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Prevalence and Distribution of Thoracic and Lumbar Compressive Lesions in Cervical Spondylotic Myelopathy

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Study Design: Retrospective cross-sectional study.

Purpose: This study analyzed the prevalence and distribution of horacic and lumbar compressive lesions in cervical spondylotic myelopathy as well as their relationships with cervical developmental spinal canal stenosis (DCS) by using whole-spine postmyelographic computed tomography.

Overview of Literature: There are few studies on missed compressive lesions of the spinal cord or cauda equina at the thoracolumbar level in cervical spondylotic myelopathy. Furthermore, the relationships between DCS, and the prevalence and distribution of thoracic and lumbar compressive lesions are unknown.

Methods: Eighty patients with symptomatic cervical spondylotic myelopathy were evaluated. Preoperative image data were obtained. Patients were classified as DCS or non-DCS (n=40 each) if their spinal canal longitudinal diameter was <12 mm at any level or ≥12 mm at all levels, respectively. Compressive lesions in the anterior and anteroposterior parts, ligamentum flavum ossification, posterior longitudinal ligament ossification, and spinal cord tumors at the thoracolumbar levels were analyzed.

Results: Compressive lesions in the anterior and anteroposterior parts were observed in 13 (16.3%) and 45 (56.3%) patients, respectively. Ligamentum flavum and posterior longitudinal ligament ossification were observed in 19 (23.8%) and 3 (3.8%) patients, respectively. No spinal cord tumors were observed. Thoracic and lumbar compressive lesions of various causes tended to be more common in DCS patients than non-DCS patients, although the difference was statistically insignificant.

Conclusions: Surveying compressive lesions and considering the thoracic and lumbar level in cervical spondylotic myelopathy in DCS patients are important for preventing unexpected neurological deterioration and predicting accurate neurological condition after cervical surgery.

Keywords: Thoracic spine; Lumbar spine; Tandem spinal stenosis; Cervical surgery

Introduction

The growing global elderly population has led toan increase in degenerative spine diseases. Cervical spondylotic myelopathy is the most common cervical spine disease. Narrowing of the spinal canal causes cervical spinal cord compression, consequently causing symptoms such as clumsiness of the hands, spastic gait, and dysesthesia,

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to name a few symptoms. Spinal canal narrowing can be caused by hypertrophy of the facet joint, thickening of the ligamentum flavum, or intervertebral disc protrusion [1]. These changes frequently affect the other mobile portions of the spine, mainly the lumbar spine. Patients with cervical myelopathy may also suffer from lumbar canal stenosis [2], which further complicates the clinical presentation.

Unexpected neurological deterioration is one of the most serious complications after spine surgery and can be due to hematoma [3] or C5 palsy [4]. However, there are few studies on missed compressive lesions of the spinal cord at the thoracic and lumbar level in cervical spondylotic myelopathy [5]. To avoid unexpected neurological deterioration due to a coexisting lower spinal compressive lesion and accurately predict a postoperative neurological condition, it is imperative to understand the prevalence and distribution of these lesions in cervical spondylotic myelopathy.

Developmental spinal canal stenosis (DCS) is an important predisposing factor for cervical myelopathy [6]. Although DCS is reported to be associated with tandems spinal stenosis, which is defined as spinal stenosis involving a cervical or lumbar lesion [2], the associations between cervical DCS and the prevalence and distribution of thoracic and lumbar compressive lesions are unknown.

The development of thin-slice postmyelographic computed tomography (CT) scanning has enabled the detailed morphological analysis of the whole spinal canal and cord. Accordingly, the present study analyzed the prevalence and distribution of thoracolumbar compressive lesions of the spinal cord in cervical spondylotic myelopathy and elucidated the relationship between cervical DCS and thoracic and lumbar compressive lesions by using whole-spine postmyelographic CT.

Materials and Methods

1. Patients

This retrospective cross-sectional study included 80 symptomatic patients with cervical spondylotic myelopathy (51 men and 29 women; mean age, 67.5 years; range, 21–87 years) who underwent whole-spine postmyelographic CT at our hospital before cervical surgery between February 2006 and September 2012. Patients with cervical spondylotic myelopathy presented with only cervical spine symptoms (e.g., clumsiness of the hands, spastic gait, etc.) and not lumbar spine symptoms (e.g., lower-extremity pain, intermittent claudication, etc.) before cervical spine surgery. Patients were classified as DCS or non-DCS (n=40 each) if their spinal canal longitudinal diameter was <12 mm at any level or ≥12 mm at all levels, respectively. Patients with recent trauma, a diagnosis of posterior longitudinal ligament or ligamentum flavum ossification, rheumatoid arthritis, infectious spondylitis, spinal tumor, or a history of fracture were excluded. All patients provided informed consent prior to participation.

2. Scanning protocol

Before cervical surgery, compressive lesions at the thoracic and lumbar levels in cervical spondylotic myelopathy were surveyed. Patients were placed in the prone position with the neck in a neutral position. After myelography, CT scans were obtained using a multi-slice scanner (Toshiba Aquilion 16, Toshiba Medical, Tochigi, Japan). Image data were obtained in 0.5-mm slices from the level of the occiput to the sacrum.

3. Image analysis

Axial images from the thoracic to lumbar level were analyzed by 2 experienced physicians. The following parameters at the thoracic and lumbar level were analyzed: compressive lesions from the anterior part (i.e., disc herniation and osteophytes) (Fig. 1A), compressive lesions from the anteroposterior part (i.e., ligamentum flavum hypertrophy) (Fig. 1B), ligamentum flavum ossification (Fig. 1C), posterior longitudinal ligament ossification (Fig. 1D), and spinal cord tumors. A compressive lesion of the spinal cord or cauda equina was defined as a lesion in contact with the anterior or posterior spinal cord or cauda equina, a morphological deformity of the spinal cord, or the disappearance of the subarachnoid space. In addition, ligamentum flavum and posterior longitudinal ligament ossification were defined as an ossified mass arising from the lamina, facet joints, or vertebrae. The numbers of affected cases as well as compressive and ossified lesions at each level of the spine were calculated.

The data were analyzed with the χ^2test. The level of significance was set at $p<0.05$. All statistical analyses were performed using SPSS ver. 13 (IBM Co., Armonk, NY,

USA). Two independent observers evaluated each parameter on a DICOM viewer and reached a consensus. The reliability of each parameter was investigated in 30 cases in a blinded fashion.The reliability of the image evaluations was estimated according to the agreement percentage and *K* statistic. *K*-values of 1 and 0 indicate absolute agreement and agreement no better than chance, respectively. Agreement wasrated as poor, fair, moderate, substantial, and excellent for *K*-values from 0–0.2, 0.21–0.4, 0.41–0.60, 0.61–0.8, and >0.81, respectively. Intra- and interobserver agreement were both excellent (*K*>0.80).

Results

1. Prevalence and distribution of compressive lesions

Compressive lesions in the anterior spinal cord were observed in 13 of 80 patients (16.3%) (Fig. 2). These lesions were mainly located at the L4–5 level (n=6), followed by the L3–4 (n=3) and L2–3 levels (n=3). Compressive lesions in the anteroposterior parts were observed in 45 of 80 patients (56.3%) (Fig. 3). These lesions were mainly located at the L4–5 level (n=34) followed by the L3–4 (n=26), L2–3 (n=17), and L1–2 (n=5) levels.

2. Prevalence and distribution of ligamentum flavum ossification and posterior longitudinal ligament ossification, and spinal cord tumors

Ligamentum flavum ossification was observed in 19 of 80 patients (23.8%) (Fig. 4). The lesions were mainly located at the T3–4 and T10–11 levels (n=5). Peaks were observed at the proximal and lower thoracic levels. Posterior longitudinal ligament ossification was observed in 3 of 80 patients (3.8%) (Fig. 5). No spinal cord tumors were observed at the thoracolumbar level.

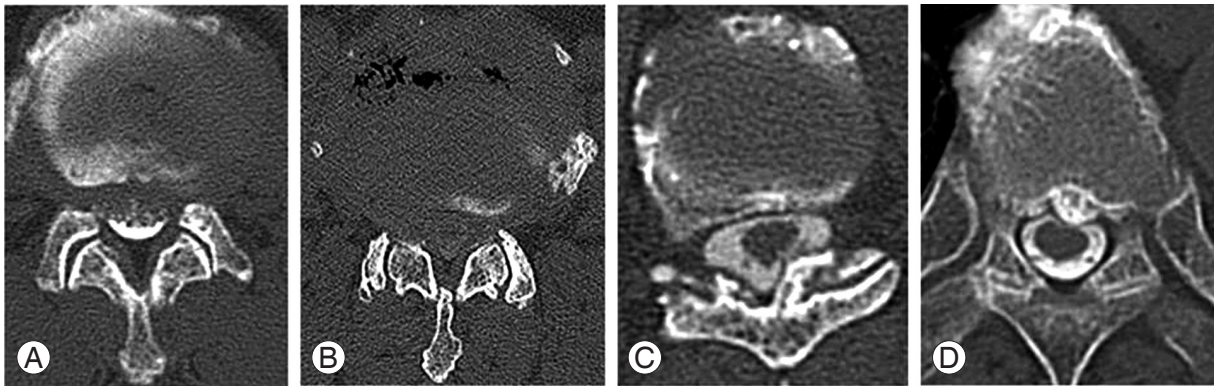

Fig. 1. (A) Compressive lesions in the anterior parts (i.e., disc herniation or osteophytes). **(B)** Compressive lesions in the anteroposterior parts (i.e., ligamentum flavum hypertrophy). **(C)** Ligamentum flavum ossification. **(D)** Posterior longitudinal ligament ossification.

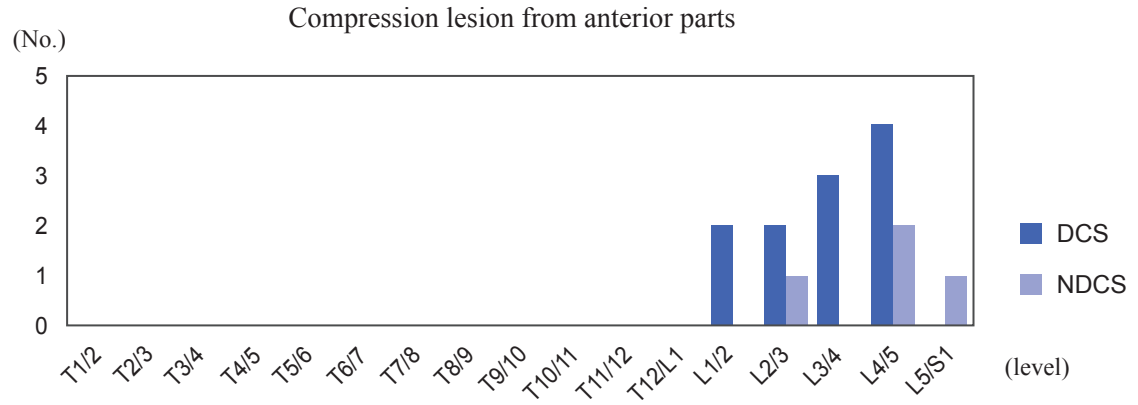

Fig. 2. Distribution of compressive lesions in the anterior parts. DCS, developmental spinal canal stenosis; NDCS, non-developmental spinal canal stenosis.

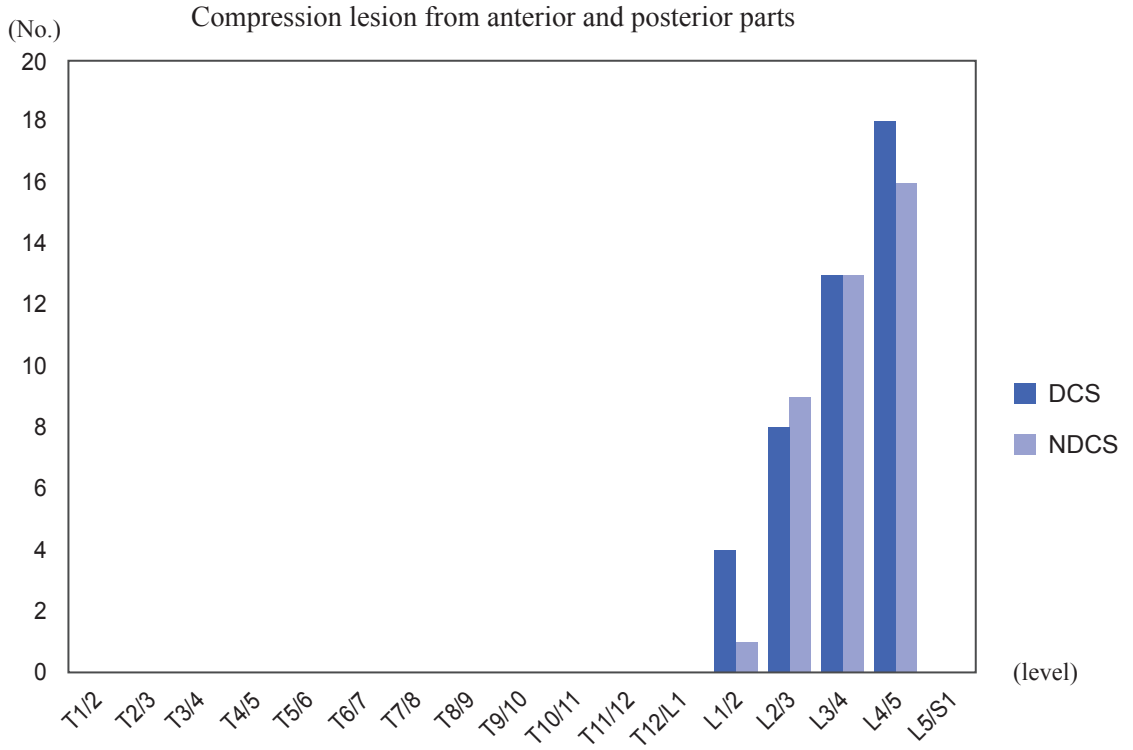

Fig. 3. Distribution of compressive lesions in the anteroposterior parts. DCS, developmental spinal canal stenosis; NDCS, non-developmental spinal canal stenosis.

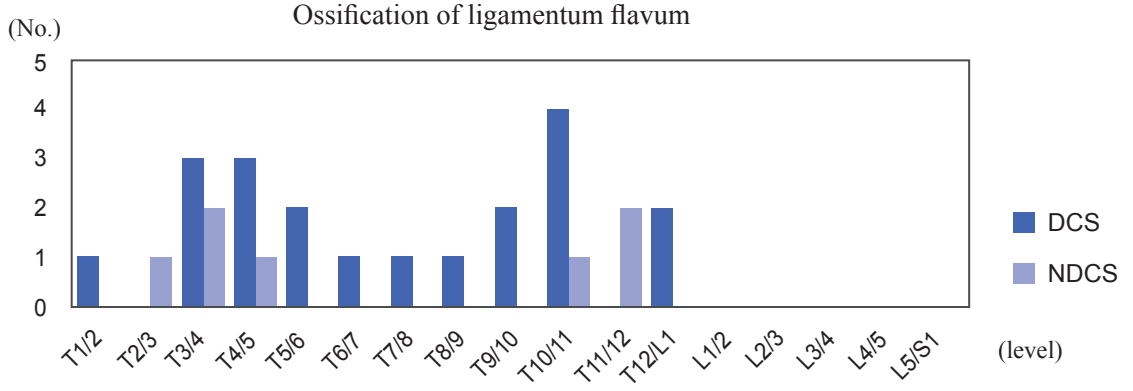

Fig. 4. Distribution of ligamentum flavum ossification. DCS, developmental spinal canal stenosis; NDCS, non-developmental spinal canal stenosis.

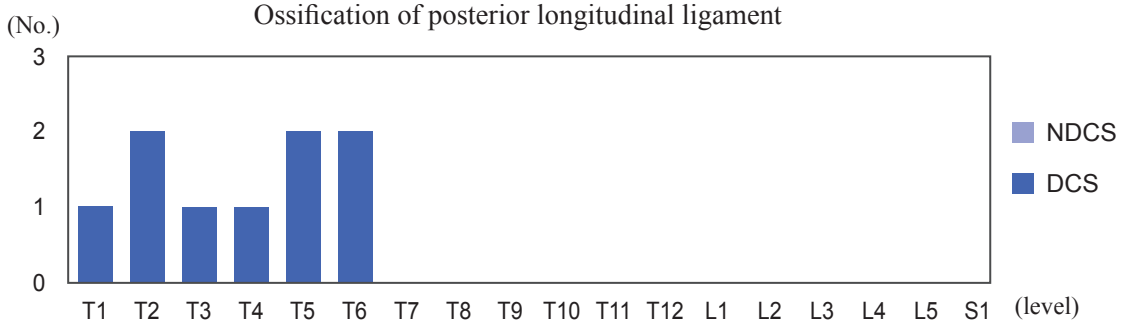

Fig. 5. Distribution of posterior longitudinal ligament ossification. DCS, developmental spinal canal stenosis; NDCS, non-developmental spinal canal stenosis.

3. Prevalence of compression lesions in DCS and non-DCS patients

Compressive lesions in the anterior parts were observed in 9 of 40 (22.5%) and 4 of 40 (10.0%) DCS and non-DCS patients, respectively (p>0.05).Compressive lesions in the anteroposterior parts were observed in 25 of 40 (62.5%) and 20 of 40 (50.0%) patients, respectively (p>0.05). Ligamentum flavum ossification was observed in 12 of 40 (30.0%) and 7 of 40 (17.5%) DCS and non-DCS patients, respectively (p>0.05). Posterior longitudinal ligament ossificationwas observed 3 of 40 (7.5%) and 0 of 40 (0%) DCS and non-DCS patients, respectively (p>0.05).

4. Case report

A 76-year-old man suffering from clumsiness of hands and limb numbness was admitted to our hospital for cervical surgery after his symptoms were aggravated despite conservative treatment. Cervical spine magnetic resonance imaging (MRI) revealed stenotic changes at the C3–4 level. Preoperative whole-spine postmyelographic CT revealed stenotic change at the C3–4 level and severe stenotic change at the L3–4 and L4–5 levels (Fig. 6). After cervical surgery, his clumsiness of hands and limb numbness were relieved and he exhibited no motor weakness. Three months postoperatively, he complained of lower extremity pain, numbness, and intermittent claudication. A lumbar operation was consequently performed to decompress the dural sac, and his neurological status improved.

Discussion

The appearance of unexpected neurological deterioration is a serious complication after spine surgery. There are numerous case reports of hematoma [3] and C5 palsy [4] causing postoperative neurological deterioration at the operative site. However, these complications can be anticipated; thus, surgeons should inform patients of the risks and obtain consent preoperatively. Meanwhile, neurological deterioration due to disorders below the operative site is unexpected; therefore, surgeons are unable to properly inform their patients of this risk before surgery, which can lead to a patient losing trust in the surgeon that often impedes consent to further treatment.

Tandem spinal stenosis is a degenerative disease that affects multiple levels of the spine [7]. This degeneration

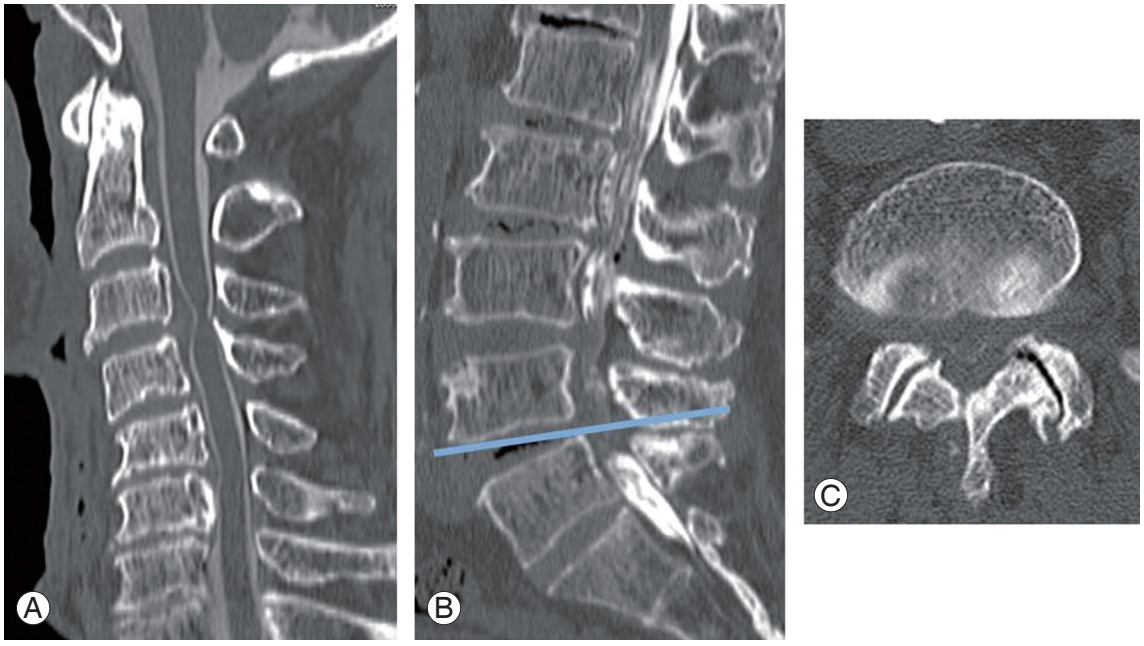

Fig. 6. Computed tomography myelography of the cervical and lumbar spine. **(A)** Sagittal reconstruction image of the cervical spine showing degenerative spinal canal stenosis at the C3–4 level. **(B)** Sagittal reconstruction image of the lumbar spine showing degenerative spinal canal stenosis at the L3–4 and L4–5 levels. **(C)** Axial image of the lumbar spine showing severe degenerative spinal canal stenosis at the L4–5 level.

is a clinical phenomenon that may cause functional loss related to neurologic compression in several areas of the spinal cord [8]. In this phenomenon, the second area of symptomatic neurologic insult is not revealed until the primary symptomatic area has been treated.

In the present study, compressive lesions from the anterior and anteroposterior areas of the spinal cord were mainly located at the L4–5 level, peaking from the middle to lower lumbar level. Matsumoto et al. [9] investigated the frequency of tandem cervical and lumbar disc degeneration in asymptomatic patients, and MRI revealed degenerative changes in the lumbar spine in 79 patients (84%). Degenerative findings were more common in older subjects at the caudal levels. Furthermore, MRI showed degenerative changes in both the cervical and lumbar spine in 78.7% of healthy volunteers.

On the other hand, ligamentum flavum ossification was predominantly observed at the lower and proximal thoracic levels. Guo et al. [10] analyzed the distribution of ligamentum flavum ossification by MRI and found that the ossified segments were mainly located in the lower thoracic spine but were not uncommon in the upper thoracic spine.

There are few reports of neurological deterioration due to a missed compressive lesion of the spinal cord or cauda equina at the thoracic and lumbar level after cervical decompressive surgery. However, as these cases tend to have poor outcomes, many go unreported. Swanson [5] reports a case of stenotic cauda equina syndrome following cervical decompression and fusion for cervical spondylotic myelopathy due to multiple levels of critical stenosis of the lower thoracic and upper lumbar spine. Preoperative neurological examination including motor, sensory, and deep tendon reflexes did not suggest thoracolumbar compressive lesions. However, after cervical surgery, the patient complained of bilateral lower extremity weakness, bowel incontinence, and saddle paresthesia, which were thought to be due to a compressive lesion in the lower spine.

DCS tends to occur in both the cervical and lumbar spine. Patients who develop lumbar degenerative disease are very likely to also suffer from cervical degenerative disease [2]. The present results show that more DCS patients suffered from thoracic and lumbar compressive lesions fora variety of reason than non-DCS patients, although the difference was not significant. Therefore, surgeons should be aware of these trends and investigate the thoracic and lumbar spine in greater detail to identify any compressive lesions at levels below the operative site before cervical surgery. Although surgery is usually not considered for non-symptomatic compressive lesions, such lesions may cause symptoms after cervical spine surgery. During posterior spine surgery, long-term changes in the position or maintenance of the prone position can cause postoperative symptoms. Furthermore, these lesions may cause symptoms in the proximate future. This information should be conveyed to patients to maintain trust.

The main limitation of the present study is its retrospective cross-sectional design and the small number of cases. Therefore, the causal relationship between compressive lesions at levels below the operative site and the appearance of symptoms cannot be confirmed; furthermore, these lesions may remain non-symptomatic. Tsutsumimoto et al. [11] report the natural progression of asymptomatic lumbar spinal canal stenosis in patients treated surgically for cervical myelopathy. Asymptomatic lumbar spinal canal stenosis was present in 32% of patients, and approximately 25% developed lumbar-related leg symptoms within 5 years after cervical surgery. Future studies should involve a larger number of cases to analyze the risk of the manifestation of symptoms at levels below the operative site in greater detail.

Conclusions

This study determined the prevalence and distribution of thoracic and lumbar compressive lesions of the spinal cord in cervical degenerative disease and investigated the relationship between cervical DCS and thoracic and lumbar compressive lesions by using postmyelographic CT. Thoracic and lumbar compressive lesions of various causes tended to be more common in DCS patients than non-DCS patients, although the difference was not statistically significant. Surveying and considering these non-symptomatic compressive lesions in DCS patients are important for preventing unexpected neurological deterioration and accurately predicting neurological conditions postoperatively.

Conflict of Interest

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

References

1. Yonenobu K. Cervical radiculopathy and myelopathy: when and what can surgery contribute to treatment? Eur Spine J 2000;9:1-7.
2. Edwards WC, LaRocca SH. The developmental segmental sagittal diameter in combined cervical and lumbar spondylosis. Spine (Phila Pa 1976) 1985;10:42-9.
3. Kou J, Fischgrund J, Biddinger A, Herkowitz H. Risk factors for spinal epidural hematoma after spinal surgery. Spine (Phila Pa 1976) 2002;27:1670-3.
4. Hasegawa K, Homma T, Chiba Y. Upper extremity palsy following cervical decompression surgery results from a transient spinal cord lesion. Spine (Phila Pa 1976) 2007;32:E197-202.
5. Swanson BT. Tandem spinal stenosis: a case of stenotic cauda equina syndrome following cervical decompression and fusion for spondylotic cervical myelopathy. J Man Manip Ther 2012;20:50-6.
6. Hinck VC, Sachdev NS. Developmental stenosis of the cervical spinal canal. Brain 1966;89:27-36.
7. Lee MJ, Garcia R, Cassinelli EH, Furey C, Riew KD. Tandem stenosis: a cadaveric study in osseous morphology. Spine J 2008;8:1003-6.
8. Kikuike K, Miyamoto K, Hosoe H, Shimizu K. One-staged combined cervical and lumbar decompression for patients with tandem spinal stenosis on cervical and lumbar spine: analyses of clinical outcomes with minimum 3 years follow-up. J Spinal Disord Tech 2009;22:593-601.
9. Matsumoto M, Okada E, Toyama Y, Fujiwara H, Momoshima S, Takahata T. Tandem age-related lumbar and cervical intervertebral disc changes in asymptomatic subjects. Eur Spine J 2013;22:708-13.
10. Guo JJ, Luk KD, Karppinen J, Yang H, Cheung KM. Prevalence, distribution, and morphology of ossification of the ligamentum flavum: a population study of one thousand seven hundred thirty-six magnetic resonance imaging scans. Spine (Phila Pa 1976) 2010;35:51-6.
11. Tsutsumimoto T, Shimogata M, Yui M, Ohta H, Misawa H. The natural history of asymptomatic lumbar canal stenosis in patients undergoing surgery for cervical myelopathy. J Bone Joint Surg Br 2012;94:378-84.