Original Article
Association of developmental canal stenosis with thoracic myelopathy induced by ossification of the ligamentum flavum

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Abstract: Developmental stenosis of the spinal canal predisposes patients to neural compression and loss of function. There is no study on the relationship between developmental stenosis and thoracic myelopathy induced by ossification of the ligamentum flavum (OLF). This study aimed to analyze thoracic myelopathy caused by OLF in an effort to study whether developmental canal stenosis could influence the age of onset and duration of symptoms. This was a case-control study of 100 patients with OLF induced thoracic myelopathy matched for age and sex with 100 controls. Thoracic canal parameters were measured based on computed tomography including spinal canal depth (SCD), spinal canal width (SCW), and spinal canal area (SCA). According to SCA values, the 100 patients were divided: group P (with developmental canal stenosis) and group N (without developmental canal stenosis). Developmental canal stenosis was found in 37% of the patients. SCD and SCA in group P were smaller than in group N (P<0.05), but SCW was similar (P>0.05). The mean age of onset was 54±8 vs. 61±7 years in group P and N, respectively (P<0.001). There were no differences for the mean duration of symptoms (P=0.410), sex (P=0.420), height (P=0.544), weight (P=0.716), and BMI (P=0.305). Thoracic OLF-induced myelopathy tends to coexist with developmental canal stenosis. Developmental stenosis of thoracic canal mainly results from decreased SCD. OLF with developmentally spinal stenosis are associated with younger age at onset of myelopathy. OLF patients with/without developmental stenosis have similar gender, height, weight, BMI, and duration of symptoms prior to treatment.
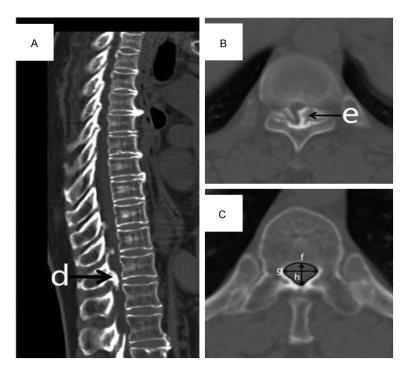
Keywords: Ossification of the ligamentum flavum, thoracic myelopathy, developmental canal stenosis, case-control study

Introduction

Thoracic myelopathy is a rare condition with an insidious development and varied neurological and clinical manifestations [1]. These manifestations are often at first misdiagnosed as spinal disorders, which leads to longer time before surgery, possible irreversible nerve damage, and poor prognosis [2]. A number of causes has been suggested to cause thoracic myelopathy, including ossification of the posterior longitudinal ligament, ossification of the ligamentum flavum (OLF), intervertebral disc herniation, and spondylosis [3-8]. A recent study revealed associations between lower muscle weakness and T10/11 anterior compression, lower limb pain and T11/12 anterior compression, and hyporeflexia and T12/L1 anterior compression [9].

Thoracic myelopathy induced by OLF has predominantly been reported in East Asian countries [9-11]. Its exact etiology and epidemiological characteristics remain unclear. Many investigations have been performed on the clinical characteristics, prognostic factor, and effective surgical management [12-15], but no study focused on the relationship between developmental stenosis and thoracic OLF-induced myelopathy.

Developmental spinal stenosis, also known as congenital spinal stenosis, is caused by growth disturbance of the dorsal spinal elements that result in an insufficient available space in the spinal canal. Its diagnosis is difficult because compared with acquired spinal stenosis, which has easily recognizable features on imaging [16], developmental spinal stenosis is diffuse

Figure 1. Computed tomography (CT) images of an OLF patient determined to be with developmental stenosis. The ossified ligamentum flavum (d and e) is shown on the (A) sagittal and (B) axial images. Radiographic measurements have been drawn on (C). f: spinal canal depth (SCD); g: spinal canal width (SCW); and h: spinal canal area (SCA).

included. The diagnosis of thoracic OLF-induced myelopathy was made by two senior (>10 years of experience) spine surgeons based on clinical and imaging findings. The control group was sex- and age-matched to the case group, and selected from thoracic fracture patients treated during the same period. For both groups, patients with ossification of posterior longitudinal ligament, thoracic disc herniation, and cervical myelopathy were excluded.

The study was approved by the ethics committee of the Affiliated Hospital of Qingdao University. The need for individual consent was waived because of the retrospective nature of the study.
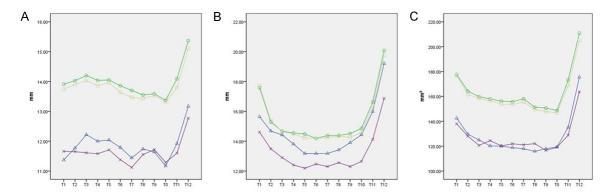
Data collection

Age, sex, body weight, body height, BMI, and duration of the symptoms were recorded. Imaging was reviewed.

Imaging data

The patients were placed in the prone position. CT scans were obtained using a multi-slice scanner (GE Healthcare, Waukesha, WI, USA). Image data were obtained using 0.5-mm slices from T1 to 12. The axial image used for measurement was the cut with the thickest pedicle diameter that also contained the vertebral body, pedicle, and lamina. Measurements in the axial scan included spinal canal depth (SCD), spinal canal width (SCW) and spinal canal area (SCA) (**Figure 1**). All images were measured using the Centricity Enterprise Web V3.0 software (GE Healthcare, Waukesha, WI, USA). Two investigators (CQY and WT), blinded to all clinical information, performed all the measurement. A consensus on the standardized method of measurements was made prior to data collection. The first and second round of measurement was performed at least one month apart. Measurement techniques and the intra and inter-observer agreements were good to excellent for each parameter (all κ>0.70).

and does not present the classical features of spinal stenosis [17, 18]. Nevertheless, the classical feature of developmental spinal stenosis is a reduced anteriorposterior lower spinal diameter to <12 mm on computed tomography (CT) or <9 mm on magnetic resonance imaging (MRI) [19].

Therefore, the aim of the present study was to retrospectively analyze thoracic myelopathy caused by OLF in an effort to study whether developmental canal stenosis could influence the age of onset and duration of symptoms.

Materials and methods

Study design and patients

This was a case-control study of 100 controls (normal thoracic spine except an incidental fracture) and 100 patients treated for thoracic OLF-induced myelopathy at our hospital from January 2003 to January 2015. All patients were Chinese. During the study period, 260 patients underwent treatment for thoracic OLF-induced myelopathy, but only those cases with complete radiological examinations were

Figure 2. Measurements in the axial scan. A: Spinal canal depth (SCD) of group P (○), group N (□), control group (Δ), and standard (×); B: Spinal canal width (SCW) of group P (○), group N (□), control group (Δ), and standard (×). C: Spinal canal area (SCA) of group P (○), group N (□), control group (Δ), and standard (×).

Table 1. Axial CT radiographic measurements of spinal canal depth (SCD) and comparison among group P, group N, and controls

Level	Group P (mm)	Group N (mm)	Controls (mm)	Standard (mm)	P value among groups	P value between groups P and N	P value between group P and controls	P value between group N and controls
T1	11.37	13.91	13.74	11.66	<0.001	<0.001	<0.001	0.242
T2	11.77	14.03	13.90	11.65	<0.001	<0.001	<0.001	0.409
T3	12.22	14.20	14.03	11.61	<0.001	<0.001	<0.001	0.329
T4	12.00	14.04	13.86	11.58	<0.001	<0.001	<0.001	0.282
T5	12.04	14.05	13.96	11.71	<0.001	<0.001	<0.001	0.644
T6	11.79	13.86	13.64	11.38	<0.001	<0.001	<0.001	0.189
T7	11.44	13.70	13.47	11.12	<0.001	<0.001	<0.001	0.196
T8	11.74	13.55	13.43	11.55	<0.001	<0.001	<0.001	0.414
T9	11.64	13.59	13.53	11.72	<0.001	<0.001	<0.001	0.891
T10	11.17	13.37	13.32	11.29	<0.001	<0.001	<0.001	0.830
T11	11.92	14.09	13.79	11.60	<0.001	<0.001	<0.001	0.092
T12	13.17	15.37	15.11	12.77	<0.001	<0.001	<0.001	0.166

Stenosis was considered in the presence of SCA values of less than 2 standard deviations (SD) below the mean SCA of the control group [20]. According to this criterion, the 100 patients with thoracic OLF-induced myelopathy were divided into two groups: Group P (Positive: thoracic OLF-induced myelopathy with developmental canal stenosis) and Group N (Negative: thoracic OLF-induced myelopathy without developmental canal stenosis).

Statistical analysis

Data was presented as mean ± standard deviation. Differences among Group P, Group N and Control Group were analyzed with analysis of variance (ANOVA) and the Tukey's post hoc test. Statistical differences (sex, height, weight,

BMI, age of onset, duration of symptoms) between groups N and P were determined by the Student t test and the chi-square test, as appropriate. Two-sided *P*-values <0.05 were considered statistically significant. Statistical analyses were performed using SPSS 19.0 (IBM, Armonk, NY, USA).

Results

Diagnosis of developmental thoracic stenosis

According to the criterion for developmental thoracic stenosis [21], the values of SCA for the diagnosis of developmental thoracic stenosis were: T1, 137.98 mm^2; T2, 128.07 mm^2; T3, 120.65 mm^2; T4, 124.50 mm^2; T5, 120.24 mm^2; T6, 121.89 mm^2; T7, 121.13 mm^2; T8,

Table 2. Axial CT radiographic measurements of spinal canal width (SCW) and comparison among group P, group N, and controls

Level	Group P (mm)	Group N (mm)	Controls (mm)	Standard (mm)	P value among groups	P value between groups P and N	P value between group P and controls	P value between group N and controls
T1	15.64	17.59	17.72	14.61	<0.001	<0.001	0.601	15.64
T2	14.68	15.28	15.39	13.49	0.001	<0.001	0.472	14.68
T3	14.42	14.67	14.64	12.90	0.420	0.252	0.807	14.42
T4	13.81	14.55	14.49	12.41	0.001	<0.001	0.734	13.81
T5	13.17	14.48	14.23	12.19	<0.001	<0.001	0.096	13.17
T6	13.17	14.19	14.18	12.47	<0.001	<0.001	0.909	13.17
T7	13.18	14.38	14.25	12.30	<0.001	<0.001	0.379	13.18
T8	13.42	14.39	14.35	12.56	<0.001	<0.001	0.762	13.42
T9	13.90	14.51	14.25	12.30	0.026	0.079	0.164	13.90
T10	14.43	14.86	14.61	12.65	0.186	0.394	0.207	14.43
T11	15.99	16.63	16.37	14.12	0.037	0.085	0.210	15.99
T12	19.21	20.08	19.75	16.87	0.020	0.059	0.172	19.21

Table 3. Axial CT radiographic measurements of spinal canal area (SCA) and comparison among group P, group N, and controls

Level	Group P (mm²)	Group N (mm²)	Controls (mm²)	Standard (mm²)	P value among groups	P value between groups P and N	P value between group P and controls	P value between group N and controls
T1	142.51	177.61	176.84	137.98	<0.001	<0.001	<0.001	0.786
T2	129.68	164.33	161.58	128.07	<0.001	<0.001	<0.001	0.238
T3	124.94	159.80	158.45	120.65	<0.001	<0.001	<0.001	0.619
T4	120.22	158.02	156.72	124.50	<0.001	<0.001	<0.001	0.575
T5	119.99	156.20	153.76	120.24	<0.001	<0.001	<0.001	0.321
T6	118.84	155.77	153.66	121.89	<0.001	<0.001	<0.001	0.352
T7	117.92	158.14	155.52	121.13	<0.001	<0.001	<0.001	0.287
T8	115.84	151.33	149.55	122.05	<0.001	<0.001	<0.001	0.383
T9	117.95	150.68	147.86	116.62	<0.001	<0.001	<0.001	0.393
T10	119.45	148.70	146.87	119.12	<0.001	<0.001	<0.001	0.373
T11	135.13	173.27	168.82	128.89	<0.001	<0.001	<0.001	0.224
T12	175.53	210.89	205.13	163.70	<0.001	<0.001	<0.001	0.083

122.05 mm²; T9, 116.62 mm²; T10, 119.12 mm²; T11, 128.89 mm²; and T12, 163.70 mm². Using this standard, groups P and N included 37 and 63 patients, respectively. SCD and SCA in group P were significantly smaller than those in group N, whereas all of them had a similar SCW (**Figure 2** and **Tables 1**-**3**).

Characteristics of the patients

The mean age was 55 ± 6 years in the control group, 57 ± 8 years in group P, and 63 ± 6 years in group N. There were 24 males and 13 females in group P, and 34 males and 29 females in group N. In group P, body weight was 68.0 ± 10.7 kg, height was 164.8 ± 6.5 cm, and

body mass index (BMI) was 24.9 ± 2.8. In group N, those values were 68.8 ± 10.1 kg, 164.0 ± 7.0 cm, and 25.5 ± 2.7. There were no differences between the two groups (all P>0.05).

The mean age of onset was 54 ± 8 years and 61 ± 7 years in groups P and N, respectively (P<0.001) (**Table 4**). The mean duration of symptoms was 26.9 ± 28.4 months and 23.6 ± 32.7 months in groups P and N, respectively (P=0.410) (**Table 4**).

Discussion

Developmental stenosis occurs when the lumen of the spinal canal is smaller than expect-

Table 4. Characteristics of groups P and N

	Group P N=37	Group N N=63	Controls N=100	P Value
Age (year)	57 ± 8	63 ± 6	55 ± 6	P<0.001
Male	23 (62.16%)	34 (53.97%)	59 (59.00%)	P=0.420
Age of onset (years)	54 ± 8	61 ± 7	-	P<0.001
Duration of symptoms (months)	26.91 ± 28.38	23.62 ± 32.69	-	P=0.410
Height (cm)	164.8 ± 6.5	164.0 ± 7.0	-	0.544
Weight (kg)	68.0 ± 10.7	68.8 ± 10.2	-	P=0.716
BMI (kg/m^2)	24.9 ± 2.8	25.5 ± 2.7	-	P=0.305

ed in the general population. It is an important predisposing factor for myelopathy [21, 22]. Compared with numerous reports regarding the effect of developmental canal stenosis on cervical or lumbar diseases [23-27], there is no study on the impact of developmental canal stenosis among patients with thoracic myelopathy caused by OLF. Therefore, this study aimed to analyze thoracic myelopathy caused by OLF in an effort to study whether developmental canal stenosis could influence the age of onset and duration of symptoms. The results showed that thoracic OLF-induced myelopathy tends to coexist with developmental canal stenosis. Developmental stenosis of thoracic canal mainly results from decreased SCD. OLF with developmentally spinal stenosis are associated with younger age at onset of myelopathy. OLF patients with/without developmental stenosis have similar gender, height, weight, BMI, and duration of symptoms prior to treatment.

Using lateral chest X-rays among 1744 people, Kudo et al. [28] observed a prevalence of OLF of 6.2% in males and 4.8% in females. Because of the high prevalence of asymptomatic thoracic OLF, early diagnosis is necessary for people with developmental thoracic stenosis. Not only because the coexistence of OLF with developmental thoracic stenosis leads to clinical presentation occurring earlier in life, but also because of the irreversible damage to the cord and the delay for treatments.

In the present study, 37% patients with thoracic myelopathy induced by OLF had significantly smaller SCA and SCD, suggesting that thoracic OLF-induced myelopathy tended to coexist with developmental canal stenosis. All the patients in group P exhibited significantly smaller canal area at all levels compared with Group N. Radiographic findings showed that the decline of SCA was primarily due to the decreased SCD

while patients with/without developmental stenosis had a similar SCW. This finding implies that the dysplastic and short pedicle results in the decline of SCD, thus exclusively causing the difference of SCA between patients in the two groups. However, because of the restriction of recognition and identifying the end point of the pedicle in axial CT images, the evaluation of pedicle length in the present study was impossible, while studies had reported that developmental stenosis was characterized by a short pedicle in the thoracic spine [29] and lumbar spine [30]. Numerous anatomic and radiographic studies of various canal parameters have been performed to establish a standard for defining spinal stenosis by inconsistent imaging modalities including radiographs, computed tomographic scans, and magnetic resonance images [20, 27, 31, 32].

In the cervical spine, the Torg-Pavlov ratio is commonly used to diagnose developmental canal stenosis [33]. In the lumbar spine, Verbiest [34] first defined developmental narrowing of the lumbar canal by an abnormally short anteroposterior diameter on plain radiographs and an absolute value of less than 10 mm as developmental stenosis. However, when it comes to the criteria of developmental thoracic canal stenosis, a universally accepted diagnostic criteria is unavailable [35]. In any given population, measurements of the spine are continuous variables, and to select an arbitrary diameter cutoff may not be useful clinically. Therefore, for the purpose of the present study, the method reported by Bajwa et al. [9] was selected to identify the presence of developmental stenosis. Patients with values that were 2 SD below the mean of the control group were considered as having thoracic developmental canal stenosis. By using this method, developmental thoracic stenosis was defined in the present study as: T1, 137.98 mm^2; T2, 128.07

mm^2; T3, 120.65 mm^2; T4, 124.50 mm^2; T5, 120.24 mm^2; T6, 121.89 mm^2; T7, 121.13 mm^2; T8, 122.05 mm^2; T9, 116.62 mm^2; T10, 119.12 mm^2; T11, 128.89 mm^2; and T12, 163.70 mm^2.

Previous studies [21, 36-38] of cervical myelopathy had reported that a subset of patients present clinical symptoms of spinal stenosis at an earlier age. These patients are typically in their late 40 s and early 50 s. In these patients, imaging studies often showed fewer degenerative changes with a markedly narrowed spinal canal involving multiple levels of stenosis. Arnoldi et al. [39] classified these patients as having congenital stenosis. In the thoracic spine, if the spinal canal is developmentally narrow, relatively little encroachment by OLF can cause spinal cord compression. Few studies had been performed to investigate the aforementioned phenomenon in patients with thoracic OLF-induced myelopathy. In the present study, there was significant difference in the average age of onset between group P and group N, suggesting that the patients with thoracic developmental stenosis presented at an earlier age. This finding is supported by Sanghvi et al. [40], who first and exclusively reported that patients with developmental canal stenosis showed significant correlation with younger age at onset of thoracic OLF-induced myelopathy. Similar findings were found in patients with cervical and lumbar myelopathy [33, 41-43]. This is the largest CT-based study on OLF-induced thoracic myelopathy conducted in a Chinese population. Patient parameters were blinded to the two observers and the measurement techniques were uniform. The novelty of this research includes 3 aspects. This work further investigates the unique relationship between thoracic OLF-induced myelopathy and developmental canal stenosis while almost all investigations have been performed on the clinical characteristics, prognostic factor, and effective surgical management. It also demonstrates the differences of age and duration between OLF patients with and without developmental canal stenosis and how the differences could be conducive to the promotion of effective diagnosis and treatment. Up to now, only 1 research simply focusing on the differences has been reported. Finally, our research presents a novel and practical approach for the standard and model of developmental canal stenosis.

Nevertheless, this study could suffer from some limitations. The cut-off values for developmental thoracic stenosis were arbitrarily based on values smaller than 2 SD lower than the mean control value, and it is unknown if this cut-off value is clinically significant. Secondly, in this retrospective study, developmental thoracic stenosis was based on CT images, which are less reliable for soft tissue components. MRI could be a better choice to directly assess spinal cord compression. Further research is required for analyzing the relationship between thoracic developmental stenosis and myelopathy caused by OLF.

In conclusion, Thoracic OLF-induced myelopathy tends to coexist with developmental canal stenosis. Developmental stenosis of thoracic canal mainly results from decreased SCD. OLF with developmentally spinal stenosis are associated with younger age at onset of myelopathy. OLF patients with and without developmental stenosis have similar gender, height, weight, BMI, and duration of symptoms prior to treatment.

Disclosure of conflict of interest

None.

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References

[1] Yonenobu K, Ebara S, Fujiwara K, Yamashita K, Ono K, Yamamoto T, Harada N, Ogino H and Ojima S. Thoracic myelopathy secondary to ossification of the spinal ligament. J Neurosurg 1987; 66: 511-518.

[2] Ohtsuka K, Terayama K, Yanagihara M, Wada K, Kasuga K, Machida T and Matsushima S. A radiological population study on the ossification of the posterior longitudinal ligament in the spine. Arch Orthop Trauma Surg 1987; 106: 89-93.

[3] Toribatake Y, Baba H, Kawahara N, Mizuno K and Tomita K. The epiconus syndrome presenting with radicular-type neurological features. Spinal Cord 1997; 35: 163-170.

[4] Yoon SH, Kim WH, Chung SB, Jin YJ, Park KW, Lee JW, Chung SK, Kim KJ, Yeom JS, Jahng TA, Chung CK, Kang HS and Kim HJ. Clinical analysis of thoracic ossified ligamentum flavum without ventral compressive lesion. Eur Spine J 2011; 20: 216-223.

[5] Aizawa T, Sato T, Sasaki H, Matsumoto F, Morozumi N, Kusakabe T, Itoi E and Kokubun S. Results of surgical treatment for thoracic myelopathy: minimum 2-year follow-up study in 132 patients. J Neurosurg Spine 2007; 7: 13-20.

[6] Matsumoto M, Chiba K, Toyama Y, Takeshita K, Seichi A, Nakamura K, Arimizu J, Fujibayashi S, Hirabayashi S, Hirano T, Iwasaki M, Kaneoka K, Kawaguchi Y, Ijiri K, Maeda T, Matsuyama Y, Mikami Y, Murakami H, Nagashima H, Nagata K, Nakahara S, Nohara Y, Oka S, Sakamoto K, Saruhashi Y, Sasao Y, Shimizu K, Taguchi T, Takahashi M, Tanaka Y, Tani T, Tokuhashi Y, Uchida K, Yamamoto K, Yamazaki M, Yokoyama T, Yoshida M and Nishiwaki Y. Surgical results and related factors for ossification of posterior longitudinal ligament of the thoracic spine: a multi-institutional retrospective study. Spine (Phila Pa 1976) 2008; 33: 1034-1041.

[7] Matsumoto M, Toyama Y, Chikuda H, Takeshita K, Kato T, Shindo S, Abumi K, Takahata M, Nohara Y, Taneichi H, Tomita K, Kawahara N, Imagama S, Matsuyama Y, Yamazaki M and Okawa A. Outcomes of fusion surgery for ossification of the posterior longitudinal ligament of the thoracic spine: a multicenter retrospective survey: clinical article. J Neurosurg Spine 2011; 15: 380-385.

[8] Ando K, Imagama S, Wakao N, Hirano K, Tauchi R, Muramoto A, Kato F, Yukawa Y, Kawakami N, Sato K, Matsubara Y, Kanemura T, Matsuyama Y and Ishiguro N. Examination of the influence of ossification of the anterior longitudinal ligament on symptom progression and surgical outcome of ossification of the thoracic ligamentum flavum: a multicenter study. J Neurosurg Spine 2012; 16: 147-153.

[9] Takenaka S, Kaito T, Hosono N, Miwa T, Oda T, Okuda S, Yamashita T, Oshima K, Ariga K, Asano M, Fuchiya T, Kuroda Y, Nagamoto Y, Makino T, Yamazaki R and Yonenobu K. Neurological manifestations of thoracic myelopathy. Arch Orthop Trauma Surg 2014; 134: 903-912.

[10] Ando K, Imagama S, Ito Z, Hirano K, Muramoto A, Kato F, Yukawa Y, Kawakami N, Sato K, Matsubara Y, Kanemura T, Matsuyama Y and Ishiguro N. Predictive factors for a poor surgical outcome with thoracic ossification of the ligamentum flavum by multivariate analysis: a multicenter study. Spine (Phila Pa 1976) 2013; 38: E748-754.

[11] Li M, Wang Z, Du J, Luo Z and Wang Z. Thoracic myelopathy caused by ossification of the ligamentum flavum: a retrospective study in Chinese patients. J Spinal Disord Tech 2013; 26: E35-40.

[12] Li Z, Ren D, Zhao Y, Hou S, Li L, Yu S and Hou T. Clinical characteristics and surgical outcome of thoracic myelopathy caused by ossification of the ligamentum flavum: a retrospective analysis of 85 cases. Spinal Cord 2016; 54: 188-196.

[13] Onishi E, Yasuda T, Yamamoto H, Iwaki K and Ota S. Outcomes of surgical treatment for thoracic myelopathy: a single-institutional study of 73 patients. Spine (Phila Pa 1976) 2016; 41: E1356-E1363.

[14] Kawaguchi Y, Yasuda T, Seki S, Nakano M, Kanamori M, Sumi S and Kimura T. Variables affecting postsurgical prognosis of thoracic myelopathy caused by ossification of the ligamentum flavum. Spine J 2013; 13: 1095-1107.

[15] Wang T, Yin C, Wang D, Li S and Chen X. Surgical technique for decompression of severe thoracic myelopathy due to tuberous ossification of ligamentum flavum. Clin Spine Surg 2017; 30: E7-E12.

[16] Yang Z, Xue Y, Dai Q, Zhang C, Zhou HF, Pan JF and Sheng D. Upper facet joint en bloc resection for the treatment of thoracic myelopathy caused by ossification of the ligamentum flavum. J Neurosurg Spine 2013; 19: 81-89.

[17] Postacchini F. Lumbar spinal stenosis and pseudostenosis. Definition and classification of pathology. Ital J Orthop Traumatol 1983; 9: 339-350.

[18] Garfin SR and Rauschning W. Spinal stenosis. Instr Course Lect 2001; 50: 145-152.

[19] Gallucci M, Limbucci N, Paonessa A, Splendiani A. Degenerative disease of the spine. Neuroimaging Clin N Am 2007; 17: 87-103.

[20] Chatha DS and Schweitzer ME. MRI criteria of developmental lumbar spinal stenosis revisited. Bull NYU Hosp Jt Dis 2011; 69: 303-307.

[21] Bajwa NS, Toy JO and Ahn NU. Establishment of parameters for congenital thoracic stenosis: a study of 700 postmortem specimens. Clin Orthop Relat Res 2012; 470: 3195-3201.

[22] Hinck VC and Sachdev NS. Developmental stenosis of the cervical spinal canal. Brain 1966; 89: 27-36.

[23] Shigematsu H, Ueda Y, Koizumi M, Takeshima T, Tanaka Y, Satoh N, Matsumori H, Oshima T, Matsuyama E, Kugai A and Takakura Y. Does developmental canal stenosis influence surgical results of bilateral open-door laminoplasty for cervical spondylotic myelopathy? J Neurosurg Spine 2008; 9: 358-362.

[24] Horne PH, Lampe LP, Nguyen JT, Herzog RJ and Albert TJ. A novel radiographic indicator of developmental cervical stenosis. J Bone Joint Surg Am 2016; 98: 1206-1214.

[25] van Eck CF, Spina Iii NT and Lee JY. A novel MRI classification system for congenital functional lumbar spinal stenosis predicts the risk for tandem cervical spinal stenosis. Eur Spine J 2017; 26: 368-373.

[26] Yu M, Tang Y, Liu Z, Sun Y and Liu X. The morphological and clinical significance of developmental cervical stenosis. Eur Spine J 2015; 24: 1583-1589.

[27] Cheung JP, Samartzis D, Shigematsu H and Cheung KM. Defining clinically relevant values for developmental spinal stenosis: a large-scale magnetic resonance imaging study. Spine (Phila Pa 1976) 2014; 39: 1067-1076.

[28] de Graaf I, Prak A, Bierma-Zeinstra S, Thomas S, Peul W and Koes B. Diagnosis of lumbar spinal stenosis: a systematic review of the accuracy of diagnostic tests. Spine (Phila Pa 1976) 2006; 31: 1168-1176.

[29] Kudo S, Ono M and Russell WJ. Ossification of thoracic ligamenta flava. AJR Am J Roentgenol 1983; 141: 117-121.

[30] Barnett GH, Hardy RW Jr, Little JR, Bay JW and Sypert GW. Thoracic spinal canal stenosis. J Neurosurg 1987; 66: 338-344.

[31] Santiago FR, Milena GL, Herrera RO, Romero PA and Plazas PG. Morphometry of the lower lumbar vertebrae in patients with and without low back pain. Eur Spine J 2001; 10: 228-233.

[32] Cheung JPY, Ng KKM, Cheung PWH, Samartzis D and Cheung KMC. Radiographic indices for lumbar developmental spinal stenosis. Scoliosis Spinal Disord 2017; 12: 3.

[33] Singh K, Samartzis D, Vaccaro AR, Nassr A, Andersson GB, Yoon ST, Phillips FM, Goldberg EJ and An HS. Congenital lumbar spinal stenosis: a prospective, control-matched, cohort radiographic analysis. Spine J 2005; 5: 615-622.

[34] Torg JS, Pavlov H, Genuario SE, Sennett B, Wisneski RJ, Robie BH and Jahre C. Neurapraxia of the cervical spinal cord with transient quadriplegia. J Bone Joint Surg Am 1986; 68: 1354-1370.

[35] Verbiest H. Results of surgical treatment of idiopathic developmental stenosis of the lumbar vertebral canal. A review of twenty-seven years' experience. J Bone Joint Surg Br 1977; 59: 181-188.

[36] Wilson ES, McKinlay S, Crawford JM and Robb HM. The influence of esmolol on the dose of propofol required for induction of anaesthesia. Anaesthesia 2004; 59: 122-126.

[37] Amundsen T, Weber H, Lilleas F, Nordal HJ, Abdelnoor M and Magnaes B. Lumbar spinal stenosis. Clinical and radiologic features. Spine (Phila Pa 1976) 1995; 20: 1178-1186.

[38] Miyazaki M, Takita C, Yoshiiwa T, Itonaga I and Tsumura H. Morphological analysis of the cervical pedicles, lateral masses, and laminae in developmental canal stenosis. Spine (Phila Pa 1976) 2010; 35: E1381-1385.

[39] Morishita Y, Naito M, Hymanson H, Miyazaki M, Wu G and Wang JC. The relationship between the cervical spinal canal diameter and the pathological changes in the cervical spine. Eur Spine J 2009; 18: 877-883.

[40] Arnoldi CC, Brodsky AE, Cauchoix J, Crock HV, Dommisse GF, Edgar MA, Gargano FP, Jacobson RE, Kirkaldy-Willis WH, Kurihara A, Langenskiold A, Macnab I, McIvor GW, Newman PH, Paine KW, Russin LA, Sheldon J, Tile M, Urist MR, Wilson WE and Wiltse LL. Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. Clin Orthop Relat Res 1976; 4-5.

[41] Sanghvi AV, Chhabra HS, Mascarenhas AA, Mittal VK and Sangondimath GM. Thoracic myelopathy due to ossification of ligamentum flavum: a retrospective analysis of predictors of surgical outcome and factors affecting preoperative neurological status. Eur Spine J 2011; 20: 205-215.

[42] Cheung PWH, Tam V, Leung VYL, Samartzis D, Cheung KM, Luk KD and Cheung JPY. The paradoxical relationship between ligamentum flavum hypertrophy and developmental lumbar spinal stenosis. Scoliosis Spinal Disord 2016; 11: 26.

[43] Kitab SA, Alsulaiman AM and Benzel EC. Anatomic radiological variations in developmental lumbar spinal stenosis: a prospective, control-matched comparative analysis. Spine J 2014; 14: 808-815.