# The influence of developmental spinal stenosis on reoperation risk at the adjacent segment after decompression surgery for lumbar spinal stenosis


## Abstract

**Aims:** To determine the influence of developmental spinal stenosis (DSS) on the risk of adjacent level reoperation.

**Patients and Methods:** This was a retrospective study of consecutive patients with decompression-only surgery for lumbar spinal stenosis and minimum 5-years follow-up. Adult deformities and spondylolisthesis were excluded. Presenting symptoms, levels operated on initially and at reoperation were studied. MRI measurements included the anteroposterior bony spinal canal diameter, degree of disc degeneration and ligamentum flavum thickness. DSS was defined by respective bony spinal canal diameter measurements. Risk factors for reoperation at the adjacent level were determined and included into a multivariate stepwise logistic regression for prediction modeling. Odds ratios (ORs) with 95% confidence intervals were calculated.

**Results:** 235 subjects were analyzed and 21.7% required reoperation at adjacent segments. Reoperation at the adjacent segment was associated with DSS ($p=0.026$), the number of operated levels ($p=0.008$) and age at surgery ($p=0.013$). Multivariate regression model ($p<0.001$) controlled for other confounders showed that DSS was a significant predictor of reoperation at an adjacent segment, with an adjusted OR of 3.93.

**Conclusions:** Adjacent nonoperated DSS levels are 3.9 times more likely of undergoing future surgery. This is a poor prognostic marker that can be identified during the index decompression surgery.

**Clinical Relevance**

- Patients with developmental narrowing of the bony spinal canal is at-risk of reoperation at adjacent levels after decompression surgery.

**Level of Evidence:** Level II Prognostic Study

**Keywords:** Developmental spinal stenosis; reoperation; adjacent level; adjacent segment; lumbar surgery; risk factor

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## Introduction

Developmental spinal stenosis (DSS) is manifested as pre-existing narrowing of the bony spinal canal.[1] This is likely a result of maldevelopment of the posterior elements during the fetal and postnatal period.[2] Several studies having identified potential candidate genes responsible for DSS as well as altered development not only in the bone but in the surrounding soft tissue.[3,4] The diagnostic definition of DSS has been studied and are level-specific for both radiographs and magnetic resonance imaging (MRI).[5,6] DSS may be identified as an apparent short pedicle on plain radiographs (**Figure 1**).

With a narrowed spinal canal, neural tissues may be more prone to compression and development of symptoms. Moreover, multilevel involvement is commonly observed and there is a possible risk of reoperation due to the predilection for symptomatic stenosis.[7] Reoperation in lumbar spinal stenosis surgery is not uncommon.[8] Previous reports suggest that up to 13% of patients require reoperation after decompression surgery, with 50% occurring at adjacent levels amounting to an average of 3.3% of patients per year requiring revision surgery.[9] Although some may attribute reoperation due to inadequate decompression, adjacent level degeneration, or unidentified hypermobility at adjacent segments, the influence of any inherent narrowing of the bony canal on the reoperation rate has not been studied. Even with successful decompression, we suspect that a patient with a developmentally narrowed canal is more likely to experience stenosis at other spinal levels despite only a mild degree of degeneration, thereby requiring another surgery. Hence, the aim of study is to determine the influence of DSS on the risk of adjacent level reoperation.

**Patients and Methods**

*Study Design and Subjects*

This was a retrospective analysis of consecutive patients who have underwent decompression-only surgery for lumbar spinal stenosis with at least 5 years of postoperative follow-up, and seen at a tertiary spine clinic from January 2010 to January 2016. All patients underwent their index operation between November 1993 and August 2010 for clinical symptoms of neurogenic claudication and/or lumbar radiculopathy both at index operation and at reoperation. Back pain was not considered as an indication for surgery. To limit the influence of external factors leading to reoperation risk, patients not operated for clinical symptoms of spinal stenosis or underwent fusion surgery, had previous spinal surgery, scoliosis deformity, spondylolisthesis, infection or spinal tumor were all excluded. Ethics approval has been obtained from the local ethics committee. Written informed consent has been obtained from all included patients.

All patients who underwent surgery had prior failed conservative treatment from 6-8 weeks including physiotherapy, analgesics or epidural injections. As we are a university unit, all patients must undergo a standardized assessment protocol prior to surgery. Patients were assessed by surgeons and therapists independently followed by a discussion and correlation with imaging. The operative levels were decided upon by matching the clinical symptomatology such as the nerve root involved with the spinal levels that required decompression. For example, a patient with L5 radiculopathy and MRI findings of L3/4 central stenosis and L4/5 lateral recess stenosis would have both levels decompressed as the L5 root could be compressed at either level. On the contrary, if a patient had L3 radiculopathy with the same MRI findings, surgery would not be offered due to the incompatibility of symptoms and further investigations were warranted.

For surgery, the standard surgical procedure adopted by our unit is a midline approach bilateral fenestration via laminotomy of the cranial lamina stopping at the level of the pars with undercutting of the cranial lamina until the ligamentum flavum is detached, and laminotomy of the caudal lamina to detach the caudal attachment of the ligamentum flavum. Medial facetectomy (both superior and inferior articular processes) and removal of the ligamentum flavum completes the decompression. More than half of the facets should be preserved during the procedure to prevent iatrogenic instability. Lateral extent of the decompression is considered complete when the traversing nerve roots are visualized.

*Data collection and imaging parameters*

At baseline, clinical parameters of symptomatology, age, gender, and levels operated were collected. Any new/recurrence of symptoms and reoperation rate were also studied. The levels where reoperation took place was specifically analyzed. Our study aimed to only assess the risk of surgery at the adjacent segments as any analysis of reoperation at the index spinal level might be construed as inadequate decompression and we would like to focus the analysis on the effects of DSS by limiting other confounding factors.

The preoperative T1- and T2-weighted sagittal and axial MRIs were read to collect imaging parameters from L1-S1 levels. The anteroposterior (AP) bony spinal canal diameter, ligamentum flavum thickness, intervertebral disc signal intensity (Pfirrmann grading system[10] and Schneiderman's classification[11]), any anterior or posterior disc bulging or herniation, and disc height were measured by two independent readers separately who were blinded to the clinical information. The AP spinal canal diameter was measured by the narrowest distance between the posterior vertebral body to the base of the laminar arch at the pedicle level (**Figure 2**) and was used for diagnosis of DSS. Ligamentum flavum thickness and the various disc degeneration phenotypes were recorded as these are important covariates that might give rise to stenosis at adjacent levels and subsequent need for surgery. The ligamentum flavum thickness was measured at the axial cut and at the thickest portion for both sides individually. Regarding the disc degeneration phenotypes, the Pfirmann grading system consists of five levels.[12] Grade I indicates a homogenous bright white disc structure with clear distinction between the nucleus and annulus, and a hyperintense or isointense signal to cerebrospinal fluid and normal disc height, while grade V indicates an inhomogenous black structure, loss of nucleus-annulus distinction, hypointense signal and collapsed disc space. For the Schneiderman's classification[11] (grade 0-3), grade 0 represents normal disc height and signal intensity while grade 3 indicates a signal void. Disc herniation was defined as any disc material that extended beyond a line drawn from the posterior vertebral body of the two adjacent vertebral levels. Disc height was measured at its midpoint per spinal level on the mid-sagittal image. Measurements were performed using the computer software, Centricity Enterprise Web 4.0 (GE Healthcare IT, Barrington, USA, 2010). Any patients without complete data were excluded from study.

3-Tesla MRI was utilized for this study. A standardized MRI protocol exists at our university unit to prevent variations in measurements. The field of view was 18x18cm for axial scans and 28x28cm for sagittal scans. Slice thickness was 4mm for both scans and slice spacing was 0mm for axial scans and 0.5mm for sagittal scans. Imaging matrix was 288x192 for axial scans and 512x224 for sagittal scans. The TR was 700-800ms and 4000-6000ms for T1 and T2, respectively and the TE was 8-10ms and 80-100ms for T1 and T2, respectively. There were 11 slices per vertebral level and parallel slices were made according to the disc and pedicle levels.

*Definition of DSS*

According to the index operated levels, their upper and lower segment and/or its adjacent level were identified and classified as having DSS or not depending on the individual AP bony spinal canal diameter and previously defined measurements.[6] Hence, any canal measurement smaller than the following measurements were considered DSS: L1: 20mm, L2: 19mm, L3: 19mm, L4: 14mm, L5: 14mm, and S1: 12mm.

*Statistical Analysis*

Descriptive statistics of the study population were calculated, including mean ± standard deviation (SD) and percentage (%). Depending on the type of variables (continuous, ordinal and categorical), intraclass correlation coefficient (ICC) and weighted kappa (wκ) were used to determine the interrater and intrarater reliability of the measurement of spinal canal diameter, ligamentum flavum thickness and disc height as well as disc degeneration. Two-way mixed effects model and one-way random effects model were used for the calculation. The reported ICC is indicative of poor, moderate, good and excellent agreement if the ICC value is of less than 0.5, between 0.5 and 0.75, between 0.75 and 0.9, and greater than 0.90 respectively, based on the 95% confidence interval of the ICC estimate.[13] Levels of agreement determined by wκ are considered slight, fair, moderate, substantial and almost perfect agreement when Cohen's kappa (κ) coefficient value is of 0.00 to 0.20, 0.21 to 0.40, 0.41 to 0.60, 0.61 to 0.80, and 0.81 to 1.00 respectively.[14]

The continuous measurements (canal diameter, ligamentum flavum thickness, disc height) were averaged if the differences were <1mm. Any deviances beyond this, as well as any for disc intensity and herniation grading, were discussed among the readers for a final agreed data point for analysis.

Univariate analysis through binary logistic regression was conducted between independent variables and the reoperation rate at adjacent segments. This allowed the examination of the association between variables, and facilitated the selection of predicting, associating factors or 'at risk' factors for reoperation at adjacent segment that were to be included in the subsequent multivariate analysis. The following independent variables at baseline were dichotomized for statistical analysis: presence of disc degeneration (in terms of anterior and posterior bulging at adjacent segments), the number of vertebral levels operated at index surgery (1-level, > 1 level), whether the cranial/caudal adjacent segment with index levels fulfilled the criteria for DSS, and whether reoperation involved the adjacent segments of index operated site. Disc height, Pfirrmann and Schneiderman classification at adjacent segments were also tested for any association with reoperation rate. Gender and age at index surgery were tested as confounding factors.

Multivariate logistic regression was performed, and odds ratios with 95% confidence intervals were calculated. Variables fulfilling a p-value <0.2 observed in the univariate analysis was included in the multivariate model.[15] Stepwise regression was applied as the appropriate measure since this study question was exploratory and previous regression models for DSS were not available. The model measured the independent effects of each of the predictor variables identified above on the outcome of reoperation at adjacent segment to index surgery levels after adjusting for any potential confounders. Statistical analyses were conducted using SPSS Windows

23.0 (IBM SPSS Inc., Chicago, IL, USA). Statistical significance was considered with a p-value less than 0.05.

**Results**

Out of a total of 332 patients who underwent surgery for lumbar spinal stenosis followed-up during the study period, 97 patients were excluded according to the criteria above leaving 235 subjects for analysis. None of the patients had missing data, or iatrogenic instability/spondylolisthesis. The mean duration of follow-up was 10.1 (± SD of 4.8) years since the index surgery. There was good interrater reliability for spinal canal diameter (ICC: 0.83, 95% CI (0.62, 0.93)) and ligamentum flavum thickness (ICC: 0.76, 95% CI (0.68, 0.88)) measurements and moderate interrater reliability for disc height measurements (ICC: 0.72, 95% CI (0.53, 0.84)). For disc bulging, Pfirmann and Schneiderman's grading, the interrater agreement ranged from fair to substantial (disc bulging – κ: 0.80, 95% CI (0.63, 0.97); Pfirrmann – κ: 0.32, 95% CI (0.13, 0.52); Schneiderman – κ: 0.39, 95% CI (0.19, 0.59)). Moderate to good (ICC ranged from 0.64 to 0.87) intrarater reliability was noted for measurement of spinal canal diameter, ligamentum flavum and disc height, and fair to moderate (κ ranged from 0.27 to 0.53) reliability for disc degeneration assessment.

Of the 235 patients studied, 203 (86.4%) had DSS at the cranial segment of the index operated level and 56 (23.8%) had DSS at the caudal segment. A total of 21.7% (n=51) of patients required reoperation at the adjacent level (**Table 1**). Among these patients, 43 (84.3%) and 10 patients (19.6%) had DSS at the cranial and caudal segments of the index operated level, respectively. The L4-5 level was most common as the index operated level with 77.4% of one-level decompression cases. These spinal segments were also most common to have adjacent levels of DSS requiring reoperation. The reoperation rate at the adjacent level of L5 (L4-5 or L5-S1) was 65.7%, whereas the reoperation rate of L4 (L3-4 or L4-5) was 62.5% (**Table 2**). **Table 3** summarizes the results for the univariate analysis of independent variables with reoperation at adjacent segment. No associations were found between reoperation and gender, nor with disc height and disc degeneration at adjacent segment. Significant associations were found between reoperation at adjacent segment and: i) DSS at the upper or lower adjacent segments of the index level, ii) the number of operated levels, iii) age at surgery. These associated factors were selected for the prediction model in the multivariate analysis.

**Table 4** entails the results of the multivariate regression model for prediction of reoperation involving the adjacent segment. The model reached statistical significance, $X^2$ (3, n = 235) = 17.938, p<0.001. The percentage of cases for which reoperation at adjacent segment correctly predicted by this model was 89.4%. The model explained 14.9% of the variance in reoperation at the adjacent segment. When considering the factor of DSS alone, the estimated odds ratio (OR) for reoperation at the adjacent level was 4.07 (95% CI : 1.18, 14.06, p=0.026). With the prediction model introducing the factors of the number of levels operated and the age at index surgery, the adjusted OR for reoperation at the adjacent segment was 3.93 (95% CI: 1.10, 14.01, p=0.035). Increasing age at surgery (β regression coefficient=-0.041) was associated with reduced likelihood of reoperation. The effects of these risk factors were demonstrated by the significant change of -2 Log likelihood if these factors were removed from the model (all at p<0.05).

## Discussion

DSS is a distinct imaging phenotype characterized by multiple vertebral level involvement, patients who are more prone to developing compression with less severity of degeneration, and an earlier age of onset as compared to degenerative stenosis (**Figure 3)**.[7,16] A pre-existing narrowed bony spinal canal leads to reduced threshold for neural compression. This feature is also a static parameter that is independent of the degenerative processes occurring in the disc, facet joints or ligamentum flavum. Our findings are consistent with these theories as our patients with DSS requiring reoperation were of younger age, independent of gender or disc degeneration factors. According to the Gerling study[17], patients with reoperation are likely to have undergone progressive degeneration. Although some form of degenerative process will still occur with age and itself will increase the risk of reoperation, we expect symptoms to arise in patients with DSS by only a milder degree of degeneration. As our study suggests, patients with DSS can experience reoperation during the early follow-up from the index operation due to only mild changes. DSS is found in our study to be an independent and highly influential factor with almost 4-fold increase in risk for reoperation at adjacent levels after lumbar spinal stenosis decompression surgery.

Compared to previous reports that 50% of patients with reoperation are performed at the adjacent levels, we only observed 21.7% in our series.[9] Our lower reoperation rate may be a result of only including patients with reoperation at adjacent levels while previous reports may have compiled all cases including those caused by inadequate decompression at the same index level requiring revision surgery. Nevertheless, our reoperation rate is more representative of the actual risk of adjacent level reoperation with reference to DSS. This relationship was more important in our prediction model as compared to features of disc degeneration like low signal intensity, bulging, loss of disc height and ligamentum flavum thickness, which are common causes of canal narrowing and symptoms. As illustrated from our model testing, the factor of DSS does significantly out-weigh the severity of disc degeneration and ligamentum flavum hypertrophy. In addition, the degree of degeneration is difficult to predict as its onset and progressive nature is often variable.

By comparison, DSS is an easier and measurable parameter that can be identified at the index operation as an accurate risk predictor for future surgery at the adjacent levels. This may influence the surgical decision at the index operation.

Nonoperated DSS levels were found have a higher risk of requiring surgery at follow-up. The significant association of reoperation with number of levels operated at index surgery reflects this relationship. A single-level surgery at the index operation is 3.6 times more likely to have reoperation at the adjacent DSS level. The relative less risk associated with multi-level surgery may simply be due more at-risk DSS levels being decompressed already at the index operation. Whether they were actually symptomatic to begin with or asymptomatic but radiologically significant enough for the surgeon to decompress prophylactically is difficult to ascertain. It is also evident that the effect of including the number of levels operated on improved the prediction model. According to Porter *et al*, the pathophysiological hypothesis of the involvement of two vertebral levels in lumbar stenosis is of degenerative nature.[18] Yet, DSS usually involves multiple levels and thus require meticulous surgical planning and may warrant a different surgical approach.

Limitations of study are unavoidable with a retrospective design as subjects have variable durations of follow-up and at the index operation, some DSS levels may not have been symptomatic but prophylactically decompressed based on the surgeon's decision. Hence, our overall reoperation rate is likely an underestimation. The definition of DSS used in this study was also based on an ethnically Chinese population and hence it may be subjected to variations in other populations. Despite these concerns, the adjusted OR for reoperation based on this DSS factor is already too significant to overlook. Although we have controlled external factors as much as possible by excluding patients with spondylolisthesis and adult spinal deformity, the effect of global spinal alignment and disc hypermobility were not studied and could have a role in determining reoperation rate.[19] It is also important to note that we specifically excluded patients who underwent spinal fusion as this significantly influences the risk of adjacent segment disease and reoperation.[20-23] Also, any differences between decompression techniques was not studied. Our unit performs a standard bilateral fenestration and medial facetectomy for decompression. However, whether our findings can be replicated with open laminectomy or endoscopic decompression for example, requires further study.

This study is a novel outlook on the effects of DSS on the risk of reoperation at the adjacent segment after lumbar spinal stenosis decompression surgery. There are significant implications on the approach to designing patient specific management strategies. Although a preliminary and controversial insight, there may be a role for pre-emptive decompression of developmentally narrowed levels at the index surgery. Scar tissues at and surrounding index surgical sites can increase the difficulty of decompression[24], leading to the risks of dural tears and nerve injuries. Along with the risks associated with repeated anesthesia, avoiding reoperation is the best solution in order to achieve the best surgical prognosis and efficacy. Hence, the canal diameter of nonoperated segments should be assessed and considered at the index operation. This is the first examination of DSS as it pertains to reoperation rate and serves as an important foundation for future prospective trials.

Nonoperated DSS levels are high risk for surgery after lumbar spinal stenosis decompression surgery. Adjacent levels should be screened for DSS prior to the index operation for risk assessment. By better prediction of the natural course of disease, avoiding future surgery may be achieved by early interventions. By extension, this improves the allocation of medical resources in terms of cost-effectiveness of a single surgery. This may optimize surgical treatment long-term prognosis, treatment outcome and overall patient care. Nevertheless, verification of these findings and theories should be performed via future trials.

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Table 1. Baseline characteristics at index surgery versus reoperation

| Parameters/ Radiological parameters | Study population n=235 | | | | | Reoperation n= 51 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Gender (F:M) | 106:129 (M:54.9%) | | | | | 22:29 (M: 56.9%) | | | | |
| Age at surgery (years, mean ± SD) | 66.8 ± 11.3 | | | | | 60.9 ± 11.4 | | | | |
| Spinal canal diameter (mm, mean ± SD) | | | | | | | | | | |
| L1 | 15.9 ± 1.6 | | | | | 15.7 ± 1.8 | | | | |
| L2 | 14.9 ± 1.8 | | | | | 14.5 ± 1.8 | | | | |
| L3 | 14.0 ± 2.1 | | | | | 14.2 ± 2.1 | | | | |
| L4 | 13.4 ± 1.8 | | | | | 13.7 ± 1.8 | | | | |
| L5 | 13.5 ± 2.1 | | | | | 13.7 ± 2.3 | | | | |
| S1 | 13.1 ± 2.3 | | | | | 13.1 ± 2.2 | | | | |
| Ligamentum flavum thickness (mm, mean ± SD) | | | | | | | | | | |
| | Right | | | Left | | Right | | | Left | |
| L1-2 | 3.1 ± 1.0 | | | 3.1 ± 1.0 | | 2.8 ± 0.9 | | | 2.7 ± 0.8 | |
| L2-3 | 3.5 ± 1.1 | | | 3.4 ± 1.2 | | 3.2 ± 1.1 | | | 3.3 ± 1.1 | |
| L3-4 | 3.7 ± 1.2 | | | 3.8 ± 1.2 | | 3.8 ± 1.7 | | | 3.8 ± 1.4 | |
| L4-5 | 3.8 ± 1.3 | | | 3.9 ± 1.4 | | 3.4 ± 1.3 | | | 3.4 ± 1.3 | |
| L5-S1 | 3.1 ± 1.3 | | | 3.1 ± 1.4 | | 2.7 ± 1.1 | | | 2.7 ± 1.2 | |
| Intervertebral disc height (mm, mean ± SD) | | | | | | | | | | |
| L1-2 | 9.6 ± 1.8 | | | | | 9.9 ± 1.6 | | | | |
| L2-3 | 10.0 ± 2.3 | | | | | 9.7 ± 2.6 | | | | |
| L3-4 | 10.6 ± 2.7 | | | | | 10.7 ± 2.5 | | | | |
| L4-5 | 10.0 ± 2.7 | | | | | 9.9 ± 2.8 | | | | |
| L5-S1 | 10.7 ± 2.8 | | | | | 10.7 ± 2.7 | | | | |
| Pffirmann (n) | | | | | | | | | | |
| | I | II | III | IV | V | I | II | III | IV | V |
| L1-2 | 0 | 1 | 72 | 90 | 4 | 0 | 1 | 17 | 18 | 1 |
| L2-3 | 0 | 1 | 63 | 93 | 12 | 0 | 1 | 17 | 16 | 3 |
| L3-4 | 0 | 0 | 50 | 106 | 12 | 0 | 0 | 16 | 19 | 2 |
| L4-5 | 0 | 0 | 36 | 114 | 18 | 0 | 0 | 14 | 20 | 3 |
| L5-S1 | 1 | 0 | 62 | 96 | 12 | 1 | 0 | 15 | 19 | 2 |
| Schneidermann (n) | | | | | | | | | | |
| | 0 | 1 | 2 | 3 | | 0 | 1 | 2 | 3 | |
| L1-2 | 0 | 73 | 90 | 4 | | 0 | 18 | 18 | 1 | |
| L2-3 | 0 | 65 | 92 | 12 | | 0 | 18 | 17 | 2 | |
| L3-4 | 0 | 51 | 103 | 14 | | 0 | 16 | 18 | 3 | |
| L4-5 | 1 | 37 | 112 | 19 | | 0 | 14 | 20 | 3 | |
| L5-S1 | 0 | 61 | 97 | 13 | | 0 | 15 | 20 | 2 | |
| Number of levels, n (%), operated at Index operation | | | | | | Reoperation Total n=51 | | | Reoperation at Adjacent level +/- index levels Total n=29 | |
| 1-level | n=116 | | | | | n=31 | | | n=21 | |
| At L3 - L4 | 5 (4.3) | | | | | 1 (3.2) | | | 1 (4.8) | |

| | | | |
|---|---|---|---|
| L4 - L5 | 100 (86.2) | 24 (77.4) | 15 (71.4) |
| L5 - S1 | 9 (7.8) | 4 (12.9) | 3 (14.3) |
| Others (L2; L4) | 2 (1.7) | 2 (6.5) | 2 (9.5) |
| 2-levels | n=84 | n=15 | n=7 |
| L2 - L4 | 3 (3.6) | 3 (20.0) | 2 (28.6) |
| L3 - L5 | 58 (69.0) | 10 (66.7) | 5 (71.4) |
| L4 - S1 | 22 (26.2) | 2 (13.3) | 0 |
| Others (at L2-L3, L4-5) | 1 (1.2) | 0 | 0 |
| ≥ 3 levels | n=35 | n=5 | n=1 |
| L1 - L5 | 3 (8.6) | 1 (20.0) | 0 |
| L2 - L5 | 15 (42.9) | 1 (20.0) | 0 |
| L2 - S1 | 3 (8.6) | 1 (20.0) | 0 |
| L3 - S1 | 12 (34.3) | 2 (40.0) | 1 (100) |
| Others (L1-S1; T12-L3) | 2 (8.6) | 0 | 0 |

F: females, M: males, SD: standard deviation, n: number

Table 2. Prevalence of developmental spinal stenosis (DSS) at defined vertebral levels

| Vertebral level | DSS at specific levels of study population n (%) | % of DSS involved in reoperation | Reoperation at adjacent DSS levels n (%) |
|---|---|---|---|
| L1 | 98 (97.0) | 90.5% (19/21) | 11 (57.9) |
| L2 | 156 (97.5) | 97.2% (35/36) | 21 (60.0) |
| L3 | 228 (99.1) | 96.0% (48/50) | 29 (60.5) |
| L4 | 152 (64.7) | 62.7% (32/51) | 20 (62.5) |
| L5 | 156 (66.4) | 62.7% (32/51) | 21 (65.7) |
| S1 | 79 (35.1) | 29.8% (14/47) | 7 (50.0) |

Note: n: number, %: percentage

Table 3. Univariate analysis for associations between independent variables and reoperation at adjacent segment

| Independent Variables | n | | $X^2$ | p-value of model prediction | Accuracy of prediction (% correct) | Odds Ratio (95% CI) |
|---|---|---|---|---|---|---|
| | Yes | No | | | | |
| Adjacent segment with index levels with DSS | 157 | 78 | 6.613 | 0.026* | 89.4 | 4.07 (1.18 – 14.06) |
| Number of levels operated<br>(Yes: 1 level; No: > 1 level) | 117 | 118 | 8.033 | 0.008* | 89.4 | 3.62 (1.39 – 9.42) |
| Disc Degeneration at adjacent levels | | | | | | |
| Anterior disc bulging | 111 | 115 | 0.274 | 0.601 | 89.4 | 1.25 (0.54 – 2.93) |
| Posterior disc bulging | 81 | 151 | 0.078 | 0.779 | 89.7 | 1.13 (0.47 – 2.72) |
| Pfirrmann grading | II<br>III<br>IV<br>V | 1<br>46<br>112<br>11 | 0.000 | 0.907 | 88.8 | - |
| Schneiderman's classification | 1<br>2<br>3 | 46<br>113<br>11 | 0.064 | 0.968 | 88.8 | - |
| Disc Height at adjacent levels | | | | | | |
| Upper adjacent segment | - | | 0.037 | 0.848 | 88.5 | 0.98 (0.82 – 1.18) |
| Lower adjacent segment | - | | 11.744 | 0.320 | 88.8 | 0.92 (0.78 – 1.09) |
| Confounding Factors | | | | | | |
| Age (Years) | - | | 5.973 | 0.013* | 89.4 | 0.96 (0.926 – 0.99) |
| Sex (M: F) | 106 : 129 | | 0.952 | 0.336 | 89.4 | 1.53 (0.645 – 3.61) |

Note: n: number of cases available for analysis, CI: Confidence interval, $X^2$: Chi-square value, DSS: developmental spinal stenosis

* Significance at p-value <0.05

Table 4. Multivariate logistic regression model for reoperation at DSS adjacent segment

| Predictor | Regression coefficient | Wald $X^2$ | p-value | Odds Ratio | 95% CI | Change in -2 Log Likelihood | Significance of change if factor is removed from model |
|---|---|---|---|---|---|---|---|
| Adjacent segment with index levels with DSS | 1.369 | 4.464 | 0.035* | 3.93 | 1.10 – 14.01 | 5.786 | 0.016* |
| Number of levels operated | 0.990 | 3.836 | 0.050* | 2.69 | 1.00 – 7.24 | 4.216 | 0.040* |
| Age | -0.041 | 4.875 | 0.027* | 0.96 | 0.93 – 0.99 | 4.856 | 0.028* |

Note: CI: Confidence interval, $X^2$: Chi-square value, DSS: developmental spinal stenosis

* Significance at p-value <0.05

## Figure Legends


**Figure 1:** Patients with developmental spinal stenosis has shorter pedicles (left) as compared to usual individuals (right) as illustrated in the lateral radiographs.

**Figure 2:** The anteroposterior bony spinal canal diameter was measured by the narrowest distance between the posterior vertebral body to the base of the laminar arch at the pedicle level.


Fig. 3a

Fig. 3b

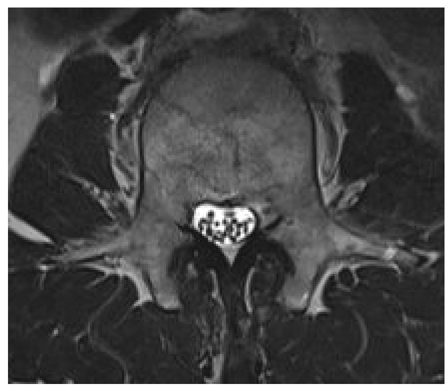

Fig. 3c

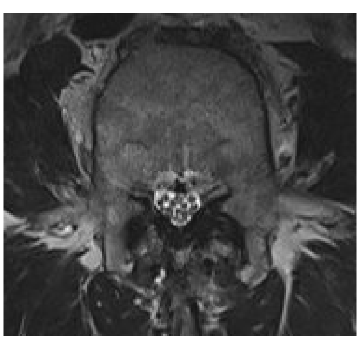

Fig. 3d

Fig. 3e

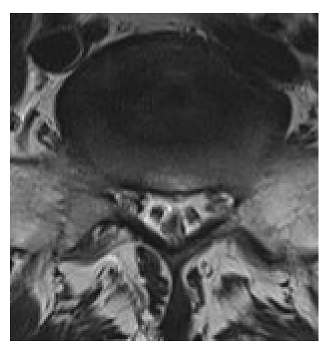

Fig. 3f

Fig. 3g

Fig. 3h

**Figure 3:** An illustrative case of a patient who presented with L5 radiculopathy contributed by lateral recess stenosis at L4/5 as shown on the T2-weighted sagittal (a) and axial (b) cuts. There was gross developmental spinal stenosis at multiple lower lumbar levels: L3 (<12mm) (c); L4 (<12mm) (d); L5 (<12mm) (e); S1 (<10mm) (f). Five years after the index surgery, the patient required another surgery due to recurrence of the L5 radiculopathy which can be contributed by L3/4 stenosis (g) despite mild degeneration only. There was facet joint hypertrophy and the sedimentation sign was observed as the nerve roots were unable to float to the posterior aspect of the dura. After the second surgery, the patient's L5 symptoms subsided. Further follow-up and observation is necessary for L5/S1 (h) closely as it is also at-risk for future compression due to its narrowed bony canal.