Although the etiology of scoliosis in elderly individuals is unclear, DLS is a form of adult-onset lumbar curve degeneration due to severe disc degeneration, without evidence of previous scoliosis. Compared with adult idiopathic scoliosis, DLS shows minimal structural vertebral deformity, advanced degenerative changes, a smaller lumbar curve, and multiple degenerated discs not associated with pain.7

Degenerative lumbar scoliosis in elderly patients with no history of idiopathic scoliosis occurs after skeletal maturity and is associated with disc and facet joint degeneration. Over a period of 10 years, scoliosis curves in elderly patients have been reported to increase 3°–18°, with no direct relationship found between scoliosis and degenerative changes.17 By contrast, DLS has been reported to be triggered by disc degeneration at the lumbar segments13 and associated with asymmetrical disc degeneration.10

Because of the increase in the average life span, the prevalence of degenerative disease is increasing. The eti-
Risk of progression of degenerative lumbar scoliosis

ology and risk factors associated with DLS progression are, however, not clear. The prediction of the progression of scoliosis curvature combined with degenerative change may help determine whether patients should be treated conservatively or surgically, as well as help decide on the extent of surgery. Instrumented fusion has been recommended for patients with DLS. In addition, patients with DLS are at higher risk for the development of multisegmental instability, leading to poor treatment outcomes. Therefore, in this study the risk factors associated with the progression of DLS were assessed.

Methods

Patients who had undergone treatment for DLS between 1987 and 2000 were identified from the medical records of our hospital. The study was performed after our institutional review board approved a retrospective chart review and outcome assessment. The inclusion criteria were the following: 1) age older than 50 years at initial visit; 2) no associated congenital, developmental, or neuromuscular spinal abnormalities; 3) no osteoporotic compression fractures of the spine; 4) no history of spinal surgery; and 5) scoliosis greater than a 10° coronal curvature at initial visit, as measured by the Cobb method. Patients with a history of idiopathic scoliosis when they were young were excluded. This study was performed using patients who had refused surgery at the initial visit or during follow-up, even though they had severe pain and claudication. During that period, 196 patients were surgically recommended for the treatment of DLS. Patients received surgical intervention within 1 year of the initial visit, and 77 patients received surgical intervention 1 year after the initial visit following the failure of conservative treatment. We were able to monitor 34 patients for a long period of time. Among 34 patients, 7 failed to maintain follow-up. Therefore, 27 patients (3 men and 24 women; mean age at the first visit 64.3 years, range 50–71 years) were enrolled in this study. All patients were treated with conservative therapy including nonsteroidal antiinflammatory drugs and physical therapy when they had symptoms during the follow-up period.

Radiographic Outcome Measurements

All patients underwent standing lumbar anteroposterior and lateral radiography at least once a year between 1987 and 2000. The mean follow-up duration was 10 ± 3.9 years (range 7–18 years). The radiological parameters measured included the following: 1) scoliosis angle as determined by the Cobb method; 2) lumbar lordosis angle; 3) the disc index (the ratio of disc height on the decreased side to the disc height on the opposite side); 4) lateral osteophyte difference (that is, the difference between the length of the lateral osteophytes on each side. [The lengths of the lateral osteophytes are the sums of the perpendicular distances measured from the reference line to the lateral ends of the osteophytes on the upper and lower endplates.]); 5) the distance from the center sacral line to the most lateral concave margin of the apical vertebra; 6) the relationship between the intercrest line and the L-5 vertebra; 7) lateral listhesis (the distances from the reference line of the laterally translated vertebral body to that of the lower vertebra); 8) degenerative listhesis anteriorly, posteriorly, or both; and 9) each segmental angle of L2–3 and L3–4 (asymmetrical disc degeneration was most prevalent at the L2–3 and L3–4 disc space above and below the L-3 apical vertebra) (Figs. 1 and 2). Each of the angles was added after measuring the L2–3 and L3–4 segmental angles; this was because there was difficulty deciding which disc level (L2–3 or L3–4) would be used for determining the extent of DLS in some patients with simultaneous asymmetrical discs at both disc levels. In addition, if the scoliosis angle was measured from the lower endplate of L-2 to the upper endplate of L-4 for the analysis of the disc index or segmental scoliosis angle, the asymmetrical vertebral body in between these discs would be included. Therefore, each disc level was measured and added to eliminate the effects of the asymmetrical vertebral body for accuracy of the measurement for each segmental scoliosis angle and the disc index. All radiographic measurements were determined by one of the coauthors. The presence of osteoporosis based on DEXA (dual energy x-ray absorptiometry) was measured in all patients at the first visit; patients were considered to have osteoporosis of the spine if their T-score for bone mineral density of the spine was greater than −2.5.

Clinical Outcome Measurements

Pain and disability were scored using the VAS for low-back pain and the ODI questionnaire, with measurements obtained yearly or every 6 months. All patients were asked to complete the VAS and ODI outcome questionnaires during clinic visits.

Statistical Analysis

SPSS 13.0 software (SPSS, Inc.) was used for the statistical analysis. The Mann-Whitney U-test was used to analyze the relationship between curve progression and direction of the scoliosis, sex, lateral listhesis, interest line, and osteoporosis. The Kruskal-Wallis test was used to analyze the relationship between curve progression and age, and the sum of the L2–3 and L3–4 segmental angles, divided into 3 groups as follows: less than 5° (Group I), between 5° and 10° (Group II), and greater than 10° (Group III). The Fisher exact test was used to analyze spondylolisthesis and curve progression. Correlation analyses were performed to determine the relationship between curve progression and the lordosis angle, the center of the sacral line, disc index, lateral osteophyte difference, segmental angle, and VAS and ODI scores with use of the Spearman correlation coefficient analysis. A p value < 0.05 was significant.

Results

There were 10 patients with 10° of progression or less, and 17 patients had more than 10° of progression with regard to their scoliosis angle (Table 1). The group with 10° of progression or less had a mean angle of scoliosis that was 14.2° ± 4.4° at the first visit and 21.8° ± 6.9° at the most recent visit. For the group with more than 10° of progression, the mean Cobb angle was 13.9° ± 5.8° at the first visit.
and 26.9° ± 9.3° at the most recent visit. However, there were no statistically significant differences in the initial Cobb angle between the 2 groups (p = 0.43). The scoliosis curves included 3 vertebrae in 4 patients, 4 vertebrae in 19, and 5 vertebrae in 4.

**Direction of Scoliosis**

Eighteen patients had left-side curves and 9 had right-side curves; their mean final scoliosis angles were 26° ± 6.9° and 24° ± 11.4°, respectively (p = 0.34) (Table 2).

**Patient Age**

When we divided the patients into 3 age groups (< 60, 60–70, and ≥ 70 years of age), the mean final scoliosis angles were 18.9° ± 7.0°, 27.4° ± 9.0°, and 24.4° ± 6.2°, respectively (p = 0.14) (Table 2).

**Patient Sex**

The mean final scoliosis angles in men and women were similar (24.2° ± 10.4° vs 25.5° ± 8.5°). Since there were fewer men (3 patients) than women (24 patients), it was not possible to determine statistical significance.

**Lateral Listhesis**

An initial lateral listhesis was observed in 14 patients (at L2–3 in 1, at L3–4 in 8, and at L4–5 in 5). The lateral

![Fig. 1. Diagram showing the measurement method. The segmental scoliosis angle, the distance from the center sacral line to the most lateral concave margin of the apical vertebra, and the relationship between the intercrest line and the L4–5 intervertebral disc space are illustrated.](image)

![Fig. 2. Diagrams. The disc index, lateral osteophyte, and lateral listhesis were measured using the tangential line through the most concave point of the lateral vertebral wall as a reference line. A: Lateral listhesis: the distances from the reference line of laterally translated vertebral body to that of the lower vertebra. B: Lateral osteophyte differences: the difference between the length of the lateral osteophytes on each side. The lengths of the lateral osteophytes are the sums of the perpendicular distances measured from the reference line to the lateral ends of the osteophytes on the upper and lower endplates. Lateral osteophyte difference = (a+b) – (c+d). C: Disc index: the ratio of disc height on the decreased side to the disc height on the opposite side. Disc index = (e+f)/(g+h).](image)
Risk of progression of degenerative lumbar scoliosis

**TABLE 1: Relationship between the sum of the segmental angles above and below the L-3 vertebra and scoliosis angle progression more than 10°**

<table>
<thead>
<tr>
<th>Initial SA</th>
<th>Progression of SA</th>
<th>≤10°</th>
<th>&gt;10°</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (SA &lt;5°)</td>
<td>4 (57.1)</td>
<td>3 (42.9)</td>
<td></td>
</tr>
<tr>
<td>Group II (SA 5°–10°)</td>
<td>4 (33.3)</td>
<td>8 (66.7)</td>
<td></td>
</tr>
<tr>
<td>Group III (SA &gt;10°)</td>
<td>2 (25.0)</td>
<td>6 (75.0)</td>
<td></td>
</tr>
</tbody>
</table>

* Values represent the number of patients with percentages in parentheses. Abbreviation: SA = the sum of the L2–3 and L3–4 segmental angles.

listhesis was at least 6 mm in 2 patients, whereas 25 had a lateral listhesis less than 6 mm or no listhesis; their final scoliosis angles were similar (31° ± 7.1° vs 24.9° ± 8.5°, p = 0.25) (Table 2). On the final plain radiographs, 19 patients had a lateral listhesis greater than 6 mm, whereas 8 had a lateral listhesis less than 6 mm; their final scoliosis angles differed significantly (30.4° ± 9.9° vs 23.2° ± 7.0°, p = 0.04).

**Degenerative Spondylolisthesis**

All patients showed degenerative spondylolisthesis anteriorly, laterally, or posteriorly on their last radiograph, compared with 12 patients for whom it was found on the initial radiograph. Among the 24 patients, 18 had anterior spondylolisthesis and 6 patients had combined anterior and posterior spondylolisthesis. All patients showed lateral listhesis on their last radiograph. There was no correlation between initial spondylolisthesis and curve progression (p = 0.12).

**Intercrest Line**

The intercrest line passed through the L-4 vertebral body in 8 patients, through the L4–5 intervertebral disc space in 9 patients, and through the L-5 vertebral body in 10 patients. The mean final scoliosis angle was 31.4° ± 7.9° in the 19 patients with intercrest lines passing through the L-5 vertebral body or L4–5 intervertebral disc space, compared with 21.8° ± 6.7° in the other 8 patients; this difference was statistically significant (p = 0.01) (Table 2).

**Lordosis Angle**

The lordosis angle between the L-1 upper endplate and the L-5 lower endplate was 29.1° ± 12.9° initially and 20.8° ± 14.7° at the last follow-up visit. Although the lumbar lordosis angle tended to decrease as the scoliosis angle increased, there was no statistical significance (p = 0.20).

**Osteoporosis**

Among the 27 patients, 14 had osteoporosis and 13 did not; their mean final scoliosis angles were similar (25.7° ± 10.4° vs 25.0° ± 6.5°, p = 0.98) (Table 2).

**Center of the Sacral Line**

On initial radiography, the mean distance from the center of the sacral line to the apical vertebra was 36.0

**TABLE 2: Results of the Mann-Whitney U-test according to parameters measured**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD (°)*</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>side of scoliosis</td>
<td>26.0 ± 6.9</td>
<td>0.34</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>24.0 ± 11.4</td>
<td>0.14†</td>
</tr>
<tr>
<td>lat listhesis (mm)</td>
<td>18.9 ± 7.01</td>
<td>0.01‡</td>
</tr>
<tr>
<td>intercrest line</td>
<td>31.0 ± 7.1</td>
<td>0.25</td>
</tr>
<tr>
<td>osteoporosis</td>
<td>25.7 ± 10.4</td>
<td>0.98</td>
</tr>
<tr>
<td>absent</td>
<td>25.0 ± 6.5</td>
<td></td>
</tr>
</tbody>
</table>

* Mean of last follow-up scoliosis angle.
† Result of Kruskal-Wallis H-test.
‡ Statistically significant.

± 9.7 mm. A significant positive correlation was detected between this distance and the final scoliosis angle (p = 0.6; p < 0.001).

**Disc Index and Lateral Osteophyte Difference**

The mean lateral osteophyte differences at L-3, L-4, and the apical vertebrae were 5.2 ± 5.2, 3.9 ± 3.2, and 5.7 ± 5.4 mm, respectively. Each of these differences and final follow-up scoliosis angle showed no statistically significant correlations (p = 0.2, p = 0.14; p = 0.12, p = 0.25; and p = 0.1, p = 0.25, respectively).

The mean disc indices at L-3, L-4, and the apical vertebrae were 0.65 ± 0.2, 0.75 ± 0.2, and 0.6 ± 0.2, respectively, on initial radiography. Each of these indices was significantly correlated to the final follow-up scoliosis angle (p = 0.7, p < 0.001; p = 0.6, p = 0.001; and p = 0.6, p < 0.001, respectively).

**Segmental Angle**

Initially, the mean segmental angles at L2–3 and L3–4 were 4.8° ± 2.1° and 3.7° ± 2.8°, respectively. There were no statistically significant correlations between any of these angles and the final follow-up scoliosis angle (p = 0.2, p = 0.67; and p = 0.1, p = 0.22, respectively). The mean of the sum of the L2–3 and L3–4 segmental angles (that is, SA23 + SA34) was 8.5° ± 4.7°. There was significant positive correlation between the sum of the L2–3 and L3–4 segmental angles and the final follow-up scoliosis angle (p = 0.6; p < 0.001).

When we divided the patients into 3 groups based on the sum of the L2–3 and L3–4 segmental angles, as the angle increased, a greater proportion of patients had progression of their scoliosis angle more than 10° (Table 1).
Visual Analog Scale and ODI Scores During the Follow-Up Period

The correlation coefficient values were low between the VAS score and the scoliosis angle ($r = 0.16$). However, a significant positive correlation was detected between ODI and the scoliosis angle ($r = 0.6, p < 0.001$). The initial VAS was $6.2 \pm 1.1$ in the group with $10^\circ$ of progression or less and $6.5 \pm 1.3$ in the group with more than $10^\circ$ of progression. The final VAS scores were $6.1 \pm 2.1$ in the group with $10^\circ$ of progression or less and $6.8 \pm 2.1$ in the group with more than $10^\circ$ of progression. There was no significance between the initial and the final VAS in the group with $10^\circ$ of progression or less ($p = 0.84$) or in the group with more than $10^\circ$ of progression ($p = 0.69$) (Fig. 3A). The initial ODI scores were $33.3 \pm 10.3$ in the group with $10^\circ$ of progression or less and $32.4 \pm 11.1$ in the group with more than $10^\circ$ of progression. The last ODI was $37.8 \pm 17.2$ in the group with $10^\circ$ of progression or less and $46.9 \pm 15.1$ in the group with more than $10^\circ$ of progression. There was no significance between the initial and the last ODI in the group with $10^\circ$ of progression or less ($p = 0.25$). However, the last ODI was significantly increased in the group with more than $10^\circ$ of progression ($p = 0.01$) (Fig. 3B).

Discussion

Robin et al.\textsuperscript{17} reported that among 160 patients with a straight spine who were observed for more than 7 years, 55 patients (34.4\%) developed DLS. Likewise, Kobayashi et al.\textsuperscript{10} found that 22 (36.7\%) of 60 patients developed de novo scoliosis of more than $10^\circ$ in their 12-year prospective cohort study; however, other authors reported a higher frequency (58\%) of DLS in patients with osteoporosis.\textsuperscript{8} Although osteoporosis, degenerative facet joint disease, degenerative disc disease, compression fractures, lateral listhesis, and menopause are factors associated with the development and progression of DLS, the exact causes of the condition remain unknown. Suspected risk factors associated with scoliosis curve progression have been suggested (Table 3).\textsuperscript{1,5,10,11,13,15,21,22} Risser\textsuperscript{46} and Wiltse\textsuperscript{23} suggested that degeneration and thinning of the intervertebral discs at points of great stress may increase scoliosis curvature in the elderly. Degenerative lumbar scoliosis may be related to metabolic bone diseases such as osteoporosis and osteomalacia\textsuperscript{22} or to a low bone mineral density.\textsuperscript{20} However, others have reported that the presence or progression of scoliosis is not directly related to osteoporosis.\textsuperscript{17} We observed no relationship between osteoporosis and the progression of DLS; however, patients with osteoporotic compression fractures of the lumbar spine were excluded from our study. The mean curve progression over 5 years was reported to average $3^\circ$ per year in 73\% of patients; moreover, Grade 3 apical rotation, a Cobb angle $\geq 30^\circ$, lateral vertebral translation 6 mm or greater, and prominence of L-5 in relation to the intercrest line were important factors for predicting curve progression.\textsuperscript{15} Disc spaces above and below L-4 may also be related to DLS.

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vanderpool et al., 1969</td>
<td>osteoporosis</td>
</tr>
<tr>
<td>Benner &amp; Ehni, 1979</td>
<td>asymmetric intervertebral osteochondrosis</td>
</tr>
<tr>
<td>Pritchett &amp; Bortel, 1993</td>
<td>curve w/ Cobb angles $&gt;30^\circ$; degree of apical rotation; relationship of L-5 to intercrest line</td>
</tr>
<tr>
<td>Murata et al., 2002</td>
<td>disc degeneration</td>
</tr>
<tr>
<td>Korovessis et al., 1994</td>
<td>lat listhesis; Harrington factor; disc index</td>
</tr>
<tr>
<td>Daffner &amp; Vaccaro, 2003</td>
<td>degeneration of intervertebral disc; osteoporosis</td>
</tr>
<tr>
<td>Tribus, 2003</td>
<td>degenerative disc disease; facet incompetence; hypertrophy of ligamenta flava</td>
</tr>
<tr>
<td>Kobayashi et al., 2006 present study</td>
<td>disc index; lat osteophyte difference</td>
</tr>
</tbody>
</table>

* $p = 0.01$. Y = year.
Risk of progression of degenerative lumbar scoliosis

The disc between L-3 and L-4 forms the apex of the lumbar lordosis curve, and the L-4 vertebra has the smallest transverse process and relatively long iliotsacral ligaments, which contribute comparatively less ligamentous support. In addition, the L-5 vertebra has broad pedicles and large transverse and accessory processes, which reflect its strong muscular and ligamentous support. In our study, patients with a sum of initial L2–3 and L3–4 segmental scoliosis angle greater than 10° showed a greater frequency of progression than patients with a scoliosis angle less than or equal to 10°, but the difference was not statistically significant. Initial lateral listhesis was not related to progression of the scoliosis angle, but patients with intercrest lines passing through the L-5 vertebra or the L4–5 disc space had a larger final scoliosis angle than patients with intercrest lines passing through the L-4 vertebra. The relationship between the L-5 vertebra and the intercrest line may affect the progression of scoliosis. Clearly, the scoliosis is likely to progress in the case of a deep-seated L-5.

Marty-Poumarat et al.12 reported that after menopause, deterioration was noted in patients with de novo scoliosis, caused by rotatory subluxation as the initial event. By contrast, in this study there was no relationship between rotational subluxation and progression of scoliosis observed, even though all of our female patients were postmenopausal at their first visit.

A study of the degree of change in scoliosis and lordosis wedging in 47 patients with scoliosis angle increases 10° or greater suggested that DLS was triggered by any disc degeneration at the lumbar level.13 Loss of segmental lordosis usually occurs at the same disc level as segmental wedging, and DLS could be followed by assessment of decreased lumbar lordosis.13,15 The maximum lumbar lordosis has been reported to negatively correlate with the Cobb angle of a major curve.9 By contrast, the results of this study showed no statistically significant correlation between the lordosis and scoliosis angles, although the lordosis angle was likely to decrease as the scoliosis angle increased.

When asymmetrical degenerative changes were evaluated by measuring the disc index and lateral osteophyte difference, the results suggested that development of de novo scoliosis was associated with the baseline radiological finding of asymmetrical disc degeneration.10 In addition, a greater than 20% asymmetrical decrease proportional to the opposite side in unilateral disc height or a longer than 5-mm osteophyte on one side led to an increased incidence of de novo scoliosis, which may affect the long-term results of spinal surgery.10 By contrast, we did not observe any relationship between the initial lateral osteophyte difference and final follow-up scoliosis angle. Degenerative discs have been classified into 3 distinct stages, with the final stage defined as stabilization.9 At this stage, the progressive development of osteophytes around the disc and facet joints leads to segmental stiffness, which stabilizes the unstable spine. Thus, osteophyte size is not related to progression, or, if related, it is not a cause of progression but is a compensatory reaction that stabilizes the instability caused by curve progression.

The initial disc indices at L-3 and the apical vertebra, but not at L-4, were related to final scoliosis angle. We also found that the sum of the L2–3 and L3–4 segmental angles was correlated with the final follow-up scoliosis angle. Angulation of the disc space induced by asymmetrical degeneration of the disc is related to DLS progression, with L2–3 and L3–4 degeneration especially affecting the progression of DLS (Table 2, Figs. 4 and 5). If the sum of the L2–3 and L3–4 segmental angles was more than 5° in this study, the progression of DLS was highly likely, with occurrence in 70% (14 of 20 patients) (Table 2). Therefore, the measurement of asymmetrical disc degeneration could be used for the radiological assessment of the progression of DLS.

Patients who showed vertebral body translation on radiographs had indications of unstable DLS. This finding suggested that the presence of lateral osteophytes was not closely correlated with the progression of DLS, but rather reflected a compensatory reaction to stabilize the unstable spinal segment. Degenerative lateral scoliosis may be regarded as an asymmetrical collapse of the disc and asymmetrical incompetence and hypertrophy of the facet joints, leading to a lateral and rotational deformity.21 The result is a deformity combined with varying degrees of central lateral recess and foraminal stenosis. The key factor for causing curves is the asymmetrical degeneration of the intervertebral disc space.2 Low pressures in the disc space would then induce angulation and high pressure in the space, thus inducing osteophyte formation.

We observed no relationship between the initial lateral listhesis and the final follow-up scoliosis angle. By contrast, the final lateral listhesis was related to the final follow-up scoliosis angle, and the initial disc index and segmental angle were linearly correlated with the final follow-up scoliosis angle. These findings suggest that asymmetrical degeneration of the disc space and lateral listhesis did not occur in parallel. It is likely that asymmetrical degeneration of the disc space occurs first followed by lateral listhesis, leading to curve progression. Thus, measuring the asymmetrical degeneration of the disc space at L2–3 and L3–4 may be useful for estimating the progression of DLS.

Everett and Patel6 suggested conservative treatment when there are no significant stenotic, radicular, and/or back pain symptoms, including curves less than 30° with less than 2 mm of subluxation with anterior osteophytes. Surgical treatment is offered when a correlation exists between clinical and specific radiographic findings, particularly, L-3 and L-4 endplate angulations, lumbar lordosis, thoracolumbar kyphosis, and lateral olisthesis.18 Silva and Lenke19 suggested treatment guidelines for the 6 distinct levels, to sort the patient’s symptoms and radiographs, of operative treatment for DLS. When surgical intervention is needed, this guideline would be very helpful in deciding the optimal surgical method. Regarding our study, however, further studies, in a larger consecutive series of patients, examining asymmetrical disc degeneration and its association with the development of DLS are needed to determine the optimal treatment for patients.

We measured the VAS and ODI scores to determine the relationships between pain and the limitations on daily living associated with the scoliosis angle. We ob-
served no direct relationship between the scoliosis angle and back pain. By contrast, we observed a correlation between the scoliosis angle and limitations on daily living. These findings suggest that patients with DLS experience limitations of daily activities because of the symptoms associated with stenosis caused by radiculopathy and/or degenerative spinal deformities. However, there are some limitations in the evaluation of pain and functional ability in this study; this is because conservative treatments with nonsteroidal antiinflammatory drugs, physical therapy, and referrals to the pain clinic were provided when patients complained of low-back pain and radicular symptoms.

The limitations of this study include the following. This study was not part of a larger consecutive series of patients. Most patients complained of severe back pain, deformity, and symptoms associated with stenosis at the first visit. The patients enrolled in this study were those who refused surgery. During the follow-up period, therefore, some patients who had severe back pain with progression of curves and radiculopathy underwent surgery, and some patients were treated with conservative therapy. Therefore, the VAS and ODI scores might not be reliable in this study because when the patients presented to our clinic, some of the symptoms were improved by conservative treatments. However, we measured VAS and ODI every year or biannually at follow-up visits whether the patient had symptoms. The results showed that functional activity was correlated with the scoliosis angle.

Conclusions

The progression of DLS appears to be affected by the relationship between the intercrest line and the L-5 vertebra. Asymmetrical degeneration of the disc above and below L-3 or the apical vertebra may be important factors that can be used to predict scoliosis curve progression. This long-term follow-up study may provide information that is useful for understanding the natural history of DLS.

Fig. 4. Radiographs demonstrating the progression of scoliosis and loss of segmental lordosis with the passing of time. A: Images in a patient in whom the sum of the L2–3 and L3–4 segmental angles was 5°, and L4–5 intervertebral disc space was located below the intercrest line with a scoliosis angle of 13°. B and C: Images showing progression of the scoliosis angle from 17° to 21°. D: Images showing a severe case of sclerosis and collapse of the intervertebral disc space of the lumbar spine with a scoliosis angle of 24°.
Risk of progression of degenerative lumbar scoliosis

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: all authors. Acquisition of data: all authors. Analysis and interpretation of data: all authors. Statistical analysis: all authors. Administrative/technical/material support: all authors. Study supervision: all authors.

**References**


*Fig. 5.* Radiographs. A: Images obtained in a patient whose L2–3 and L3–4 segmental angles were summed to 7.8°. In addition, the L4–5 intervertebral disc space was located below the intercrest line with a scoliosis angle of 10°. B and C: At later time points, these angles were 16° and 33°, respectively. D: The scoliosis angle with loss of lumbar lordosis progressed to 38.6°.

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