Evidence that impaired motor conduction in the bilateral ulnar and tibial nerves underlies cervical spondylotic amyotrophy in patients with unilateral deltoid muscle atrophy

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Abstract:

Introduction: The clinical entity of cervical spondylotic amyotrophy (CSA) is characterized by severe muscle atrophy in the upper extremities with insignificant sensory deficits in patients with cervical spondylosis. However, the pathogenesis of CSA is still unclear.

Methods: We assessed electrophysiological motor conduction through the corticospinal tract and ulnar and tibial nerves, which do not supply the deltoid or biceps muscles, of 18 patients with CSA, 12 patients with compressive cervical myelopathy, and 18 control subjects with cervical spondylotic radiculopathy. Motor evoked potentials following transcranial magnetic stimulation and M-waves and F-waves following electrical stimulation were measured from the bilateral abductor digiti minimi muscles (ADMs) and abductor hallucis muscles (AHs). The peripheral conduction time (PCT) was calculated from the latencies of the CMAPs and F-waves as follows: (latency of CMAPs + latency of F-waves - 1) / 2. The central motor conduction time (CMCT) was calculated by subtracting the PCT from the onset latency of the MEPs.

Results: The M-wave (M) latency and minimum F-wave (Fmin) latency from the ADM, and Fmin-M latency from the ADM/AH were significantly longer in the CSA group than in the other groups, on both the affected (p = 0.000-0.007) and unaffected sides (p = 0.000-0.033). F-wave persistence from the bilateral ADMs was significantly lower in the CSA group than in the other groups (p = 0.000-0.002). Among the CSA patients, there were no significant differences in these parameters between the affected and unaffected sides. The CMCT showed no significant differences between the CSA and control groups, but significant differences between the CSA and CCM groups (p = 0.000-0.004).

Conclusions: CSA patients with unilateral deltoid muscle atrophy had subclinical impairments of lower motor neurons and/or peripheral axons in the ulnar nerve, and subclinical impairments of peripheral axons in the tibial nerve. These motor impairments may have originally existed in these individuals before the onset of CSA.

Keywords:

central motor conduction time, cervical spondylotic amyotrophy, cervical spondylotic radiculopathy, F-wave, motor evoked potentials

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Introduction

Keegan first reported dissociated motor loss in the upper extremity of a patient with cervical spondylosis in 1965[1]. Thereafter, several authors reported the clinical entity of cervical spondylotic amyotrophy (CSA), which is characterized by severe muscle atrophy in the upper extremities with insignificant sensory deficits in patients with cervical spondylosis[2,3].

Two different mechanisms have been proposed for the pathophysiology of CSA. Keegan demonstrated that selective ventral motor root lesions were the cause of dissociated motor loss in the upper extremity of patients with cervical spondylosis[1]. On the other hand, others found by magnetic resonance imaging (MRI) that an anterior horn lesion in the spinal cord was the cause of CSA[4,5]. Shinomiya and colleagues found four types of neural injury to a cervical root or the spinal cord among patients with CSA by examining

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neuroradiologic and intraoperative electrophysiologic data[2]. However, the pathogenesis of CSA is still unclear. An interesting question is why severe atrophy of the deltoid muscles occurs in patients with CSA, but does not commonly occur among patients with acute cervical spondylotic radiculopathy (CSR), even though cervical root compression exists. Patients with acute CSR usually have radicular pain[6] without severe muscle atrophy, despite cervical nerve root compression.

We hypothesized that patients with CSA have distinctive impairment of the motor pathway in lower motor neurons, such as the anterior horn in the spinal cord or peripheral nerve. We quantitatively assessed the electrophysiological motor conduction of the corticospinal tract and F-waves in the ulnar and tibial nerves, which do not supply the deltoid or biceps muscles, of patients with CSA.

Materials and Methods/Case Material

2.1. Patients with CSA or control subjects

Eighteen patients (all men) with CSA (CSA group) and twelve patients (6 women and 6 men) with compressive cervical myelopathy (CCM, CCM group), who underwent surgical decompression in the Department of Orthopaedic Surgery at Hiroshima University Hospital between 2010 and 2014 and exhibited neurological improvement after surgery, were included in this study, along with eighteen control subjects (8 women and 10 men). One subject each in the CSA and control groups, and two patients in the CCM group, had diabetes; none of these patients had polyneuropathic symptoms. The age and height of the CSA, CCM, and control groups were 61 ± 8.6 (46-78), 67 ± 6.9 (54-77), and 59 ± 9.8 (38-72) years and 164 ± 6.1 (153-176), 163 ± 6.8 (150-170), and 160 ± 9.3 (143-175) cm, respectively [mean ± SD (range)], with no significant differences between the three groups. All patients provided written informed consent after a full explanation of the treatment. The patients and their families were informed that data from the case would be submitted for publication, and gave their consent. Approval was granted by the institutional review board and informed consent was obtained from each patient or candidate. This study was conducted before any surgical treatments were administered.

The patients with CSA had severe unilateral muscle weakness, less than manual muscle testing (MMT) grade 3, muscle atrophy of the deltoid and/or bicep muscle as confirmed by neurological testing, the presence of positive sharp wave and fibrillation potentials on needle electromyography (Fig. 1A), decreasing amplitude of compound muscle action potentials (CMAPs) recorded in the affected deltoid and biceps muscles following brachial plexus stimulation (Fig. 1B), and T2-weighted magnetic resonance imaging (MRI). The amyotrophy was found to have been due to cervical spondylosis in all 18 CSA patients. The CSA patients did not have hypoesthesia in their four limbs, spastic gait disturbance, or weakness in their hands and feet. Four patients with other thoracic spinal cord, cauda equina, or peripheral nerve disorders were excluded. All patients had a routine examination (such as needle electromyography, a motor/sensory nerve conduction study, and/or blood exam) by a neurologist in our institution to exclude other neurological diseases, such as chronic inflammatory demyelinating polyneuropathy, multifocal motor neuropathy, or motor neuron disease. In all of the CSA patients, the compression site was a unilateral C4, C5, and/or C6 spinal nerve root, and/or anterior horn in the spinal cord at the C3-4, C4-5, and/or C5-6 disc level as observed on MRI, without compression to the C8 or T1 nerve root. Table 1 shows the compression sites as observed on MRI and characteristics of the 18 CSA patients. In nine of the 18 patients with CSA, an intramedullary high-intensity zone (HIZ) was detected on T2-weighted MRI (Fig. 1C, Table 1).

The patients in the CCM group presented with symptoms of myelopathy, such as numbness or sensory disturbance in the upper and/or lower limbs, clumsiness in the hand, spasticity in the lower limbs, and/or bladder and bowel dysfunction, but no muscle weakness less than MMT grade 3 in the upper limbs. Myelopathy in each patient was confirmed by physical examination, and cord compression was present between C4-5 disc level on myelography findings. T2 weighted MRI demonstrated intramedullary HIZ at C4-5 disc level in all patients in the CCM group. The CCM was due to cervical spondylosis (9 patients), or OPLL (3 patients). Five patients were excluded from the study due to presenting with no HIZ on the T2-weighted MRI, muscle weakness less than MMT grade 3 in the upper limb, cauda equina, or entrapment peripheral nerve disorders.

2.2. Measurement of F-wave and calculation of central motor conduction time

The F-wave was measured and central motor conduction time (CMCT) was calculated according to a previously-reported method[7-12]. Transcranial magnetic stimulation (TMS) was delivered using a round coil (Model 200; Magstim, Whitland, UK) with 20% above the threshold intensity. The motor evoked potentials (MEPs) following TMS were recorded from the bilateral abductor digiti minimi muscles (ADMs) and abductor hallucis muscles (AHs; Fig. 2A). MEPs were recorded at least 4 times from each muscle; the responses were superimposed and the shortest latency was determined.

Continuous current stimulation at supramaximal intensity (0.2 ms square wave pulses; Viking IV, Nicolet Biomedical, Madison, WI, USA) was applied to the ulnar and tibial nerves at the wrist and ankle, respectively, and CMAPs (M-waves) and F-waves were recorded from the ADMs and AHs. Thirty-two serial responses were obtained from each muscle. The M-wave latency (M latency) and the shortest F-wave latency among the 32 F-wave responses (Fmin latency) were determined (Fig. 2B). The Fmin-M latency was defined as the shortest latency measured from the onset of

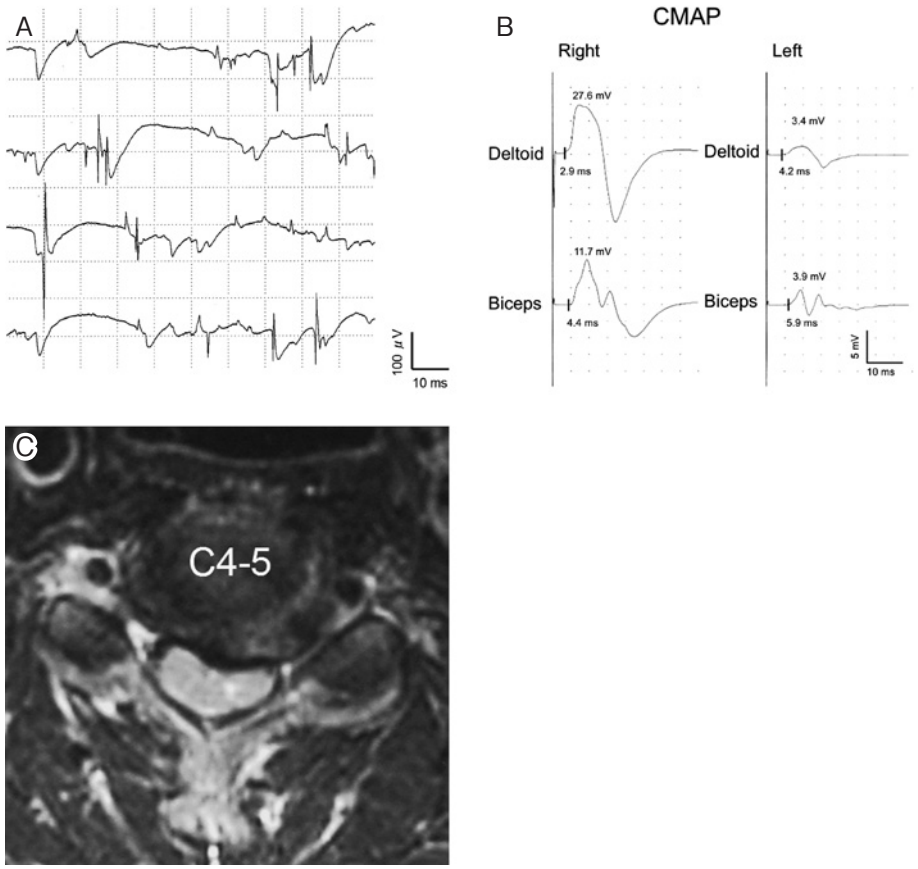

Figure 1. Waveform of needle electromyography in the left deltoid muscle (A), waveforms of CMAPs recorded from the right/left deltoid and biceps muscles following brachial plexus stimulation (B), and axial T2-weighted MR image of the spine at the C4-5 level (C) of a representative patient with weakness of the left deltoid and biceps muscles in the CSA group (Patient 8). (A) Positive sharp waves are seen in the waveform under resting conditions. (B) The amplitudes of CMAPs recorded from the left deltoid and biceps muscles are lower than those from the right side. (C) Compression of the left anterior horn due to posterior prominence of a vertebral bone spur and an intramedullary high-intensity zone in the left anterior horn are seen in the MR image.

the M-wave to the onset of the following F-wave. The persistence (i.e., the occurrence of F-wave responses to 32 consecutive stimuli) was assessed and was expressed as a ratio, with 1.00 indicating the occurrence of 32 F-wave responses to all 32 consecutive stimuli. The sensitivity for the evaluation of F-waves was set at 200 μV per division, and an amplitude of 50 μV or higher was considered acceptable for further calculations. All muscle responses were recorded after they traversed a bandpass filter of 0.5-2000 Hz using a commercially available system (Viking IV; Nicolet Biomedical). An epoch of 100 ms elapsed after the stimulation was digitized at a sampling rate of 5 kHz.

The peripheral conduction time from the spinal cord to the muscle was calculated from the M- and F-wave latencies as follows: (latency of M-wave + latency of F-wave - 1) / 2. The CMCT, i.e., the conduction time from the motor cortex to the spinal motor neuron, was calculated by subtracting the peripheral conduction time from the latency of onset of the MEPs.

2.3. Statistical analysis

The F-wave and CMCT values were compared among the CSA, CCA, and control groups using a multivariate analysis of variance followed by the Tukey-Kramer post-hoc test. The F-wave and CMCT values were calculated from the right and left side separately. The patients in the CSA group had severe unilateral muscle weakness and atrophy of the deltoid and/or biceps, and thus the F-wave and CMCT values were split into values from the affected side and those from the unaffected side. However, the subjects in the CCM and control groups did not have significant muscle weakness less than MMT grade 3. Therefore, the F-wave and CMCT values from each side in the CSA group were compared with those from both sides in the CCM group or control group. The F-wave and CMCT values determined from the ADMs and AHs were compared between the affected and unaffected sides in the CSA group using the paired t-test for nonparametric statistical analysis. Statistical significance was determined at $p < 0.05$.

Results

Table 2 shows the F-wave parameters and CMCTs determined from the ADMs and AHs of the CSA, CCM and control groups. The Fmin, M, and Fmin-M latencies from the ADM, and Fmin-M latencies from the AH were significantly longer in the CSA group than in the CCM or control group, on both the affected (ADM, p = 0.000-0.026; AH, p = 0.007-0.026) and unaffected sides (ADM, p = 0.000-0.018; AH, p = 0.002-0.006). Among the CSA patients, there were no significant differences in the Fmin latency, M latency, and Fmin-M latency determined from the ADM between the affected and unaffected sides (Fmin latency, p = 0.332; M latency, p = 1.000; Fmin-M latency, p = 0.481), nor were there differences in the Fmin latency, M latency, and Fmin-M latency determined from the AH between the affected and unaffected sides (Fmin latency, p = 0.238; M latency, p = 0.629; Fmin-M latency, p = 0.096). The persistence of the F-wave from the ADM was significantly lower in the CSA group than in the CCM or control group, on both the affected (p = 0.001 vs. CCM group, p = 0.002 vs. control group) and unaffected sides (p = 0.000). The persistence of the F-wave from the AH on both the affected and unaffected sides was 1.000 across the CSA, CCM, and control groups.

The CMCT from the ADM on both the affected and unaffected sides, and from the AH on the unaffected side in the CSA group was significantly shorter than that in the CCA group (ADM, p = 0.000; AH, p = 0.004). The CMCT from the ADM and AH on both the affected and unaffected sides showed no significant differences between the CSA and control groups (p = 0.116-0.909).

Table 1. Characteristics of the Patients with CSA[a].

Case	Age (yr)	Gender	Affected side	Compression site		HIZ[b]
				nerve root	anterior horn	
1	46	male	Right	none	C3-4, 4-5	C4-5
2	60	male	Left	C5	C4-5	none
3	53	male	Right	C5, 6	C4-5	none
4	58	male	Left	C5	C4-5	C4-5
5	62	male	Right	C5	none	none
6	58	male	Right	C4, 5, 6	C4-5	C4-5
7	67	male	Left	C5	none	none
8	51	male	Left	C5	C4-5	C4-5
9	67	male	Left	C5	C3-4, 4-5	C3-4
10	58	male	Left	none	C5-6	C5-6
11	64	male	Left	C6	none	none
12	69	male	Right	none	C4-5	none
13	63	male	Right	C5	C3-4	C3-4
14	77	male	Right	none	C4-5	C4-5
15	51	male	Left	C5	C4-5	C4-5
16	59	male	Right	C5, 6	C4-5	none
17	78	male	Right	C4	C4-5	none
18	62	male	Left	none	C4-5	none

[a]CSA, cervical spondylotic amyotrophy

[b]HIZ, level at which the intramedullary high-intensity zone appears on T2-weighted MRI

Discussion

We found that the F-wave parameters from the bilateral ADMs and AHs, and the distal motor latency (M latency) from the ADMs of the CSA patients, were significantly prolonged compared with those of the control subjects or patients with CCM at the C4-5 level, even though our CSA patients had no atrophy nor weakness in these muscles. In the CSA patients, prolongation of F-wave latency was observed in all four limbs and not just in the affected limb. The F-wave persistence from the ADM was significantly lower in the CSA group than in the control group, on both the affected and unaffected sides. F-wave latencies elicited by distal stimulation represent the motor conduction time to and from the spinal cord along the entire motor nerve axon[13-16]. These results showed a similar pattern as that previously reported in patients with amyotrophic lateral sclerosis[17-19].

The ADMs and AHs are predominantly supplied by the ulnar and tibial nerves, respectively. The ulnar and tibial

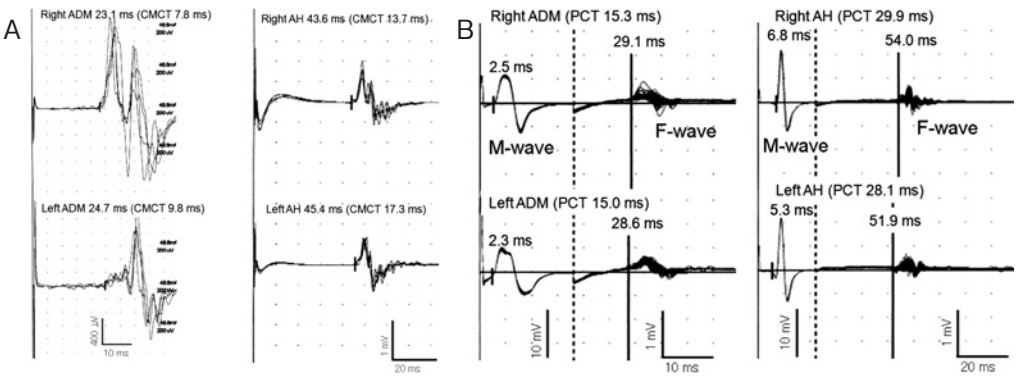

Figure 2. Representative MEPs, M-waves, and F-waves recorded from the patient with CSA presented in Figure 1. (A, B) Waveforms of MEPs recorded from the right/left ADM and AH following TMS (A), and M-waves and F-waves recorded from the right/left ADM and AH following electric stimulation of the ulnar and tibial nerves at the wrist and ankle (B), respectively, are shown.

Table 2. F-wave and CMCT Values of the CSA, CCM and Control Groups[a, b].

		CSA group (n=18)						CCM group (n=12)	Control group (n=18)
		Affected side	*p* value		Unaffected side	*p* value			
			vs. CCM	vs. Control		vs. CCM	vs. Control		
ADM[c]									
	Fmin latency[d]	28.1±1.5 (26.2-31.6)	0.007**	0.026*	27.8±1.7 (24.8-31.2)	0.002**	0.006**	25.4±2.5 (20.9-30.0)	25.8±2.0 (21.3-30.2)
	M latency[e]	3.0±0.6 (1.8-4.1)	0.000**	0.000**	3.0±0.4 (2.2-3.4)	0.000**	0.000**	2.4±0.4 (1.8-3.1)	2.2±0.3 (1.7-2.9)
	Fmin-M latency[f]	25.1±1.5 (23.1-28.3)	0.007**	0.025*	24.9±1.8 (22.1-28.3)	0.018**	0.069	23.0±2.4 (19.0-27.6)	23.5±1.9 (19.0-27.7)
	Persistence[g]	0.80±0.13 (0.63-1.00)	0.001**	0.002**	0.77±0.11 (0.63-1.00)	0.000**	0.000**	0.94±0.05 (0.81-1.00)	0.90±0.07 (0.72-1.00)
	CMCT	7.9±2.1 (5.1-13.2)	0.000**	0.826	7.8±1.7 (4.9-10.8)	0.000**	0.909	11.9±3.3 (7.1-17.9)	7.4±1.3 (5.2-10.1)
AH									
	Fmin latency	49.7±4.0 (44.3-59.9)	0.359	0.033*	50.0±4.1 (44.8-60.7)	0.212	0.013*	46.4±3.0 (41.1-50.2)	46.8±3.1 (39.6-52.0)
	M latency	4.6±1.0 (2.9-6.5)	0.937	0.519	4.4±0.9 (2.9-6.8)	0.753	0.789	4.7±1.0 (3.5-6.4)	4.2±0.7 (3.3-6.2)
	Fmin-M latency	45.1±3.5 (40.8-54.5)	0.007**	0.026*	45.6±3.6 (40.4-55.0)	0.002**	0.006*	41.7±2.4 (37.0-45.3)	42.5±2.9 (34.9-47.5)
	Persistence	1.00±0.00 (1.00-1.00)	1.000	1.000	1.00±0.00 (1.00-1.00)	1.000	1.000	1.00±0.00 (1.00-1.00)	1.00±0.00 (1.00-1.00)
	CMCT	15.9±2.1 (12.6-19.4)	0.059	0.116	15.1±1.8 (12.9-19.0)	0.004**	0.650	18.0±3.9 (11.6-26.6)	14.2±1.9 (10.5-18.1)

[a]CMCT, central motor conduction time; CSA, cervical spondylotic amyotrophy: CCM, compressive cervical myelopahty

[b]Data are shown as mean±SD (range).

[c]ADM, abductor digiti minimi muscle; AH, abductor hallucis muscle

[d]Fmin latency, minimum F-wave latency among 32 F-wave responses.

[e]M latency, M-wave latency.

[f]Fmin-M latency, latency from M-wave to the onset of F-wave.

[g]Persistence, the occurrence of F-wave responses to 32 consecutive stimuli.

*p<0.05 vs. CCM or control group, **p<0.01 vs. CCM or control group.

nerves are mainly supplied by nerve roots that are caudal to C7, while the compression level in our patients was rostral to or at the C5-6 level of the spinal cord and/or rostral to or at the C6 nerve root. The compression of a cervical ventral root and/or anterior horn could not be directly involved in the motor impairment of the bilateral ulnar and tibial nerves in our CSA patients. Thus, our results suggest that subclinical impairment of motor neuron excitability and axonal conductivity in levels that are caudal to the compression site existed in our CSA patients. Possible pathophysiological causes of these motor impairments in our CSA patients are secondary impairment of lower motor neuron excitability and peripheral axonal degeneration due to compression of the spinal cord. Among our 18 CSA patients, MRI demonstrated compression of the anterior horn in 15 patients and an intramedullary high intensity zone in 9 patients.

Recently, several groups reported motor neuron excitability below the level of spinal cord injury[20-23]. Curt and colleagues[20] studied the F-wave parameters in the median and ulnar nerves of healthy subjects and patients with spinal cord injury in the acute or chronic phase. A difference was not observed in the F-wave latency, but increased frequency of F-wave production was observed in the patients with spinal cord injury. Their results in patients with paralysis of all four limbs differ from the F-wave parameters in our CSA patients. Although there was greater deterioration of the spinal cord in the patients with paralysis of all four limbs than in our CSA patients, no prolongation of F-wave latency was demonstrated in the patients from the study by Curt and colleagues. Thus, the alterations in F-wave parameters may not have occurred secondary to compression of the spinal cord in our CSA patients.

Another possibility is that impairment of lower motor neuron excitability and peripheral axonal degeneration had originally existed in the CSA patients before compression of spinal nerve root or anterior horn. In other words, individuals with subclinical impairment of nerve conductivity in all four limbs may readily develop CSA upon compression of a cervical ventral root and/or anterior horn. If this is the case, it may be possible to screen patients preoperatively and provide more accurate counseling with regard to the risk of developing deltoid and/or bicep muscle weakness. A few stud-

ies reported electrophysiological data obtained from untreated patients with CSA. Shibuya and colleagues[24] reported electrophysiological data in three patients with CSA. They calculated CMCT values from the latency of the MEP and the tendon reflex from the deltoid and bicep muscles, and the F-wave latencies from the abductor pollicis brevis muscles (APBs) and ADMs. The F-wave latencies determined from the APBs and ADMs of their CSA patients were longer than those in the healthy subjects in the study by Puksa and colleagues[25], although Shibuya and colleagues did not discuss the possible causes of prolongation of F-wave latency. The results of Shibuya and colleagues were similar to the results of the present study. The mechanisms of the development of these impairments could not be clarified in the present study and are still unclear. However, if Fmin-M latency of the upper and lower limbs is prolonged, attention should be paid to the onset of CSA.

One limitation to this study is that we only investigated F-wave parameters in the ulnar and tibial nerves as a peripheral nerve conduction, because this study was retrospective. Further study is needed to investigate sensory nerve conduction as well as motor conduction in the median and peroneal nerves for patients with CSA. Another limitation is that we could not investigated differences in electrophysiological parameters between CSA patients and CCM patients with motor weakness. Further study is also needed to compare between those patient groups.

In conclusion, our results suggest that CSA patients with unilateral deltoid muscle atrophy had subclinical impairment of the lower motor neurons and/or peripheral axons in the ulnar nerve, and subclinical impairments of peripheral axons in the tibial nerves, which by themselves would not cause unilateral deltoid muscle atrophy. These motor impairments may have originally existed in these individuals before the onset of CSA.

Conflicts of Interest: The authors have no conflict of interest to declare.

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References

1. Keegan JJ. The cause of dissociated motor loss in the upper extremity with cervical spondylosis. J Neurosurg. 1965;23(5):528-36.
2. Shinomiya K, Komori H, Matsuoka T, et al. Neuroradiologic and electrophysiologic assessment of cervical spondylotic amyotrophy. Spine. 1994;19(1):21-5.
3. Ebara S, Yonenobu K, Fujiwara K, et al. Myelopathy hand characterized by muscle wasting. A different type of myelopathy hand in patients with cervical spondylosis. Spine. 1988;13(7):785-91.
4. Kameyama T, Ando T, Yanagi T, et al. Cervical spondylotic amyotrophy. Magnetic resonance imaging demonstration of intrinsic cord pathology. Spine. 1998;23(4):448-52.
5. Mizuno J, Nakagawa H, Hashizume Y. Cervical amyotrophy caused by hypertrophy of the posterior longitudinal ligament. Spinal Cord. 2002;40(9):484-8.
6. Abbed KM, Coumans J-VCE. Cervical radiculopathy: pathophysiology, presentation, and clinical evaluation. Neurosurgery. 2007;60 (1 Supp1 1):S28-34.
7. Kimura J, Yamada T, Stevland NP. Distal slowing of motor nerve conduction velocity in diabetic polyneuropathy. J Neurol Sci. 1979;42(2):291-302.
8. Maertens de Noordhout A, Remacle JM, Pepin JL, et al. Magnetic stimulation of the motor cortex in cervical spondylosis. Neurology. 1991;41(1):75-80.
9. Di Lazzaro V, Restuccia D, Colosimo C, et al. The contribution of magnetic stimulation of the motor cortex to the diagnosis of cervical spondylotic myelopathy. Correlation of central motor conduction to distal and proximal upper limb muscles with clinical and MRI findings. Electroencephalogr Clin Neurophysiol. 1992;85(5): 311-20.
10. Kaneko K, Taguchi T, Morita H, et al. Mechanism of prolonged central motor conduction time in compressive cervical myelopathy. Clin Neurophysiol. 2001;112(6):1035-40.
11. Nakanishi K, Tanaka N, Fujiwara Y, et al. Corticospinal tract conduction block results in the prolongation of central motor conduction time in compressive cervical myelopathy. Clin Neurophysiol. 2006;117(3):623-7.
12. Nakanishi K, Tanaka N, Kamei N, et al. Significant correlation between corticospinal tract conduction block and prolongation of central motor conduction time in compressive cervical myelopathy. J Neurol Sci. 2007;256(1-2):71-4.
13. Conrad B, Aschoff JC, Fischler M. [The diagnostic value of the F wave latency (author's transl)]. J Neurol. 1975;210(3):151-9.
14. Kimura J. Principles and pitfalls of nerve conduction studies. Ann Neurol. 1984;16(4):415-29.
15. King D, Ashby P. Conduction velocity in the proximal segments of a motor nerve in the Guillain-Barré syndrome. J Neurol Neurosurg Psychiatr. 1976;39(6):538-44.
16. Panayiotopoulos CP, Scarpalezos S, Nastas PE. F-wave studies on the deep peroneal nerve. J Neurol Sci. 1977;31(3):319-29.
17. Mills KR, Nithi KA. Peripheral and central motor conduction in amyotrophic lateral sclerosis. J Neurol Sci. 1998;159(1):82-7.
18. Argyropoulos CJ, Panayiotopoulos CP, Scarpalezos S. F- and M-wave conduction velocity in amyotrophic lateral sclerosis. Muscle Nerve. 1978;1(6):479-85.
19. Bradley WG. Recent views on amyotrophic lateral sclerosis with emphasis on electrophysiological studies. Muscle Nerve. 1987;10 (6):490-502.
20. Curt A, Keck ME, Dietz V. Clinical value of F-wave recordings in traumatic cervical spinal cord injury. Electroencephalogr Clin Neurophysiol. 1997;105(3):189-93.
21. Hiersemenzel LP, Curt A, Dietz V. From spinal shock to spasticity: neuronal adaptations to a spinal cord injury. Neurology. 2000;54 (8):1574-82.
22. Mangold S, Keller T, Curt A, et al. Transcutaneous functional electrical stimulation for grasping in subjects with cervical spinal cord injury. Spinal Cord. 2005;43(1):1-13.
23. Dietz V, Curt A. Neurological aspects of spinal-cord repair: promises and challenges. Lancet Neurol. 2006;5(8):688-94.
24. Shibuya R, Yonenobu K, Yamamoto K, et al. Acute arm paresis with cervical spondylosis: three case reports. Surg Neurol. 2005;63 (3):220-28.
25. Puksa L, Stålberg E, Falck B. Reference values of F wave parameters in healthy subjects. Clin Neurophysiol. 2003;114(6):1079-90.

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