Risk of Progression in De Novo Low-Magnitude Degenerative Lumbar Curves: Natural History and Literature Review

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Abstract

Natural history studies have focused on risk for progression in lumbar curves of more than 30°, while smaller curves have little data for guiding treatment. We studied curve progression in de novo degenerative scoliotic curves of no more than 30°.

Radiographs of 24 patients (17 women, 7 men; mean age, 68.2 years) followed for up to 14.3 years (mean, 4.85 years) were reviewed. Risk factors studied for curve progression included lumbar lordosis, lateral listhesis of more than 5 mm, sex, age, convexity direction, and position of intercrestal line.

Curves averaged 14° at presentation and 22° at latest follow-up and progressed a mean of 2° (SD, 1°) per year. Mean progression was 2.5° per year for patients older than 69 years and 1.5° per year for younger patients. Levoscoliosis progressed 3° per year and dextroscoliosis 1° per year ($P<.05$). Forty-six percent of patients had lateral listhesis of more than 5 mm at L3 and L4.

Curve progression was not linear and might occur rapidly, particularly in women older than 69 with lateral listhesis of more than 5 mm and levoscoliosis. Small curves can progress and therefore should be individualized in the context of other risk factors.

Degenerative scoliosis tends to present in the lumbar spine of patients older than 50 years with low back pain, neurogenic claudication, and sciatica.[1-32] These symptoms are often associated with advanced disc degeneration, asymmetric facet arthrosis, osteoporosis, compression fractures, hypertrophy of ligamentum flavum, segmental instability, and foraminal and central stenosis.[6,10,11,13,17,24-26,28,31,32] Nonsurgical management is usually first-line treatment, but, in patients with disabling pain and progressive deformity, surgery might be needed to relieve symptoms.[1,3,8,9,12,14,15,19-23,27,30] However, the decision to perform surgery is often complicated by advanced age and variable life expectancy, osteoporosis, and multiple medical comorbidities that commonly characterize this patient population. Complications after surgery range from 20% to 40% in most series.[1,3,8,9,12,14,15,19,21,23,27,30]

There is lack of consensus for surgical management of lumbar degenerative scoliosis because of the heterogeneous nature of the disorder and the afflicted patient population, the multiple surgical options, and the lack of rigorous evidence-based outcomes. Fusion is usually recommended in symptomatic patients with significant lumbar curves considered at risk for progression. Natural history studies have therefore attempted to determine objective criteria for risk and rate of curve progression as a variable that might help the surgeon and the patient make a more informed decision regarding surgery.[13,24-26,28,31,32]

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Radiographic follow-up studies have identified certain risk factors for pain and curve progression. These factors include Cobb angles of more than 30°, loss of lumbar lordosis, apical rotation higher than Nash-Moe grade II, lateral listhesis of more than 5 mm, and intercrestal line through or below the L4–L5 disc space.[13,24-26,28,31,32]

Natural history studies have therefore focused on risk for progression in curves of more than 30°, and lower magnitude curves, particularly those less than the 15° criteria of the Scoliosis Research Society (SRS), have little data for guiding treatment.

To generate additional data on risk for curve progression, we retrospectively assessed the rate of progression in a patient population with de novo low-magnitude but measurable degenerative coronal curvature of the lumbar spine and documented potential associated risk factors. These

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Table. Degenerative Scoliosis Measurements

Patient	Age (y)	Sex	Presenting Curve (°)	Follow-Up Curve (°)	Total Change (°)	Change/ Year (°)	Follow-Up (y)	Follow-Up Lordosis (°)	Apex		Intercrestal Line	Short-Segment Curve (°)	Lateral Listhesis
1	66	F	30	36	5	0.8	3.8	34	Right	L2	L4	—	Right (L3)
2	64	F	7	7	0	0	2	30	Left	L4	L4–L5	—	—
3	69	F	6	8	2	0.2	11	27	Right	L3	L4–L5	—	Right (L4)
4	50	F	18	30	12	2	6	26	Left	L2	L4–L5	—	—
5	74	F	20	46	26	3.4	7.8	22	Left	L3	L4	—	—
6	70	F	8	15	7	2.8	2.5	32	Left	L3	L4–L5	12	—
7	81	F	12	21	10	9	1	21	Left	L4	L4	—	Left (L3)
8	71	M	7	16	9	1.4	6.3	19	Left	L3	L4–L5	—	—
9	69	M	14	21	7	1.2	5.8	32	Left	L3	L4	—	—
10	74	F	25	29	4	0.7	6	31	Right	L1	L4	21	Left (L4)
11	51	F	9	20	11	1.8	6.1	27	Left	L2	L5	—	Left (L3)
12	76	F	18	23	5	4.3	1.2	30	Left	L3	L4–L5	—	Left (L3)
13	66	F	15	37	22	6.6	3.3	30	Left	L2	L4	—	Left (L3)
14	63	F	3	10	7	1.7	4.1	28	Right	L3	L4–L5	—	Right (L3)
15	77	M	10	15	5	3.2	1.6	30	Right	L3	L4	—	Right (L3)
16	50	M	21	21	0	0	2.3	35	Left	L3	L4–L5	—	—
17	72	F	9	13	4	1.1	3.8	24	Right	L4	L4	10	—
18	73	F	4	4	0	0	2	6	Right	L2	L4	—	—
19	68	F	12	20	8	0.8	10.7	7	Right	L1	L4–L5	15	—
20	65	M	13	25	12	0.8	14.3	27	Right	L3	L4–L5	—	Right (L4)
21	76	M	25	32	7	1.8	4	27	Right	L3	L4–L5	—	Right (L4)
22	73	F	22	22	0	0	2.6	13	Right	L3	L4	—	Right (L3)
23	66	F	20	27	7	1.5	4.8	39	Right	L1	L4	20	Left (L4)
24	73	M	11	20	9	2.5	3.7	11	Left	L3	L4–L5	—	—

risk factors included curve magnitude at presentation, lumbar lordosis, lateral listhesis of more than 5 mm, sex, age, convexity direction, and position of intercrestal line. We hypothesized that de novo low-magnitude degenerative lumbar curves would also be at risk for progression.

Materials and Methods

We retrospectively reviewed the medical records of 24 patients (17 women, 7 men) found on presentation at a spine clinic to have de novo degenerative scoliosis on routine standing plain anteroposterior radiographs. These patients were followed nonoperatively for a minimum of 12 months. Mean age was 68.2 years (range, 50-81 years) for all patients, 68 years (range, 50-81 years) for the women, and 68.7 years (range, 50-77 years) for the men. Treatment included formal physical therapy programs and use of anti-inflammatory medications. Patients with radicular symptoms were recommended for epidural injection. No patient was treated with a brace.

Patients with lumbar curves surgically treated within 12 months after evaluation were excluded. Inclusion criteria were measurable degenerative lumbar curves nonoperatively treated and availability for follow-up radiographs since first presentation for treatment. Patients presented with low back and/or leg pain and underwent routine radiographic evaluation. All radiography was performed at the same facility. To not be limited by prior reports on the natural history of degenerative scoliosis and to include all measurable curves, we set no minimal criteria for curves; as a result, the angles in this study ranged from 3° to 30°. We recorded patient demographics, curve magnitude at presentation and follow-up, lumbar lordosis measured from T12 to sacrum, lateral listhesis of more than 5 mm, position of intercrestal line, location of apical vertebra and direction of convexity, and presence of a short-segment or compensatory curve below the major curve. These data points were chosen because they could be objectively measured, in contrast to others, such as degree of apical rotation, which is more subjective. All patients had varying degrees of apical rotation. Measurements were done on the first and most recent standing plain radiographs taken before any surgical intervention. The Cobb technique was used to measure curve magnitude. Dr. Bohlman and Dr. Bouchard recorded Cobb angles for each patient.[3] Dr. Chin performed all chart reviews and remeasured the Cobb angles for each patient. Interobserver variability between at least 2 different measurements per radiograph were determined. To assess interobserver reliability, we used a 15-patient overlap to determine the correlation coefficient between the investigators and found $r = 0.95$. When there is intraobserver variation with one type of measurement, no consistent bias is expected, rendering the correlation coefficient an effective measurement.[33]

Statistical analysis was performed with Microsoft Excel to assess for significant differences. A 2-tailed, 2-sample, unequal-variance Student t test was used. $P < .05$ was considered significant. Because of the limited sample size, measurements are reported as means and SDs.

Results

The data are summarized in the Table. We compared risk for progression in patients who had curves of 15° or more (SRS criterion for scoliosis) and patients who had curves of less than 15°. Curves averaged 14.2° (SD, 7.4°; range, 3°-30°) at presentation and 21.6° (SD, 10°; range, 4°-46°) at latest follow-up. Mean progression was 7.3° (SD, 6.3°; range, 0°-26°) over 1.2 to 14.3 years (mean, 4.85 years). Curves of 15° or more progressed 2.1° (SD, 1.3°) per year compared with 1.9° (SD, 1.2°) for curves of less than 15°. However, progression was unpredictable. We were surprised to find that a 3° curve

progressed to 10° after 4.1 years and that a 20° curve progressed only 7° over 4.8 years. Overall mean progression was 2° (SD, 0.9°; range, 0°-9°) per year. Four curves, measuring 4°, 7°, 21°, and 22° at presentation, did not progress over 2, 2, 2.3, and 2.6 years, respectively.

We assessed risk for curve progression by age. Mean progression was 2.2° (SD, 1.2°; range, 0°-9°) per year for women compared with 1.6° (SD, 1.7°; range, 0°-3.2°) for men. The most progression, 26°, occurred over 7.75 years in a 74-year-old woman who had a 20° curve at presentation. The most significant incremental progression was 9° over 1 year in an 81-year-old woman with a 12° curve at presentation. One curve, in a 66-year-old woman, was 30° at presentation and 36° about 4 years later.

"Degenerative lumbar curves tend to be left-convexed and centered around L3 or L4 and involve a mean of 4 segments.[13,24,26]"

In trying to define the age above which curve progression was clearly more likely, we found mean progression to be 2.5° (SD, 1.4°; range, 0°-9°) per year for patients older than 69 years compared with 1.5° (SD, 1.0°; range, 0°-6.6°) for younger patients; 2.7° (SD, 3°; range, 0°-9°) for women older than 69 compared with 1.7° (SD, 2°; range, 0°-6.6°) for younger women; and 2.2° (SD, 0.7°; range, 1.4°-3.2°) for men older than 69 compared with 0.7° (SD, 0.5°; range, 0°-1.2°) for younger men.

Mean lordosis was 25.3° (SD, 8.7°; range, 6°-39°) at latest follow-up. Mean lordosis in curves of less than 20° was 24.5°, similar to the 25.8° in curves of 20° or more.

We also assessed the risk for progression in patients with lateral listhesis. Thirteen curves (54%) had lateral listhesis of more than 5 mm at L4 and L3. Of these curves, 5 were at L4, and 8 were at L3. The curves with lateral listhesis of more than 5 mm increased a mean of 2.5° (SD, 1.5°; range, 0°-9°) per year, whereas the other curves increased a mean of 1.4° (SD, 0.7°; range, 0°-3.4°) per year.

Location of intercrestal line was assessed as a risk factor. This line went through the L4 body in 11 cases, through the L4–L5 disc space in 12 cases, and through the L5 body in 1 case. Of the 4 patients with no progression, 2 had intercrestal lines at the L4 body and the other 2 at the L4–L5 disc space. Mean progression was 2.5° (SD, 1.7°) per year for the patients with the line at the L4 body and 1.5° (SD, 0.7°) for those with the line at or below the L4–L5 disc space.

Direction of apex of curve was evaluated as a risk factor. Of the 24 curves, 12 were convexed to the left and 12 to the right. Mean annual progression was 2.9° (SD, 1.5°) for the left-convexed curves and 1° (SD, 0.5°) for the right-con-

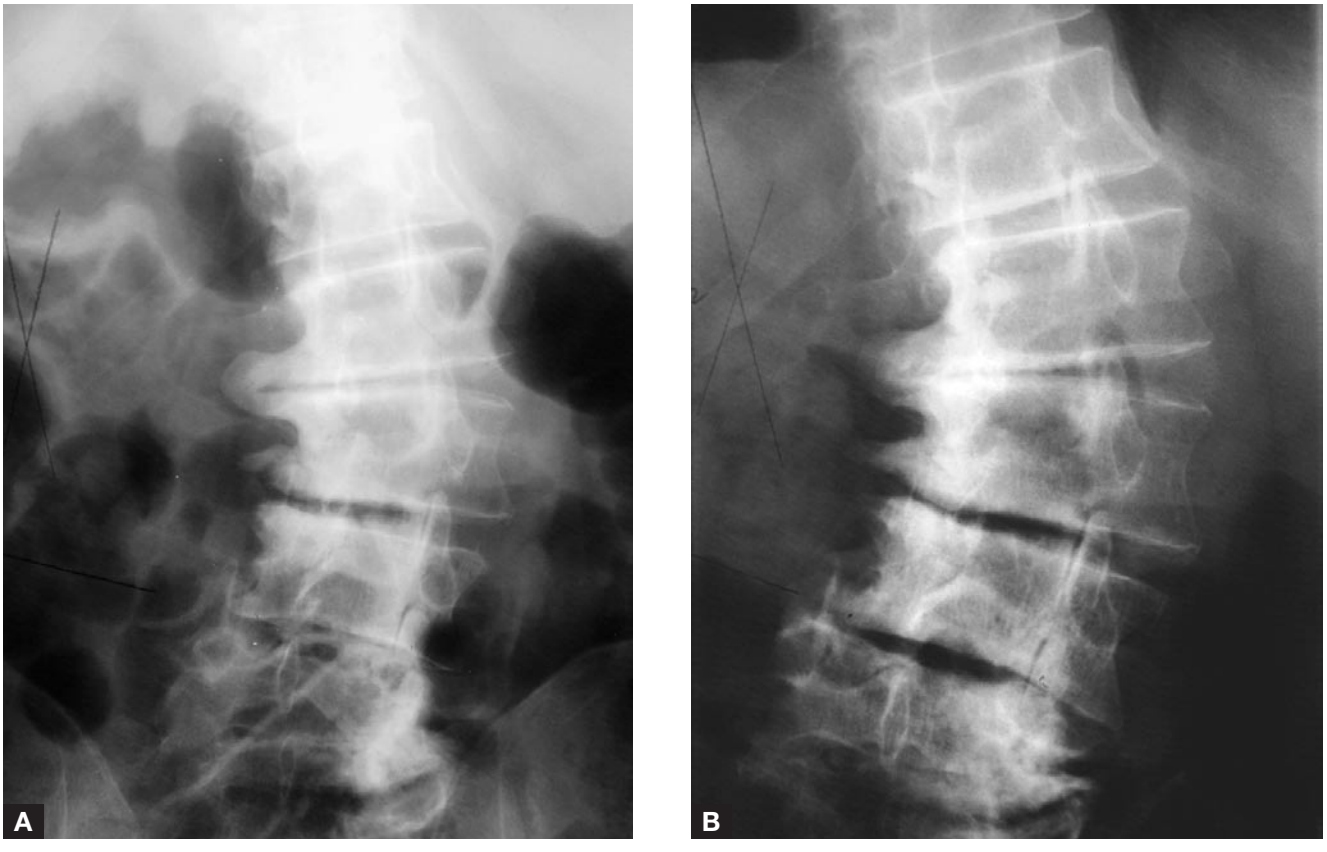

Figure. Patient is a man in his mid-70s. (A) Anteroposterior plain radiograph shows 25° degenerative lumbar curve on presentation. (B) Four-year follow-up anteroposterior radiograph shows increased curvature (32°).

vexed curves ($P<.05$). However, of the 4 curves that did not progress, 2 were left-convexed, and 2 were right-convexed. The apical vertebra was L3 in 13 cases, L4 in 3 cases, L2 in 5 cases, and L1 in 3 cases. The apical vertebra was at L3 in 2 of the 4 cases that did not progress and at L4 and L2 in the other 2 cases.

There were 3 short-segment curves, all between L4 and the sacrum and all convexed to the left opposite the major curve. The apex of each short-segment curve was at the L4–L5 disc space. The intercrestal line was at the L4 body in 2 cases and at the L4–L5 disc space in the third case. These curves, which were not seen at presentation but developed over 6, 10.67, and 4.75 years (mean, 7.14 years), measured 21°, 15°, and 20°, respectively, at latest follow-up. The 15° curve was the only one not associated with lateral listhesis of L4 of more than 5 mm.

"...our data suggest that any lumbar scoliotic curve that is undergoing radiographic degeneration can progress and, therefore, that patients who present with symptomatic curves should be followed."

Six patients (25%) failed nonoperative treatment and underwent lumbar decompression and uninstrumented fusion a mean of 5 years (range, 1-11 years) after presentation. Indications for surgery included disabling low back and leg pain, claudication, and curve progression. Preoperative curve progression was more rapid for this group (mean, 2.9° per year; SD, 3.3°; range, 0°-9°) than for the group treated only nonoperatively (mean, 1.2° per year; SD, 1.7°; range, 0°-6.6°). No other factors (eg, compression fractures) were identified as possible reasons for faster progression in the surgical group.

Discussion

Patients with degenerative lumbar scoliosis present with a wide range of symptoms, comorbidities, and ages.[1-32] When curves are more than 30°, surgeons are likely to be more concerned with risk for progression and are more inclined to follow these cases more closely.[13,24-26,28,31,32] In contrast, when curves are less than 30°, surgeons are likely to treat them nonoperatively and schedule less frequent follow-ups. However, there is still a lack of consensus regarding risk factors for progression, and there is sparse natural history data to guide treatment of de novo low-magnitude degenerative curves.

The impact of age and sex on curve progression has not been clearly defined, but there is a tendency for progression of larger curves in elderly patients.[25,26,31,32] This observation might be attributed to the likelihood of osteoporosis with advanced age and females' increased tendency to develop osteoporosis. Osteoporosis has not been shown to cause degenerative scoliosis,[10-13,22,28,30] but Vanderpool and colleagues[31] reported a 36% incidence of degenerative scoliosis in patients with osteoporosis and a 38% incidence in patients with osteomalacia—an approximately 6-fold higher incidence than that found in age- and sex-matched controls. According to Vanderpool and colleagues,[31] the high incidence of scoliosis in patients older than 50 years with osteoporosis implicated osteoporosis as the cause of deformity. In the present study, there was also a tendency for a higher rate of curve progression in patients older than 69 independent of sex, but progression tended to be faster in women. In addition, the most significant incremental curve progression was 9° over 1 year in an 81-year-old woman with a 12° curve at presentation. Her rapid progression was unusual, but we could not identify a specific reason for it (eg, compression fracture) other than presence of the risk factors evaluated in this study. Her most glaring risk factors were age, her sex, and presence of osteopenic bone. However, Thevenon and colleagues[28] demonstrated only a weak correlation between development of lumbar scoliosis and decreased bone mineral density of the femoral neck in a group of 56 patients older than 60.

Degenerative lumbar curves tend to be left-convexed and centered around L3 or L4 and involve a mean of 4 segments.[13,24,26] It is unclear if convexity direction has prognostic value for progression, but in the present study there were equal numbers of left- and right-convexed curves, and the progression rate for the left-convexed curves was significantly ($P<.4$) higher. The L3 vertebra was the most common apical vertebra.

In multiple studies, lateral listhesis of more than 5 mm has been found to be prognostic for curve progression.[13,24,26] The L3 and L4 vertebrae were most likely to have lateral listhesis. Grubb and Lipscomb[13] noted an 89% incidence of L3 or L4 lateral listhesis in 55 patients, and Pritchett and Bortel[24] noted a 39% incidence, also at L3 or L4. Curve progression was noted in all cases. These findings are similar to ours—that 54% of patients had lateral listhesis at L3 or L4 and that their progression rate tended to be approximately 2 times that of the patients without lateral listhesis.

Our data demonstrated that progression is not limited to curves of more than 30°. Robin and colleagues[25] conducted a follow-up study of 554 patients (age range, 50-84 years) to determine presence, appearance, and progression of scoliosis in the elderly and the relationship of scoliosis to osteoporosis and back pain. These 554 patients were a subset of 3600 patients who had been examined 7 to 13 years earlier. Seventy percent of patients had scoliosis; 30% had curves of

10° or more. Fifty-five (10%) of the 554 patients developed de novo scoliosis during follow-up. No curve was more than 20°. Forty-six percent of patients had curve progression. Mean progression was 7° (range, 3°-18°) for curves of 10° or more. Robin and colleagues[25] observed development of de novo curves and progression in low-magnitude curves, but they found no relationship between progression of scoliosis and osteoporosis, between scoliosis and back pain, or between scoliosis and degenerative changes.

Curve magnitude at presentation might affect progression rate, particularly for curves of more than 30°. In the 200-patient study conducted by Pritchett and Bortel,[24] curves ranged from 14° to 60° (mean, 24°), and 43 patients had curves of more than 35°. Forty-one patients had follow-up radiographs over 10 years, and 73% of curves progressed a mean of 3° per year. All curves of 30° or more showed progression. In the present study, smaller curves had a lower progression rate. Twenty of 24 curves progressed at a combined rate of 2° (SD, 0.9°) per year, and curves of 15° or more showed an increased trend for progression. However, progression was unpredictable. We noted it in curves as small as 3°, and yet some larger curves did not progress. Although a curve of 3° might not even be considered a true curve in a symptomatic patient, all curves start out as a low-magnitude curve, and a 3° curve today might be a 31° curve in a few years.

Location of the intercrestal line between or below the L4–L5 disc space has been considered a risk factor for progression.[24,26] Pritchett and Bortel[24] noted that, of the 41 patients followed in their study for more than 10 years, all those who did not have curve progression had an intercrestal line that passed through the L4 body. Sapkas and colleagues[26] reported similar results in a study of 162 women (mean age, 65 years) followed for 8 years (range, 5-30 years). In the present study, curves progressed in patients with an intercrestal line through the L4 body, and there was a trend for a higher rate of progression in these patients compared with patients whose intercrestal line was below L4. The reason for this is unclear but speaks to the multifactorial etiology of curve progression. In addition, the intercrestal line probably should not be the sole determinant of progression, and its location at L4 is not protective against progression.

Patients with degenerative lumbar scoliosis tend to have lumbar lordosis of less than 30°, but this might be an observation rather than a cause. Grubb and Lipscomb[13] found that 17 (31%) of 55 patients with adult scoliosis had lumbar lordosis of 30° or less. Pritchett and Bortel[24] noted a mean lordosis of 18°. In the present study, mean lumbar lordosis was 25° (SD, 3.5°). There was no significant difference in lordosis between larger and smaller curves. Loss of lordosis was more likely caused by advanced disc degeneration and loss of disc height and pain.

As the etiology of lumbar degenerative scoliosis is likely multifactorial, and it is difficult to adequately control for each variable, attempts to study the natural history are challenging. Therefore, the present study has some of the same limitations as other retrospective radiographic studies on the topic. We selected only symptomatic curves, so we cannot generalize to the risk for curve progression in patients with similar asymptomatic curves. Although we found few interobserver differences in measurements, in some instances the rate of curve progression was small and might have been within the error of the Cobb measurements. Curves of less than 10° do not meet the SRS definition for true scoliosis, but we wanted to include curves of all degrees visible on standing radiographs, as would be observed in a clinic treating spinal disorders. The data showed that even symptomatic curves of less than 10° should be followed regarding risk for progression. Additional data points (eg, apical rotation, sagittal and coronal imbalance) are arguably sequelae of degenerative scoliosis and not causes, but they could affect progression. We did not address these measurements in this study. Detailed clinical outcomes data were not included, as this study was focused on assessing the radiographic risk factors for curve progression. However, the majority of patients improved with nonoperative treatment, and only 25% of patients underwent surgery. Indications for surgery in this group were failure of nonoperative treatment for more than 6 months, disabling low back and leg pain, claudication, and curve progression. Finally, the relatively modest sample size might preclude us from drawing absolute conclusions from the data, but progression was noted in these smaller curves, and thus this study has value in raising awareness and, taken cumulatively with other studies, might allow more substantiated conclusions to be drawn.

Despite these limitations, our study results showed that symptomatic patients with degenerative lumbar scoliosis are at increased risk for curve progression if they are female and older than 69 and have a left-convexed curve, L3 or L4 lateral listhesis of more than 5 mm, and an intercrestal line at or below the L4 body. Although we cannot propose an absolute curve magnitude that would reliably predict which curves will progress, our data suggest that any lumbar scoliotic curve that is undergoing radiographic degeneration can progress and, therefore, that patients who present with symptomatic curves should be followed (Figure). We recommend close clinical and radiographic follow-up of all patients with symptomatic degenerative lumbar scoliosis with the consideration that curve progression is unpredictable and might not be linear. These data should be considered when managing and counseling patients with degenerative scoliosis.

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The authors report no actual or potential conflict of interest in relation to this article.

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References

1. Aebi M. Correction of degenerative scoliosis of the lumbar spine: a preliminary report. *Clin Orthop*. 1988;(232):80-86.
2. Benner B, Ehni G. Degenerative lumbar scoliosis. *Spine*. 1979;4(6):548-552.
3. Bouchard JA, Bohlman HH. Posterior decompression and in situ fusion for the treatment of degenerative scoliosis. *Orthop Traumatol*. 1995;4(1):54-59.
4. Briard JL, Jegou D, Cauchoix J. Adult lumbar scoliosis. *Spine*. 1979;4(6):526-532.
5. Epstein JA, Epstein BS, Jones MD. Symptomatic lumbar scoliosis with degenerative changes in the elderly. *Spine*. 1979;4(6):542-547.
6. Farfan HF, Huberdeau RM, Dubow HI. Lumbar intervertebral disc degeneration: the influence of geometrical features on the pattern of disc degeneration--a post mortem study. *J Bone Joint Surg Am*. 1972;54(3):492-510.
7. Fowles JV, Drummond DS, L'Ecuyer S, Roy L, Kassab MT. Untreated scoliosis in the adult. *Clin Orthop*. 1978;(134):212-217.
8. Frazier DD, Lipson SJ, Fossel AH, Katz JN. Associations between spinal deformity and outcomes after decompression for spinal stenosis. *Spine*. 1997;22(17):2025-2029.
9. Gelalis ID, Kang JD. Thoracic and lumbar fusions for degenerative disorders: rationale for selecting the appropriate fusion techniques. *Orthop Clin North Am*. 1998;29(4):829-842.
10. Grubb SA, Lipscomb HJ, Coonrad RW. Degenerative adult onset scoliosis. *Spine*. 1988;13(3):241-245.
11. Grubb SA, Lipscomb HJ, Guilford WB. The relative value of lumbar roentgenograms, metrizamide myelography, and discography in the assessment of patients with chronic low-back syndrome. *Spine*. 1987;12(3):282-286.
12. Grubb SA, Lipscomb HJ, Suh PB. Results of surgical treatment of painful adult scoliosis. *Spine*. 1994;19(14):1619-1627.
13. Grubb SA, Lipscomb HJ. Diagnostic findings in painful adult scoliosis. *Spine*. 1992;17(5):518-527.
14. Hanley EN Jr. Indications for fusion in the lumbar spine. *Bull Hosp Jt Dis*. 1996;55(3):154-157.
15. Hanley EN Jr. The indications for lumbar spinal fusion with and without instrumentation. *Spine*. 1995;20(24 suppl):143S-153S.
16. Jackson RP, Simmons EH, Stripinis D. Incidence and severity of back pain in adult idiopathic scoliosis. *Spine*. 1983;8(7):749-756.
17. Kirkaldy-Willis WH, Farfan HF. Instability of the lumbar spine. *Clin Orthop*. 1982;(165):110-123.
18. Kostuik JP, Bentivoglio J. The incidence of low-back pain in adult scoliosis. *Spine*. 1981;6(3):268-273.
19. Marchesi DG, Aebi M. Pedicle fixation devices in the treatment of adult lumbar scoliosis. *Spine*. 1992;17(8 suppl):S304-S309.
20. Nachemson A. Adult scoliosis and back pain. *Spine*. 1979;4(6):513-517.
21. Nasca RJ. Surgical management of lumbar spinal stenosis. *Spine*. 1987;12(8):809-816.
22. Ogilvie JW. Adult scoliosis: evaluation and nonsurgical treatment. *Instr Course Lect*. 1992;41:251-255.
23. Postacchini F. Surgical management of lumbar spinal stenosis. *Spine*. 1999;24(10):1043-1047.
24. Pritchett JW, Bortel DT. Degenerative symptomatic lumbar scoliosis. *Spine*. 1993;18(6):700-703.
25. Robin GC, Span Y, Steinberg R, Makin M, Menczel J. Scoliosis in the elderly: a follow-up study. *Spine*. 1982;7(4):355-359.
26. Sapkas G, Efstathiou P, Badekas AT, Antoniadis A, Kyratzoulis J, Meleteas E. Radiological parameters associated with the evolution of degenerative scoliosis. *Bull Hosp Jt Dis*. 1996;55(1):40-45.
27. Simmons ED Jr, Simmons EH. Spinal stenosis with scoliosis. *Spine*. 1992;17(6 suppl):S117-S120.
28. Thevenon A, Pollez B, Cantegrit F, Tison-Muchery F, Marchandise X, Duquesnoy B. Relationship between kyphosis, scoliosis, and osteoporosis in the elderly population. *Spine*. 1987;12(8):744-745.
29. Tribus CB. Degenerative lumbar scoliosis: evaluation and management. *J Am Acad Orthop Surg*. 2003;11(3):174-183.
30. Vaccaro AR, Ball ST. Indications for instrumentation in degenerative lumbar spinal disorders. *Orthopedics*. 2000;23(3):260-271.
31. Vanderpool DW, James JI, Wynne-Davies R. Scoliosis in the elderly. *J Bone Joint Surg Am*. 1969;51(3):446-455.
32. Winter RB, Lonstein JE, Denis F. Pain patterns in adult scoliosis. *Orthop Clin North Am*. 1988;19(2):339-345.
33. Bland JM, Altman DG. Applying the right statistics: analyses of measurement studies. *Ultrasound Obstet Gynecol*. 2003;22(1):85-93.