A new perspective on dysplastic spondylolisthesis: imaging analysis and hypothesis on the pathogenesis

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Background: Dysplastic spondylolisthesis (DS) is a common spine disease in children and adolescents, and its pathogenesis remains unclear. Few previous studies have focused on the cauda equina in patients with DS. This retrospective case-control study evaluated the imaging findings of patients with DS from a novel perspective and attempted to speculate on its underlying etiology.

Methods: 126 young patients (16–24 years) with DS were selected, and the lumbar spinal canal ratio (LSCR) and disc height index (DHI) of nondiseased segments (L1–L5) were measured. The morphology of the cauda equina was observed via magnetic resonance imaging (MRI). We also measured the filum terminale detection rate (FTDR), filum terminale diameter (FTD), and the relative position of the conus medullaris on MRI. The DS group was compared with the control group. The control group consisted of 126 healthy young individuals who were age- and sex-matched to the DS group.

Results: Compared with that in the control group, the cauda equina in the DS group was more likely to experience excessive posterior displacement (EPD) ($P<0.05$). DS patients showed a lower FTDR and smaller FTD at the L4–L5 and L5–S1 segments ($P<0.05$), and exhibited a higher conus medullaris position ($P<0.05$). The LSCR in L3–L5 segments was significantly greater in the DS group than in the control group ($P<0.05$). There was no significant difference in the DHI between the two groups at all lumbar segments.

Conclusions: Compared with healthy young individuals, patients with DS demonstrate thinner filum terminale, a relatively higher position of the conus medullaris, a wider spinal canal, and a higher incidence of cauda equina EPD. Based on these imaging differences, we explored the potential causes of DS and put forward the compensatory spondylolisthesis hypothesis.

Keywords: Dysplastic spondylolisthesis (DS); etiological speculation; cauda equina; filum terminale; conus medullaris

Submitted Mar 11, 2026. Accepted for publication Sep 08, 2026. Published online Sep 29, 2026.

doi: 10.21037/qims-2026-0591

View this article at: https://dx.doi.org/10.21037/qims-2026-0591

Introduction

Dysplastic spondylolisthesis (DS) is a common spine disease in children and adolescents, mostly occurring in the lumbosacral region, and its pathogenesis remains unclear. In 1997, Marchetti and Bartolozzi (1) proposed the concept of DS and described the radiological features of diseased segments in high-dysplastic

spondylolisthesis (HDS), such as wedging of the L5 vertebra, doming of the S1 endplate and lumbosacral kyphosis. Mac-Thiong *et al.* (2) subsequently proposed diagnostic criteria for HDS and low-dysplastic spondylolisthesis (LDS). Our team has always adhered to the concept of the "holistic-spine", which suggests that the vertebral column and nervous tissue of the spine should be considered as a whole and that the etiology of all spinal diseases should be analyzed from a holistic perspective. However, most previous studies of DS focused on the bony structure of the diseased segment. We believe that the nondiseased segment of the DS and cauda equina also have great research value.

Our team diagnosed and treated several patients with DS in clinical work and observed several interesting phenomena: on routine supine sagittal lumbar magnetic resonance imaging (MRI) of the lumbar spine, the spinal canal of the DS patients appears wider, and the cauda equina is attached to the posterior wall of the spinal canal. Initially, we thought that this might be caused by local kyphosis and intervertebral slippage. However, as the number of patients increased, we found that a considerable number of patients with low-grade dysplasia showed no obvious local kyphosis or intervertebral slippage, but the cauda equina were still closely attached to the posterior wall of the spinal canal. Furthermore, this posterior displacement is different from the sedimentation of the cauda equina, and it is an excessive posterior displacement (EPD). This study investigated the imaging features of DS and the cause from a new perspective. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-2026-0591/rc).

Methods

Group of patients with DS

Patients diagnosed with DS at the Third Hospital of Hebei Medical University from January 2014 to January 2024 were enrolled in this study, and their medical records and relevant imaging data were retrospectively reviewed. The diagnostic criteria of DS by Mac-Thiong *et al.* (2) were used. Moreover, to reduce the influence of growth and degenerative diseases on the measurement results, this study included only young patients aged 16–24 years. A total of 126 patients with complete imaging data were included. Patients with a history of cancer, trauma, congenital malformation, infection, or spine surgery were excluded.

Control group

As the control group, 126 healthy young individuals matched to the DS group in age and sex were recruited from the physical examination center of the Third Hospital of Hebei Medical University during the same study period. All participants had complete imaging data including lumbar MRI, which revealed no organic spinal lesions. Subjects with a history of cancer, trauma, congenital malformation, infection, or spine surgery were excluded. None of the participants in the control group presented any clinical symptoms related to the cauda equina. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. The study was approved by the Ethics Committee of Third Hospital of Hebei Medical University (No. W2024-017-1) and informed consent was obtained from all individual participants and legal guardians.

Imaging parameters and measurement methods

In this study, MRI data acquisition of the lumbar spine was performed on a 1.5 T magnetic resonance scanner (MAGNETOM Avanto; Siemens Healthineers, Erlangen, Germany). The scanning coverage extended from the superior endplate of T11 to the superior endplate of S2. All subjects were scanned in

the supine position, with detailed MR sequences and imaging parameters listed in *Table 1*, and identical scanning parameters were applied to both the DS group and the control group. All MRI scans were performed by one qualified and trained radiology technician. Raw DICOM images were archived on the picture archiving and communication system (PACS) for subsequent post-processing and analysis. All radiographic images were evaluated and quantified independently in a double-blinded manner by two senior radiologists. Mean values of paired measurements were recorded. In cases of significant inter-reader disagreement, a third senior radiologist was consulted to make the final adjudication.

Table 1 Magnetic resonance imaging sequences and parameters

Parameters	Sagittal T2-weighted	Axial T2-weighted
Sequence type	Turbo spin echo	Turbo spin echo
Repetition time (msec)	3,500–3,600	3,500–3,600
Echo time (msec)	110–120	110–120
Flip angle (°)	150	150
Echo train length	26	26
Slice thickness (mm)	4	4
Acquisition matrix	320×256	512×256
Field of view (mm)	280×280	200×220
Number of excitations	2	4

Lumbar spinal canal ratio (LSCR)

The LSCR refers to the ratio of the lumbar spinal canal diameter measured on MRI images of the vertebral body center to the corresponding vertebral body diameter (*Figure 1*). A larger numerical value indicates a relatively larger spinal canal. The L1–L5 segments of the lumbar spine were chosen for measurement.

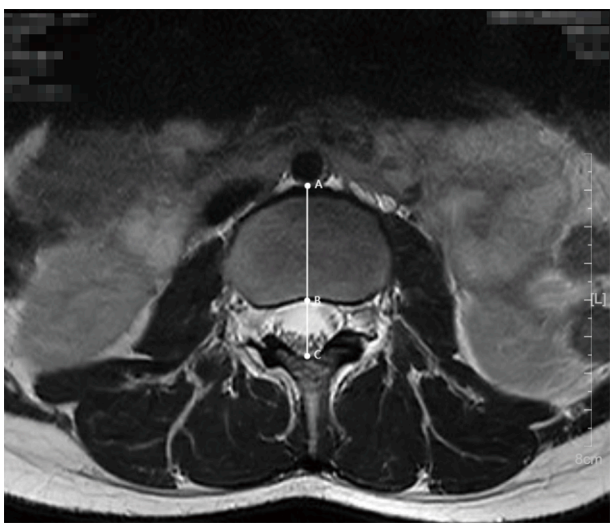


Figure 1 In the view of the center of the vertebral body, BC/AB is the LSCR. Point A is the midpoint of the anterior vertebral border, point C is the base of the spinous process, and point B is the intersection of line AC and the posterior border of the vertebral body. LSCR, lumbar spinal canal ratio.

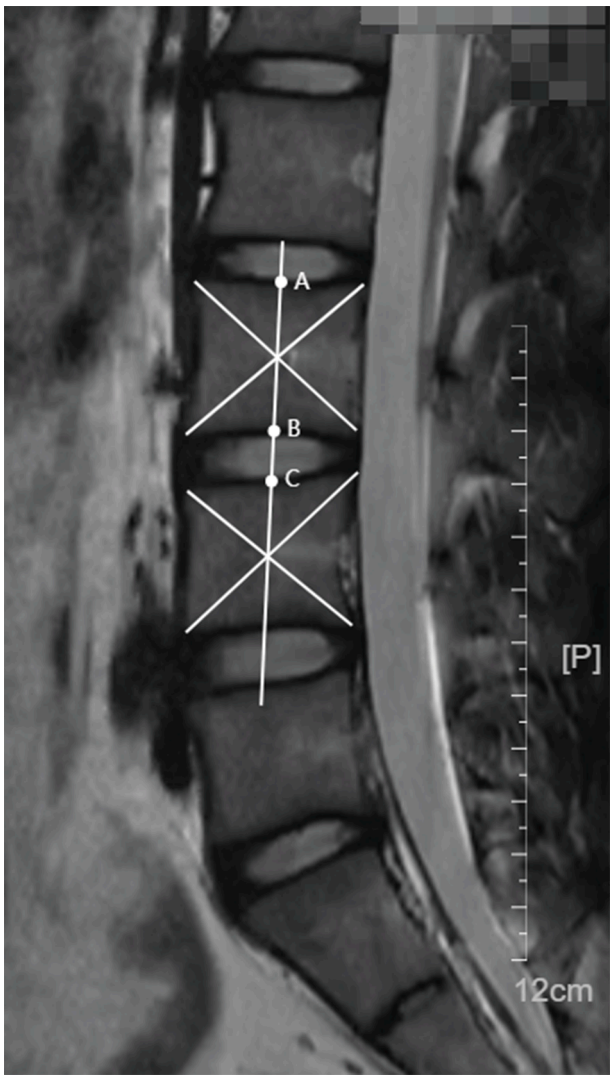


Figure 2 DHI measured via MRI, where BC/AB is the DHI. The intersections of the midvertebral line with the superior border of the upper vertebral body, the inferior border of the upper vertebral body, and the superior border of the lower vertebral body are A, B and C, respectively. DHI, disc height index; MRI, magnetic resonance imaging.

Disc height index (DHI)

DHI is the ratio of the height of the intervertebral disc to the height of the upper vertebral body. The disc height and vertebral body height were measured via the midvertebral line. The line connecting the intersections of the diagonals of the two vertebral bodies was the midvertebral line (*Figure 2*). Since narrowing of the intervertebral space in the L5–S1 segment has been confirmed by many previous studies (3), this study measured only the L1–L5 segments.

EPD of the cauda equina

Because MRI examinations are performed in the supine position, the cauda equina settles on the dorsal side of the spinal canal under the action of gravity. To determine whether the cauda equina nerve is normal sedimentation or EPD, we use the ellipse method—drawing an ellipse that is tangent to the inner wall

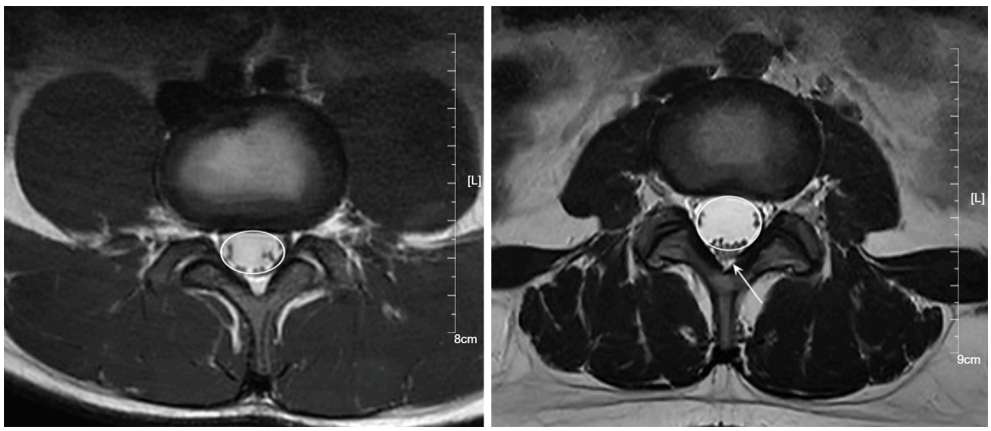


Figure 3 Assessment of posterior displacement of the cauda equina on MRI. An ellipse tangent to the inner wall of the spinal canal was drawn. The dural sac and the cauda equina of a 19-year-old individual in the control group are contained within the ellipse (left). The dural sac and part of the cauda equina of a 19-year-old individual in the DS group breach the boundary of the ellipse (right). DS, dysplastic spondylolisthesis; MRI, magnetic resonance imaging.

of the spinal canal. Under normal circumstances, the dural sac and cauda equina should be contained within the ellipse or overlap with the boundary of the ellipse (*Figure 3* left). If the cauda equina breaches the boundary of the ellipse dorsally, it is considered that EPD of the cauda equina has occurred (*Figure 3* right).

The filum terminale diameter (FTD)

The diameter of the filum terminale was measured on T2-weighted axial MRI. Due to the presence of cauda equina, the dot of the filum terminale may not be clearly distinguishable from the dots of the cauda equina, making it difficult to measure (*Figure 4*). However, as the number of cauda equina decreased, the filum terminale detection rate (FTDR) significantly increased at the L4–5 and L5–S1 levels. We first calculated the FTDR at the L4–5 and L5–S1 levels, then measured the diameter of clearly visualized filum terminale.

The conus medullaris position index (CMPI)

The CMPI refers to the ratio of the vertical distance from the tip of the conus medullaris to the inferior border of the T11 vertebral body, to the straight-line length from the T11 to the S1 vertebral body. It is used to eliminate the interference of height and spinal length on the measurement of conus position, and to objectively reflect the relative position of the conus medullaris in the lumbosacral segment of the spine. A smaller ratio indicates a higher position of the conus medullaris, whereas a larger ratio indicates a lower position.

Statistical analysis

All continuous parameters are expressed as the mean ± standard deviation (SD), and categorical parameters are expressed as proportions. Data analysis was performed via an independent sample t-test and a Chi-squared test. All analyses were performed via SPSS 22.0 software (SPSS Inc., Chicago, IL, USA). $P<0.05$ was considered statistically significant.

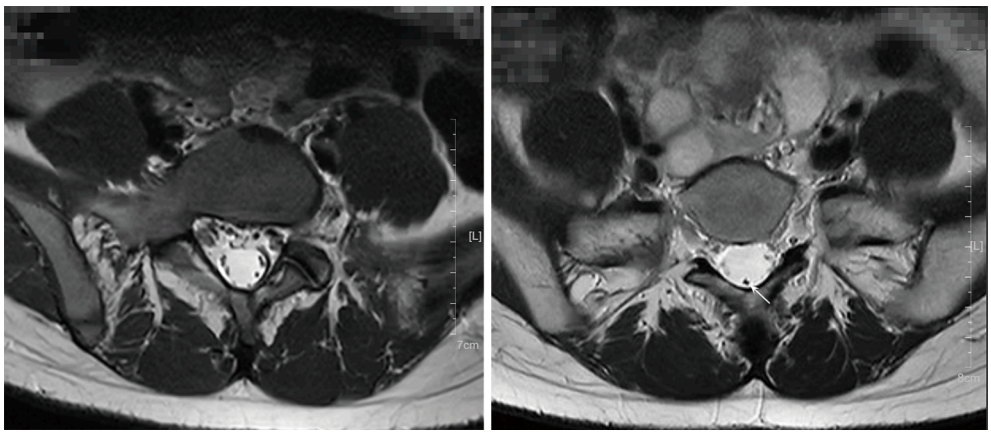


Figure 4 In axial T2-weighted MRI studies of a 19-year-old individual in the control group, the filum terminale is not clearly visualized in L4–5 segment (left). In axial T2-weighted MRI studies of a 19-year-old individual in the DS group, the filum terminale is clearly visualized in L4–5 segment (right). DS, dysplastic spondylolisthesis; MRI, magnetic resonance imaging.

Results

A total of 126 patients were included in the DS group, consisting of 56 (44.44%) with HDS and 70 (55.56%) with LDS. Among them, 58 (46.03%) were male and 68 (53.97%) were female, with a mean age of 19.94±2.69 years. A total of 126 patients were included in the control group, including 58 (46.03%) males and 68 (53.97%) females, with a mean age of 20.08±2.59 years. In the DS group, 73 patients exhibited EPD of the cauda equina, which was significantly different from that in the normal young population (P<0.05). The differences in LSCR between the DS group and the control group are presented in *Table 2*. The results suggest that LSCR in the DS group was significantly greater than that in the control group at the L3–L5 segments (P<0.05). There was no significant difference in DHI between the two groups at any lumbar segment. FTDR in the DS group was significantly lower than that in the control group, and the corresponding diameters were also significantly smaller. CMPI indicated that the relative position of the conus medullaris in the DS group was higher than that in the control group (*Table 2*).

Table 2 Comparison of imaging parameters in the DS group and control group

Parameters	DS group	Control group	P value
LSCR			
L1–L2	0.374±0.061	0.371±0.072	0.703
L2–L3	0.384±0.069	0.372±0.070	0.179
L3–L4	0.406±0.075	0.356±0.061	0.000
L4–L5	0.448±0.076	0.352±0.070	0.000
DHI			
L1–L2	0.335±0.043	0.327±0.044	0.146
L2–L3	0.364±0.041	0.355±0.047	0.112

Table 2 *(continued)*

Table 2 *(continued)*

Parameters	DS group	Control group	P value
L3–L4	0.385±0.062	0.373±0.055	0.111
L4–L5	0.431±0.056	0.438±0.058	0.403
EPD of cauda equina			
L1–L2	5 (3.97%)	0	0.024
L2–L3	12 (9.52%)	0	0.000
L3–L4	33 (26.19%)	3 (2.38%)	0.000
L4–L5	73 (58.73%)	9 (7.14%)	0.000
FTDR			
L4–5	76 (60.3%)	97 (77.0%)	0.004
L5–S1	75 (59.5%)	95 (75.4)	0.007
FTD (mm)			
L4–L5	1.11±0.23	1.22±0.25	0.005
L5–S1	1.03±0.18	1.11±0.22	0.016
CMPI	0.20±0.09	0.23±0.05	0.003

CMPI, conus medullaris position index; DHI, disc height index; DS, dysplastic spondylolisthesis; EPD, excessive posterior displacement; FTD, filum terminale detection; FTDR, filum terminale detection rate; LSCR, lumbar spinal canal ratio.

Among the 70 LDS patients in this study, 26 (37.14%) presented with low back pain, while none of the LDS patients exhibited neurological symptoms. Among the 56 HDS patients, 51 (91.07%) had low back pain, and 7 (13.73%) patients exhibited L5/S1 neurological deficit, with one case accompanied by mild cauda equina syndrome. The 7 patients with neurological deficits underwent surgical treatment using in situ fusion. At the 12-month follow-up, all patients showed significant improvement in neurological function.

Discussion

DS can cause significant morbidity in teenagers Compared with degenerative spondylosis, the characteristics of DS are as follows (2,3): (I) the main diseased segment is L5–S1; (II) the dysplasia of the vertebral surface or lamina (Wedge-shaped deformation of the L5 vertebral body, L5–S1 articular surface dysplasia or spina bifida); (III) it is often accompanied by kyphosis angulation; (IV) L5–S1 disc height reduction (L5/S1 disc height <25% of L4/L5 disc height on lateral radiograph). Because patients with HDS mostly suffer from severe lumbosacral kyphosis and spino-pelvic sagittal imbalance (4-7), HDS has a great impact on their lives.

Currently, the majority of scholars postulate that the etiology of DS is potentially attributed to defects in the pars interarticularis and stress fractures (8-10). In this study, only a few participants had a history of professional sports or physical activity that caused strain on the lumbar spine, with 2 subjects in the DS group and 3 in the control group. Statistical comparison of the proportion of individuals with excessive lumbar load showed no intergroup difference (P>0.05). Current bone-based hypotheses deduce the pathogenesis of DS backward from final isthmic weakness or spondylolysis, and thus cannot explain the unique clinical symptoms and imaging paradoxes of this disease, such as mild symptoms in patients with severe isthmic injury.

The results of this study suggested that, compared with healthy young people, DS patients had significant differences in cauda equina, filum terminale, conus medullaris and the spinal canal. The results revealed that EPD of the cauda equina occurred in some DS patients. The affected nerves were primarily the sacral nerve and the coccygeal nerve. This phenomenon was more likely to occur in the L4–L5 segment, was often accompanied by the disappearance of epidural fat, and was sometimes accompanied by abnormalities in the dural sac and even the vertebral lamina morphology. DS patients had wider spinal canals than normal young people, especially in the L3–L5 segment. On sagittal MRI, the upper end of the spinal canal in these patients was relatively narrow, and the lower end was wider, with a shape similar to that of an eggplant. We termed this the "eggplant-shaped spinal canal" (*Figure 5*). In patients with an eggplant-shaped spinal canal, the cauda equina still coursed closely to the posterior wall of the spinal canal. Moreover, patients with DS have a lower detection rate and a smaller diameter of the filum terminale, and the position of the conus medullaris is correspondingly higher. Although a low FTDR does not directly equate to anatomical thinning or dysplasia of the filum terminale, insufficient image resolution can obscure its clear visualization and introduce bias during image interpretation. Nevertheless, given the unified standardized MRI scanning protocol and double-blind multi-reader quality control that were implemented in this study, FTDR still carries meaningful clinical and statistical reference value.

To further explore the relationship between various imaging parameters and the degree of dysplasia, we compared the imaging parameters of LDS and HDS patients (*Table 3*). The results showed compared with LDS patients, HDS patients were more prone to EPD of the cauda equina, and the difference was statistically significant ($P<0.05$). This indicated that the severer the dysplasia was, the more prone the cauda equina was to EPD.

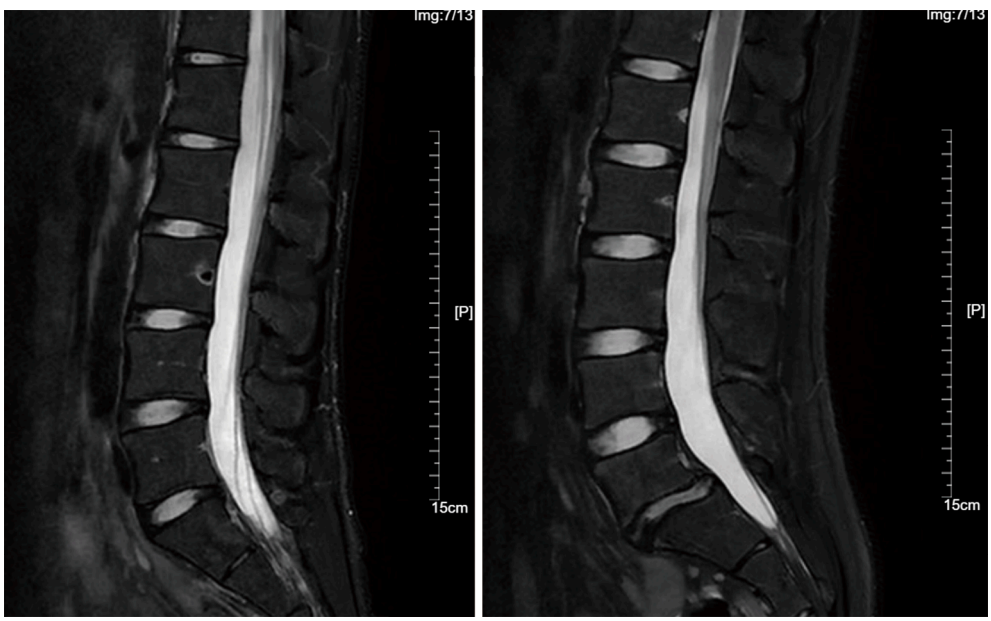


Figure 5 Compared with the spinal canal of an 18-year-old individual in the control group (left), the spinal canal of an 18-year-old DS patient is eggplant shaped (right). DS, dysplastic spondylolisthesis.

Table 3 Comparison of imaging parameters in the HDS group and LDS group

Parameters	HDS group	LDS group	P value
LSCR			
L1–L2	0.370±0.050	0.377±0.068	0.497
L2–L3	0.378±0.066	0.389±0.071	0.404
L3–L4	0.400±0.073	0.411±0.076	0.461
L4–L5	0.451±0.070	0.445±0.081	0.676
DHI			
L1–L2	0.331±0.040	0.338±0.046	0.354
L2–L3	0.361±0.044	0.366±0.039	0.527
L3–L4	0.389±0.062	0.381±0.062	0.496
L4–L5	0.426±0.060	0.436±0.053	0.304
EPD of cauda equina			
L1–L2	4 (7.14%)	1 (1.43%)	0.103
L2–L3	9 (16.07%)	3 (5.36%)	0.025
L3–L4	21 (37.50%)	12 (17.14%)	0.010
L4–L5	40 (71.43%)	33 (47.14%)	0.006
FTDR			
L4–5	30 (53.6%)	46 (60.5%)	0.166
L5–S1	30 (53.6%)	45 (59.2%)	0.223
FTD (mm)			
L4–L5	1.14±0.23	1.09±0.22	0.330
L5–S1	1.06±0.15	1.02±0.20	0.417
CMPI	0.19±0.04	0.21±0.11	0.334

CMPI, conus medullaris position index; DHI, disc height index; EPD, excessive posterior displacement; FTD, filum terminale detection; FTDR, filum terminale detection rate; HDS, high-dysplastic spondylolisthesis; LDS, low-dysplastic spondylolisthesis; LSCR, lumbar spinal canal ratio.

It is difficult to decide whether the EPD of the cauda equina is related to the causation or is a result of the local kyphosis and intervertebral slippage, but there are still reasons to believe the former: firstly, there is a considerable proportion of LDS patients who show minimal local kyphosis and intervertebral slippage, yet they still experience EPD of the cauda equina. This indicates that the changes in the cauda equina occur prior to the development of local kyphosis and intervertebral slippage. Secondly, when LDS develops into HDS, as the L5–S1 intervertebral space narrows and the L5 vertebral body slips forward and downward, the length of the spinal canal decreases, causing the cauda equina to become loose or even redundant. However, conversely, no loose or redundant cauda equina is observed in DS patients. This suggests that spondylolysis is merely a result, not the cause of EPD of the cauda equina.

Then, it is necessary to clarify the cause of EPD of the cauda equina. For patients with DS who require surgical treatment, reduction or fusion in situ is still controversial, but it is undeniable that in situ fusion has the advantage of protecting nerve function (11-13). Owing to the large amplitude of vertebral slip and the obvious narrowing of the intervertebral space, reduction can lead to elongation of the spinal canal, which pulls the cauda equina and causes neurological symptoms. This finding indicates that the length

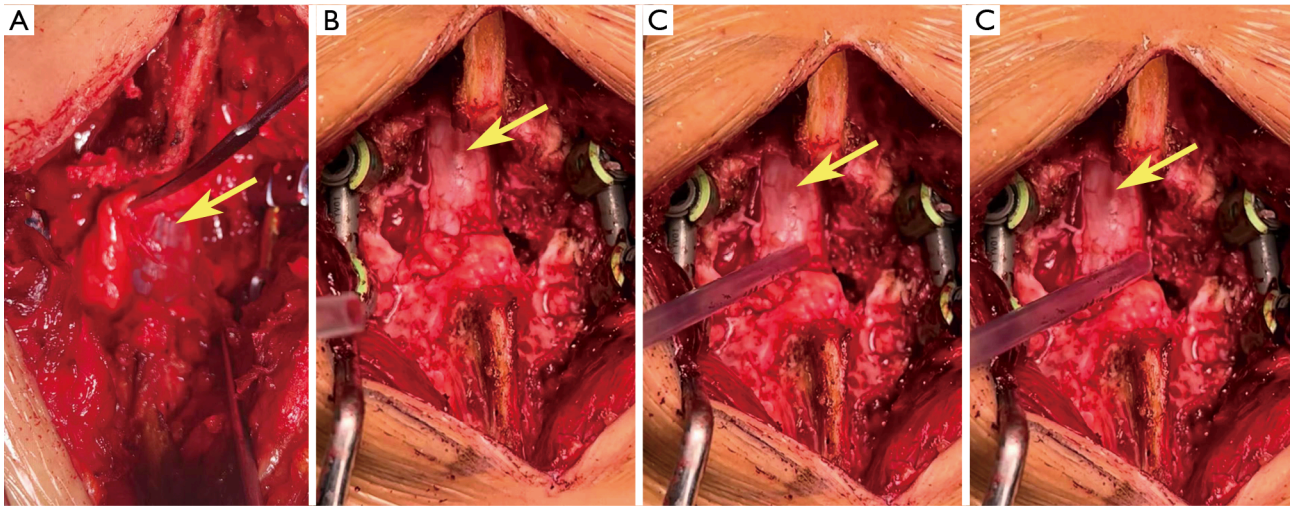


Figure 6 Intraoperative findings of EPD of the cauda equina. Intraoperative view in a 21-year-old individual in the prone position, clearly showing the cauda equina closely apposed to the posterior wall of the dural sac (A). Intraoperative view in a 23-year-old individual, also demonstrating the cauda equina adherent to the posterior dural wall (B). Upon application of slight downward pressure, the cauda equina disappears (C); it rebounds immediately after the pressure is released (D). EPD, excessive posterior displacement.

of the cauda equina in some DS patients cannot match the length of the spinal canal after reduction. In other words, before the occurrence of spondylolisthesis, the cauda equina and the lumbosacral vertebral column are already uncoupled. The cauda equina may be taut, forcing it to choose the shortest path, which ultimately leads to EPD. Furthermore, this viewpoint was corroborated during surgery. Among the 7 HDS patients in this study who required surgical treatment, intraoperative findings revealed taut cauda equina in 5 cases. Even in the prone position, the cauda equina remained closely opposed to the posterior wall of the dural sac. Upon application of a slight downward pressure, the cauda equina disappeared, and they rebounded once the pressure was relieved (*Figure 6*).

However, we believe that besides above factor, the filum terminale has a greater impact on the tension of the cauda equina. The filum terminale stabilizes and fixes the lower end of the spinal cord. It is well known that a thick and tight filum terminale pulls the spinal cord and results in a tethered spinal cord (14). This study demonstrates that DS patients exhibit a lower detection rate and smaller diameter of the filum terminale compared to the control group. It is possible that when the filum terminale is thin, the conus medullaris cannot be properly fixed, resulting in a higher position. The pulling force that should have acted on the filum terminale may act on the cauda equina and increase the axial tension of the cauda equina, perhaps we can call it the "tethered cauda equina".

DS patients often show severe imaging manifestations but mild clinical symptoms, with the majority of patients primarily presenting mechanical midline low lumbar pain. Even among HDS patients, severe neurological deficit is uncommon (15,16). In this study, only 7 (13.73%) patients exhibited L5/S1 neurological deficit, which further supports this viewpoint. We speculate that whether it is the anterior and downward movement of the L5 vertebral body or the narrowing of the L5–S1 intervertebral space, these changes serve to shorten the length of the spinal canal and thereby reduce the axial tension on the cauda equina. In addition, a widened vertebral canal and L5/S1 spina bifida provide ample space behind the cauda equina, indirectly reducing its axial tension and, consequently, protecting the function of the cauda equina.

On the basis of the above findings, we propose the hypothesis of compensatory spondylolisthesis: when the cauda equina is subjected to excessively high axial tension, the lumbosacral vertebrae will undergo spondylolisthesis to reduce this tension, ultimately protecting nerve function. In other words, DS is the expression of bones and discs adapting to nerves or the body's protection and maintenance of the function of nerves. We define this as "compromised regulation", where regenerable tissues with less important functions and simple structures are sacrificed to preserve nonrenewable tissues with crucial functions and complex structures.

In fact, it is not uncommon for nerves to affect bones; for example, fetal vertebral canal size and growth are closely related to the size of the spinal cord (17). The brain determines the size of the skull, and in older children, the bony vertebral canal adapts to accommodate intramedullary tumors. However, the influence of nerves on bone only seems to occur during the growth and development stages of the body. We speculated that growth hormone may play an important role in this regulation.

DS seems to be correlated with idiopathic scoliosis (IS) (15,18,19). Among the 126 patients included in this study, 22 (17.46%) were complicated with IS. This correlation between DS and the IS further confirmed the feasibility of the holistic-spine concept. R. W. Porter (20) proposed the concept of uncoupled neuro-osseous growth to explain the etiology of adolescent IS, and this hypothesis of increased neural axial tension leading to spinal deformity also applies to DS. We speculated that IS and DS may be different manifestations of the same cause in different parts of the spine.

Royo-Salvador *et al.* (21) summarized and outlined the etiopathogenic relationships between Arnold-Chiari syndrome type I, idiopathic syringomyelia and IS and other associated pathologies, such as spinal cord filum disease and neurocranio-vertebral syndrome. We very much agree with the point of view of Royo-Salvador *et al.* because they regard the bony structure and neural structure of the spine as a whole and define the disease and symptoms from a holistic perspective, which is exactly the ideal that we insist on. According to Royo-Salvador *et al.*, the caudal traction force applied to the nervous system by means of the filum terminale is expressed in the form of the descent of the cerebellar tonsils and the spine, which seeks to minimize trauma to the spinal cord, bends and creates abnormal spinal curvatures. However, we argue that owing to the existence of nerve roots, the traction force exerted by the filum terminale on the nervous system cannot be evenly distributed throughout the entire nervous system, nor can it be concentrated at both ends of the nervous system. Inevitably, there will be variations in the strength of this traction force at different parts of the nervous system. We speculate that when the axial caudal force is concentrated on the upper cervical spine, it manifests as Arnold-Chiari type I syndrome and skull base lesions. When the axial caudal force is concentrated on the thoracic spinal cord, it manifests as IS. When the axial caudal force is concentrated on the conus spinal cord, it manifests as a tethered spinal cord. When the filum terminale is thin, the cauda equina nerve bears the reverse pulling force of the nervous system, which manifests as DS. These pathological changes may occur alone or simultaneously according to the location of the nervous system being stretched. This is a reasonable speculation on the basis of the present study and a summary of the previous literature.

This paper is a retrospective study with a small number of samples and a high level of speculation, rendering the results less convincing. Furthermore, only supine MRI protocols were adopted, and prone scanning was not performed routinely. Owing to the absence of dedicated prone positioning devices, prone sequences were compromised by respiratory motion artifacts, thus preventing us from evaluating the reversibility of EPD of the cauda equina in the prone posture. Moreover, the tension of the filum terminale and cauda equina cannot be directly quantified *in vivo*. In the present study, neural tension could only be indirectly inferred from imaging morphological characteristics. Finally, a reasonable explanation cannot be given for the specific mechanism by which the cauda equina affects bone.

Conclusions

Compared with the control group, patients with DS exhibit thinner filum terminale, a relatively higher conus medullaris position, a wider spinal canal, and a greater prevalence of cauda equina EPD. Based on the above morphological correlations, we put forward an exploratory compensatory spondylolisthesis hypothesis to interpret the observed phenomenon.

Acknowledgments

None.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-2026-0591/rc

Data Sharing Statement: Available at https://qims.amegroups.com/article/view/10.21037/qims-2026-0591/dss

Funding: This work was supported by Provincial Government Funding for the Cultivation of Outstanding Talents in Clinical Medicine of China (No. ZF2024088).

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-2026-0591/coif). X.M. reports that this work was supported by Provincial Government Funding for the Cultivation of Outstanding Talents in Clinical Medicine of China (No. ZF2024088). The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. The study was approved by the Ethics Committee of Third Hospital of Hebei Medical University (No. W2024-017-1) and informed consent was obtained from all individual participants and legal guardians.

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Cite this article as: Liu K, Chang H, Li H, Zhang Z, Meng X. A new perspective on dysplastic spondylolisthesis: imaging analysis and hypothesis on the pathogenesis. Quant Imaging Med Surg 2026;16(11):862. doi: 10.21037/qims-2026-0591