerve swelling progresses more in the entrapment region of the nerve when DPN is present and reflects the increased susceptibility of patients with DPN to entrapment neuropathy development [17]. Moreover, the ulnar nerve-swelling ratio was significantly greater in the UNE_e group than in the DPN group. This finding is because DPN influences nerve swelling at non-compressive sites (e.g., D4) than UNE_e does.

Although the precise pathophysiological implications of nerve enlargement in DPN have not been fully elucidated, it has been suggested that increased sorbitol levels in patients with diabetes may contribute to increased intracellular hydration, which subsequently affects swelling of the nerves [18]. Furthermore, the ulnar nerve may become enlarged owing to repetitive undetected trauma and external compression, which can increase endoneurial edema, perineural thickening, and vascular changes in the vasa nervorum, causing nerve ischemia [19,20]. Simon et al. [21] reported that even in healthy individuals, the ulnar nerve was larger at the ME level than at more proximal and distal sites of the nerve. This pattern of sonographic change was also observed in the present study, although the extent of nerve swelling at the ME was different among the groups. The authors of the previous study hypothesized that the precipitation of symptomatic UNE can reflect an exacerbation of an existing asymptomatic ulnar nerve lesion [21]. Moreover, diabetes can increase the susceptibility of the ulnar nerve to injury [22,23]. Therefore, early detection of UNE_sc in patients with diabetes can be important for preventing symptomatic UNE. For diagnosing UNE_sc in the present study, the cut-off value of the CSAmax in patients without DPN was 9.85 mm^2. This value was within the range of previously published cut-off values of CSAmax (8.3 to 11.0 mm^2) for diagnosing symptomatic UNE from ultrasonographic studies [7–9]. This suggests that CSAmax in patients with diabetes without DPN is mainly influenced by UNE that is similar in healthy controls [16].

This study has several limitations. First, a healthy control group was not included. Moreover, because the presence of DPN was determined by electrophysiological criteria, it is likely that patients with neuropathic symptoms such as small fiber neuropathy were not included in the DPN group. However, because electrophysiological diagnosis is an objective indicator of neurologic morbidity, it can serve as a reference. Finally, there is a lack of definite diagnostic criteria for UNE in patients with DPN; thus, subsequent electrodiagnostic and ultrasonographic studies that include patients with definite UNE symptoms of DPN are required.

Conclusion

Ultrasonographic evaluations of ulnar nerve swelling are useful for diagnosing UNE_sc in patients with diabetes, although the ability to diagnose UNE based on ultrasonographic abnormalities such as nerve swelling appears to be limited in patients with DPN. Our data supported that a patient with diabetes should be diagnosed with UNE_sc when the CSAmax of the ulnar nerve is greater than 9.8 mm^2 around the ME or when the swelling ratio is greater than 1.4. In such cases, education and treatment to prevent progression to symptomatic UNE is recommended.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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CASE REPORT

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Guillain-Barré Syndrome Following the ChAdOx1-S/nCoV-19 Vaccine: A Case Report

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Serious neurological complications following coronavirus disease 2019 (COVID-19) vaccination, such as cerebral venous sinus thrombosis or acute disseminated encephalomyelitis, have rarely been reported. Here, we report a case of Guillain-Barré syndrome (GBS) that occurred within 2 weeks of receiving the AstraZeneca ChAdOx1-S/nCoV-19 vaccine. A 61-year-old man presented with weakness and hypesthesia of the legs 9 days after the first dose of the ChAdOx1-S vaccine. The patient progressed to respiratory failure and severe quadriparesis. Nerve conduction studies showed markedly reduced amplitudes of compound muscle action potentials and sensory nerve action potentials in both upper and lower extremities, without definitive evidence of demyelination. The patient was diagnosed with axonal GBS and received intravenous immunoglobulin treatment, with a poor response. This is a rare case of GBS after AstraZeneca ChAdOx1-S/nCoV-19 vaccination in Korea. Physicians should be aware of this rare but serious complication of the COVID-19 vaccine to effectively manage such a situation in clinical practice.

Keywords: Guillain-Barré syndrome; COVID-19; Vaccines

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Introduction

The coronavirus disease 2019 (COVID-19) pandemic has been ongoing for more than a year and a half, and COVID-19 vaccines obtained through emergency use approval are becoming an important factor in the stabilization of the pandemic [1]. As of July 26, 2021, approximately 16.9 million of the Korean population had received the COVID-19 vaccine.

In Korea, from February 2021, reports of various side effects related to the vaccine started surfacing, mainly among healthcare workers who were administered the vaccine at the beginning of the immunization program [2,3]. Recently, serious complications such as thromboembolic events arising from the Oxford AstraZeneca ChAdOx1nCov-19 vaccine and myocarditis from the Pfizer-BioNTech COVID-19 vaccine have been reported [3].

As more and more people are being vaccinated in order to achieve herd immunity within the general population, reports of rare but serious adverse events are increasing, and the demand for the analysis of vaccine related disease and causality is increasing. Guillain-Barré syndrome (GBS) is one of the rare adverse events reported after COVID-19 vaccine and has been reported in some parts of the world [4]; however, no such case has been reported in Korea to date. To the best of our knowledge, we report a rare case of GBS manifesting as ascending quadriparesis 2 weeks after receiving the Oxford AstraZeneca COVID-19 vaccine in Korea.

The study was approved by the Ethics Committees of the Inje University Busan Paik Hospital. Informed consent was obtained from the patient.

Case Report

A 61-year-old male patient without significant comorbidities presented with progressive paraparesis. The patient had nausea, vomiting, headache, and dizziness at presentation which started 3 days ago. There was no known history of COVID-19 exposure. The COVID-19 reverse-transcriptase polymerase chain reaction test performed nine months prior to this presentation was negative. He received his first dose of the Oxford AstraZeneca ChAdOx1-S/nCoV-19 vaccine nine days prior to paraparesis presentation. After the initial paralysis of the lower extremities, muscle strength in the arms also began to decrease gradually, and on the day before the visit to the hospital, the patient was having difficulty in speaking and swallowing accompanied by respiratory problems. At the time of his arrival to the emergency room, respiratory failure had progressed, and thus, intubation was performed.

Neurological examination revealed dysarthria and dysphagia. There was severe weakness in the upper extremities (Medical Research Council grade 2/2) and the lower extremities (grade 0-1/0-1). Deep tendon reflexes in the upper and lower extremities were absent.

Thereafter, routine blood investigations, including serological tests, assays for paraneoplastic markers, and cerebrospinal fluid (CSF) analysis, were performed, which ruled out several etiologies. CSF examination revealed albuminocytologic dissociation (white blood cell [WBC] count, 0/mm^3; protein, 87.6 mg/dL). Laboratory tests revealed normal WBC counts, C-reactive protein levels, and erythrocyte sedimentation rate, thus pointing toward an absence of inflammation. The screening tests for systemic vasculitis were also normal. No paraproteins were detected using serum protein electrophoresis. Testing for respiratory viruses, such as influenza and COVID-19, that can cause respiratory failure was carried out and yielded negative results. Brain computed tomography scan showed no significant abnormality.

Nerve conduction studies (NCS) performed on the sixth day of initial presentation revealed acute motor sensory axonal neuropathy (Table 1). There was mild slowing of conduction velocities and severely decreased compound muscle action potentials without definite evidence of demyelination in both forearms and legs. Sensory NCS showed decrease in sensory nerve action potential amplitude and conduction velocities of both the upper extremities and sural nerves. There was an absence of the H-reflex in both lower limbs and an absence or delay of F-waves in all 4 extremities, along with a bilateral delayed response in the blink reflex pathway. All the above findings led to the diagnosis of axonal GBS.

Treatment with intravenous immunoglobulins was started, but the symptoms gradually worsened and progressed to quadriplegia. One week after admission, bilateral abducens nerve palsy progressed to bilateral complete ophthalmoplegia, facial diplegia, and bilateral ptosis.

Autonomic nervous system symptoms presented with cardiovascular dysautonomia, including fluctuations in blood pressure and heart rate, and gastrointestinal dysmotility leading to constipation.

Antiganglioside antibody analysis, including immunoglobulin (Ig)G and IgM antibodies against gangliosides GM1, GD1b, and GQ1b revealed negative results.

Two months after the onset of symptoms, severe ptosis, ophthalmoplegia, and bilateral facial palsy resolved moderately, and quadriparesis also began to improve gradually (upper extremities: grade 1-2/1-2; lower extremities: grade 1/1). Tracheostomy with mechanical ventilation was still maintained.

Discussion

GBS has been reported as one of the neurological manifestations that can occur in patients with COVID-19 [5]; however, it has rarely been reported since the initiation of COVID-19 vaccination program worldwide [6]. In Korea, healthcare workers were the first to receive COVID-19 vaccines, and starting in February 2021, local or systemic adverse effects related to the vaccine, such as pain, fever, chills, and myalgia, were reported in some cases [2]. However, reports on rare and serious complications such as thrombosis and cardiomyositis are scarce. As of July 26, 2021, approximately 16.9 million Korean people (32.9%) had been vaccinated with the first dose of the COVID-19 vaccine, and one reason behind the fewer number of reports on side effects may be related to the fact that a major part of the population is not vaccinated. Another reason may be limited interest in serious complications or difficulty in explaining the relationship between the vaccine and the complications.

Axonal GBS, one of the subtypes of GBS, has more severe symptoms and poorer prognosis than acute inflammatory demyelinating polyradiculopathy, and is mainly related to *Campylobacter jejuni* infection [7]. Neurological adverse effects that may occur after immunization are mainly demyelinating diseases, and association between influenza vaccines and GBS has been reported [7]. Although the reported risk of GBS after influenza vaccine is moderate, the vaccine is generally recommended because it outweighs the risk of H1N1 influenza morbidity and mortality [8]. Some studies suggest that post-vaccination demyelinating events are likely to trigger clinical disease expression in individuals who already

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Table 1. NCS and Blink Reflex Findings

Nerve sites	Latency (ms)		Amplitude (mV)		CV (m/s)	
	Right	Left	Right	Left	Right	Left
Motor NCS						
Median						
Wrist	7.62*	5.25*	0.19*	0.24*		
Elbow			0.16*	0.23*	48.6*	49.1*
Axilla			0.15*	0.22*	47.5*	47.0*
F-wave	40.2*	32.9*				
Ulnar						
Wrist	3.25*	3.25*	1.78*	1.68*		
Below elbow			1.59*	1.24*	48.8*	45.2*
Above elbow			1.35*	0.93*	44.6	43.4
Axilla			1.28*	0.83*	47.1*	53.8
F-wave	NP*	NP*				
Peroneal						
Ankle	NP*	NP*	NP*	NP*		
Fibular head			NP*	NP*		
Knee			NP*	NP*		
F-wave	NP*	NP*				
Tibial						
Ankle	4.61	5.01	0.94*	0.65*		
Popliteal fossa			0.57*	0.53*	36.0*	34.9*
F-wave	NP*	NP*				
Sensory NCS						
Median						
Index-wrist			1.49*	NP*	42.3	NP*
Wrist-elbow			17.6	5.6*	48.8*	47.0*
Elbow-axilla			89	29.1	47.2*	59.5
Ulnar						
Finger-wrist			NP*	NP*		
Wrist-elbow			12.0	16.5	44.7*	46.4*
Elbow-axilla			37.5	36.3	52.9	49.7
Sural						
Lower leg	2.88	3.17	4.9*	5.5*	43.4	41.0
H-reflex	NP*	NP*				
Cranial						
Facial nerve motor NCS						
Orbicularis oculi	2.73	2.83	2.3	2.6		
Blink reflex						
Ipsilateral R1	21.0*	15.7*				
Ipsilateral R2	45.6*	47.0*				
Contralateral R2	46.6*	49.4*				

NCS, nerve conduction study; CV, conduction velocity; NP, no potential.
*Abnormal.

have an underlying disease process [9]. Even in cases of GBS, there are conflicting opinions regarding the association with vaccination. In many studies conducted in the early days of the pandemic, it was noted that the antigenicity of severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) itself is not involved in the causation of GBS, and therefore, ganglioside antibody is not found in most GBS cases after COVID-19 infection [5]. In addition, in post-vaccination GBS, ganglioside antibody in post-infectious GBS was not found. In the present case, GM1, GD1b, and GQ1b ganglioside antibodies were not detected.

However, recently, it has been shown that the COVID-19 spike protein binds to the glycolipoprotein-containing sialic acid present in coronavirus as well as peripheral nerve myelin, suggesting the possibility of cross-reactivity between SARS-COV-2 and peripheral nerves [10]. Although it is difficult to explain the mechanism of development of GBS following COVID-19 vaccination [4], if GBS develops within a few days after inoculation and cannot be explained by any other disease, the relationship between GBS and vaccine cannot be completely ruled out.

In this case, it was difficult to determine whether GBS was an isolated occurrence caused by an asymptomatic infection or related to the COVID-19 vaccine. However, in this case, there were no respiratory or gastrointestinal symptoms before onset, and no significant abnormalities were found in laboratory tests carried out for diagnosing the other possible causes. Therefore, we speculate that vaccination may have acted as a trigger in the development of GBS, as there was a history of receiving ChAdOx1-S/nCoV-19 vaccination 2 weeks prior to the onset, and the likelihood of other causes was low.

The annual incidence rate of GBS in Korea was 1.48 patients per 100,000 population through a nationwide epidemiological study from 2010 to 2016 [11]. Also, according to the Korean National Health Insurance Service claims data, a causal relationship between the occurrence of GBS within 6 weeks (42 days) and the vaccine was not found in the elderly who were mainly vaccinated against influenza [12]. Since it is still difficult to explain the causal relationship between GBS and the COVID-19 vaccine, there are not enough studies on the report itself or the relationship between the occurrence of GBS after the COVID-19 vaccine in Korea and around the world. Therefore, since more than 80% of the total population has completed the first dose of vaccination by the end of October in Korea, it is possible to study the causal relationship between GBS and the COVID-19 vaccine in a follow-up study based on nationwide vaccination, and the difference between the influenzae vaccine and the COVID-19 vaccine in the occurrence of GBS can also be compared.

The classification of GBS subtypes or NCS in patients who developed GBS after the COVID-19 vaccine has not yet been established. It can be seen that both the demyelinating type and the axonal type occur in GBS after COVID-19 infection [5], and it can be seen that both the demyelinating type or axonal type occurred in

GBS that occurred after the corona 19 vaccination [13]. Future studies on the neurophysiology of GBS associated with the COVID-19 vaccine will be needed through a study that recruited patients with a high causal relationship with the COVID-19 vaccine.

Although vaccines approved for emergency use have some rare but serious complications in addition to the minor side effects, the benefits far outweigh these risks; therefore, vaccination programs are continuing around the world [1].

Because it targets the general population without disease, serious complications such as GBS may occur that require intensive care unit treatment due to neuromuscular respiratory failure. Efforts should be made to keep the immunization program stable through a good medical emergency management system.

In conclusion, although it is not yet possible to explain the direct relationship between the COVID-19 vaccine and GBS, this report raises interest in the occurrence of such diseases as a complication of vaccines. Further studies are needed in this area to thoroughly explore this relationship and determine the possible link between COVID-19 vaccination, GBS, and the related long-term neurological sequelae. A complete investigation of adverse events associated with COVID-19 vaccines will not only help in gathering accurate information about the vaccine but also guide the clinicians toward a better management of such events in practice.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Delayed Bilateral Facial Nerve Palsy Manifesting 3 Months after Traumatic Brain Injury: A Case Report

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Facial nerve palsy (FNP) can be a challenging medical condition, and bilateral FNP is an uncommon occurrence that is potentially fatal and warrants urgent medical intervention. We report a rare case of bilateral FNP that developed after traumatic brain injury (TBI), which we approached through an electromyographic study. A 23-year-old male patient had experienced a fall-down injury from height of 4 meters while on his military service. Computed tomography of the brain and facial bone showed acute TBI and multiple skull base fractures. Limited facial expression and dysarthria started at the time of cranioplasty, which was about 3 months after the patient's initial presentation, and these symptoms gradually deteriorated over time. An electrodiagnostic study demonstrated incomplete bilateral facial nerve lesions, which were strongly indicated as lower motor neuron lesions. An early diagnosis based on electrodiagnostic study should be considered for proper treatment, which can achieve optimal functional recovery after bilateral FNP.

Keywords: Traumatic brain injury; Facial paralysis; Electromyography

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Introduction

Facial nerve palsy (FNP) can be a challenging medical condition, and patients with facial nerve disorders often faces various psychological obstacles [1]. Wide variety of etiologic factors exists causing facial paralysis, including Bell palsy, underlying medical conditions such as Guillain-Barre syndrome, leukemia, or infectious mononucleosis, and trauma [2]. Unilateral FNP is relatively common, reporting incidence of 20 to 25 per 100,000 population, whereas bilateral FNP is a rare clinical occurrence, which accounts for approximately 0.3 to 2 percent in facial palsy cases [2,3]. Bilateral FNP is usually found in systemic medical conditions, which are often potentially fatal and frequently warrant urgent medical intervention [4]. Among these causes, trauma is an extremely rare entity of bilateral facial paralysis [5].

We report an uncommon case of bilateral FNP proposed after traumatic brain injury (TBI), using electromyographic study to approach this medical condition.

Case Report

A 23-year-old male patient who had no medical comorbidity had experienced a fall down injury from height of 4 meters while on his military service. On the initial neurological examination, his mental status was stuporous, and he had been admitted to the emergency department. According to the medical record, there was no skin laceration or muscle injury in the face other than head trauma, and no treatment history involved as well. Initial Glasgow Coma Scale was rated for 9, eye response for 3, verbal response for 2, and motor response for 4, respectively. Computed tomography (CT) of brain and cranium showed acute subarachnoid hemorrhage and acute epidural hemorrhage on right

frontotemporal lobe and left occipital lobe, accompanied with multiple skull base fractures in both sides (Fig. 1). No other facial bone fractures were seen in the initial CT. Magnetic resonance imaging (MRI) was taken after 1 month from initial injury, which showed multiple T2 hyperintensities in left cerebral hemisphere and brainstem, suggesting diffuse axonal injury (Fig. 2). He had been transferred to the department of neurosurgery, and decompressive craniectomy and hematoma removal had been done. After about 2 months later, cranioplasty was conducted. He has received comprehensive rehabilitation therapy including physiotherapy, occupational therapy, and pulmonary rehabilitation throughout several hospitals. After 2 years from initial onset, he was transferred to our hospital to receive continuous rehabilitation therapy.

On admission day of our hospital, he showed limited facial expression and often spilled food residue on both side of while eating. Symptoms had been developed after cranioplasty, which was about 3 months later from initial onset, and gradually got worsened over time. Furthermore, owing to his severe dysarthria, it was often difficult to understand even at the word level without contextual clues, and controlling articulation in all consonants was incomplete. Videofluoroscopic swallowing study revealed incomplete laryngeal closure and elevation, as well as cricopharyngeal dysfunction. He showed obvious bilateral facial weakness with disfiguring asymmetry, which was more prominent on left side, evaluated as grade IV for left side and grade II for right side, according to House-Brackmann (H-B) grading system. Specifically, there was no movement for wrinkling forehead, incomplete eye closure and asymmetry for grinning mouth with maximal effort for left side, and slight weakness for wrinkling forehead and mouth movement, and complete eye closure with minimal effort for right side. There was no facial sensory change or hearing loss on both sides. Prior to our hospital, diagnostic approaches associated with these symptoms have never been conducted.

We performed electrodiagnostic approach for his bulbar symptoms, which can discriminate the etiology of bilateral facial palsy. According to the result, facial motor nerve conduction study stimulating bilateral facial nerves at preauricular area showed prolonged onset latency in nasalis branches, and low amplitude of combined motor action potential in frontalis, oculi, nasalis, and oris branches of bilateral facial nerves (Table 1). Needle electromyography showed abnormal spontaneous activities in the bilateral orbicularis oculi and orbicularis oris muscles, and it also showed decreased interference patterns at all examined muscles (Table 2). Blink reflex study stimulating both supraorbital nerves showed no responses for both R1, ipsilateral R2 and contralateral

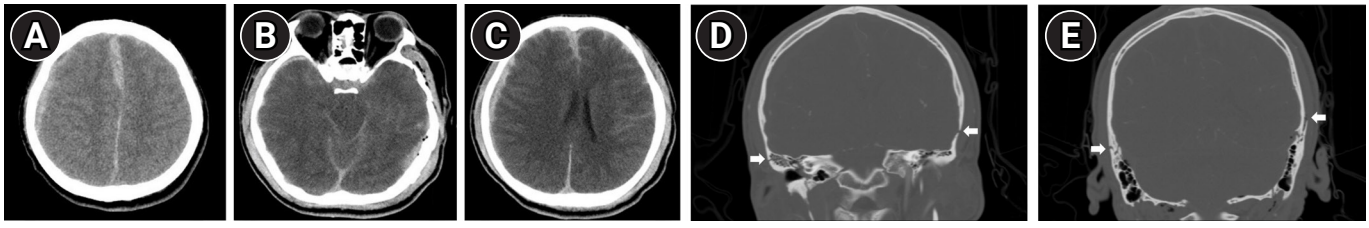

Fig. 1. Initial brain computed tomography (CT) of the patient. Axial view of brain CT (A-C) showed traumatic epidural hemorrhage in the right frontotemporal lobe and left occipital lobe, and acute subarachnoid hemorrhage. Coronal view of facial bone CT (D, E) demonstrated multiple skull base fractures on both sides (white arrows).

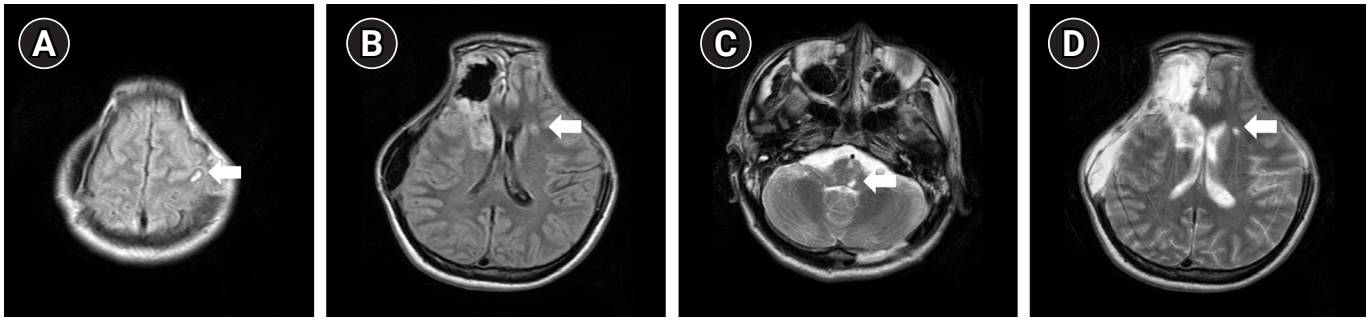

Fig. 2. Brain magnetic resonance imaging of the patient taken 1 month after the initial injury. T2-weighted images including fluid-attenuated inversion recovery (A, B) and turbo spin echo (C, D) sequences showed hyperintensities in the left cerebral hemisphere and brainstem, indicating lesions of subcortical white matter, which demonstrated diffuse axonal injury (white arrows).

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Table 1. Facial Motor Nerve Conduction Studies

Side	Motor nerve	Stimulation	Recording	Latency (ms)	Amplitude (mV)
Left	Facial nerve	Periauricular area	Frontalis muscle	3.3	0.1
			Nasalis muscle	4.3	0.1
			Orbicularis oculi muscle	3.0	0.6
			Orbicularis oris muscle	2.0	1.0
Right	Facial nerve	Periauricular area	Frontalis muscle	3.0	1.4
			Nasalis muscle	6.0	0.2
			Orbicularis oculi muscle	3.0	1.9
			Orbicularis oris muscle	2.1	1.6

Table 2. Needle Electromyography

Side	Muscle	Insertional activity	Spontaneous activity	Motor unit action potentials			IP
				Polyphasia	Amplitude	Duration	
Right	Frontalis	N	N	N	N	N	P
	Orbicularis oculi	I	F&P(+)	+	N	N	P
	Orbicularis oris	I	F&P(+)	+	N	N	P
Left	Frontalis	N	N	N	N	N	S
	Orbicularis oculi	I	F&P(++)	+	N	N	S
	Orbicularis oris	I	F&P(++)	+	N	N	S

IP, interference pattern; N, normal; P, partial; I, increased; F&P, fibrillation potentials & positive sharp waves; S, single.

R2 (Table 3). These results corresponded with incomplete bilateral facial nerve lesions, which highly indicates lower motor neuron lesions. Despite consecutive rehabilitation program including speech therapy, occupation therapy and transcutaneous electrical stimulation therapy, severity of FNP showed no interval change in a 6-month follow-up at outpatient basis.

The patient was informed that data concerning the cases would be submitted, and he provided informed written consent for publication of this case report and accompanying images.

Table 3. Blink Reflex Study

Side	R1	R2 ipsilateral	R2 contralateral
Left	No response	No response	No response
Right	No response	No response	No response

Discussion

Post-traumatic FNP usually results from fracture of the base of the skull or acoustic trauma [5]. To our knowledge, the present case is unique in that, delayed onset and persistent bulbar symptoms appeared after TBI were diagnosed as bilateral FNP via electrodiagnostic approach. Compared with the previously reported cases of bilateral FNP after trauma [6–8], there is a difference between the timing of symptom onset and electrodiagnostic study. One of the primary causes of bulbar symptoms such as decreased facial expression and dysarthria after cranioplasty in our patient was bilateral temporal bone fracture, which was initially confirmed by CT. Delayed onset of facial paralysis may be the result of bleeding into the surrounding structures of facial nerve, leading to gradual edematous status which can induce external compression of the nerve [8]. Moreover, existence of blood clots in an artery or vein can lead to ischemic damage and a compressive effect [8]. Considering the gradual deterioration after cranioplasty, the possibility of facial nerve damage during surgery cannot be excluded.

Electrodiagnostic approaches for bilateral FNP are performed through facial nerve conduction and blink reflex study. No responses for blink reflex study can be observed as in our case, where the severity of the damage and the prolonged symptom onset may have affected the test results. Additional evaluation such as brain imaging might be required depending on the results. Furthermore, it is also important in determining whether to perform treatment targeting peripheral nerve lesions regarding the rehabilitation strategies, such as electrical stimulation therapy. Obtaining degeneration ratio through electroneurography is significant to predict recovery from facial palsy. It was suggested that sequelae of facial palsy might persist, and effect of the treatment might not be favorable for our patient. Additional tests such as trigeminal somatosensory evoked potential might have been required, which was unfeasible due to poor cooperation of

the patient.

When bulbar symptoms affecting quality of life emerge from the patient, an early electrodiagnostic study can be performed for the prompt initiation of rehabilitation treatment through early diagnosis in the patient with bilateral FNP. Physicians should get acquainted that delayed onset bulbar symptoms may occur in patients with TBI, despite the absence of bilateral temporal bone fractures, and symptoms may not appear in the early stages. Outcomes of TBI are often fatal especially when parenchymal injury is present, which leads to failure of early detection of bilateral FNP. Early diagnostic intervention may be necessary if patients exhibit incomplete eye closure or labile affect at the time of admission. Since an early electrodiagnostic study can be significant evidence to determine the possibility of surgery, being alert to these symptoms might be the attitude that physicians dealing with TBI patients should have.

Our case has a limitation regarding the measurement tool. We used H-B scale for evaluation tool, and since it is a scoring system that comprehensively grades each section of the face, overall scoring may be inaccurate if a large deviation exists for each section. Furthermore, bilateral FNP would not be easily recognizable in H-B scale due to difficulty of comparing both sides. In our case, however, left side showed more severe feature compared to right side, which might have been acceptable for applying H-B scale.

In conclusion, our case presents rare medical condition of delayed onset bilateral FNP occurred after TBI, which can be a diagnostic challenge. Early diagnosis based on electrodiagnostic study should be considered for proper treatment, which can achieve optimal functional recovery after bilateral FNP.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Brachial Plexopathy Due to Upper-Extremity Deep Vein Thrombosis: A Case Report

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Upper-extremity deep vein thrombosis (DVT) accounts for 1% to 4% of DVT cases. Brachial plexus injury can result from venous distention due to thrombosis in the upper extremity, leading to neurologic deficits. We report a patient diagnosed with brachial plexopathy caused by venous thrombosis in the upper extremity. A 66-year-old female patient with a medical history of Parkinson disease and dementia presented to the emergency department with drowsy mental status. She was diagnosed with pre-renal acute kidney injury with multi-organ dysfunction. On the second hospital day, right upper extremity edema and muscle weakness were observed. On the sixth hospital day, the patient's overall medical condition improved after conservative treatment, but severe edema was noticed, and muscle weakness did not show significant improvement in the right upper extremity. Computed tomography showed multifocal pulmonary thromboembolism and DVT in the pulmonary arteries, superior vena cava, and popliteal veins. After anticoagulant administration, the swelling subsided, but the weakness in the entire right upper extremity did not improve. Electromyography demonstrated right brachial pan-plexopathy involving the upper, middle, and lower trunks. A follow-up examination showed no significant improvement of muscle strength and function in the right upper extremity at 3 months after the first hospital day.

Keywords: Upper extremity; Brachial plexus neuropathies; Venous thrombosis

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Introduction

Upper-extremity deep vein thrombosis (DVT) accounts for 1% to 4% of all DVT, and the incidence is much lower than that of the lower extremities, which is reported as approximately 9.1% to 11.1% [1]. Eighteen to sixty-seven percent of upper-extremity DVT occurs most frequently in the subclavian vein, followed by the axillary vein and the brachial vein [2]. Risk factors for upper-extremity DVT include hypercoagulable state, intravenous catheter, trauma, malignancy, vigorous upper extremity movement, and anatomical abnormalities of the thoracic outlet structure [3].

When DVT of upper extremities results in venous congestion, the patient complains of discomfort or changes in skin color. In severe cases, the brachial plexus is compressed and neurological symptoms such abnormal sensation and muscle weakness may occur. A case is reported that excessive flexion of the neck during cranial incision surgery causes the compression of internal jugular vein [4], which lead to swelling of the neck and upper extremities and brachial plexus injury occurs due to venous congestion in upper extremity.

Although cases of brachial plexus injury due to venous congestion have been reported; however, a case of brachial plexus injury due to upper-extremity DVT is rare, and the extent as well as prognosis of brachial plexus injury are not well known. In this case, we report clinical manifestations and electromyographic

characteristics of a patient diagnosed with brachial plexus injury due to upper-extremity DVT.

Case Report

A 66-year-old female patient with medical history of Parkinson disease and dementia presented to the emergency department with the chief complaint of deterioration of consciousness. She was admitted to nephrology department with diagnosis of acute renal failure and multiple organ failure due to bacterial sepsis, *Staphylococcus capitis* found on peripheral bloody culture study, and fluids and antibiotic (piperacillin/tazobactam) treatment began.

D-dimer was 5.66 mg/L fibrinogen equivalent unit (FEU) (normal range, 0-0.55 mg/L FEU) at the first laboratory result. At the 2nd hospital day, mild edema and muscle weakness were observed in right upper extremity. At the 6th hospital day, the patient showed increase in consistency with following and responding to simple verbal commands, but severe edema in whole upper extremity including shoulder girdle was noticed and muscle weakness did not show significant improvement in the right upper extremity. At the 8th hospital day, we assessed possibility of compartment syndrome, but the radial pulse was intact and pain, pallor, or abnormal sensation was not found. In manual muscle strength test, muscle strength was measured as 1 out of 5 for shoulder flexion, abduction, adduction and external rotation, elbow and wrist flexion and extension, finger flexion, extension, adduction and abduction, and skin ulceration on the lateral side of the right shoulder was observed. Imaging studies were performed to exclude central nervous system disease in brain, cervical spine, shoulder, and rib, and blood coagulation test was also performed, which all showed no abnormal findings. However, D-dimer, 9.57 mg/L FEU, was elevated in comparison to the first laboratory result. At the 14th hospital day, computed tomography (CT) venography showed multiple thrombosis in bilateral pulmonary arteries, superior vena cava, and popliteal vein. Central line was inserted, starting with anticoagulant treatment (rivaroxaban 15 mg) (Fig. 1). The multilayer bandage treatment in right upper extremity was started at the 18th hospital day and continued before transfer to Department of Rehabilitation Medicine.

At the 30th hospital day, she was transferred to the Department of Rehabilitation Medicine for edema management. The right upper extremity edema disappeared, but muscle weakness and function in the right upper extremity did not show any significant improvement, although we could not assess sensory function in right upper extremity due to dementia. At the 47th hospital day, the electromyography was performed to assess the possibility of neuropathy in upper extremity. The sensory nerve conduction study showed that there was no sensory nerve action potential (SNAP) in right median and ulnar nerve, and the amplitude of SNAPs in right radial, lateral and medial antebrachial nerve was decreased. In motor nerve conduction study, there was no compound motor action potential (CMAP) in right median nerve, and amplitude of CMAPs in the ulnar nerve, radial nerve, axillary nerve, musculocutaneous nerve, and suprascapular nerve was decreased (Table 1). In needle electromyography, abnormal spontaneous activity potentials were found in infraspinatus, pectoralis major, deltoid, biceps brachii, triceps, brachioradialis, flexor carpi radialis, extensor carpi radialis, first dorsal interosseous, flexor carpi ulnaris, abductor pollicis brevis and extensor indicis muscles, which led to the diagnosis of right brachial panplexopathy (Table 2).

At 3 months after the first hospital day, the muscle strength in right upper extremity was 3 out of 5 in elbow flexion and extension. However, there was no change in shoulder flexion, abduction, adduction and external rotation, finger flexion, extension, adduction and abduction, and wrist flexion and extension, with a score of 1 out of 5, and no significant change was observed in follow-up electromyography study.

Discussion

Upper-extremity DVT is classified into primary and secondary on the basis of pathogenesis. Primary upper-extremity DVT refers either to effort thrombosis (so-called Paget-Schroetter syndrome) or idiopathic, which account for about 20% of all upper DVT. Secondary DVT results from intravenous catheter, tumors, and hypercoagulation [5].

Although upper-extremity DVT is very rare compared to the

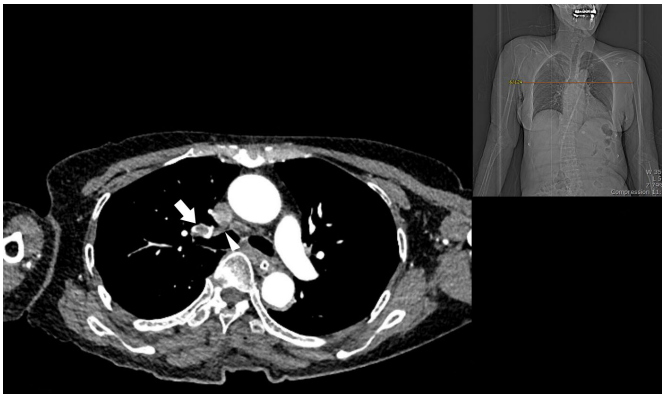

Fig. 1. Computed tomography venography showed multiple thrombi in the pulmonary arteries (arrow) and superior vena cava (arrowhead).

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Table 1. Nerve Conduction Studies

Nerve	Simulation site	Recording site	Latency (ms)	Amplitude	Conduction velocity (m/s)
Motor nerve conduction					
Rt. median	Wrist	APB	NR	NR*	NA
	Elbow	APB	NR	NR*	NA
Rt. ulnar	Wrist	ADM	1.85	0.3	NA
	Elbow	ADM	NR	NR*	NA
Rt. MSC	EP	Biceps	5.45	0.2*	NA
Lt. MSC	EP	Biceps	4.55	6.7	NA
Rt. axillary	EP	Deltoid	3.2	0.4*	NA
Lt. axillary	EP	Deltoid	4.2	3.3	NA
Rt. radial	Upper arm	EIP	5.55	2.7*	NA
Lt. radial	Upper arm	EIP	4.95	7.4	NA
Rt. suprascapular	EP	IF	5.45	0.3*	NA
Lt. suprascapular	EP	IF	4.35	2.4	NA
Sensory nerve conduction					
Rt. median	Wrist	Hand	NE	NE*	NA
Rt. ulnar	Wrist	Hand	NE	NE*	NA
Rt. radial	Wrist	Hand	2.4	16.5*	NA
Lt. radial	Wrist	Hand	2.5	43.5	NA
Rt. LABC	Elbow	Elbow	1.7	4.4*	NA
Lt. LABC	Elbow	Elbow	1.95	23.4	NA
Rt. MABC	Elbow	Elbow	2.1	7.3*	NA
Lt. MABC	Elbow	Elbow	2.1	12.3	NA

Amplitudes are measured in millivolt (mV, motor) and microvolt (μV, sensory).
Rt., right; APB, abductor pollicis brevis; NR, no response; NA, not assigned; ADM, abductor digiti minimi; MSC, musculocutaneous; EP, Erb's point; Lt., left; EIP, extensor indicis proprius; IF, infraspinatus; NE, not evoked; LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous.
*Abnormal findings are represented with asterisks; an abnormal finding was defined by a greater than 50% reduction of amplitude or 30% delay of latency compared to the unaffected side, or non-evoked sensory nerve action potential and compound motor action potential on the affected side.

Table 2. Needle Electromyography

Muscle	IA	Spontaneous		MUAP			Recruitment pattern/IP
		Fib/PSW	Other	Amplitude	Duration	Polyphasic	
B. C5-T1 PSP		Poor resting					
Rt. SERR	N	None	None	N	N	N	Disc*
Rt. RHOMB	N	None	None	N	N	N	Single*
Rt. INFR	N	1+/2+	None	N	N	N	Single*
Rt. PEC	N	2+/0*	None	N	N	N	Single*
Rt. DELTOID	N	2+/2+*	CRD1+*	N	N	N	Single*
Rt. BB	N	0/1+*	None	N	N	INC*	Disc*
Rt. TRICEPS	N	0/1+*	None	N	N	N	Disc*
Rt. BR	N	0/1+*	None	N	N	N	Disc*
Rt. FCR	N	0/4+*	None	N	N	N	Disc*
Rt. ECRB	N	0/2+*	None	N	N	N	Single*
Rt. FDI	N	0/3+*	None	N	N	N	Single*
Rt. FCU	N	0/3+*	None	N	N	N	Single*
Rt. APB	N	0/3+*	None	N	N	N	Single*
Rt. EIP	N	0/3+*	None	N	N	N	Single*

IA, insertional activity; Fib, fibrillation; PSW, positive sharp wave; MUAP, motor unit action potentials; IP, interference pattern; B., both; PSP, paraspinal; Rt., right; SERR, serratus anterior; N, normal; Disc, discrete; RHOMB, rhomboid major; INFR, infraspinatus; PEC, pectoralis major; CRD, complex repetitive discharges; BB, biceps brachii; INC, increased; BR, brachioradialis; FCR, flexor carpi radialis; ECRB, extensor carpi radialis brevis; FDI, first dorsal interosseous; FCU, flexor carpi ulnaris; APB, abductor pollicis brevis; EIP, extensor indicis proprius.
*Abnormal values.

lower extremity DVT, it can induce severe clinical symptoms that cause disability in upper extremity due to complications such as pulmonary embolism, superior vena cava syndrome, phlebitis, pain, and edema. Pulmonary artery embolism, which can lead to fatal clinical course, occurs in about one third of patients with upper-extremity DVT [6].

Considering the clinical characteristics of DVT in upper extremity, anticoagulant administration is urgently required to relieve acute symptoms caused by venous compression and to prevent complications [7]. The patient was diagnosed with multiple organ failure due to bacterial sepsis, which may be the potential factor to induce multiple thrombosis in bilateral pulmonary arteries, superior vena cava, and popliteal vein.

Superior vena cava occlusion secondary to thrombosis, potentially life-threatening medical emergency, may induce superior vena cava syndrome presenting with dyspnea, facial swelling, neck distension. In our case, DVT computed tomography showed multiple thrombosis in bilateral pulmonary arteries, superior vena cava, and popliteal vein, but total occlusion was not found. The multiple thrombosis may lead to venous congestion which worsened edema in right upper extremity. After multiple thrombosis were found, anticoagulant treatment and multilayer bandage were performed, which relieved edema, but the muscle weakness in right upper extremity continued after edema disappeared.

In the previous case reports about upper-extremity DVT, venous thoracic outlet syndrome occurred due to thrombus in the axillary subclavian vein [8] or brachial plexopathy occurred secondary to endovascular stent [9]. But specific clinical characteristics or extent of brachial plexus injury caused by venous congestion have not been reported. This case showed the brachial panplexopathy, in which all 3 branches of brachial plexus were damaged. It indicates that brachial plexus injury was more widespread, unlike thoracic outlet syndrome having injury in lower trunk of brachial plexus due to venous congestion, because it failed to detect upper-extremity DVT and initiate anticoagulation early.

Upper-extremity DVT is accompanied by pain and limitations in performing activities of daily living due to post-thrombotic syndrome, which can cause upper extremity dysfunction and significantly lower quality of life [10]. At 3 months after the first hospital day, in this case, the upper extremity muscle strength showed no improvement except for elbow flexion and extension, and severe upper extremity dysfunction continued. Although the incidence of upper-extremity DVT is much lower than that of the lower extremities DVT, this case demonstrates that brachial plexus injury due to DVT causes more significant impairment in daily activities in lower extremity DVT. It is important that upper-extremity DVT should require early diagnosis and treatment.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Instructions for Authors

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Journal of Electrodiagnosis and Neuromuscular Diseases (*J Electrodiagn Neuromuscul Dis*, JEND), an official journal of the Korean Association of EMG Electrodiagnostic Medicine, is published Three times a year. It regards all aspects of EMG, electrodiagnostic medicine, and neuromuscular diseases, including clinical practice, experimental and applied research, and education, and its formal abbreviated journal name is J Electrodiagn Neuromuscul Dis.

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Conclusions should avoid unqualified statements that are not adequately supported by the data and describe briefly novel findings of the study, according to the purpose of the study.

Acknowledgment

If necessary, persons who have made contributions to the study, but who are not eligible for authorship may be named in this section. Their contribution must be specified, such as data collection, financial support, statistical analysis, or experimentation.

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Sample References

1) Journals:

Authors: full title of the article. journal name year;volume:the first and last page number.

(e.g., Curr A, Dietz: Traumatic cervical spinal cord injury: relation between somatosensory evoked potentials, neurologic deficit and hand function. Arch Phys Med Rehabil 1996;77:48–53.)

2) Book:

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(e.g., Cailliet R: Shoulder pain. 3th ed. Philadelphia: FA Davis; 1991, pp32–35.)

3) Book chapter

Authors: title of the chapter. In: editor. The book title. edition. place: publisher; year, the first and last page number.

(e.g., Kottke FJ: The neurophysiology of motor function. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: Saunders; 1990, pp234–269.)

4) Online resource

National Library of Medicine: Fact sheet: AIDS information resources [Internet]. Bethesda: National Library of Medicine; 2003 [cited 2007 Mar 26]. Available from: http://www.nlm.nih.gov/pubs/factsheets/aidsinfs.html.

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