Thoracic myelopathy due to enlarged ossified yellow ligaments

Case report and review of the literature

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Enlarged ossified yellow ligaments are a rare and poorly understood cause of thoracic myelopathy. The authors report the case of a patient in whom thoracic myelopathy was caused by enlarged ossified yellow ligaments.

KEY WORDS • thoracic spine • myelopathy • ossification • ligamentum flavum

Enlarged ossified yellow ligaments are a rare and poorly understood cause of thoracic myelopathy. During the past two decades the condition has been reported mainly in Japanese patients, although recently six cases involving Caucasian and two involving African–American patients have been documented (Table 1). We present the case of another Caucasian patient with thoracic myelopathy due to enlarged ossified yellow ligaments.

In cases of this disease, the lower thoracic spine is the most common site affected. Symptomatic patients usually begin to develop paraspastic gait disturbance, often in combination with reduced depth sensation. Nerve root compression with intercostal neuralgic pain may be present but is not typical. Axial computerized tomography or MR imaging is used to establish diagnosis. Management generally entails performing a decompressive laminectomy, although both laminoplasty and, recently, foraminotomy or extended partial laminectomy have been suggested.

Case Report

Presentation and Examination. This 58-year-old Caucasian man had a 4-year history of progressive electrifying pain in both legs, paraspastic and atactic gait disturbance, loss of proximal leg strength, and bladder and sexual dysfunction. Neurological abnormalities included increased deep tendon reflexes and bilateral positive Babinski’s sign, virtually absent depth sensation of the lower legs and tactile as well as pain-related hyposensitivity below the level of T-12. Computerized tomography and MR imaging revealed concentric narrowing of the spinal canal between T-10 and T-12 with spinal cord compression due to enlarged calcified ossified yellow ligaments and hypertrophy of articular processes (Fig. 1). Advanced osteochondroses and spondylarthroses, without spinal cord or nerve root compression, were observed radiologically in the lumbar and cervical spine. No other skeletal abnormalities or related systemic disease were found.

First Operation. A T10–12 laminectomy was performed. Filiform narrowing of the dural sac and a thinned dura found at these spinal levels were caused by sclerotic thickened laminae and partially dura adherent ossified hypertrophic yellow ligaments, hard like stone. These were removed. Two 40-mg doses of methylprednisolone were administered intravenously during the operation, continued at the same dosage for the following 2 days, and then tapered over 5 days (40, 28, 20, 12, 8 mg, respectively).

Follow-Up Course. During a 1.5-year postoperative period, gait disturbance improved slightly as did the sensitivity of the left leg. The electrifying pain in the legs was no longer present. Within 2 years postoperatively, however, the patient developed increased spasticity and weakness of the right leg and tactile hyposensitivity below T-10. An MR image revealed narrowing of the spinal canal with...
medullary compression at T-8 and T-9 (Fig. 2), as had been demonstrated 2 years previously at T10–12.

**Second Operation and Follow-Up Course.** We performed T-8 and T-9 laminectomies, and the intraoperative findings were identical to those of the first intervention. Methylprednisolone was administered in the same fashion as in the first operation. Strength and sensitivity of the right leg were improved 1 year postoperatively, the spasticity persisted, and bladder function was slightly improved. Ascending progressive narrowing of the thoracic spinal cord due to continuing ascending ossification of the yellow ligaments was feared. One year after the second operation, MR imaging revealed no stenosis at higher vertebral levels, although a moderate restenosis had developed between T-9 and T-10.

Histological examination of the initial surgical specimen revealed sclerotic, partially necrotic bone fragments with focal signs of enchondral ossification. The surgical specimen obtained at the second operation was shown to have fragments of yellow ligaments with focal pseudocystic and mucoid degeneration, chondroid metaplasia, and enchondral ossification resulting in lamellar bone formation (Fig. 3).

Serum levels of calcium (2.37 mmol/L; normal range 2.20–2.55 mmol/L), parathyroid hormone (43 ng/L; normal < 150 ng/L), alkaline phosphatase (64 U/L, normal range 45–122 U/L), osteocalcin (2.5, μg/L; normal range 3–8 μg/L), and urinary excretion of pyridinoline/creatinine (53 nmol/mmol, normal range 30–60 nmol/mmol) and deoxypyridinoline (14 nmol/mmol, normal range 4–17 nmol/mmol) were within the reference range.

**Discussion**

Thoracic compressive myelopathy secondary to ossification of the yellow ligaments has mainly been observed in Japanese patients (Table 1). In recent reports the

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**Fig. 1.** Imaging studies. *Left:* Sagittal T₁-weighted MR image revealing posterior narrowing of the spinal cord at T10–12; note that there is no relevant narrowing at T8–9. *Right:* Axial computerized tomography scan demonstrating hypertrophied ossified yellow ligaments at T10–11.

**Fig. 2.** Sagittal T₂-weighted MR image revealing recently developed posterior narrowing of the spinal canal at T-8 and T-9.
Myelopathy due to enlarged ossified yellow ligaments

Authors have described the same disorder in six Caucasian, one African–Caribbean, and one African–American patients (Table 1). The lower thoracic spine is the predominant site affected. The pathogenesis of ossified yellow ligaments is not clear. The proposed mechanisms include mechanical stress of the lower thoracic spine and the thoracolumbar junction, causing yellow ligament degeneration. It remains to be clarified, however, why ossification of the yellow ligaments occurs less frequently in the lumbar spine, despite the fact that, compared with the lower thoracic spine, it is subjected to higher mechanical forces causing more pronounced degenerative changes of the facet joints and intervertebral discs.

Histological examination of surgically obtained specimens typically reveals ossification along the superficial layer of the hypertrophied yellow ligaments, an increased number and size of collagen fibers, fibrocartilaginous cells between these collagen fibers, and a decreased number of elastic fibers, as well as premature osteons or osteoblasts. The adjacent articular processes and laminae are also surrounded by compact lamellar bone, mainly as a result of enchondral ossification of the hypertrophied yellow ligaments. In our patient, we likewise found focal mucoid and pseudocystic degeneration of yellow ligament fragments, with chondroid metaplasia, and enchondral ossification as well as sclerotic bone fragments.

There was no evidence of increased bone remodeling, as revealed by normal serum levels of alkaline phosphatase and osteocalcin and normal urinary excretion of pyridinoline and deoxypyridinoline. This does not, however, rule out focal increased bone turnover. Because of the normal serum levels of calcium and parathyroid hormone, hyperparathyroidism was discounted. Serum levels of fibronectin were not determined in the present study. Fibronectin is thought to be responsible for the proliferation of fibroblasts in the yellow ligaments, and levels of fibronectin were reported to be raised in some Japanese patients. However, it is difficult to interpret the increased levels in the absence of other parameters that indicate increased bone turnover.

Decompressive laminectomy and removal of the enlarged ossified yellow ligaments is the most commonly performed surgical procedure in patients with compressive thoracic myelopathy due to enlarged and ossified yellow ligaments. Okada and colleagues have recommended performing laminoplasty after decompression because they observed unsatisfactory results after laminectomy alone: later deterioration due to the recurrence of ossification of the yellow ligaments at the same site or increased kyphotic deformity of the spine (Table 1). This was also reported by Yonenobu and associates. Nishiura, et

### TABLE 1

**Review of the literature on patients with symptomatic thoracic myelopathy due to enlarged ossified yellow ligaments**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>Race</th>
<th>Therapy</th>
<th>Follow Up (mos)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Omojola, et al., 1982</td>
<td>1</td>
<td>C</td>
<td>decomp</td>
<td>1.5</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Miyasaka, et al., 1983</td>
<td>18</td>
<td>J</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Yonenobu, et al., 1987</td>
<td>8</td>
<td>J</td>
<td>decomp</td>
<td>52–64</td>
<td>53% good or fair</td>
</tr>
<tr>
<td>Enomoto, et al., 1988</td>
<td>1</td>
<td>J</td>
<td>decomp</td>
<td>ND</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Vossen, et al., 1988</td>
<td>1</td>
<td>C</td>
<td>decomp</td>
<td>2</td>
<td>ND</td>
</tr>
<tr>
<td>Hanakita, et al., 1990</td>
<td>16</td>
<td>J</td>
<td>decomp</td>
<td>ND</td>
<td>30% satisfactory</td>
</tr>
<tr>
<td>Okada, et al., 1991</td>
<td>14</td>
<td>J</td>
<td>decomp;</td>
<td>86</td>
<td>75% satisfactory</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>decomp &amp; lam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kojima, et al., 1992</td>
<td>1</td>
<td>J</td>
<td>decomp</td>
<td>6</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Arifat, et al., 1993</td>
<td>1</td>
<td>C</td>
<td>decomp</td>
<td>0.3</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Miyamoto, et al., 1993</td>
<td>24</td>
<td>J</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Shiraiishi, et al., 1995</td>
<td>1</td>
<td>C</td>
<td>selective</td>
<td>7</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Chana &amp; Afshar, 1996</td>
<td>1</td>
<td>AC</td>
<td>decomp</td>
<td>2.5</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Shenoi, et al., 1997</td>
<td>1</td>
<td>J</td>
<td>decomp</td>
<td>3</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Vera, et al., 1997</td>
<td>1</td>
<td>C</td>
<td>decomp</td>
<td>12</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Pascal-Mousselard, et al., 1998</td>
<td>1</td>
<td>AA</td>
<td>decomp</td>
<td>12</td>
<td>neuro improve</td>
</tr>
<tr>
<td>van Oostenbrugge, et al., 1999</td>
<td>1</td>
<td>C</td>
<td>decomp</td>
<td>2</td>
<td>neuro improve</td>
</tr>
<tr>
<td>Nishiura, et al., 1999</td>
<td>37</td>
<td>J</td>
<td>FPL</td>
<td>ND</td>
<td>ND</td>
</tr>
</tbody>
</table>

* AA = African American; AC = African Caribbean; C = Caucasian; decomp = decompressive laminectomy; decomp & lam = decompressive laminectomy and laminoplasty; FPL = foraminotomy/partial laminectomy; J = Japanese; ND = not discussed; neuro improve = neurological improvement.
al. performed foraminotomy or an extended partial laminectomy and resection of the ossified yellow ligaments in their series. We have performed multilevel laminectomies and removal of the ossified enlarged yellow ligaments in combination with perioperative intravenous corticosteroid administration in our patient and have achieved temporary neurological improvement.

Analysis of the postoperative neurological outcomes in patients with this disease reveals that neurological improvement was demonstrated in most cases, albeit with different follow-up periods (Table 1). Ascending narrowing at other thoracic levels due to spontaneous progression was observed by Yonenobu and associates and in our case, and late deterioration after decompression has also been reported. Therefore, the long-term prognosis is probably poor.

References


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