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Abdominal Aortic Calcification Is a Significant Poor Prognostic Factor for Clinical Outcomes After Decompressive Laminotomy for Lumbar Spinal Canal Stenosis

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Abstract

Study Design: Retrospective study.

Objective: To examine whether the presence of chronic kidney disease (CKD) or advanced abdominal aortic calcification (AAC) negatively affects clinical outcomes after decompression surgery for lumbar spinal canal stenosis (LSCS).

Methods: The subjects comprised 143 patients who underwent decompressive laminotomy for LSCS and were followed for $\geq$2 years. Fifty-five patients had CKD (Stage 3-4). Clinical outcome was assessed using the Japanese Orthopaedic Association (JOA) score before surgery and at 2-year postoperatively. According to the scoring system by Kauppila et al, the AAC score (a surrogate marker of systemic atherosclerosis) was assessed using preoperative lateral radiographs of the lumbar spine.

Results: Patient age had weak but significantly negative correlations with both the preoperative JOA score and the JOA score at 2 years after surgery, but did not have a significant correlation with the recovery rate of the JOA score at 2 years postoperatively. The JOA score before surgery, the JOA score at 2-year follow-up, and the recovery rate of the JOA score were slightly lower in the CKD patients than in those without CKD, although there were no significant differences between the 2 groups. On the contrary, the AAC score had a weak but significantly negative correlation with the preoperative JOA score, and had relatively strong and significantly negative correlations with both the JOA score at 2 years after surgery and the recovery rate of the JOA score.

Conclusions: At 2 years after surgery, advanced AAC was a significant poor prognostic factor for clinical outcomes of decompression surgery for LSCS.

Keywords

chronic kidney disease, abdominal aortic calcification, lumbar spinal canal stenosis, decompressive laminotomy, surgical outcomes

Introduction

Among risk factors for systemic atherosclerosis such as hyperlipidemia, hypertension, diabetes mellitus, and chronic kidney disease (CKD), we have recently reported that CKD, a risk factor for both poor bone quality and systemic atherosclerosis, and/or the presence of advanced abdominal aortic calcification (AAC), a surrogate marker of systemic atherosclerosis, negatively affects surgical outcomes after posterior lumbar interbody fusion (PLIF).[1] We have also reported that the presence of

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aortic arch calcification, another surrogate marker of systemic atherosclerosis, is a significant independent poor prognostic factor for surgical outcomes after cervical laminoplasty.[2] CKD is reportedly one of the most significant risk factors for systemic atherosclerosis.[3] Silverstein et al recently reported that CKD was a significant independent predictor of diminished improvement in health-related quality of life after lumbar decompression surgery.[4] Poor circulation resulting from advanced atherosclerosis could aggravate degeneration of the lumbar spine and the neural tissue. Therefore, CKD and/or systemic atherosclerosis may have negative impacts on clinical results after decompression surgery for lumbar spinal canal stenosis (LSCS). To the best of our knowledge, however, there has been no report of a study evaluating negative impacts of advanced AAC on surgical outcomes after surgical decompression for LSCS. The purpose of this study was thus to examine whether the presence of CKD or advanced AAC negatively affects clinical outcomes after decompressive laminotomy for LSCS.

Table 1. Japanese Orthopaedic Association Scoring System for Assessment of the Results of Treatment for Low Back Pain.

Item	Point Range
Subjective symptoms (9 points)	
Low back pain	3, 2, 1, 0
Leg pain and/or tingling sensation	3, 2, 1, 0
Walking ability	3, 2, 1, 0
Objective symptoms (6 points)	
Straight leg raising test	2, 1, 0
Sensory disturbance in the lower extremities	2, 1, 0
Muscle weakness in the lower extremities	2, 1, 0
Restriction of activities of daily living (14 points)	
Turning over while lying	2, 1, 0
Keeping standing	2, 1, 0
Face-washing	2, 1, 0
Kneeling position	2, 1, 0
Keeping sitting (about 1 hour)	2, 1, 0
Lifting or holding heavy object	2, 1, 0
Walking	2, 1, 0
Urinary bladder function	0, −3, −6
Total	29 to -6

Materials and Methods

Patients

Of consecutive 214 patients who underwent conventional open bilateral decompressive laminotomy with medical facetectomy and removal of the hypertrophied ligamentum flavum without fusion for LSCS between January 2014 and December 2015, one patient died during postoperative follow-up, 1 patient was lost to follow-up due to unknown reason, and 69 patients with a history of previous spine surgery, peripheral artery disease, rheumatoid arthritis, and/or undergoing hemodialysis were excluded in this study. Thus, the subjects comprised 143 patients (95 men, 48 women; mean age at the time of surgery, 70.3 years; age range, 34–91 years) who were followed for ≥2 years after surgery. Diagnostic criteria for CKD (Stage 3-4) were positive uric protein persisting for at least 3 months and/or estimated glomerular filtration rate <60 mL/min/1.73 m^2 persisting for at least 3 months. Based on these criteria, 55 patients had CKD (Stage 3-4). The number of surgically decompressed segments was as follows: 1 segment in 50 patients; 2 segments in 48; 3 segments in 35; and 4 segments in 10. All patients were considered for surgery due to unresponsiveness to conservative treatment such as medication and/or epidural block.

The protocol was approved by the institutional review board of the hospital, and written informed consent was obtained from all participants.

Clinical and Radiologic Evaluations

Clinical status was assessed using the Japanese Orthopaedic Association (JOA) scoring system for assessment of the results of treatment for low back pain before surgery and at 2 years after surgery.[5] The JOA score consists of the rating of subjective symptoms (low back pain, leg pain and/or tingling sensation, and walking ability), objective symptoms (straight leg raising test, sensory disturbance, and muscle weakness in the lower extremities), restriction of activities of daily living (7 items), and urinary bladder function. The total JOA score is 29 points in normal populations (Table 1). The postoperative recovery rate of the JOA score was calculated by the following formula:

$$\begin{aligned}&\text{Postoperative recovery rate of the JOA score }(\%)\\&= (\text{postoperative score} - \text{preoperative score})\\&\quad /(29 - \text{preoperative score}) \times 100\end{aligned}$$

As a surrogate marker of systemic atherosclerosis, AAC was assessed by the first author using preoperative lateral radiographs of the lumbar spine according to the scoring system by Kauppila et al[6]: Calcified deposits in the abdominal aorta adjacent to each lumbar vertebral segment (L1 to L4) were assessed separately for the anterior and posterior walls of the aorta. Each wall at each segment was graded for the presence of calcified deposits with a score from 0 to 3 points (0 point: no deposit; 1 point: <1/3 of the aortic wall length; 2 points: from ≥1/3 to ≤2/3 of the aortic wall length; and 3 points: >2/3 of the aortic wall length covered with calcified deposits). The sum total of the scores at each segment for both the anterior and posterior walls was taken as the AAC score. The maximum AAC score is thus 3 (0-3 points) × 2 (the anterior and posterior walls of the aorta) × 4 (L1-L4 segments) = 24 points.

Statistical Analysis

The Mann-Whitney *U* test, Kruskal-Wallis test, and Spearman's rank correlation were used for statistical analysis with JMP 5.0.1 software (SAS Institute, Cary, NC), as appropriate. Values of $P < .05$ were considered significant.

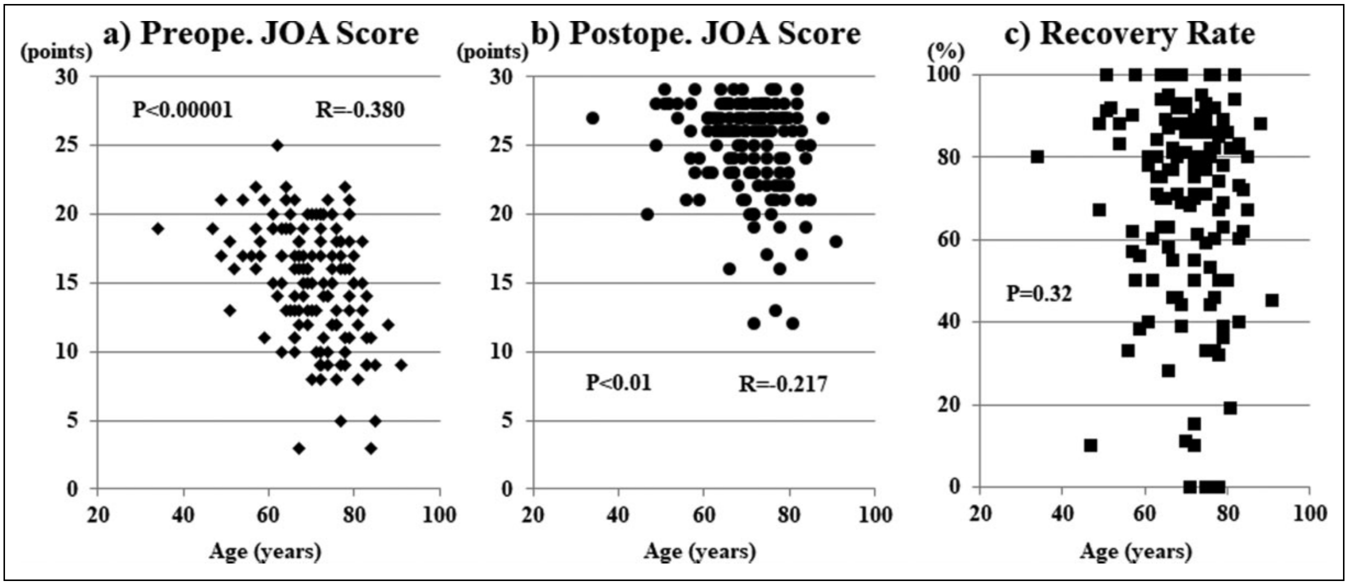

Figure 1. Correlation between patient age and severity of clinical symptoms. (a) Patient age has a relatively weak but significantly negative correlation with the Japanese Orthopaedic Association (JOA) score before surgery. (b) Patient age has a weak but significantly negative correlation with the JOA score at 2 years after surgery. (c) Patient age has no significant correlation with the recovery rate of the JOA score at 2 years after surgery.

Results

The JOA score at 2-year follow-up and the postoperative recovery rate of the JOA score tended to be worse in patients with multilevel LSCS than in those with single-level LSCS, but none of the preoperative JOA score, the JOA score at 2-year follow-up, and the postoperative recovery rate of the JOA score had any statistically significant correlations with the number of surgically decompressed segments ($P = .31$).

Patient age had weak but significantly negative correlations with both the JOA score before surgery and the JOA score at 2 years after surgery ($P < .00001$, $r = -0.380$, and $P < .01$, $r = -0.217$, respectively; Figure 1a and b), but did not have a significant correlation with the recovery rate of the JOA score at 2 years postoperatively ($P = .32$; Figure 1c).

The preoperative JOA score, the JOA score at 2-year follow-up, and the postoperative recovery rate of the JOA score were slightly lower in the CKD patients than in those without CKD, although there were no significant differences between the 2 groups ($P = .072$, $P = .28$, and $P = .43$, respectively; Table 2).

The AAC score tended to be higher in patients with multilevel LSCS than in those with single-level LSCS, but the AAC score did not have a statistically significant correlation with the number of surgically decompressed segments ($P = .22$). On the contrary, the AAC score had a weak but significantly negative correlation with the JOA score before surgery ($P < .001$, $r = -0.280$; Figure 2) and had relatively strong and significantly negative correlations with both the JOA score at 2 years postoperatively and the postoperative recovery rate of the JOA score ($P < .00000000001$, $r = -0.587$, and $P < .0000000000000001$, $r = -0.520$, respectively; Figures 3 and 4).

Table 2. Clinical Outcomes in Patients With or Without Chronic Kidney Disease.[a]

	JOA Score Before Surgery (Points)	JOA Score at 2 Years After Surgery (Points)	Recovery Rate of JOA Score at 2 Years After Surgery (%)
Chronic kidney disease (stage 3-4; n = 55)	14.1 ± 4.3	24.1 ± 4.2	67.3 ± 25.7
Chronic kidney disease (Stage 0-2; n = 88)	15.5 ± 4.3	25.1 ± 3.0	70.5 ± 22.0

Abbreviation: JOA, Japanese Orthopaedic Association.
[a]Data is mean ± standard deviation.

Discussion

Chronic Kidney Disease

CKD results in systemic atherosclerosis as well as poor bone quality.[7] The PLIF procedure consists of both neural decompression and interbody fusion. Therefore, the presence of CKD was significantly associated with worse JOA scores at 2 years after PLIF and a worse JOA score recovery rate.[1] On the other hand, in the present study, the preoperative JOA score, the JOA score at 2-year follow-up, and the postoperative recovery rate of the JOA score were slightly lower in the CKD patients than in those without CKD, although there were no significant differences between the 2 groups. Therefore, CKD alone did not have a significant negative impact on clinical outcomes after decompressive laminotomy for LSCS. This result may be because the presence of CKD itself does not necessarily reflect

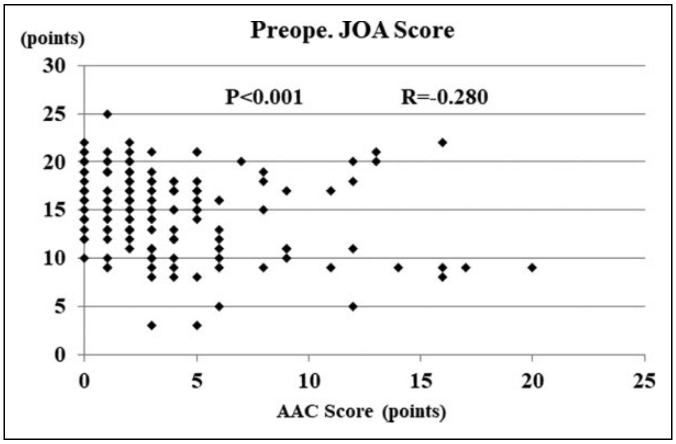

Figure 2. Correlation between the abdominal aortic calcification (AAC) score and the Japanese Orthopaedic Association (JOA) score before surgery. The AAC score has a weak but significantly negative correlation with the preoperative JOA score.

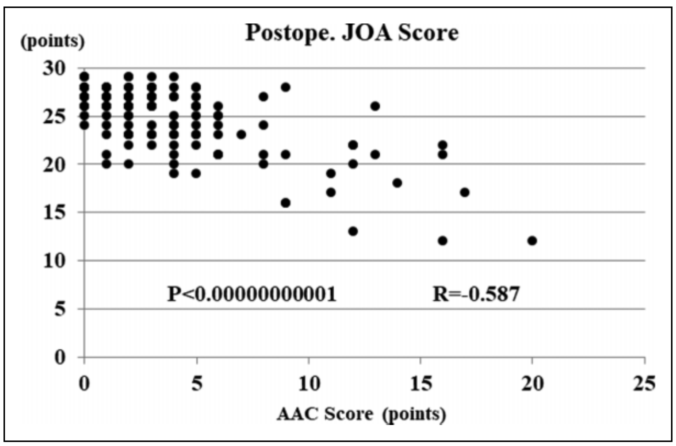

Figure 3. Correlation between the abdominal aortic calcification (AAC) score and the Japanese Orthopaedic Association (JOA) score at 2 years after surgery. The AAC score has a relatively strong and significantly negative correlation with the JOA score at 2 years postoperatively.

severity of systemic atherosclerosis, although CKD has been reported to be one of the most significant risk factors for systemic atherosclerosis.[3]

Abdominal Aortic Calcification

In our previously reported studies, the AAC score had relatively weak but significantly negative correlations with the preoperative JOA score, the JOA score at 2 years after PLIF, and the recovery rate of the JOA score, and advanced AAC significantly affected fusion status at 2 years after PLIF.[1] Moreover, the recovery rate of the JOA score at 2 years after cervical laminoplasty for cervical spondylotic myelopathy was significantly much lower in patients with aortic arch calcification than in those without aortic arch calcification.[2] In the present study, the AAC score had a weak but significantly negative correlation with the JOA score before surgery and had relatively strong and significantly negative correlations with both the postoperative JOA score and the postoperative recovery rate of the JOA score at 2 years after decompressive laminotomy for LSCS. The AAC score is reportedly one of the significant surrogate markers of systemic atherosclerosis.[8] Moreover, some researchers have reported that the extent of AAC is significantly associated with both low back pain and degeneration of the lumbar spine.[9-12] These results in the previously reported clinical studies indicated that poor circulation due to advanced AAC could aggravate degeneration of the lumbar spine and the neural tissue. On the other hand, in the present study, patient age had weak but significantly negative correlations with both the preoperative JOA score and the JOA score at 2 years after surgery. As one of the possible causes of this result, some items of the JOA score such as lifting or holding heavy objects and urinary bladder function can reflect normal aging processes. However, patient age did not have a significant correlation with the recovery rate of the JOA score at 2 years postoperatively.

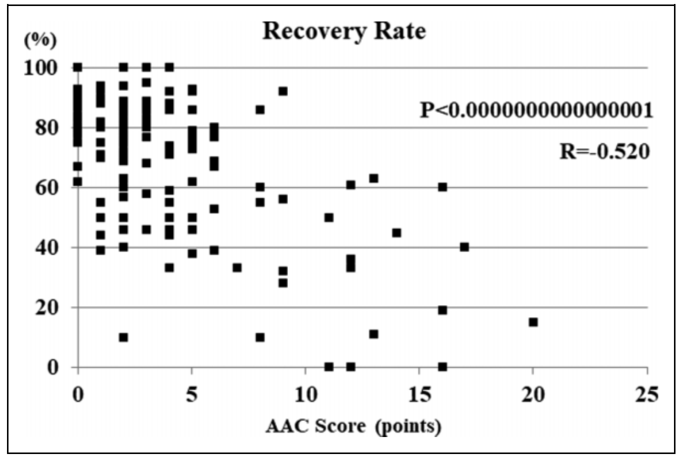

Figure 4. Correlation between the abdominal aortic calcification (AAC) score and the recovery rate of the Japanese Orthopaedic Association (JOA) score at 2 years after surgery. The AAC score has a relatively strong and significantly negative correlation with the recovery rate of the JOA score at 2 years after surgery.

Taken together, although neither severity of systemic atherosclerosis nor blood flow in cauda equina and lumbar spinal nerve roots was not directly evaluated in this study, these results in the present study indicated that advanced AAC was a significant poor prognostic factor for clinical outcomes after decompression surgery in patients with LSCS.

Limitations

There are several limitations in this retrospective study. (1) The number of patients was relatively small. (2) As described above, this study lacked direct evidences about both severity of systemic atherosclerosis and blood flow in cauda equina and lumbar spinal nerve roots. (3) As one of the significant risk factors for systemic atherosclerosis, smoking history was unclear. (4) AAC was assessed by the first author using preoperative lateral radiographs of the lumbar spine according to the scoring system by Kauppila et al. However, AAC assessment would be ideally completed by a blinded radiologist who has more experience in evaluating AAC on lateral radiographs of the lumbar spine. Thus, a further prospective study in a larger patient population including direct evaluations of both severity of systemic atherosclerosis and blood flow in cauda equina and lumbar spinal nerve roots, and smoking history will be needed to precisely analyze whether the AAC score or other confounders such as smoking history is a significant independent poor prognostic factor for clinical outcomes after decompression surgery for LSCS using the multivariate analysis.

Conclusion

In conclusion, although surgical treatments for degenerative lumbar spinal disorders have clinical significance even in patients with advanced AAC because the JOA score improved after surgery in all patients enrolled in this study, advanced AAC was a significant poor prognostic factor for clinical outcomes of decompressive laminotomy for LSCS.

Declaration of Conflicting Interests

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