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*Research Article*

# Relationship between Scheuermann disease and symptomatic thoracic spinal stenosis: A retrospective study

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ABSTRACT

***Objective:*** This study aimed to investigate the possible relationship between Scheuermann disease (SD) and the pathophysiological factors of thoracic spinal stenosis (TSS), including ossification of the ligamentum flavum (OLF), ossification of the posterior longitudinal ligament (OPLL), and thoracic disc herniation (TDH) in patients with symptomatic TSS.

***Methods:*** Demographic and radiological data from 66 consecutive patients diagnosed with symptomatic TSS from 2013 to 2018 were retrospectively collected and divided into 3 groups depending on the underlying pathomechanism of TSS: TDH group (18 patients; 6 women; mean age ± standard deviation [Sd] = 59.89 ± 11.34), OPLL group (12 patients; 8 women; mean age ± Sd = 56.08 ± 14.74), and OLF group (36 patients; 20 women; mean age ± Sd = 58.69 ± 9.77). A total of 41 age-matched healthy individuals (19 women; mean age ± Sd = 54.88 ± 13.63) were designated as the control group. In each group, both typical and atypical SD criteria were radiologically examined. The demographic data and presence of SD between the control group and 3 subgroups of TSS pathomechanisms were evaluated.

***Results:*** SD characteristics were identified in 83.33% (15/18) of patients in the TDH group, 44.44% (16/36) in the OLF group, 25% (3/12) in the OPLL group, and 17.07% (7/41) of the control individuals. When analyzed by the chi-squared test and logistic regression analysis, the presence of SD was significantly associated with TDH ($P < 0.01$) and OLF ($P < 0.05$) but not OPLL ($P > 0.05$). Patients with TDH and OLF showed peak involvement of T10/11, and patients with OPLL did not. Furthermore, we determined that age, sex, body-mass index, and smoking status were not the risk factors for TDH, OPLL, and OLF ($P > 0.05$). SD was found to be a risk factor for TDH ($P < 0.01$) and OLF ($P < 0.05$) but not for OPLL ($P > 0.05$).

***Conclusion:*** Evidence from this study indicated that SD might be a risk factor for OLF and TDH but not for OPLL.

***Conclusion:*** Level III, Diagnostic Study

## Introduction

Thoracic spinal stenosis (TSS) is a common disorder that causes pain, sensory disturbances, myelopathy, and lower-extremity weakness.[1] Various pathological factors, such as ossification of the ligamentum flavum (OLF), ossification of the posterior longitudinal ligament (OPLL), and thoracic disc herniation (TDH), may result in a reduction in the capacity of the thoracic spinal canal, further inducing TSS.

Scheuermann disease (SD) was first defined as a structural thoracic kyphosis with more than 3 wedged vertebrae (WV), and it mainly affects adolescents.[2] Its pathological changes affect the vertebral endplates and discs and are associated with radiological features, such as Schmorl's nodes (SN), irregular vertebral endplates (IE), and disc lesions. The widely accepted diagnostic criteria for "typical SD" were initially proposed by Sorensen in 1964 and were defined as ≥ 3 consecutive WV with an angle of at least 5°.[3] Patients presenting with endplate or disc lesions with only 1 or 2 WV and no notable kyphosis were classified as having "atypical SD".[4-6] SD usually develops during adolescence but may not manifest until adulthood[7]; thus, expert opinions are apt to define SD as a broader concept, including the patients showing radiographic characteristics of SD without clinical symptoms.[7,8] Because SD can induce disc degeneration, endplate lesions, and an abnormal spinal curvature, previous studies have related SD to low-back pain.[9] Moreover, some studies have indicated that SD can cause TDH.[10-14]

We noted that SD is commonly observed in our hospital among patients with TDH and OLF but not in those with OPLL. However, studies analyzing the relationship between SD and other pathophysiological factors, including OLF and OPLL, are lacking. Therefore, this study aimed to investigate whether SD is related to TDH, OLF, and/or OPLL.

## Materials and Methods

This retrospective study included patients who were at least 18 years old and met the diagnostic criteria for TSS.[15] Those with a history of spinal trauma, infection, or other congenital abnormalities (i.e., scoliosis) were excluded to avoid false-positive WV results. In total, 66 consecutive patients diagnosed with symptomatic TSS (18 with TDH, 12 with OPLL, and 36 with OLF) from October 2013 to October 2018 at our spine center were included. At the same time, we enrolled

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**Table 1.** Diagnostic criteria of thoracic spinal stenosis and its exact pathomechanisms

| | |
|---|---|
| TSS criteria | Reduction in the capacity of the thoracic spinal canal with associated compression of the spinal cord and/or nerve roots; different pathological factors of capacity reduction are recorded |
| TDH criteria | CT findings of soft compressing structures originating from the anterior spinal cord after the diagnosis of TSS |
| OPLL criteria | CT findings of hard compressing structures originating from the anterior spinal cord after the diagnosis of TSS |
| OLF criteria | CT findings of hard compressing structures originating from the posterior spinal cord after the diagnosis of TSS |

CT, computed tomography; OLF, ossification of the ligamentum flavum; OPLL, ossification of the posterior longitudinal ligament; TDH, thoracic disc herniation; TSS, thoracic spinal stenosis

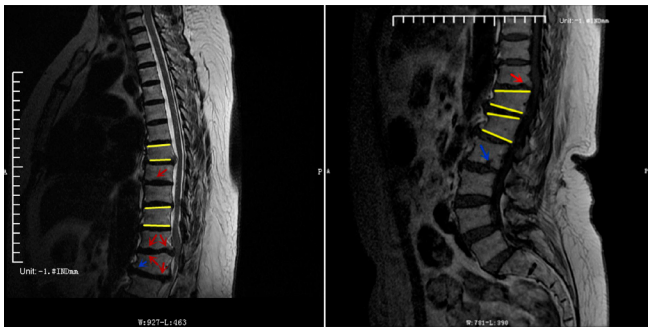

**Figure 1.** Magnetic resonance imaging of patients with atypical SD with multiple-level thoracic disc herniation
T2- weighted imaging and T1- weighted imaging of a 62-year-old woman's thoracic spine and lumbar spine, respectively, showing multiple-level inconsecutive wedged vertebrae (yellow line, T7, T10, T12, and L1), Schmorl's nodes (blue arrows, T12 and L2), and irregular vertebral endplates (red arrows, T8, T11, and T12)
MRI, magnetic resonance imaging; SD, Scheuermann disease; TDH, thoracic disc herniation; WI, weight imaging; WV: wedged vertebrae, SN: Schmorl's nodes, IE: irregular vertebral endplates

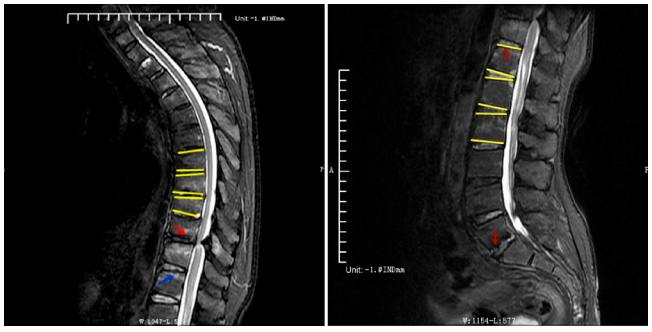

**Figure 2.** Magnetic resonance imaging of patients with typical Scheuermann disease with T10/11 ossification of the ligamentum flavum
T2- weighted imaging of a 60-year-old man's thoracic spine and lumbar spine, showing 3 consecutive wedged vertebrae (yellow line, T7-T9 and T12-L2), Schmorl's nodes (blue arrows, T12), and irregular vertebral endplates (red arrows, T10, T12 and L5)
MRI, magnetic resonance imaging; SD, Scheuermann disease; OLF, ossification of the ligamentum flavum; WI, weight imaging; WV, wedged vertebrae; SN, Schmorl's nodes; IE, irregular vertebral endplates

41 age-matched normal healthy volunteers, without any symptoms, in the control group. Demographic (including age, sex, body-mass index [BMI], and current smoking status) and imaging data (X-ray, computed tomography [CT], and magnetic resonance imaging [MRI]) from radiological images of all 66 patients and 41 normal control volunteers were collected and analyzed. To diagnose TSS and determine the exact pathomechanism, MRI findings were analyzed first,

**HIGHLIGHTS**

- Thoracic spinal stenosis is a common disorder, resulting in complications, such as ossification of the ligamentum flavum (OLF), ossification of the posterior longitudinal ligament (OPLL), and thoracic disc herniation (TDH).
- Our findings indicate that Scheuermann disease (SD) may be a risk factor for TDH and OLF but not OPLL.
- Patients with TDH and OLF showed a peak involvement of T10/11, and patients with OPLL did not. This could explain why SD was associated with TDH and OLF. However, the exact mechanism is unknown and warrants further research.

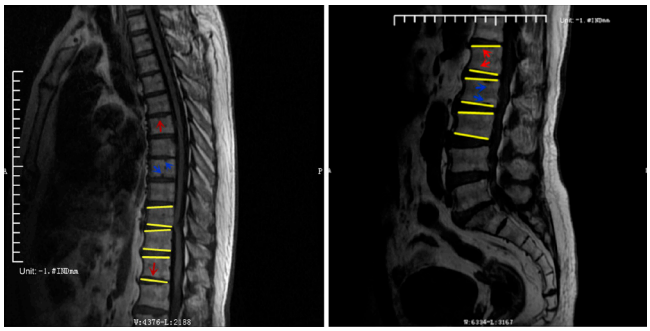

**Figure 3.** Magnetic resonance imaging of patients with typical Scheuermann disease with T10/11 ossification of the ligamentum flavum
T1- weighted imaging of a 77-year-old man's thoracic spine and lumbar spine, showing 3 consecutive wedged vertebrae (yellow line, T10-T12, and L1-L3), Schmorl's nodes (blue arrows, T8 and L2), and irregular vertebral end plates (red arrows, T6, T12, and L1).
MRI, magnetic resonance imaging; SD, Scheuermann disease; OLF, ossification of the ligamentum flavum; WI, weight imaging; WV, wedged vertebrae; SN, Schmorl's nodes; IE, irregular vertebral end plates

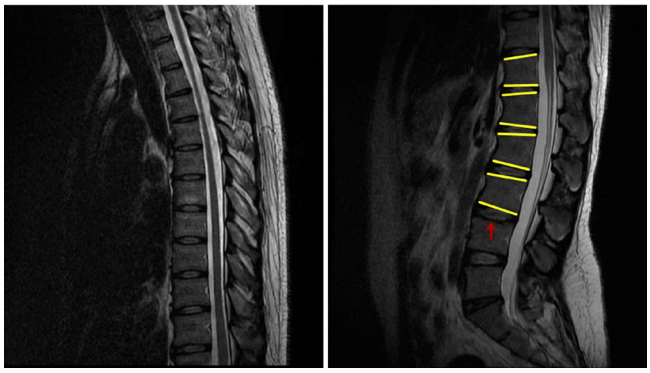

**Figure 4.** Magnetic resonance imaging of the control group individuals with typical Scheuermann disease
T2- weighted imaging of a 37-year-old man's thoracic spine and lumbar spine, showing 4 consecutive wedged vertebrae (yellow line, T12-L3) and irregular vertebral end plates (red arrows, L4).
MRI, magnetic resonance imaging; SD, Scheuermann disease; WI, weight imaging; WV, wedged vertebrae; IE, irregular vertebral end plates

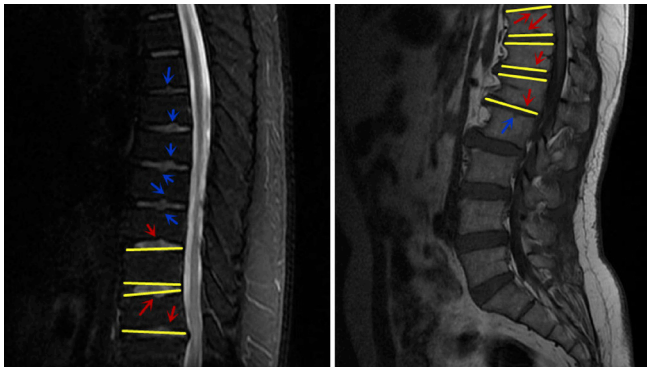

**Figure 5.** Magnetic resonance imaging of control group individuals with typical Scheuermann disease
T2- weighted imaging and T1- weighted imaging of a 60-year-old man's thoracic spine and lumbar spine, respectively, showing 3 consecutive wedged vertebrae (yellow line, T11-L1), multiple Schmorl's nodes (blue arrows, T6-T10 and L2), and irregular vertebral endplates (red arrows, T11, T12, and L1).
MRI, magnetic resonance imaging; SD, Scheuermann disease; WI, weight imaging; WV, wedged vertebrae; SN, Schmorl's nodes; IE, irregular vertebral endplates

followed by the CT scans, to assess whether the pathological lesions causing spinal cord compression were ossified. The diagnostic criteria are shown in Table 1. After analyzing the radiological images, only those with a single pathomechanism leading to TSS were included; those involving more than 1 pathomechanism were excluded. Patients with ligamentum flavum hypertrophy but no ossification were also excluded. To diagnose SD in the control group, the X-ray images of the thoracic and lumbar spine were first analyzed to determine whether the features aligned with the characteristics of typical SD. If the overall result was negative but some vertebral bodies showed WV, the MRI findings were analyzed again to determine if

**Table 2.** Number of involved levels for different pathomechanisms

| | Total number | 1 level | 2 levels | 3 levels | 4 levels | 5 levels | 6 levels |
|---|---|---|---|---|---|---|---|
| TDH | 18 | 7 | 6 | 2 | 2 | 1 | 0 |
| OPLL | 12 | 3 | 7 | 0 | 2 | 0 | 0 |
| OLF | 36 | 15 | 14 | 6 | 0 | 0 | 1 |

OLF, ossification of the ligamentum flavum; OPLL, ossification of the posterior longitudinal ligament; TDH, thoracic disc herniation

**Table 3.** Values and statistical analysis of demographic data as well as the presence of Scheuermann disease between the control group and the 3 different pathomechanisms

| | Total Number | Age (mean ± standard deviation) | Sex | BMI (mean ± standard deviation) | Currently Smoking | SD |
|---|---|---|---|---|---|---|
| Control group | 41 | 54.88 ± 13.63 | F 19 | 23.41 ± 3.10 | (+) 14 | (+) 7 |
| | | | M 22 | | (−) 27 | (−) 34 |
| TDH | 18 | 59.89 ± 11.34 | F 6 | 23.57 ± 2.51 | (+) 7 | (+) 15 |
| | | | M 12 | | (−) 11 | (−) 3 |
| $T/X_2$ | | 1.364 | 0.867 | 0.195 | 0.123 | 23.485 |
| *P* | | 0.178 | 0.352 | 0.846 | 0.726 | 0.000** |
| OPLL | 12 | 56.08 ± 14.74 | F 8 | 22.41 ± 3.42 | (+) 3 | (+) 3 |
| | | | M 4 | | (−) 9 | (−) 9 |
| $T/X_2$ | | 0.265 | 1.535 | 0.954 | 0.356 | 0.381 |
| *P* | | 0.792 | 0.215 | 0.345 | 0.550 | 0.537 |
| OLF | 36 | 58.69 ± 9.77 | F 20 | 24.10 ± 2.97 | (+) 11 | (+) 16 |
| | | | M 16 | | (−) 25 | (−) 20 |
| $T/X_2$ | | 1.424 | 0.651 | 1.001 | 0.113 | 6.856 |
| *P* | | 0.159 | 0.420 | 0.320 | 0.737 | 0.009** |

***P*<0.05, SD, Scheuermann disease; OLF, ossification of the ligamentum flavum; OPLL, ossification of the posterior longitudinal ligament; TDH, thoracic disc herniation; BMI, body-mass index; $T/X_2$,the value of t-test or Chi-squared test

**Table 4.** Results of logistic regression analysis identifying potential risk factors for predicting thoracic disc herniation

| | OR | 95% CI | *P* |
|---|---|---|---|
| Age | 0.966 | 0.903-1.034 | 0.322 |
| Sex | 0.599 | 0.087-4.113 | 0.602 |
| BMI | 0.181 | 0.864-1.615 | 0.296 |
| Current smoking | 1.663 | 0.244-11.317 | 0.603 |
| SD | 47.949 | 6.334-362.990 | 0.000* |

**P*<0.01
OR, odds ratio; CI, confidence interval; BMI, body-mass index; SD, Scheuermann disease

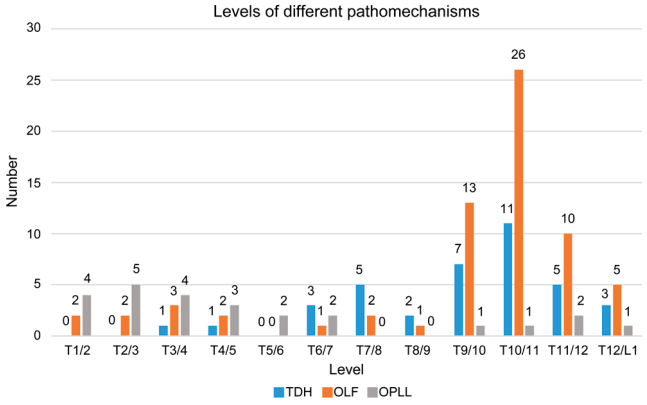


**Figure 6.** Levels of different pathomechanisms
OLF, ossification of the ligamentum flavum; OPLL, ossification of the posterior longitudinal ligament; TDH, thoracic disc herniation

the features aligned with those of atypical SD. The images of patients with typical SD are shown in Figures 1-5.

A senior doctor who was blinded to the aim of the research collected the demographic data; 2 doctors who were blinded to the research and experienced in treating spinal conditions analyzed all the radiographic images and decided whether the images met the criteria for SD, either the classic Sorensen's criterion (at least 3 consecutive WV showing > 5° anterior wedging, irrespective of whether occurring at the thoracic or thoracolumbar spinal regions) to define typical SD or the modified Heithoff's criterion (simultaneous presence of less than 3 WV with multiple SN and IE at the thoracic and lumbar spinal regions) to define atypical SD.[2-8] If there were any controversial radiographs, a more experienced doctor, also blinded to the research, was consulted to decide the final diagnosis.

The study was approved by an appropriate ethics committee (approval No. 2020-008). The requirement for obtaining informed consent was waived owing to the retrospective nature of the study; however, in cases where images or clinical data of a patient were used, consent for publication was obtained from the patient or an authorized signatory.

### Statistical analysis

We used descriptive statistics to record and analyze the levels of TDH, OPLL, and OLF. The independent sample t-test was used to analyze the role of age and BMI, and the Chi-squared test was used to compare sex, current smoking status, and the presence of SD between the 3 different clinical groups and the control group. Furthermore, logistic regression analysis was performed to evaluate various factors. The Statistical Package for the Social Sciences version 25.0 (IBM SPSS Corp., Armonk, NY, USA) was used for all the statistical analyses. The significance level was set at $P < 0.05$.

## Results

In most cases, the number of involved levels was ≤ 2, and very few cases involved > 3 levels. The number of involved levels in all cases is shown in Table 2. The number of patients, according to the different pathomechanisms and the specific affected levels, is shown in Figure 6. From these data, it was apparent that the most frequently involved level in patients with TDH and with OLF was the thoracolumbar region, whereas that in patients with OPLL was the upper thoracic region.

The values and statistical analysis of the demographic data and SD characteristics between the control and 3 different pathomechanisms are shown in Table 3. There were no significant differences in age, sex, BMI, and smoking status between the control group and the 3 different pathomechanisms ($P > 0.05$). SD characteristics appeared more frequently in patients with TDH (15/18, 83.33%) and OLF (16/36, 44.44%)

**Table 5.** Results of logistic regression analysis identifying potential risk factors for predicting ossification of the posterior longitudinal ligament

| | OR | 95% CI | *P* |
|---|---|---|---|
| Age | 0.992 | 0.943-1.043 | 0.744 |
| Sex | 0.416 | 0.084-2.057 | 0.282 |
| BMI | 1.117 | 0.864-1.444 | 0.397 |
| Currently smoking | 1.737 | 0.269-11.218 | 0.562 |
| SD | 4.165 | 0.513-33.804 | 0.182 |

OR, odds ratio; CI, confidence interval; BMI, body-mass index; SD, Scheuermann disease

**Table 6.** Results of logistic regression analysis identifying potential risk factors for predicting ossification of the ligamentum flavum

| | OR | 95% CI | *P* |
|---|---|---|---|
| Age | 0.970 | 0.924-1.019 | 0.229 |
| Sex | 0.409 | 0.114-1.469 | 0.170 |
| BMI | 0.968 | 0.813-1.152 | 0.713 |
| Current smoking | 0.861 | 0.210-3.537 | 0.835 |
| SD | 4.462 | 1.424-13.977 | 0.010* |

*$P < 0.05$
OR, odds ratio; CI, confidence interval; BMI, body-mass index; SD, Scheuermann disease

but not in patients with OPLL (3/12, 25%). SD was significantly associated with TDH and OLF ($P < 0.01$) but not OPLL ($P > 0.05$).

The logistic regression analysis performed between the control group and the 3 different pathomechanisms causing TSS is shown in Tables 4, 5, and 6, respectively, including the odds ratio, 95% confidence interval, and p values. We found that age, sex, BMI, and smoking status were not the risk factors for TDH, OPLL, and OLF ($P > 0.05$). However, SD was found to be a risk factor for TDH ($P < 0.01$) and OLF ($P < 0.05$) but not for OPLL ($P > 0.05$).

## Discussion

Our findings suggest that SD characteristics appear more often in patients with TDH and OLF but not in those with OPLL. The demographic parameters (including age, sex, BMI, and current smoking status) were not found to be the risk factors for TDH, OPLL, and OLF. Therefore, SD may be a risk factor for TDH and OLF but not for OPLL. To the best of our knowledge, this is the first study to state that SD is a risk factor for OLF.

Guo et al. performed a case-control study and found that the symptomatic TDH group had more SD features than the low-back pain group and proposed that TDH was associated with SD.[11] Our results agree with those of the aforementioned study. Kale et al. found an increase in the thoracic kyphosis in patients diagnosed with SD and found that these changes could lead to spinal cord compression.[16] The possible biomechanical mechanism underlying this compression may be that patients with SD have an increased angle of thoracic or thoracolumbar kyphosis, which can increase the longitudinal stress on the posterior annulus of the thoracolumbar intervertebral discs, which could then accelerate posterior disc herniation.[12] In our study cohort, most of the affected vertebral levels in the symptomatic TDH group were in the thoracolumbar region, where both disc herniation and WV commonly result in the greatest kyphosis in patients with SD. A kyphosis occurring in this region results in an increase in the longitudinal stress, which may contribute to TDH. In contrast, after TDH occurs, the narrowing of the intervertebral space because of disc herniation or degeneration will appear, which may further cause an increase in the degree of kyphosis.[11] Briefly, the increased kyphosis may contribute to the generation of disc herniation, which can further aggravate the kyphosis,[11] generating a negative pathophysiological loop. Another mechanism underlying the increase in the degree of kyphosis involves the IE seen in patients with SD.[17] It is well known that blood and nutrition are supplied to the discs via the endplate and that patients with SD have a weaker endplate. Under the same mechanical stress, the weaker endplate is more easily avulsed by the posterior annulus ring, which can lead to disc herniation.[18] Histological studies have demonstrated that the cartilage matrix of patients with SD develops structural weakness.[19] SN is one of the most common features of SD and also plays a role in disc herniation.[20,21]

Although OPLL mainly affects the cervical spine, the thoracic spine may also be affected in some patients.[22] The prevalence of thoracic OPLL has been reported to be 0.8%.[23] OPLL is a multifactorial disease caused by ectopic hyperostosis and calcification of the posterior longitudinal ligament.[24] Its etiology has been suggested to be related to both genetic and environmental factors.[25] Because the etiology of OPLL is based on ossification, which is different from dysplasia and degeneration caused by SD, this may explain why there is no correlation between OPLL and SD. Furthermore, as shown by our results, the most involved vertebral levels in OPLL are in the upper thoracic spine,[26] whereas the most involved levels in SD are in the lower thoracic spine or thoracolumbar region. Even when SD kyphosis occurs, it is unlikely to affect the upper thoracic spine, which might be another reason why OPLL is not associated with SD.

Although OLF has been identified in only 3.8% of the Chinese population,[6] some Korean researchers have reported incidence rates as high as 16.9%.[20] Although the exact pathophysiology has not yet been fully elucidated, 2 factors are considered particularly important in the development of OLF.[27] These include intrinsic causes, such as genetic and dietary factors,[28-30] and extrinsic causes, such as mechanical stress.[31] Frequent squatting by East Asians may partially explain why OLF is more frequent in East-Asian countries.[32] Recently, multiple studies have indicated that mechanical stress plays an important role in the onset and progression of spinal ligament ossification.[32] Kim et al. demonstrated that the occurrence of thoracic kyphosis was higher in a group of patients with thoracic OLF than in a group of patients with non-thoracic OLF; this aligns with the characteristics of SD, which also presents with an increase in the thoracic kyphosis.[33] Our study showed a peak OLF occurrence at the T10/11 level, which has been confirmed in other studies as well.[6,33] This result is likely owing to the fact that the lower thoracic spine region receives the maximum tensile force.[34] Thus, increases in the tensile forces on the ligamentum flavum because of an enhanced kyphotic angle, as seen in the patients with SD, may underlie the observed association between SD and OLF.

The aforementioned explanations may be the potential mechanisms explaining why SD is associated with TDH and OLF but not OPLL. Because the pathomechanisms of both SD and TSS (TDH, OPLL, and OLF) are not clear, some scholars have focused on genetic contributions.[2,25,29,35] Genetics may play an undeniable key role. Genes associated with the development of both SD and TDH or OLF may exist; however, to the best of our knowledge, these have not been identified yet, and further research is required to identify the genes associated with these conditions.

Other than the effects of various biomechanisms and genetics, only a few studies have evaluated the effect of demographic parameters, such as age, sex, and BMI on SD and TSS development. The chronic progressive nature of TSS makes it challenging to determine its association with demographic parameters because these parameters are constantly changing.

Our study had several limitations. First, this was a retrospective study based on the review of medical records; thus, there is a potential risk of bias because of misinterpretation of the descriptions. Second, the number of patients was small, and the study was conducted in a single spine center. Thus, a multicenter study with a larger sample size should be conducted to further verify our conclusions. Third, we did not investigate the relationship between the clinical symptoms and the different diseases that can cause TSS.

In conclusion, our preliminary findings suggest that the high proportion of patients with SD with symptomatic OLF and TDH indicates a close relationship of SD with OLF and TDH but not with OPLL. Therefore, SD may be a risk factor for OLF and TDH but not for OPLL. Further studies are needed to investigate the precise mechanisms.

**Ethics Committee Approval:** Ethics committee approval was received for this study from the Ethics Committee of Yantaishan Hospital (Approval No. 2020-008).

**Informed Consent:** The requirement for obtaining informed consent was waived due to the study's retrospective nature; however, in cases where images or clinical data of a patient were used, consent for publication was obtained from the patient or an authorized signatory.

**Author Contributions:** Concept - Y.D., S.L., Y.C.; Design - Y.D., Y.C.; Supervision - S.L.; Data Collection and/or Processing - Y.D., S.D., J.C., Z.C.; Analysis and/or Interpretation - Y.D., S.D.; Literature Review - Y.D., Z.C.; Writing - Y.D.; Critical Review - S.L., Y.C.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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