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Cervical restenosis caused by progressive ossification of the posterior longitudinal ligament in patients following laminoplasty: Two case reports☆

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ABSTRACT

We report two cases of restenosis caused by the progression of thickness of ossification of the posterior longitudinal ligament (OPLL) seven and more years after laminoplasty, resulting in neurological deterioration needed for revision anterior decompressive surgeries. Neurological recovery after revision anterior excision of OPLL was poor. In both cases, the patients had progressive OPLL, with a non-ossified segment of the ossification foci, in common. After laminoplasty, they also both exhibited osseous fusion of the elevated laminae, but there was discontinuity at the interlaminar space at the peak level of OPLL. Discontinuity of the osseous fusion in the elevated laminae might cause mechanical stress increases at the non-ossified segment of the OPLL and could lead to the progression of OPLL. The present cases showed that long-term progression of OPLL can induce neurological deterioration even after sufficient posterior decompression by laminoplasty. Therefore, when considering risk factors that may be predictive of the progression of OPLL after laminoplasty, it is important to perform strict follow-up examination to check for progression to reduce the risk of myelopathy symptoms that are indicative of neurological deterioration.

1. Introduction

Ossification of the posterior longitudinal ligament (OPLL) is one of the major causes of compressive myelopathy in Japan [1]. Once myelopathy develops in patients with OPLL, surgical decompression is strongly recommended to avoid the exacerbation of myelopathy symptoms [2]. In Japan, expansive laminoplasty is widely accepted surgical procedure for cervical OPLL [3]. Laminoplasty has several advantages: it can achieve simultaneous decompression in multilevel compression lesions, it can be performed without spinal fusion, the surgical procedure is relatively easy to perform and it can provide a long-term stable clinical outcome. However, there is a possibility that the ossification foci can progress after laminoplasty in exchange for preservation of range of motion. There are only a few reports about revision surgery for late neurological deterioration caused by progression of OPLL after laminoplasty [4,5].

Here we report two cases of restenosis caused by the progression of OPLL seven and more years after laminoplasty resulting in neurological deterioration needed for revision anterior decompressive surgeries.

2. Case presentations

2.1. Case 1

A 70-year-old man complained of bilateral hand clumsiness and gait disturbance caused by cervical OPLL. The patient had a history of laminoplasty for cervical OPLL 24 years prior, resulting in moderate recovery. Magnetic resonance imaging (MRI: Fig. 1C) and computed tomography (CT: Fig. 1B) scans revealed that the patient's OPLL had reached a peak at the C5/6 level and was compressing his spinal cord. His elevated laminae were fused from C2 to C5 and from C6 to C7 (Fig. 1A), motion at C5/6 was evident on flexion-extension radiographs, and the CT scan revealed the segment at C5/6 was non-ossified (Fig. 1B).

We performed an anterior decompression with an iliac bone graft using an anterior locking plate (Fig. 1D). Surgery only achieved a slight

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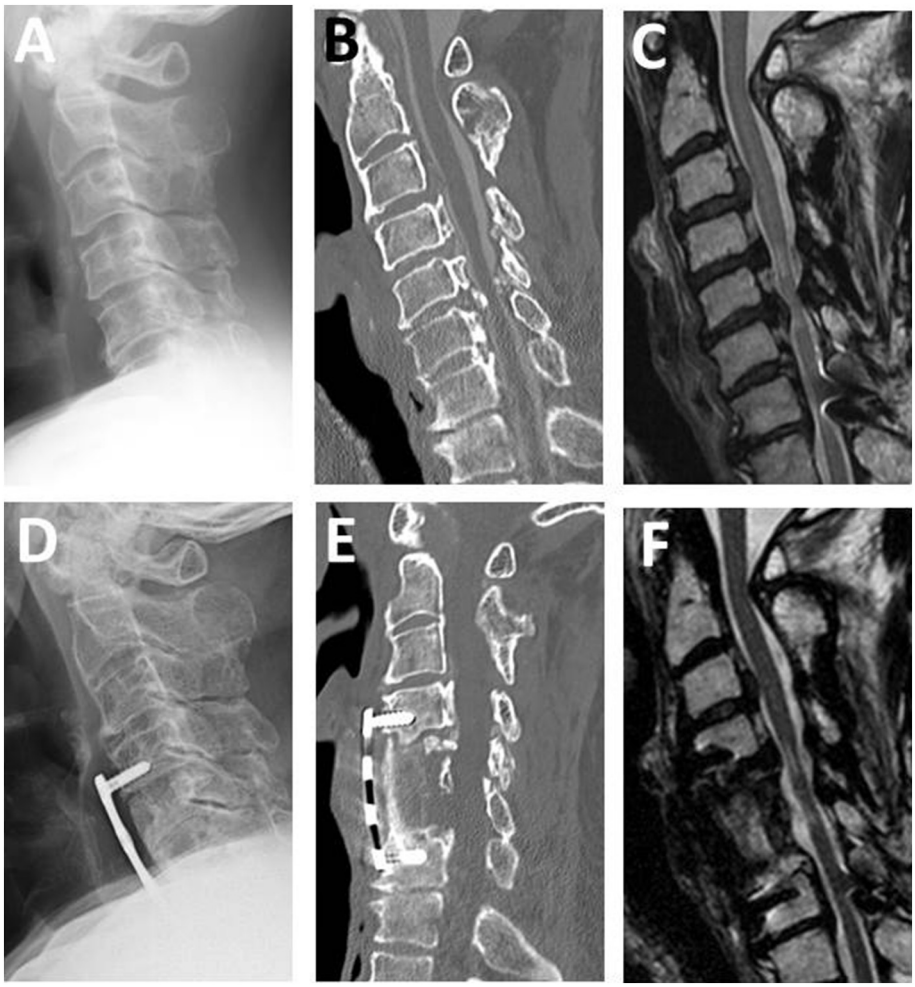

Fig. 1. Pre- and post-operative images of Case 1. Although sufficient enlargement of spinal canal was obtained by the initial laminoplasty (A), OPLL compressed spinal cord at C5/6 level (B, C). Spinal cord was decompressed by revision C4-7 anterior decompression and fusion was performed (D, E, F).

attenuation of bilateral hand clumsiness (improvement of Japanese Orthopedic Association (JOA) score was only 1 point (8.5 to 9.5 points)) although sufficient spinal cord decompression was obtained (Fig. 1E, F).

2.2. Case 2

A 62-year-old man complained of bilateral hand clumsiness and gait disturbance caused by cervical OPLL. The patient had a history of laminoplasty for cervical OPLL 7 years prior. Sufficient spinal cord decompression was obtained after initial surgery (Fig. 2A, B) resulting in moderate neurological recovery. Apparent OPLL progression was revealed CT obtained 7 years after the initial surgery at the time point with neurological deterioration (Fig. 2C, D) compared with CT immediately after the initial surgery (Fig. 2A, B). CT scan (Fig. 2C, D) revealed that the OPLL reached a peak at the C4/5 level and was compressing his spinal cord. His elevated laminae were fused from C2 to C4 and from C5 to C7 (Fig. 2D), motion at C4/5 was evident on flexion-extension radiographs, and the CT scan revealed the segment at C4/5 was non-ossified (Fig. 2D).

We performed anterior decompression and a fibula autograft using an anterior pedicle screw (Fig. 2E). Surgery only achieved a slight improvement of gait disturbance (improvement of JOA score was only 1 point (8.5 to 9.5 points)) although sufficient spinal cord decompression was obtained (Fig. 2F, G).

3. Discussion

In both cases, the patients had progressive OPLL, with a non-ossified segment of the ossification foci, in common. After laminoplasty, they also both exhibited osseous fusion of the elevated laminae, but there was discontinuity at the interlaminar space at the peak level of OPLL. Reports have indicated that OPLL can progress at the non-ossified segment of the ossification foci [6,7]. Discontinuity of the osseous fusion in the elevated laminae might cause mechanical stress increases at the non-ossified segment of the OPLL and could lead to the progression of OPLL. In addition, stress concentration to the non-ossified segment of the OPLL concomitant with discontinuity between fused laminae could induce proliferation of posterior element including laminae and remaining ligamentum flavum as well as progression of OPLL, possibly resulting in aggravation of spinal cord compression (Fig. 2C, D).

The present cases showed that long-term progression of OPLL can induce neurological deterioration even after sufficient posterior decompression by laminoplasty in contrast to the previous reports by Kawaguchi describing no revision surgery even though progression of OPLL after laminoplasty was observed [8,9].

The patients in these two case studies exhibited poor neurological recovery after revision surgery for progressive OPLL. The possible cause of these poor neurological recoveries may be attributed to long-term compression of the spinal cord by the OPLL, similar to previous reports, which caused irreversible cord damage [5,10]. Therefore, it is important to perform strict follow-up examination to check for neurological deterioration. Further exploration of long-term strategies for

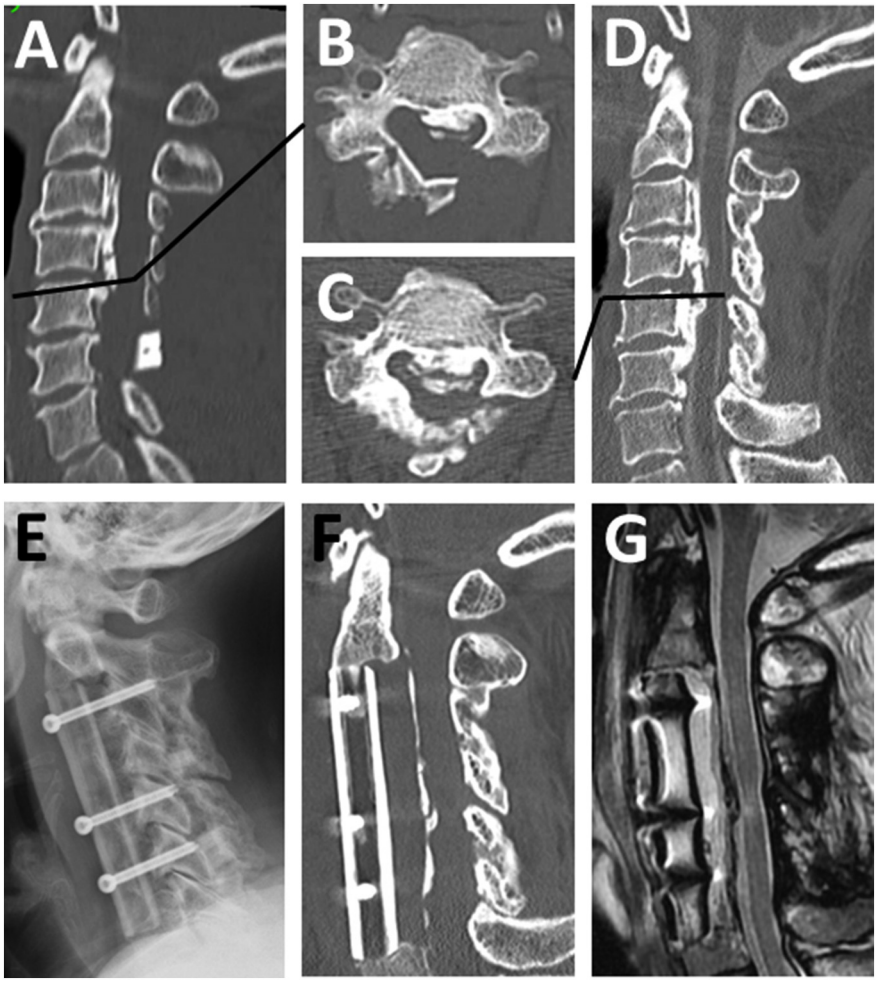

Fig. 2. Images showing OPLL progression after laminoplasty and revision anterior surgery in Case 2. Sufficient decompression was obtained immediately after initial laminoplasty (A, B). Seven years later, OPLL progression was observed, resulting in recurrent spinal cord compression (C, D). Sufficient spinal cord decompression was obtained by revision anterior decompression and fusion surgery was performed (E, F, G).

maintenance of neurological recovery after laminoplasty is needed to clarify this issue.

4. Conclusion

Long-term progression of cervical OPLL can induce neurological deterioration, even after sufficient decompression by laminoplasty.

Notes

The authors have no conflict of interest.

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