Case Report

Delayed segmental motor paralysis following laminoplasty: two case reports

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Study design: Two patients who experienced the onset of segmental motor paralysis several years after laminoplasty are presented.

Objectives: To discuss the mechanism of development of delayed segmental motor paralysis following laminoplasty.

Setting: A department of orthopaedic surgery in Japan.

Methods: One patient experienced motor weakness in his deltoid and biceps muscles on the left side 5 years after laminoplasty. The other patient noticed motor weakness in his deltoid and biceps on the right side 7 years after laminoplasty. CT myelography revealed posterior spur formation and hypertrophic facet joints on the hinged side at the C4–C5 level in both patients. **Results:** Posterior foraminotomy was performed at the C4–C5 level on the hinged side in both patients. Postoperatively, motor weakness in the deltoid and biceps muscles was improved in both patients.

Conclusions: Although spondylotic changes, including spur formation and disc herniation, have occasionally developed in operated segments after laminoplasty, few patients have required additional surgery for treatment of relapse of neurological deficits. It has been believed that spinal cord is rarely compressed by the spondylotic changes since it shifts posteriorly in the enlarged spinal canal. However, laminoplasty disturbs the facet joints since the medial portion of dorsal cortex and cancellous bone in facet joints is drilled out to make a trough. Facet joints disturbed in this fashion undergo degenerated facet joints and spurs that have developed at the entrance of root canal, resulting in segmental motor paralysis several years after laminoplasty. Careful long-term observation is necessary after this procedure.

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Introduction

Laminoplasty has been the treatment of choice for selected patients with cervical myelopathy due to multisegmental cervical spondylosis. The reported long-term results of this procedure have been considered satisfactory.^{1–12} However, a few patients have exhibited late neurological deterioration after good recovery immediately after surgery. Numerous studies have been performed to elucidate the causes of late deterioration after this procedure.^{7,10,12,13} In these studies, several factors, including progression of ossified masses, development of spondylotic changes, diminished sagittal spinal diameter, progression of instability and severe kyphosis, were considered to be associated with worsening of clinical symptoms.^{7,10,12,13} Although spondylotic changes including spur formation and disc herniation have occasionally developed in operated segments after laminoplasty, few patients have required additional surgery for treatment of relapse of neurological deficits.^{1–4,6–12} It is believed that the spinal cord is rarely compressed by spondylotic changes since it is shifted posteriorly in the enlarged spinal canal after laminoplasty.

In this report, two cases of segmental motor paralysis that developed several years after laminoplasty are presented. Thereafter, the mechanism of development of this paralysis is discussed and the possibility of

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degeneration of facet joints induced by this procedure as a cause of this motor paralysis is proposed.

Case reports

Case 1

A 69-year-old man noticed clumsiness in his finger motion and numbress in both his upper and lower extremities, and was admitted to our hospital. On admission, he presented with moderate quadriparesis and urinary disturbance. Plain lateral radiography of the cervical spine disclosed decrease of disc heights at C4-C5, C5-C6 and C6-C7 levels, retrolisthesis of the C4 vertebral body, posterior spur formation at the C5–C6 and C6-C7 levels, and developmental canal stenosis (Figures 1a and 2). According to White's criteria,¹⁴ no spinal segmental instability was observed. Open door laminoplasty using the Hirabayashi method,4,10 with C3-C7 right side open, was performed. Postoperatively, symptoms were markedly reduced, and the sensory abnormality and motor weakness in both upper and lower extremities were eliminated.

At 5 years and 6 months after the surgery, the patient experienced motor weakness in his deltoid and biceps on the left side and was readmitted to our hospital. On readmission, there was no sensory abnormality or motor weakness in either lower extremity, and no urinary disturbance or gait disturbance was observed. Motor weakness of MMT Grade 2 in the deltoid and of Grade 1 in the biceps was present on the left but not the right side. Sensory abnormality was observed in neither upper extremity. The biceps tendon reflex was diminished on the left side but normal on the right side. Other deep tendon reflexes were normal bilaterally in the upper and lower extremities. Spurling's neck compression test was negative bilaterally. On plain radiography of the cervical spine, although enlargement of the spinal canal was maintained from C3 to C7 level, spondylotic changes including decrease of disc heights, retrolisthesis of the C4 vertebral body and posterior spur formations were noted (Figure 1b). However, no spinal segmental instability was observed at the operated levels. CT myelography revealed posterior spur formation and a hypertrophic facet joint on the right side at C4–C5 level (Figure 2b).

Posterior foraminotomy was performed at the C4–C5 level on the left side. Postoperatively, motor weakness in the deltoid and biceps muscles was improved.

Case 2

A 62-year-old man suffered from sensory abnormality in both upper and lower extremities, gait disturbance and urinary dysfunction, and was admitted to our hospital. Open door laminoplasty using the Hirabayashi method,^{4,10} with C3–C7 left side open, was performed. His neurological symptoms disappeared after surgery.

At 7 years after the surgery, the patient suffered from numbness and pain in his right upper extremity, which became increasingly severe. Thereafter, he noticed motor weakness in his deltoid and biceps on the right side, and was readmitted to our hospital. On readmission, there was no sensory abnormality or motor weakness in either lower extremity, and no urinary



Figure 1 Plain lateral radiograms of the cervical spine in Case 1. (a) Before first operation. (b) At 5 years after first operation. Before first operation, decrease of disc heights at C4–C5, C5–C6 and C6–C7 levels, retrolisthesis of the C4 vertebral body, posterior spur formations at C5–C6 and C6–C7 levels, and developmental canal stenosis were observed. Although enlargement of the spinal canal was maintained from C3 to C7 level, spondylotic changes, including decrease of disc heights, retrolisthesis of C4 vertebral body, and posterior spur formations, were developed 5 years after the first operation

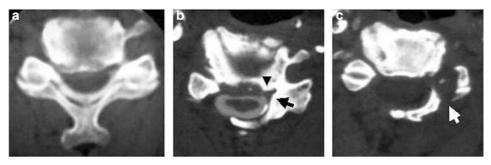


Figure 2 CT findings at C4–C5 level in Case 1. (a) CT myelogram before first operation. (b) CT myelogram 5 years after first operation. (c) CT 1 month after second operation. CT myelography revealed posterior spur formation (arrowhead) and hypertrophic facet joint (arrow) at the hinge side 5 years after the first operation. Posterior forminotomy was performed on the left side (white arrow)

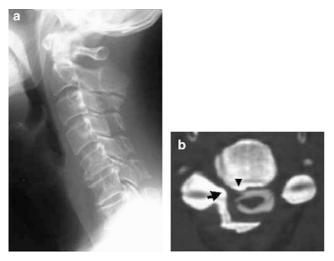


Figure 3 Plain lateral radiogram of the cervical spine and CT findings at C4–C5 level 7 years after first operation in Case 2. (a) Plain lateral radiogram of the cervical spine. (b) CT myelogram of the C4–C5 level. On the plain lateral radiogram, enlargement of the spinal canal, decrease of disc heights at C5–C6 and C6–C7 levels and posterior spur formations at C4–C5 level revealed posterior spur formation (arrowhead) and hypertrophic facet joint (arrow) at the hinge side

disturbance or gait disturbance was observed. Motor weakness of MMT Grade 3 in the deltoid and biceps and sensory abnormality in the lateral aspect of the upper arm were observed on the right but not the left side. The biceps tendon reflex was diminished on the right side but normal on the left side. Other deep tendon reflexes were normal bilaterally in the upper and lower extremities. Spurling's neck compression test was positive on the right side. On plain radiography of the cervical spine, although enlargement of the spinal canal was maintained from C3 level to C7 level, decrease of disc heights at C5–C6 and C6–C7 levels and posterior spurs at C4–C5 and C5–C6 levels were observed (Figure 3a). According to White's criteria,¹⁴ however, no spinal segmental instability was observed at the operated levels. CT myelography revealed posterior spur formation and hypertrophic facet joints on the right side at C4–C5 level (Figure 3b).

Posterior foraminotomy was performed at the C4–C5 level on the right side. Postoperatively, motor weakness in the deltoid and biceps muscles was improved.

Discussion

Segmental motor paralysis mainly involving C5 is occasionally seen in patients who have undergone laminoplasty.^{7,10,16–18,20–22} This paralysis develops until 2 weeks after surgery and usually disappears spontaneously within 2 years after surgery.^{10,17} Segmental motor paralysis is therefore considered to be one of the early complications of this procedure. It had long been believed that this paralysis was due to nerve root lesions caused either by inadequate surgical technique including trauma by high-speed burrs or Kerrison rongeurs or by compression resulting from dropping of a detached laminar hinge into the spinal canal.¹⁰ However, these types of intraoperative trauma are likely to damage the posterior root rather than the anterior root, and the sensory disturbance should therefore be marked. Nevertheless, sensory disturbance was absent in most cases showing segmental paralysis after laminoplasty. Although the etiology of this paralysis remains unclear, several investigators have recently supported the hypothesis that it involves tethering of the nerve root induced by excessive posterior shift of the spinal cord after decompression.^{10,15–22} In the present patients, on the other hand, degenerated facet joint and spur formation were considered as causes of paralysis and resulted in late deterioration following laminoplasty.

In general, degeneration of adjacent motion segments occurs less often after laminoplasty than after corpectomy.^{17,18,20,21,23} The decreased incidence of adjacent segment degeneration observed after laminoplasty may result from maintenance of segmental motion.²³ However, preserved segmental motion may play a role in the development of spondylotic changes in operated motion segments after laminoplasty. This procedure disturbs facet joints since the medial portion of dorsal cortex and 463

cancellous bone in facet joints is drilled out to make a trough. Disturbed facet joints undergo degeneration over time after surgery. In patients who have undergone laminoplasty, degeneration of facet joints and uncovertebral joints may be exaggerated by preservation of segmental motion. Although the spinal cord is rarely compressed by spondylotic changes that develop because it is shifted posteriorly in the enlarged spinal canal, nerve roots might occasionally be compressed by degenerated facet joints and uncovertebral spurs that have developed at the entrance of the root canal, resulting in segmental motor paralysis several years after surgery. Therefore, concomitant foraminotomy combined with laminoplasty may be recommended for initial surgery for patients exhibiting root canal stenosis before surgery, even if they did not complain of symptoms of radiculopathy before surgery. For patients exhibiting segmental instability before surgery, fusion with posterior decompression may be selected.

In conclusion, delayed segmental motor paralysis may be considered as one of the late complications of laminoplasty. Disturbance of facet joints by drilling and development of spondylotic changes may result in development of root canal stenosis after surgery, occasionally associated with neurological deterioration. Careful long-term observation is thus necessary after this procedure.

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