Quantitative Analysis of High-voltage-electrical Spinal Cord Injury Using Diffusion Tensor Imaging

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Quantitative Analysis of High-voltage-electrical Spinal Cord Injury Using Diffusion Tensor Imaging

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ABSTRACT

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The aim of this study was to investigate the relationships between clinical outcomes and the diffusion as well as electrical conduction characteristics of the spinal cord in patients with high-voltage-electrical injury but showing no abnormal radiological findings. We recruited eight high-voltage-electrical injury patients and eight healthy subjects matched for age and sex. Diffusion tensor imaging (DTI) and central motor conduction time (CMCT) were acquired in both patient and control groups. We obtained the patient and control group DTI indices according to the spinal cord levels (from C2 to C7) and parts (anterior, lateral, and posterior). The DTI indices of fractional anisotrophy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD) were inter-group-compared; additionally, they

were intra-group-compared in relation to spinal cord level and part. The correlations among the DTI indices, CMCT, and neurological status of spinal cord injury also were analyzed. In the patient group relative to the control group, the FA value decreased and the MD and RD values increased in all of the regions of interest (ROIs), with statistical significance ($p<0.05$); the AD value did not differ. Within the patient group, comparing the anterior spinal cord with the lateral and posterior spinal cords, the FA decreased in the ROIs ($p<0.05$). The DTI indices did not differ by level. The ASIA motor score tended to correlate with the FA of the anterior spinal cord ($p=0.16$) and with CMCT ($p=0.12$). In conclusion, DTI revealed myelopathy in patients with high-voltage-electrical injury, and corroborated high-voltage-electrical spinal cord injury's reported pathophysiology, including myelinopathy and ascending homogeneous involvement more typically affecting the anterior spinal cord.

Key words: High-voltage electrical spinal cord injury, Diffusion tensor imaging

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I. INTRODUCTION

In the case of high-voltage-electrical injury, any structures between the entry and exit points of the electrical current can be injured, though low-resistance tissues such as muscles, nerves and blood vessels are less vulnerable to electrical burn. The reported incidence of spinal cord damage following high-voltage-electrical injury ranges between 2 and 8%.[1-4] Delayed spinal cord injury occurs a few days to a few months after high-voltage-electrical injury, and usually becomes permanent as

incomplete spinal cord injury.[5]

Magnetic resonance imaging (MRI) plays an essential role in the diagnosis of spinal cord lesions. However, in many reports, the MRI findings on high-voltage-electrical spinal cord injury have been "normal".[1,6-9] Even in a case of demyelination confirmed by pathologic diagnosis, no spinal cord lesion was found on conventional MRI.[6] This can be attributed to the low signal-to-noise ratio in conventional spinal cord MRI, which makes isolation of demyelinating lesion difficult.[7,10-13]

Electrophysiologic study yields accurate information necessary for diagnosis of high-voltage-electrical spinal cord injury. Slow central conduction time, as well as delayed latency of motor-evoked potential (MEP) or somatosensory-evoked potential (SEP), have been reported.[7,14-16] However, application of electrophysiologic study is sometimes difficult to apply to high-voltage-electrical-injury patients, owing to the painfulness of the procedures as applied to patients' burned skin or muscle injury.

Recently, several investigators have assessed the feasibility of DTI for various spinal cord lesions.[17-19] DTI, an advanced MR technique that is sensitive to the underlying neural microstructure, identifies alterations to the white-matter tract and quantifies them according to fractional anisotrophy (FA), mean diffusivity (MD), axial diffusivity (AD) and radial diffusivity (RD) indices. Patholgic findings on white-matter injury, whether axonopathy or myelinopathy, also can be inferred from DTI indices, though they are derived primarily from mathematical assumption.[20] If

there is accurate measurement to quantify myelopathy following high-voltage electrical injury, physicians would have important information to guess neurological outcome and establish proper rehabilitation plans.

We hypothesized that high-voltage electrical spinal cord injury would change diffusion characteristics compared to controls, leading to differences even in the patients with normal conventional MRI. In addition, there would be relationship among the DTI indices with the clinical status and electrophysiological findings, which might indicate its clinical significance. To prove these hypotheses, we studied the cervical spinal cord DTI in patients with cervical myelopathy following high-voltage electrical injury. If there were accurate quantification of myelopathy following high-voltage-electrical injury, physicians would have important information on which to base predictions of neurological outcomes and formulations of effective rehabilitation plans.

We hypothesized that high-voltage-electrical spinal cord injury would change diffusion characteristics relative to controls, leading to differences even in patients with normal conventional MRI, and, further, that there would be correlations among the DTI indices, the clinical status and the electrophysiological findings that might indicate its clinical significance. To prove these hypotheses, we studied the cervical spinal cord DTI results for patients with cervical myelopathy following high-voltage-electrical injury.

II. MATERIALS AND METHODS

1. Subjects

The subjects included eight delayed cervical spinal cord injury (following high-voltage-electrical injury) patients and eight injury-free control-group members matched for age and sex. The inclusion criteria were (1) age 50 or younger, (2) high-voltage-electrical injury by more than 1,000 volts, (3) electrical-current flow crossing cervical spinal cord, (4) upper-extremity weakness associated with hyperreflexia, or sensory impairment, (5) delayed CMCT or lack of MEP response, and (6) normal conventional MRI (T1- and T2-weighted images) taken when the patient had cervical myelopathy symptoms. The exclusion criteria were (1) previous history of cervical spine disease, (2) spinal cord injury occurring immediately after high-voltage-electrical injury, (3) abnormal lesion on brain CT taken after development of upper-limb weakness, (4) abnormal lesion on conventional spine MRI taken at same time as DTI, (5) cognitive dysfunction, and (6) contraindication for MRI. All of the patients' were male with ages ranging from 35 to 50 years (mean age: 42.0±5.3 years). The age- and gender-matched controls' ages ranged from 39 to 50 years (mean age: 42.9±4.2 years). The patients and controls all provided written informed consent of their participation in this study, which was approved by the Institutional Review Board of Hallym University Sacred Heart Hospital.

2. Clinical Evaluation of Spinal Cord Injury

Using the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI, 2009 version), 28 dermatomes on the bilateral side of each patient were evaluated for sensitivity to light touch and pin prick via tests performed upon DTI by one physiatrist.[21] The sensitivities were determined on the three-point scale: 0 (absent), 1 (impaired), or 2 (normal). Each American Spinal Injury Association (ASIA) sensory score (ASS) for light touch and pin prick is a summation, within a 0 – 112 range, of the scores for all 28 dermatomes. Additionally, the strengths of ten key muscles were evaluated and graded on a six-point scale between 0 (complete paralysis) and 5 (normal active movements, full range of motion against full resistance). These scores were then aggregated to generate an ASIA motor score (AMS) within a 0 – 100 range.

3. Electrophysiological Evaluation of Spinal Cord Injury

In both patient and control groups, each participant's MEP, F latency and M latency were recorded within seven days prior to DTI. The central motor conduction time (CMCT) of the corticospinal tract between the motor cortex and cervical spinal motor neuron was measured using methods detailed and employed in a previous study.[14]

To record the MEP, the participants were seated comfortably in a reclining armchair with both hands pronated on a pillow. Electromyography (EMG) data were collected from the first dorsal interosseus muscle of the dominant hand via surface electrodes placed over the muscle in a belly-tendon montage. The EMG activity was amplified using the Synergy EMG/EP system (Neuroscreen® Plus, Toennies, Germany), and the data were band-pass filtered at 10 to 1,000 Hz. The optimal scalp location ("hot spot") was determined using a transcranial magnetic stimulation system (Magstim Rapid1 Stimulator, Magstim Co., UK) with a 70 mm figure-of-eight coil. The handle of the coil was oriented 45° posterior to the midline so that the electromagnetic current would flow perpendicular to the central sulcus; the stimulator was moved over the scalp in 1 cm increments.[22] Once the hot spot was identified, single-pulse transcranial magnetic stimulation (TMS) was delivered to the location to obtain consistent maximum-amplitude MEPs with the muscle in slight contraction. The average latency of the shortest of four constant MEP responses was accepted. The M latency was recorded by conduction of the ulnar nerve, and an F wave analysis of the first dorsal interosseus muscle was performed. The minimum latencies of the F and M responses were obtained, and the CMCT was calculated as follows: MEP latency – (F latency + M latency -1)/2 (msec).[23]

4. MRI Acquisition

All MRI scanning was performed using the 3T Achieva® MRI scanner

(Phillips Medical Systems, USA). The MRI acquisition protocol consisted of an initial T2-weighted sagittal scan of the cervical spinal cord, a T1-weighted 3D axial scan, and DTI. T2-weighted MRI was obtained for following parameters: TR/TE = 3,000/120 msec, field of view = 250 (AP) x 250 (FH) x 36 (RL) mm. Next, a T2-weighted sagittal image was used to prescribe T1-weighted 3D MRI, which was obtained for the following parameters: TR/TE = 8.3/4.6 msec, field of view = 150 (AP) x 150 (FH) x 150 (RL) mm.

DTI data were obtained from the same anatomic location prescribed for the T2-weighted images, that is, from the second cervical spine to the first thoracic spine using the 3T Achieva® MRI Scanner (Phillips Medical Systems, US). Single-shot echo-planar imaging was used for the transverse DTI in the cervical spinal cord, according to the following parameters: number of diffusion gradient directions = 15, b value = 500 s/mm^2, TR/TE = 6,300/63 msec, field of view = 120(AP) x 120(FH) x 145(RL) mm, voxel size = 1.25(AP) x 1.5(RL) mm, slice number = 40, slice thickness = 3 mm with no gap.

5. DTI Analysis

For DTI analysis, the raw image files were converted to nifti files using MATLAB 2009b software (MathWorks, USA). The nifti files were split using FSL software (University of Oxford, UK) and then realigned according to their gradient directions using SPM8 software (College of London, UK). These preprocessed files

were loaded onto the MedINRIA 1.6 software (INRIA Sophia Antipolis, France), and the diffusion tensors were calculated. For evaluation of the DTI indices, the regions of interest (ROIs) were positioned on the individual axial FA images (C2-C7) by Thurnher`s method (Figure 1).[24] The T2-weighted sagittal image served as a reference for positioning of axial DTI and obtaining diffusion tensor data. Six ROIs were drawn manually onto the white matter on both the right and left sides of the anterior, lateral, and posterior spinal cords at the middle level of each spine body from C2 to C7, by one researcher blinded to the subject of the respective DTI. Each ROI contained three or four voxels. Thirty-six ROIs (6 ROIs/one spinal cord level x 6 levels) were obtained from each of the participants, and in each ROI, the DTI indices (FA, MD, AD (Lambda 1) and RD ((Lambda 2 + Lambda 3)/2)) were measured.

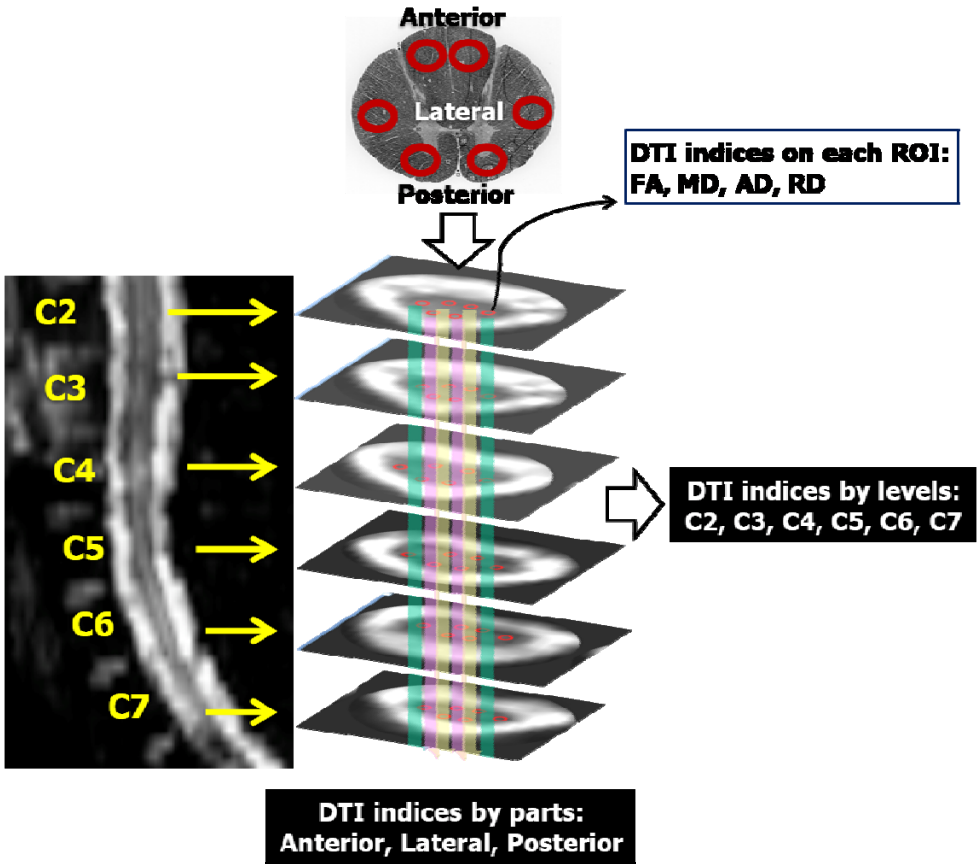

Figure 1. DTI indices in each ROI. Thurnher's technique was employed. On each axial FA map matching cervical spinal cords from C2 to C7, six ROIs in the anterior, lateral, and posterior spinal cords were assigned. The DTI indices (FA, MD, AD, and RD) of the ROIs located in the same area at each spinal level were averaged and compared between the patient and control groups.

We analyzed the differences among the DTI indices from 288 patient ROIs (36 ROIs/participant x 8 participants) and 288 control ROIs (36 ROIs/participant x 8 participants) by independent t-test with SPSS 13.0 (SPSS, USA). Next, the ROIs were classified according to their spinal cord levels and parts. The DTI indices by spinal cord level were calculated with six ROIs on one axial plane for each participant; those by spinal cord part were calculated with 12 ROIs (2 ROIs/level x 6 levels) located in the same part on each axial plane. The two sets of DTI indices were inter-group-compared by independent t-test; they were then intra-group-compared for level and part by one-way ANOVA and post-hoc Tukey test. $P<0.05$ was considered statistically significant.

6. Correlations among Clinical Parameters, Electrophysiologic Parameters and DTI Indices

Pearson's correlation coefficient was used to assess the correlations among the clinical parameters (ASS to light touch and pin prick; AMS), CMCT and DTI indices (FA, MD, AD, RD). In two patients, neither the AMS nor the ASS could be evaluated, due to arm amputation (case 6: transhumeral amputation; case 7: transradial amputation). Accordingly, these individuals were excluded from the correlation analysis (see Figure 2).

III. RESULTS

1. Neurological Status

Eight patients' ASIA impairment scale (AIS), AMS, and ASS to light touch and pin prick results are summarized in Table 1. All of the patients had incomplete spinal cord injury. The CMCT was delayed in six patients, and in two patients, it could not be calculated because the MEP was not evoked.

2. DTI Indices

In the inter-group comparison, FA decreased and MD and RD increased in the ROIs for all levels (C2~C7) and all parts (anterior, lateral and posterior) in the patient group relative to the control group, with significance ($p<0.05$) (Table 2 and Table 3). AD did not differ between the groups. In the intra-group comparison, FA decreased in the ROIs for the anterior spinal cord relative to the lateral and posterior spinal cords in the patient group ($p<0.05$) (Table 3). MD, AD and RD did not differ by level or part of spinal cord in the patient group. In the control group, none of the DTI indices differed by spinal cord level or part.

3. Correlation of AMS, CMCT, and DTI Indices

The patients' motor functions showed a tendency to be affected by the FA of the anterior spinal cord (R^2=0.427, p=0.16) and by CMCT (R^2=0.493, p=0.12), but without significance (Figure 2).

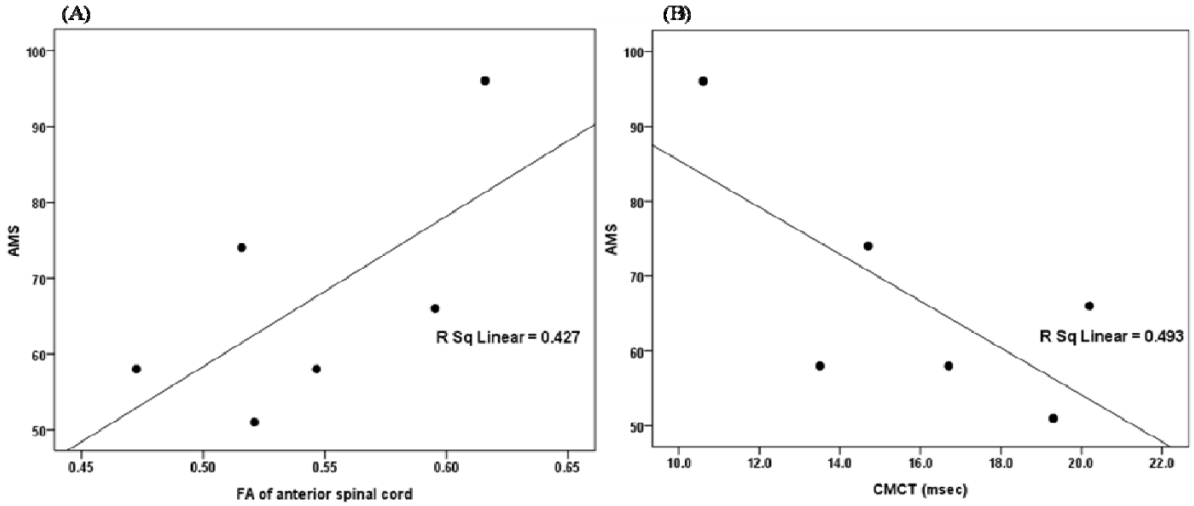

Figure 2. Correlation between ASIA motor score and FA of anterior spinal cord (A) and central motor conduction time (msec) (B)

Table 1. Patients' characteristics, neurological status and comorbidities

No	Age[a]	Duration[b]	Electrical flow (in→out)	AIS	AMS	ASS to light touch	ASS to pin prick	CMCT (msec)	Combined Injury
1	38	5mo	Both hands → Scalp	D	58	60	9	13.5	-
2	50	1yr 2mo	Scalp → Rt arm, Back	C	58	91	94	16.7	-
3	35	8mo	Rt arm → Scalp	D	96	112	112	10.6	-
4	36	9mo	Both hands → Both feet	D	74	96	96	14.7	-
5	44	6mo	Scalp → Both hands	C	51	103	103	19.3	Neuropathy of bilateral median and ulnar nerve
6	40	14yr	Scalp → Rt arm	UC[c]	UC[c]	UC[c]	UC[c]	MEP: not evoked	Rt transhumeral amputation
7	43	1yr 9mo	Both hands → Both feet	UC[c]	UC[c]	UC[c]	UC[c]	MEP: not evoked	Rt transradial amputation
8	50	2yr 5mo	Rt hand → Scalp	D	66	62	62	20.2	Neuropathy of Rt median and ulnar and nerve

Table 2. DTI indices by spinal cord levels

	Patient (n=8)				Control (n=8)			
	FA	MD	AD	RD	FA	MD	AD	RD
C2	0.59±0.11*	0.61±0.16*	1.07±0.25	0.38±0.13*	0.81±0.05	0.43±0.08	0.96±0.16	0.16±0.05
C3	0.61±0.11*	0.58±0.18*	1.04±0.31	0.35±0.13*	0.79±0.06	0.43±0.08	0.93±0.16	0.17±0.06
C4	0.60±0.14*	0.56±0.18*	0.98±0.28	0.35±0.15*	0.80±0.05	0.42±0.07	0.93±0.14	0.17±0.05
C5	0.58±0.11*	0.58±0.18*	1.00±0.28	0.37±0.15*	0.83±0.06	0.40±0.07	0.92±0.15	0.14±0.05
C6	0.59±0.08*	0.59±0.16*	1.02±0.26	0.37±0.12*	0.79±0.05	0.45±0.08	0.97±0.14	0.18±0.06
C7	0.61±0.08*	0.67±0.22*	1.18±0.39	0.41±0.15*	0.80±0.06	0.48±0.11	1.05±0.20	0.19±0.08
Ave	0.60±0.10*	0.60±0.18*	1.05±0.30	0.37±0.14*	0.80±0.06	0.43±0.09	0.96±0.16	0.17±0.06

*p<0.05 relative to control group by independent t-test

Table 3. DTI indices by spinal cord parts

	Patient (n=8)				Control (n=8)			
	FA	MD	AD	RD	FA	MD	AD	RD
Ant	0.55±0.10*†	0.61±0.18*	1.01±0.31	0.40±0.14*	0.80±0.06	0.40±0.09	0.89±0.18	0.16±0.06
Lat	0.61±0.10*	0.59±0.18*	1.04±0.29	0.36±0.14*	0.80±0.05	0.45±0.09	0.98±0.16	0.18±0.06
Post	0.63±0.09*	0.60±0.19*	1.09±0.31	0.36±0.14*	0.81±0.06	0.45±0.07	1.01±0.13	0.18±0.06

*p<0.05 relative to control group by independent t-test

†p<0.05 relative to other intra-group areas by ANOVA test and post-hoc comparison with Tukey test

IV. DISCUSSION

High-voltage-electrical-injury patients usually manifest skin burn, muscle injury, and atypical neurologic symptoms; therefore, confirming spinal cord injury only by clinical findings is difficult. Although electrophysiologic studies such as SEP and MEP are used in the diagnosis of myelopathy, they are not easily applied to high-voltage-electrical-injury patients, as the external wound makes their use painful and uncomfortable. MRI using T1- and T2-weighted sequences is the first choice for diagnosis of myelopathy including high-voltage-electrical spinal cord injury; however, MRI sometimes lacks sensitivity in detecting or characterizing cord lesions.[18,25] Indeed, in some cases of high-voltage-electrical spinal cord injury, MRI has failed to isolate spinal cord lesions.[1,6-9] DTI is a newly developed MR technique that has been utilized for direct assessment of spinal cord integrity.[20,26-28] The present report is the first on the application of DTI to cases of delayed high-voltage-electrical spinal cord injury.

The possible pathogenesis of myelopathy by high-voltage-electrical injury are ischemic injury, heating effect, and trauma.[5] Ischemic injury, occurring secondarily to thrombosis by endothelial injury or vasospasm, is the most probable mechanism; in fact, it is a common cause of delayed spinal cord injury.[29,30] The heating effect is generated in proportion to the electrical intensity, tissue resistance and duration of electrical contact ($H=0.238I^2RT$), and is a common cause of immediate spinal cord injury.[8] Traumatic spinal cord injury occurs by fracture or dislocation resulting from

intense muscle spasm, and is a also common cause of immediate spinal cord injury.

In studies on delayed spinal cord injury, proper patient selection is very important, as the condition's diagnosis is sporadic and can be missed. Moreover, an age limitation is necessary in order to eliminate natural age-related spinal cord deterioration. According to Maier and Mamata's correlation of DTI indices with age, the lowest apparent diffusion coefficient (ADC) values are observed in adults between the ages of 30 and 50 years; in subjects older than 50, the ADC typically is higher.[31] For the purposes of the present study, the cervical spinal cord injury patients were chosen on the basis of (1) electrical-current flow crossing the cervical spinal cord (which was deduced from the locations of the entry and exit wounds on the scalp, neck or upper limb; (2) hyperreflexia-associated weakness or sensory impairment of the upper limb, and (3) delayed CMCT or lack of MEP response. To rule out not-evoked MEP and upper-limb hyperreflexia by brain injury, the patients with abnormal lesions on brain CT or cognitive dysfunction were excluded. The limitation of normal conventional MRI was to investigate specific findings with DTI to overcome the limitation of conventional MRI.

By DTI, we confirmed ascending paralysis and predominant motor deficit with relative sensory preservation. These symptoms, distinct features of high-voltage-electrical spinal cord injury, originate from the spinal cord's vascular supply:[5,8] the cervical spinal cord is supplied by the anterior and posterior spinal arteries;[32] the anterior two-thirds of the spinal cord are supplied by the one anterior spinal artery, whereas the posterior one-third is supplied by the two posterior spinal

arteries. The anterior spinal artery coalesces out of the two vertebral arteries and travels along the anterior sulcus, where it is supplied by the sulcal branches; its diameter is thinnest in the thoracic cord where the ischemic change most commonly occurs, and then widens to upper cervical spinal cord. As it is supplied only by the sulcal branches of the anterior spinal artery, it is more susceptible to ischemic injury.

Ascending paralysis can be surmised with reference to identical changes of DTI indices in all ROIs. In the present case, both a decrease of FA and increases of MD and RD were observed in all ROIs without exception. This homogeneous change of the entire spinal cord is indeed good evidence of ascending paralysis, though we did not follow up on the progress of paralysis. Predominant motor deficit with relative sensory preservation also can be explained according to the pattern of change of DTI indices. In our patient group, the FA decrease was more severe in the anterior spinal cord than in the lateral or posterior spinal cord. The anterior spinal cord is predominantly a motor area consisting of the anterior corticospinal tract and the extrapyramidal rubrospinal, reticulospinal, olivospinal and vestibulospinal tracts. Injury to these motor tracts in the anterior spinal cord typically induces mainly motor deficits. The lateral corticospinal tract is the main tract, generating muscle power and controling movement of the ipsilateral limb.[33] Therefore, partial sparing of the lateral corticospinal tract would contribute to incomplete motor paralysis after high-voltage-electrical injury. In the present study, patients 2, 4 and 5 had definite motor deficits and relatively spared sensory function.

We also used DTI to confirm the pathologic findings on myelopathy. RD

increases in myelinopathy and AD decreases in axonopathy. Correspondingly, in our investigation, we found that RD increased in all ROIs but that AD did not change. Myelinopathy's role in the pathology of high-voltage-electrical spinal cord injury thus was proved.[6,8,34]

Some correlations among neurological status, CMCT and DTI were found, though they were not significant. The AMSs were high in the patients with high FA in the anterior spinal cord (R^2=0.427, p=0.16), which good motor status had been the result of well preserved white matter in the anterior spinal cord, composed significantly of many motor tracts. The AMSs were low in the patients with long CMCT (R^2=0.493, p=0.12), which finding mirrored our previous studies.[14] If we had examined enough cases, we probably would have found strong correlations among the neurological status, CMCT, and DTI findings.

The DTI findings also were helpful in explaining cases. Both patients 6 and 7 had severe high-voltage-electrical injury and had undergone upper-limb amputation, due to which it was impossible to measure the AMS or ASS. Though the MEP was not evoked, conventional MRI failed to show spinal cord lesion in either case. In patient 6, the mean FA of the anterior, lateral, and posterior spinal cords were 0.54, 0.61, and 0.59, respectively. In patient 7, those values were 0.57, 0.74, and 0.70, respectively. Both patients represented an FA decrease relative to the control group (FA of anterior, lateral, and posterior spinal cords: 0.78, 0.79, and 0.79, respectively). Patient 3 had definite hyperreflexia in all four limbs, and positive Hoffmann's sign and ankle clonus were observed. Slight weakness was manifested

in the bilateral legs (AMS=96), and the sensory functions were normal (ASS to light touch=122; ASS to pin prick=122). Conventional MRI was normal, and no peripheral neuropathy was found in a nerve conduction study on the four limbs. In this case, the mean FA of the anterior, lateral, and posterior spinal cords were 0.62, 0.62, and 0.61, respectively.

After delayed spinal cord injury by high-voltage-electrical injury, many deficits persist throughout life.[35-37] DTI is helpful as a means of directly assessing spinal cord integrity. This study had some methodological limitations. The first is the accuracy of neurological evaluation. We evaluated the patients' neurological status using ISNCSCI, though comorbidities including muscle injury and peripheral neuropathy might also affect motor and sensory evaluation. The second limitation is the ROI method employed to evaluate the integrity of the white matter. When using the ROI method, it is crucial to include a sufficient number of voxels. Unlike the case of the brain, it is impossible to include many voxels in the spinal cord white matter, given that the average diameter of the cervical spinal cord from MRI is 7~9 mm (anteroposterior) x 11~14 mm (transverse) and that the white matter surrounds gray matter.[38] The voxel size used in the present study was 1.25 (AP) x 1.5 (RL) mm, and we drew each ROI to contain three or four voxels. FA, MD, AD, and RD did not differ at different levels of the cervical cord in the control group. Most previous investigations of the FA of the cervical spine by 3T MRI systems in healthy volunteers have reported 0.70±0.08,[39] which is consistent with our control group FA (0.80±0.06). The third limitation of the present study is the small sample

size. The incidence of high-voltage-electrical spinal cord injury is 2-8%. But, we imposed many required conditions, including a normal conventional MRI. In order to confirm the correlations with the clinical and electrophysiological findings, we will have to recruit more patients.

V. CONCLUSION

Subtle structural changes of the spinal cord were quantitatively analyzed. With reference to the FA, MD, AD and RD values measured by DTI, myelopathy was revealed in patients who had been suspected of clinical spinal high-voltage-electrical spinal cord injury. DTI indices corroborate high-voltage-electrical injury's reported pathophysiology, including ascending homogeneous involvement more typically affecting the anterior spinal cord, and myelinopathy.

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국문 요약

확산텐서영상을 이용한 고압전기 척수손상의 분석

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온 석 훈

본 연구는 고압전기에 의한 척수손상이 의심되는 환자에게 확산텐서영상을 이용하여 척수 변화의 특징을 알아보고자 하였다. 고압전기에 의한 척수손상이 의심되는 환자 8명을 대상으로 경수 확산텐서영상, 중추운동전도시간을 검사하였고 척수손상을 ASIA 운동지표점수, ASIA 감각지표점수로 측정하였다. 환자군과 나이 및 성별이 맞는 8명의 정상인을 대조군으로 하여 경수 확산텐서영상, 중추운동전도시간을 검사하였다. 확산텐서영상에서 제 2 경수에서 제 7경수까지 각 경수 수준에 해당하는 축면을 하나씩 선택하여, 각 축면의 좌우 앞 쪽, 가 쪽, 뒷 쪽 6개의 관심영역에서 확산텐서영상 지표(분할 비등방성, 겉보기 확산 계수, 축성 확산, 방사 확산)를 측정하였다. 두 군의 확산텐서영상 지표를 비교하였

고, 환자군 및 대조군 각 군에서 척수 수준별 및 영역별 확산텐서영상 지표를 비교하였으다. 환자군에서 확산텐서영상 지표, 중추운동전도시간, 척수손상의 상관관계를 분석하였다. 환자군의 제 2경수에서 제 7 경수까지 모든 경수 수준의 관심영역, 앞 쪽, 가 쪽, 뒷 쪽의 모든 관심영역에서 분할 비등방성은 감소하고, 겉보기 확산 계수, 방사 확산은 대조군에 비해 통계학적으로 유의하게 증가하였고($p<0.05$), 축성 확산은 차이가 없었다. 환자군의 앞쪽 척수 관심영역에서 가 쪽 및 뒷 쪽 척수에 비해 분할 비등방성이 감소하였다($p<0.05$). 환자군에서 경수 수준에 따른 관심영역에서 확산텐서영상 지표의 차이는 없었다. 상관관계 분석에서 통계학적 의의는 없었으나, 환자군에서 앞 쪽 척수 관심영역의 분할 비등방성이 높을수록, 중추운동전도시간이 빠를수록 ASIA 운동지표점수가 높은 경향이었다. 결론적으로, 확산텐서영상을 이용하여 임상적으로 고압전기 척수손상이 의심되는 환자에서 척수손상이 있음을 확인할 수 있었고, 고압전기 척수손상의 특징적 증상인 상행성 마비 및 흔한 운동 손상 및 감각 보존, 수초병의 병태생리를 뒷받침 할 수 있을 것이다.

핵심되는 말: 고압전기 척수손상, 확산텐서영상