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Sagittal balance analysis and treatment rationale for young patients with symptomatic lumbosacral transitional vertebrae

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Lumbosacral transitional vertebrae (LSTV) are a common anomaly in 7–36% of the population. LSTV can contribute to mechanical low back pain and increase the risk of early degeneration in cranial segments due to hypermobility and stress. This study analyzed sagittal balance in young symptomatic LSTV patients and tried to identify compensatory mechanisms that may explain early degeneration. Nineteen symptomatic and skeletally mature subjects with LSTV were retrospectively identified. Imaging included standing biplanar spine radiographs and supine lumbar MRI. Sagittal balance parameters were measured, and LSTV were classified using the Castellvi classification. Vertical mid-vertebral angle differences were calculated using MRI and lateral radiographs. The cohort included 17 females and 2 males (mean age 16 ± 3 years). Mean pelvic incidence was 67°±8°. L1-S1 lordosis averaged 61°±10°, L4-S1 lordosis was 10° lower than expected, and L4-L5 lordosis was higher than literature values. Thirteen patients had L4-L5 discopathy, with nine showing additional abnormalities such as interspinous ligament edema or posterior facet hypertrophy. In our study, LSTV was associated with L5-S1 disc hypoplasia and altered lumbar lordosis, leading to compensatory L4-L5 hyperextension. These findings suggest early degeneration may result from abnormal lordosis distribution. Treatment should aim to optimize lordosis distribution to reduce stress on adjacent segments.

Keywords Lumbosacral transitional vertebrae, Sagittal balance, Lordosis restoration, Surgical planning

Lumbosacral transitional vertebrae (LSTV) is common throughout the general population, with a prevalence ranging from 7 to 36%[1,2]. LSTV are defined by a morphological variation in the lumbosacral junction, with two categories, depending on the spinal level of the anomaly. These include sacralisation of L5 (the most caudal lumbar segment) or lumbarisation of S1 (the most cranial sacral vertebra). In the former case, transverse processes are enlarged and can articulate with the sacrum or the ilium to varying degrees, from partial (pseudarthrosis) to complete fusion. In the latter case, an intervertebral disc develops between the first and second sacral segments, resulting in a sixth lumbar vertebra[1,3].

Mario Bertolotti was the first to describe the morphological characteristics of LTSV and their association with low back pain, especially in young patients, thus lending his name to *Bertolotti syndrome*[4]. Classifications and diagnostic tools have been described previously in the literature. The Castellvi classification divides LTSV into four types according to how the last lumbar vertebra's enlarged transverse process (commonly > 19 mm) is linked to the sacrum[1]. *A* and *B* variants specify whether the pattern is unilateral or bilateral[5,6]. Farshad recently described the most caudal vertebra's vertical mid-vertebral angle (VMVA) as a helpful measurement for diagnosing LSTV using sagittal magnetic resonance imaging (MRI) or a lateral view X-ray[7].

Spinal sagittal balance is a key concept in spinal pathology. A harmonious alignment of the spine's shape in the sagittal and coronal planes is associated with improved quality of life[8,9]. Relationships between spinopelvic parameters can suggest non-operative treatment strategies and are essential for surgical planning in patients with degenerative disease. Recent studies have evaluated spinopelvic parameters in adults with LSTV[10–12] and have compared them with asymptomatic subjects without LSTV. Most of these studies included large age ranges and sagittal balance evaluations can sometimes be distorted by other degenerative processes. To the best of our

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knowledge, no recent studies have described how LSTV affect the spine's sagittal balance and the compensation mechanisms on adjacent levels that could explain early symptoms in young patients.

The present study aimed to analyse sagittal balance in young patients presenting with LSTV with sacralisation of the most caudal lumbar vertebra associated with low back pain. Our hypothesis was that these patients had an altered distribution of lumbar lordosis due to a lack of lordosis at the transitional level and the need to use hyperextension compensation mechanisms at adjacent levels to keep overall lumbar lordosis within normal ranges. The L4-L5 segment is particularly vulnerable due to its proximity to the transitional level and is the segment that must hyperextend the most. This scenario would explain early symptoms in young patients even before the appearance of segmental degeneration.

Materials and methods

Subjects

We identified young patients with LSTV evaluated for pain in the lower lumbar area at Geneva University Hospitals' Paediatric Orthopaedics Unit (Geneva, Switzerland) and by an independent spine surgeon from La Tour Hospital (Meyrin, Switzerland) between November 2011 and July 2024. Symptomatic subjects were defined using a visual analogue scale score for pain > 4/10 on the day of their initial evaluation and their history of functional impairment. Inclusion criteria were the presence of LSTV with sacralisation of the most caudal lumbar vertebra and radiological skeletal maturity. Exclusion criteria were a history of trauma or a tumour, previous spinal surgery, age > 30 years old, a coronal plane spinal deformity and refusal to participate. The study was approved by the cantonal ethics committee (2023 – 00978) as all the subjects had provided a written informed consent form allowing the use of their data in research projects. All methods and analysis were performed in accordance with the relevant guidelines and regulations.

Imaging analysis

Radiological data were extracted from the two hospitals' Picture Archiving and Communication Systems (PACS), and Osirix MD software (Pixmeo SARL, CH-1233 Bernex, Switzerland) was used for radiological analysis. Radiological measurements were performed independently by an orthopaedic surgery resident and a senior spine surgeon. Patients' radiological assessments included standing full-spine biplanar low-dose imaging (EOS) and lumbar MRI. All participants had been imaged in a standard position with fists on clavicles[13,14] to achieve reliable and functional positioning (Fig. 1).

LSTV were diagnosed on MRI and X-ray images using the Castellvi classification and measurements of the VMVA[7]. A VMVA difference < 10° was used as a cut-off for the presence of LSTV. Skeletal maturity was defined using the Risser classification[15]. It was previously described in literature that incidence of L5 (L5I) could be used instead of Pelvic Incidence (PI) in subjects with sacralisation of L5 and consider L5 as the base of pelvis to adapt the new pelvic morphology[16]. Because this concept is not universally accepted, it was decided to keep S1 as a reference for the base of the pelvis. The spinopelvic parameters of PI, Sacral Slope (SS) and Pelvic Tilt (PT) were also measured. The Sagittal Vertical Axis (SVA) was measured at T1 and T12 levels (mm). L1-S1, L1-L4, L1-L5, L4-S1 and L5-S1 lordosis were measured in every subject using the Cobb angle method. Theoretical values for L1-S1, L1-L4 and L4-S1 lordosis were calculated using the formulas described in the literature and based on PI[2,16]. Segmental lordosis was defined by the angle between the superior endplate at a given spinal level and the superior endplate of the level below[17]. Each intervertebral disc's lordotic angle was measured between the endplates above and below it. The lumbar spine's apex was determined by measuring the maximal perpendicular distance between the midpoint of the posterior wall of each lumbar vertebra and the tangent line between the posterosuperior angle of L1 and the posterior angle of S1[18]. The modified Pfirrmann classification was used to assess disc degeneration[19]. Annular tears and posterior disc protrusions were documented. The presence of oedema in the pedicles, facet joints, isthmus or interspinous ligaments was recorded. The presence of spondylolisthesis above the transitional vertebra was identified using MRI and/or the lateral radiograph[20] and evaluated using software's dedicated tool "Perpendicular Lines" to limit measuring error.

Statistical analysis

Descriptive statistics were used to summarise the data. Continuous variables were reported as mean ± standard deviation, median and range, whereas categorical data were reported as proportions. The normality of continuous variable distributions was assessed using the Shapiro-Wilk test, and the normality of the residuals was assessed visually on a Q-Q plot. Differences between paired continuous data were examined using either paired Student t-tests or the Wilcoxon signed-rank test. Inter-rater reliability for radiographic measurements was evaluated using intra-class correlation coefficients ($ICC_{2,1}$) based on single raters (A.T and A.F), absolute-agreement, 2-way random-effects model. Values < 0.5 are indicative of poor reliability, values between 0.5 and 0.75 indicate moderate reliability, values between 0.75 and 0.9 indicate good reliability, and values greater than 0.90 indicate excellent reliability. All analyses were performed using R software (version 3.6.2, R Foundation for Statistical Computing, Vienna, Austria), and p-values < 0.05 were considered significant.

It has been previously published that ≥ 5 degrees of lordosis can be considered as an indicator or true radiographic change[21,22]. Furthermore, Pesenti et al. reported a standard deviation of 8° when evaluating the cumulative lordosis at the L4-S1 level[17]. An a priori sample size calculation was then performed before the study using a one-tailed paired Student t-test (statistical power of 0.80 and a significant alpha level of 0.05) and indicated that 18 patients were needed to significantly detect a minimum difference of 5° (± 8°) between the cohort and theoretical L4-S1 cumulative lordosis.

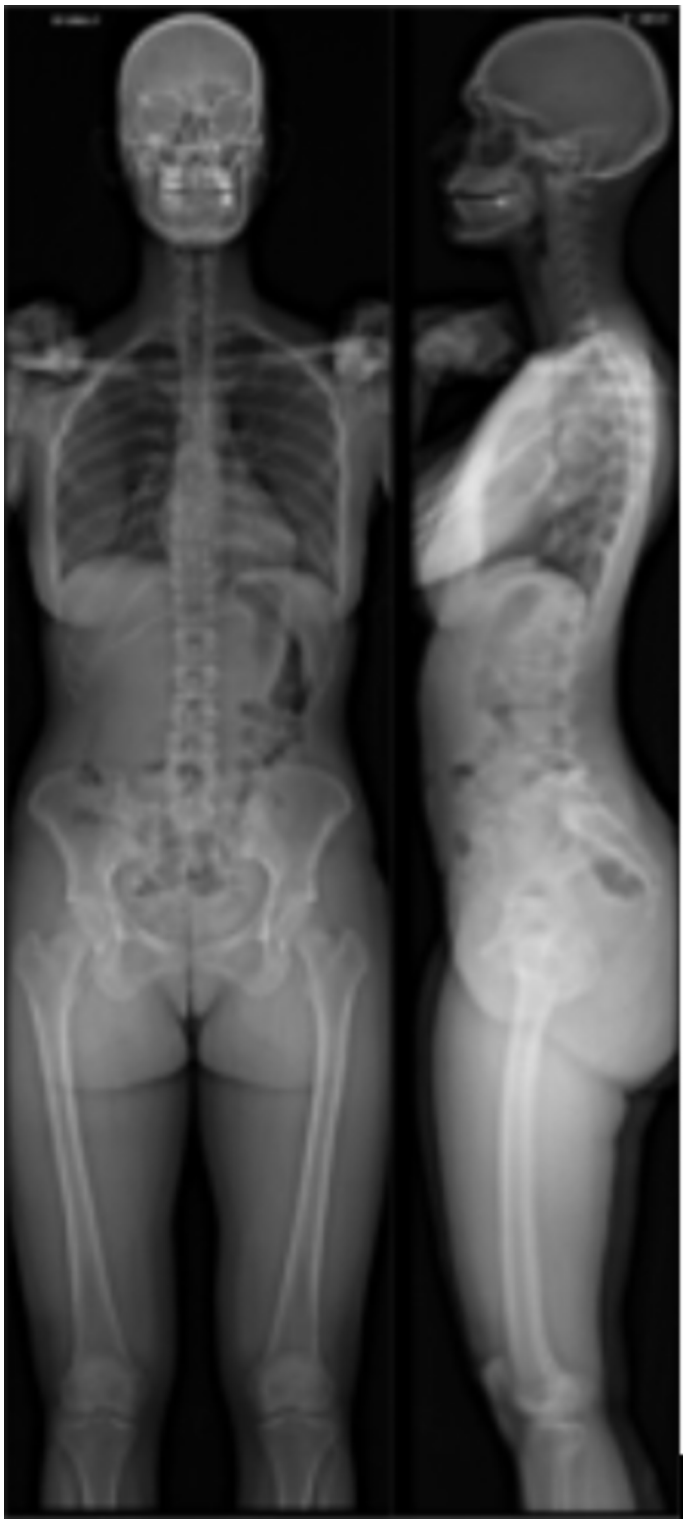

Fig. 1. The standard biplanar low-dose imaging position used for every subject, with feet 10–15 cm apart and fingertips resting upon the ipsilateral clavicles, aiming for a reproducible body position and fewer inaccurate measurements secondary to posture variations.

Results

Of the forty-two subjects evaluated with LSTV during the study period, 19 finally met the criteria for further analysis (Fig. 2). The study examined 19 subjects (17 females, 2 males) with a mean age of 16 ± 3 years old (Table 1). The reliability of radiographic measurements was moderate for the lordosis at L5-S1 disc (ICC, 0.72). The reliability was good for the lordosis measurement at L3-L4 (ICC, 0.80), L1-L2 (ICC, 0.83) and L4-L5 discs

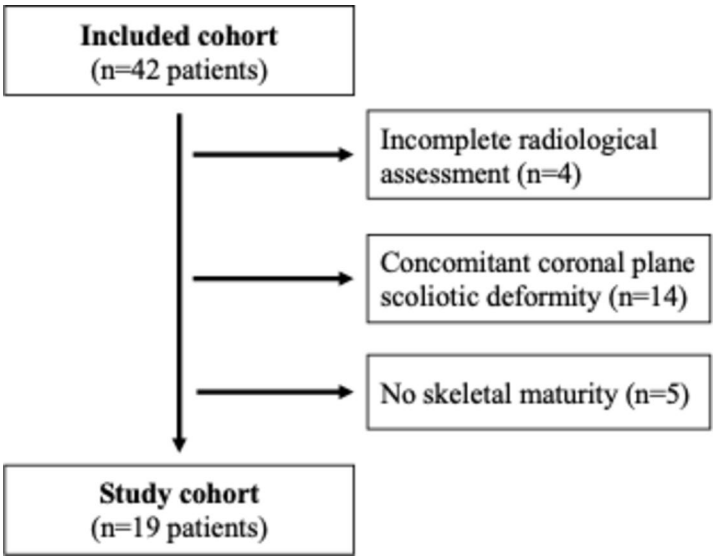

Fig. 2. Study flowchart.

	N	(%)		
	Mean	**±SD**	**Med**	**(Range)**
Sex				
Female	17	(89)		
Male	2	(11)		
Age (years)	16	3	16	(13, 27)
Height (cm)	158	7	159	(143, 172)
Weight (kg)	55	11	52	(45, 77)

Table 1. Demographic data. *SD* standard deviation, Med median.

(ICC, 0.89) as well as excellent for all remaining radiographic measurements (ICC ≥ 0.90): pelvic incidence (ICC, 0.95), sacral slope (ICC, 0.94), pelvic tilt (ICC, 0.97), global lordosis at L1-S1 (ICC, 0.96), L1-L5 (ICC, 0.97), L1-L4 (ICC, 0.92) and L4-S1 (ICC, 0.92) levels, as well as for the lordosis at L2-L3 disc (ICC, 0.92). 11 subjects (58%) had a type IIA Castellvi anomaly and 8 (42%) had a type IIB anomaly. All subjects had a VMVA difference < 10°. Subjects had a mean PI of 67°±8°, a mean SS of 45°±7° and a mean PT of 21°±7° (Table 2). They had a mean L1-S1 lordosis of 61°±10°, a mean L1-L5 lordosis of 46° ± 8°, a mean L4-S1 lordosis of 34°±7° and a mean lordosis at the L5-S1 disc of 1°±2° (Table 2).

Figure 3 illustrates the distribution of lordotic angles by lumbar disc (°). The lumbar lordosis apex was located at the L3 level in 12 patients (63%), at L2 in 4 (21%) and at L4 in 3 (16%). Segmental lordosis at the L4-L5 and L5-S1 levels accounted for 32%±8% and 23%±12% of overall lordosis, respectively.

Seven subjects (37%) presented with a Roussouly type III spine shape and 12 presented with a Roussouly type IV (63%). Lordotic disc angles (°) were compared to the literature according to the Roussouly morphotype (Table 3; Fig. 4)[23].

The mean L1-S1 lordosis of our LSTV subjects did not differ statistically from values calculated (61°±10° vs. 65°±4°, $p = 0.136$). However, their segmental lordosis was significantly different from comparative theoretical values. Their mean L1-L4 lordosis was higher than theoretical values by approximately 5° (27°±6° vs. 22°±2°, $p = 0.004$) and their mean L4-S1 lordosis was significantly lower by almost 10° (34°±7° vs. 43°±3°, $p = 0.004$) (Table 4).

An MRI evaluation was available for 16 subjects, revealing anomalies in 13 (81%). Most presented with mild L4-L5 retrolisthesis ($n = 12$, 63%), one (5%) had spondylolytic spondylolisthesis. Thirteen subjects (81%) presented with a discopathy. The discopathies were located at the L4-L5 level for eight subjects (61.5%), at the L3-L4 and L4-L5 levels for four subjects (31%), and at every level from L1 to L5 for one subject (7.5%). One subject (7.5%) had stage III disc degeneration (on the modified Pfirrmann classification), five (38%) had a stage IV and two had a stage V at the L4-L5 disc segment. Nine examinations (56%) revealed a mild posterior central disc protrusion. Two subjects (12.5%) had radicular compression at the L4-L5 segment in addition to concomitant posterior disc protrusion at the L3-L4 level. Nine subjects presented with additional abnormalities: four (44.5%) with oedema of an interspinous ligament, three (33.5%) with posterior facet hypertrophy and oedema, one (11%) with an isthmus oedema and one (11%) with a pedicular oedema.

	Study cohort (n = 19)			
	Mean	± SD	Med	(Range)
Pelvic incidence (°)	67	± 8	67	(52, 80)
Sacral slope (°)	45	± 7	45	(35, 62)
Pelvic tilt (°)	21	± 7	21	(2, 33)
L1–S1 lordosis (°)	61	± 10	61	(48, 80)
Cumulative lordosis (°)				
L1–L5	46	± 8	46	(32, 62)
L1–L4	27	± 6	28	(15, 39)
L4–S1	34	± 7	33	(24, 46)
Segmental lordosis (°)				
L5–S1	14	± 7	11	(8, 34)
L4–L5	20	± 5	18	(13, 30)
Lordosis proportion (%)				
L1–L4	44	± 8	46	(28, 55)
L4–S1	55	± 8	52	(45, 72)
L5–S1	23	± 12	21	(13, 56)
L4–L5	32	± 8	31	(21, 53)
Lordosis (°) per intervertebral disc				
L5–S1	1	± 2	1	(0, 7)
L4–L5	14	± 4	13	(9, 20)
L4–L3	10	± 2	10	(7, 16)
L3–L2	7	± 3	6	(2, 13)
L2–L1	4	± 2	4	(2, 9)
Sagittal vertical axis (mm)				
T1-S1	−0.7	± 17	3.6	(−29.1, 24.4)
T12-S1	7.5	± 20	7.4	(−29.1,44)

Table 2. Spinopelvic parameters. *SD* standard deviation; *Med* median.

Discussion

The principal finding of this study was that segmental lordosis at the L4-S1 level was reduced by approximately 10° in LSTV patients compared to theoretical values. It confirms our initial hypothesis that these patients would have a significantly altered lumbar lordosis distribution due to a lack of lordosis at the transitional level. The expected hyperextension compensation mechanism in LSTV patients was also revealed by our results given we observed a higher cumulative lordosis at L1-L4 segment by approximately 5°.

Previous studies have demonstrated that LSTV protect subjects from disc degeneration at the transitional segment but that the adjacent levels are prone to greater and earlier disc degeneration or spondylolisthesis[24–27]. This is observed among younger individuals in particular[4,24]. The greater the osseous bridging of the LSTV, the less mobile that level is, and thus the mobility and stress on the lumbar spine are transferred to the adjacent mobile levels[5]. In our knowledge, our study is the first analysing sagittal balance in young symptomatic patients with LSTV and trying to extrapolate findings for a better understanding of the symptoms as well as pathogenesis of accelerated degeneration on adjacent levels. Patients with LSTV present with hypoplasia of the L5-S1 disc[24]. In our study, this was observed as the inferior endplate of L5 and the superior endplate of S1 were almost parallel (1°±2° of lordotic angle of the L5-S1 disc. See Figs. 3 and 4). These values were significantly lower (> 10° lower) than those described in the literature concerning asymptomatic patients without LSTV (Table 2)[23]. The lordosis at the L4-L5 and L5-S1 levels, which accounted for 32% and 23% of the overall L1-S1 lordosis, respectively, were considerably different from that described in the literature on asymptomatic patients without LSTV (27% at L4-L5 and 35% at L5-S1)[17].

There was no statistical difference between the L1-S1 lordosis measured and the theoretical lordosis based on PI when using the formula 0.56*PI + 27.6, as previously described by Le Huec et al.[2]. However, when the upper (L1-L4) and lower (L4-S1) lumbar arcs were compared, lordosis was found to be significantly higher than theoretical values for the upper arc (by approximately 5°; $p = 0.004$) and significantly lower in the lower arc (by approximately 10°; $p < 0.001$)[17].

The findings shown above confirmed our initial hypothesis. The study population's overall L1-S1 lordosis matched the expected values. The distribution of their lordosis was abnormal, however, with significantly less lordosis in the inferior lumbar arc than expected. All of our patients had high PI values, with a mean PI of 67°, concordant with the findings of Verhaegen et al.., who also showed higher PI values in subjects with LSTV[10–12]. The hypoplastic L5-S1 disc is probably the main structure responsible for this lack of lordosis. In our opinion, the levels above the transitional vertebra, especially the L4-L5 level, must compensate by hyperextending to keep the L1-S1 lordosis stable, leading to increased stress at these levels (L4-L5 lordosis accounted for 32% of overall lordosis in our study compared to 27% in the literature). MRI studies confirmed the presence of hyperextension

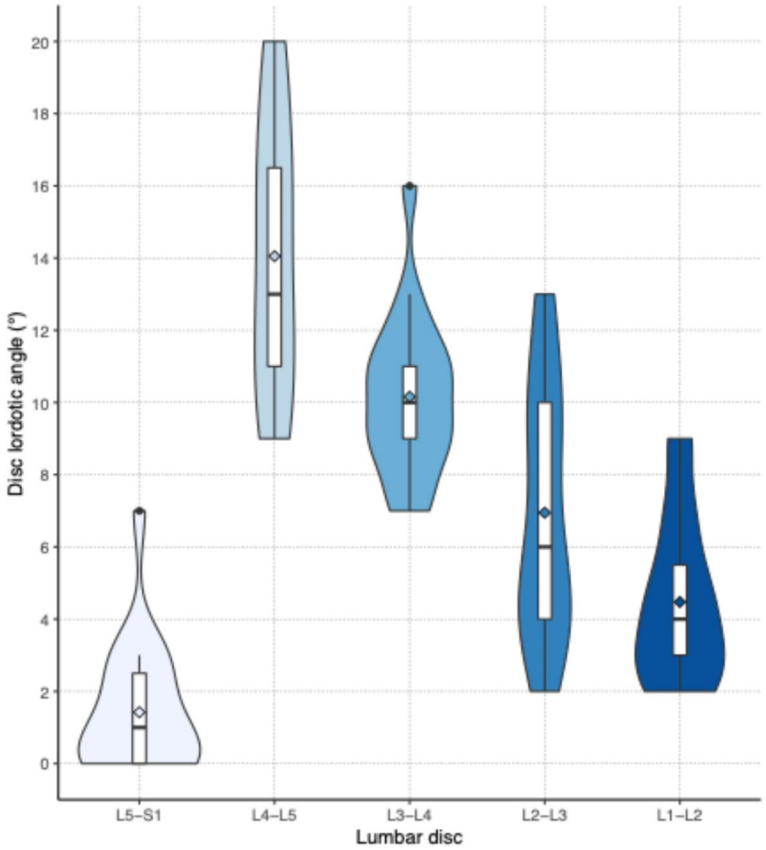

Fig. 3. Distribution of lordotic angles by lumbar disc (°).

	Study cohort				Literature [23]	
	Mean	± SD	Med	(Range)	Mean	± SD
Roussouly type III (*n* = 7)						
L1–L2	3	± 1	2	(2, 5)	4	± 3
L2–L3	7	± 4	4	(2, 13)	7	± 3
L3–L4	11	± 3	11	(7, 16)	10	± 3
L4–L5	16	± 3	16	(11, 20)	14	± 4
L5–S1	1	± 1	0	(0, 2)	12	± 4
Roussouly type IV (*n* = 12)						
L1–L2	5	± 2	5	(3, 9)	5	± 3
L2–L3	7	± 3	7	(4, 13)	8	± 3
L3–L4	10	± 2	10	(7, 12)	12	± 2
L4–L5	13	± 4	13	(9, 20)	16	± 3
L5–S1	2	± 2	1	(0, 7)	15	± 3

Table 3. Lordosis (°) at each intervertebral disc space by Roussouly type. Cohort values versus literature findings. *SD* standard deviation, *Med* median.

mechanisms alongside retrolisthesis, posterior facet oedema, hypertrophy and interspinous ligament oedema. Moreover, L4-L5 disc degeneration with posterior disc protrusion was found in most of our subjects, with five patients having simultaneous L3-L4 disc degeneration and two presenting with two-level posterior disc protrusion. Finally, it has been previously established, and also confirmed by our study, that subjects with high PI angles have an increased risk of spondylolisthesis and posterior facet degeneration[10,26,28]. In our group, 12 subjects presented with the Roussouly type IV morphotype, meaning that their spines had recreated large lordotic curves to match the PI with L1-S1 lordosis.

LSTV are a developmental variation that interferes with lumbar spine development, especially the L5-S1 disc segment, and induces increased stress on adjacent levels that lead to early degenerative changes, starting at

	Cohort real values				Cohort theoretical values				
	Mean	±SD	Med	(Range)	Mean	±SD	Med	(Range)	*p*-value
L1–S1	61	± 10	61	(48, 80)	65	± 4	65	(57, 72)	0.136
L1–L4	27	± 6	28	(15, 39)	22	± 2	22	(19, 25)	*0.004*
L4–S1	34	± 7	33	(24, 46)	43	± 3	43	(37, 48)	*< 0.001*

Table 4. Study cohort's real lordosis vs. theoretical cumulative lordosis (°). *SD* standard deviation, *Med* median. Underlined *p*-values indicate those below 0.05.

paediatric or adolescent ages. We tried to develop a rationale that clinicians could use when treating patients with LSTV, even in paediatric populations. Early non-operative treatment should always be attempted by adapting the patient's everyday activities combined with physiotherapy to reinforce the erector spinae, anterior abdominal wall muscles and stretch the iliopsoas[29,30]. The patient's pelvis will then have a greater capacity for retroversion and will be able to partially compensate for their lumbar hyper-lordosis[31]. This approach could avoid overuse and fatigue of the spinal erector muscles that can contribute to painful symptoms[32]. Unfortunately, LSTV appear early and have consequences on spinal development, especially the L5-S1 disc. Once subjects become symptomatic, non-operative treatment can be overwhelmed by the development of a degenerative discopathy with instability that is sometimes associated with compressive herniations at levels cranial to the transitional vertebra. In such cases, surgery may be necessary to decompress and restore local lordosis.

Figure 5 shows the radiograph of a 27-year-old female patient from our study group. She was evaluated for low back pain and L5 radicular pain. The radiological assessment showed a Castellvi type IIB LSTV, and MRI demonstrated a modified Pfirrmann stage V degenerative discopathy at the L4-L5 level with posterior protrusion and L4-L5 retrolisthesis. She presented with degeneration at the L3-L4 level and, again, a Pfirrmann stage V discopathy. Initial nonoperative treatment aiming reinforcement of abdominal wall and erector spinae muscles was conducted for two years. Because of persistent pain and progression of radiological disc degeneration at

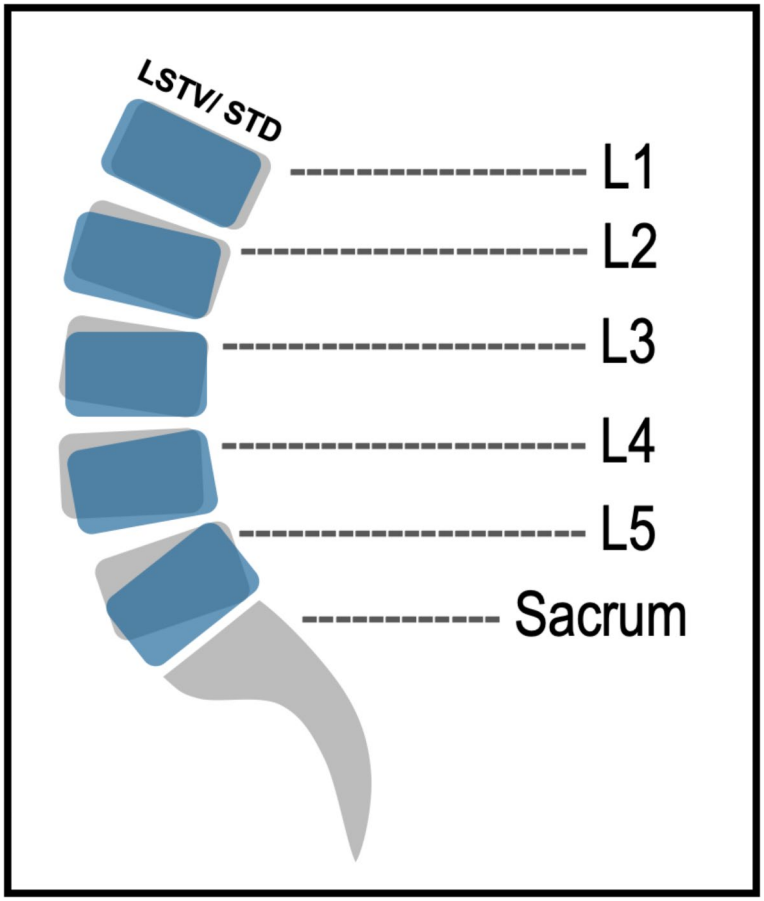

Fig. 4. Illustration of the relative lordosis differences at each intervertebral disc space between the present study's LSTV subjects (in blue) and the standard values of asymptomatic patients without LSTV (in grey) published in the scientific literature[23]. Our subjects presented with excessive lordosis at the L4–L5 level to compensate for L5 and S1 being parallel. The overall L1–S1 lordosis angles matched, however.

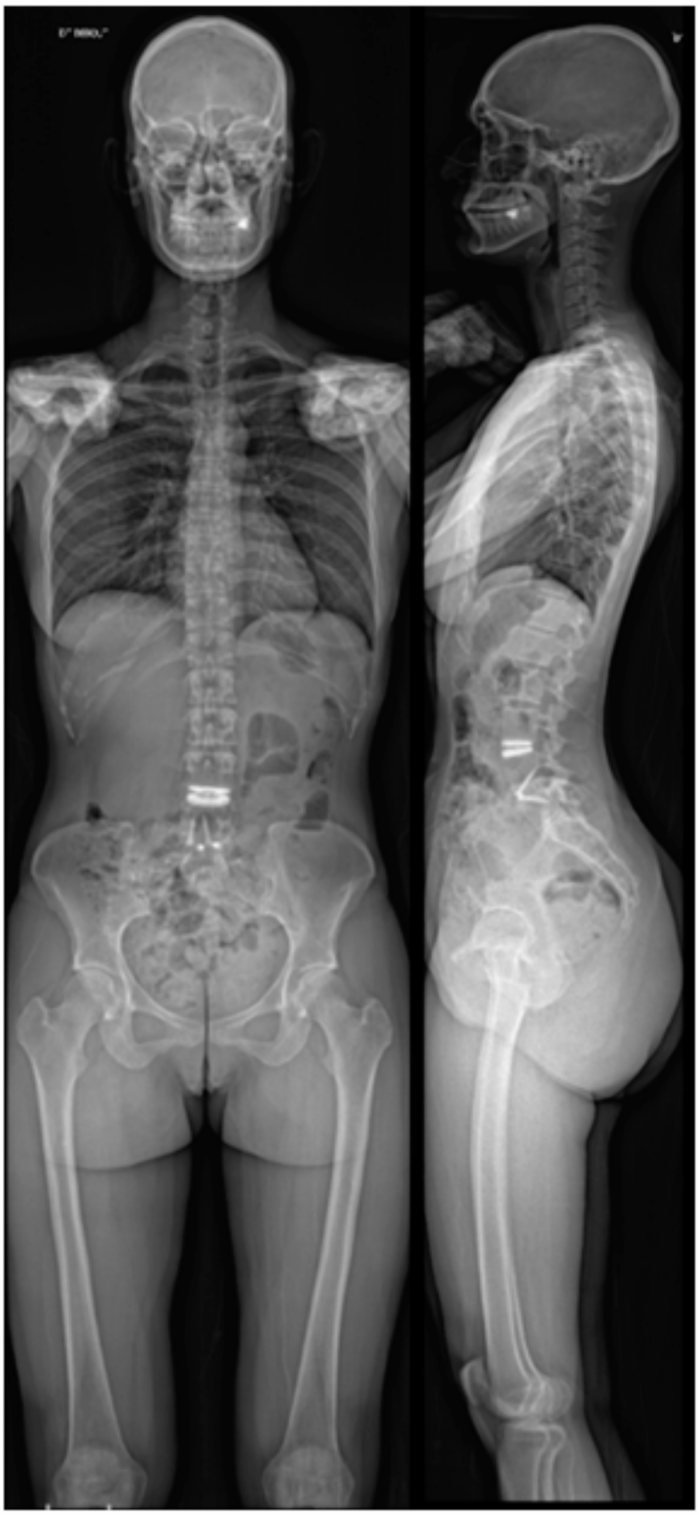

Fig. 5. Illustration of a subject with LSTV and advanced discopathy who underwent an L4-L5 ALIF using a hyper-lordotic cage and an L3-L4 disc prosthesis.

the L3-L4 and L4-L5 levels, surgery was proposed. She underwent Anterior Lumbar Interbody Fusion (ALIF) at the L4-L5 level and simultaneous disc prosthesis at the L3-L4 level. The surgical strategy was to maximise the lordotic correction at the L4-L5 level and harmonise the distribution of overall lumbar lordosis by concentrating it in the L4-S1 levels. This approach should decrease stress on adjacent cranial levels due to hyperextension compensation. After surgery, her overall L1-S1 lordosis matched with theoretical values based on PI, and 66% of her lordosis was located at the L4-S1 level. Post-operative assessment and follow-up showed good clinical and

radiological results with no significant pain (pre-operative visual analogue scale pain scores fell from 5/10 in the lumbar area and 4/10 for leg pain to 0/10 for lumbar pain and 2/10 for leg pain 2 years post-operatively) and no functional impairment (Oswestry Disability Index at 42% pre-operatively and 6% 2 years post-operatively). This clinical case supports the rationale for treating patients with LSTV and degenerative spinal alterations, even at young ages.

This study had some limitations. Firstly, symptomatic subjects were defined based on mechanical pain in the lower lumbar area with a visual analogue scale score > 4/10, but no more extended evaluations of pain scales or clinical scores were available for the quantification of functional impairment. Secondly, the patient group included in the study was small, and more subjects would be needed to confirm our hypothesis and enable subgroup comparisons or advanced statistical analyses. We decided not to include a control group of subjects without LSTV and instead used the theoretical values and formulae available in the literature to calculate lordosis distributions across the global lumbar area and the superior (L1-L4) and inferior (L4-S1) lumbar arcs. Finally, care should be taken when diagnosing LSTV, as incomplete or inaccurate spine imaging can lead to under- or over-estimations of sagittal balance analyses and mislead clinicians planning both non-operative and operative treatments.

Conclusion

The present study enabled us to understand the biomechanical effects of LSTV in young patients. Compensation mechanisms appear early in the disease in the cranial spinal levels, especially hyperextension of the L4-L5 segment. LSTV are a morphological change present from childhood, and they influence the development of the spine during growth. Patients with LSTV may become symptomatic earlier in life than subjects without LSTV because of the inherent mechanical disorder and stress. When treating patients with LSTV, efforts should be made to restore harmony to the lumbar curve, especially the lower L4-S1 lumbar arc. Further investigations into spinopelvic parameters and their correlations in LSTV patients, as well as the definition of the base of the pelvis and reference taken between L5 and S1 in sacralisation subjects, will need larger samples to help validate the present work. Finally, a longitudinal study comparing LSTV subjects to controls and examining the behavior of spino-pelvic parameters over time could help understanding the pathology.

Data availability

The data that support the findings of this study are not openly available due to reasons of sensitivity. They are available from the corresponding author upon reasonable request. Data are located in controlled access data storage at Geneva University Hospital and Hopital de La Tour.

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Author contributions

A.T., R.D. and A.F. collected data. H.B. performed statistical analysis. A.T. prepared the main manuscript and figures. All authors reviewed and approved the manuscript.

Declarations

Competing interests

The authors declare no competing interests.

Human ethics and consent to participate declaration

The study was approved by the cantonal ethics committee (2023 – 00978) as all the subjects, or their parent and legal guardian had provided a written informed consent form allowing the use of their data in research projects. Moreover, the authors affirm that human research participants provided informed consent for publication of the images in (Figs. 1 and 5). All methods and analysis were performed in accordance with the relevant guidelines and regulations.

Additional information

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