Reversed adjacent-segment degeneration after posterior lumbar interbody fusion

Case report

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One of the most important sequelae affecting long-term results is adjacent-segment degeneration (ASD) after posterior lumbar interbody fusion (PLIF). Although several reports have described the incidence rate, there have been no reports of repeated ASD. The purpose of this report was to describe 1 case of repeated ASD after PLIF. A 62-year-old woman with L-4 degenerative spondylolisthesis underwent PLIF at L4–5. At the second operation, L3–4 PLIF was performed for L-3 degenerative spondylolisthesis 6 years after the primary operation. At the third operation, L2–3 PLIF was performed for L-2 degenerative spondylolisthesis 1.5 years after the primary operation. Vertebral collapse of L-1 was detected 1 year after the third operation, and the collapse had progressed. At the fourth operation, 3 years after the third operation, vertebral column resection of L-1 and replacement of titanium mesh cages with pedicle screw fixation between T-4 and L-5 was performed. Although the patient’s symptoms resolved after each operation, the time between surgeries shortened. The sacral slope decreased gradually although each PLIF achieved local lordosis at the fused segment.

Key Words • posterior lumbar interbody fusion • adjacent-segment degeneration • postoperative complications • sacral slope • vertebral column resection

Abbreviations used in this paper: ASD = adjacent-segment degeneration; PLIF = posterior lumbar interbody fusion.
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Bone union of L4–5 was confirmed 4 months after the primary operation. Although L-3 slippage was observed starting at 3 years after the primary operation, there was no complaint for 5 years after the primary operation. However, her condition gradually deteriorated as the L-3 slippage progressed (Fig. 1B). A myelogram obtained just before the second operation showed an incomplete block at the L3–4 segment, with L-3 slippage.

At the second operation, 6 years after the primary operation, when the patient was 67 years old, PLIF was performed at L3–4 and her symptoms resolved. Although bone union of L3–4 was confirmed 6 months after the second operation, L-2 slippage was detected 9 months after the second operation, and her symptoms again deteriorated gradually (Fig. 1C).

At the third operation, 1.5 years after the second operation, when the patient was 68 years old, PLIF was performed at L2–3 and the pathology of the intervertebral disc tissue was inspected, but no infection or tumor was detected. Although her symptoms resolved after the third operation, and bone union was confirmed 9 months after the third operation, vertebral collapse of L-1 was detected 1 year after the third operation (Fig. 1D). At that time, she was postmenopausal but did not demonstrate generalized osteoarthritis and secondary osteoporosis (rheumatoid arthritis– or steroid-induced). The bone mineral density of the femoral neck and young adult mean were 0.847 g/cm² and 98%, respectively. No osteoporosis was detected.

Conservative treatment such as medication and orthotic therapy was selected because her symptoms were not severe and an operation for valvular heart disease was performed after the third operation. However, back pain and lower-limb pain deteriorated as the L-1 vertebral collapse progressed. Local alignment of L1–2 deteriorated from 2° lordosis just after the third operation to 37° kyphosis, and the thoracic spine below T-7 showed compensatory lordosis just before the fourth operation (Fig. 2A–C).

At the fourth operation, 3 years after the third operation, when the patient was 71 years old, vertebral column resection of L-1 and replacement of titanium mesh cages with pedicle screw fixation between T-4 and L-5 was performed (Fig. 2D and E). Pathological analysis of the intervertebral disc tissue was also performed, but no infection or tumor was detected. After the fourth operation, her symptoms resolved.

Lumbosacral parameters such as sacral slope, pelvic tilt, pelvic incidence, and lumbar lordosis angle (angle between the L-1 upper endplate and the S-1 upper endplate) were measured retrospectively. The pelvic incidence of this patient was 53°. Before the first, second, third, and fourth operations, and after the fourth operation, sacral slopes were 36°, 33°, 29°, 23°, and 34°, respectively; pelvic tilts were 15°, 21°, 26°, 30°, and 17°, respectively; and lumbar lordoses were 45°, 18°, 11°, −7°, and 33°, respectively. Sacral slope decreased gradually, while pelvic tilt increased. Furthermore, lumbar lordosis decreased with the loss of sacral slope (Fig. 3). The sagittal vertical axis lengths at those times were 35 mm, 110 mm, 111 mm, 120 mm, and 35 mm, respectively.

Discussion

Although several reports have described ASD,1,3,7,8 there have been no reports of repeated ASD. We previously reported on surgical complications of PLIF in 251 patients between 1996 and 2002 and reviewed previous reports.4 In that study, the reoperation rate for ASD was 4.4%, and there were no repeat cases; other studies reported reoperation rates ranging from 1.4% to 16.8%. In subsequent research, we performed PLIF in 913 patients between 1996 and 2012, and reoperations for ASD were conducted in 75 patients (8.2%). In these reoperations, 9 patients (1.0%) developed ASD twice, and 1 patient (0.1%) developed ASD three times. The ASD rate, as well as repeat cases, will change as the follow-up periods extend. Furthermore, we reported that there were 2 peaks of ASD occurrence, within 2 years and more than 5 years after the primary operation.7 In the current case, the first ASD occurred 6 years after the primary operation, but the second and third ASDs were detected within 1 year.

In terms of risk factors for ASD, Park et al. identified the following factors: older age, osteoporosis, female

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**Fig. 1.** Lateral standing radiographs obtained before the first (A), second (B), third (C), and fourth (D) operations. Circle indicates the center of the femoral head.
sex, postmenopausal state, preexisting disc degeneration, addition of instrumentation, injury to the adjacent facet joint, fusion length, and sagittal alignment. These factors applied partially to the current case, but age and sex were also risk factors for spondylolisthesis. As for facet injury due to an implant, each postoperative CT scan did not demonstrate the pedicle screw malposition and facet violation in the present case. We have also previously reported that ASD tended to occur at the cranial fusion segment under the same conditions as those at primary surgery, and the coexistence of facet tropism of more than 5° and lamina inclination more than 130° adjacent to the fusion segment were risk factors for ASD. Furthermore, Kaito and colleagues reported that excessive disc height reduction, more than 6 mm, was a risk factor for ASD after PLIF. In terms of risk factors for ASD, lamina inclination of more than 130° and facet tropism more than 5° were not detected at each level. Furthermore, disc height reduction of more than 6 mm was not detected at any PLIF level. Although these risk factors did not apply to the current case, spondylolisthesis at the cranial fused segment under the same conditions as at primary surgery occurred with the first and second ASD, as in our previous report. However, the third ASD showed L-1 vertebral collapse, not spondylolisthesis. Obvious osteoporosis was not detected. Furthermore, bone union was confirmed at each PLIF level, and no infection or tumor was detected in the pathological inspection of the intervertebral disc tissue. The pathology of the third ASD was considered to be proximal junctional kyphosis with adjacent vertebral collapse after multilevel fusion surgery. In fact, the sacral slope decreased gradually, as well as the lumbar lordosis. A decrease in sacral slope implies loss of lumbar lordosis. Although each PLIF achieved local lordosis at the fused segment, lumbar lordosis decreased gradually due to kyphosis of the cranial adjacent segment. A decrease in sacral slope might cause kyphotic mechanical force adjacent to the fused segment. With the increase in the fused segment, the time to revision surgery shortened, and the L-1 vertebra finally collapsed. A decrease in sacral slope and lumbar lordosis may be one of the major risk factors for ASD after PLIF.

The choice of surgery for the third ASD, whether anterior, posterior, or anterior and posterior combined, as well as the extent of the fusion length, was debatable. Although compression factors consisted mainly of anterior factors, posterior surgery was selected due to the necessity of implant removal before decompression and extension of the posterior fusion area, its advantage for kyphosis correction, and the need for moderately invasive surgery following cardiac surgery. In terms of fusion area, the cranial end was extended to T-4 beyond the kyphosis apex and the caudal end to L-5 to acquire the anchoring force. It was debatable whether the L5–S1 segment should be included in the fusion area. In the present case, the L5–S1 segment was not included because the anchoring force was sufficient, and no stenosis or instability such as excessive dynamic motion or spondylolisthesis was detected at the L5–S1 segment. Further observation will be necessary to confirm bone union at the L-1 level, implant

**Fig. 2.** Images obtained before (A–C) and after (D and E) the fourth operation. A: Lateral standing radiograph of the whole spine. B and C: Axial and sagittal CT scans of the L-1 vertebra. D and E: Standing radiographs of the entire spine.

**Fig. 3.** Graphs of lumbar lordosis, sacral slope, and pelvic tilt before each operation and after the last operation. Sacral slope decreased gradually, while pelvic tilt increased. Furthermore, lumbar lordosis decreased with loss of sacral slope.
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failure, proximal junctional kyphosis, and progression of L5–S1 degeneration.

Disclosure

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

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