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흉추 척수증을 유발한 황색인대 골화증의 골절 - 증례 보고 -

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Fracture of Ossification of the Yellow Ligament Causing Thoracic Myelopathy - A Case Report -

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– Abstract –

Ossification of the yellow ligament (OYL) is a rare disease that causes myeloradiculopathy at the thoracic spine. The advances in radiographic imaging techniques have made a diagnosis of this pathological entity relatively simple. Surgical decompression and excision of the OYL is the treatment of choice if there are neurological complications from the OYL. We describe a 50-year-old male with thoracic myelopathy caused by a fractured OYL at T10-11, and suggest possible mechanism of the fracture of the OYL at the thoracolumbar spine.

Key Words: Thoracic myelopathy, Yellow ligament, Ossification, Fracture

Ossification of the yellow ligament (OYL) of the thoracic spine is relatively rare cause of thoracic myelopathy. Since Yamaguchi et al[1] first described OYL inducing thoracic myelopathy, only 40 cases have been reported[2,3]. In contrast to most of the cases reported, we experienced a patient with myelopathy caused by a fractured fragment from the OYL in the thoracolumbar spine. The myelopathic symptoms suddenly aggravated after hyperflexion of the back. The patient underwent decompressive laminectomy with removal of the fracture fragment and OYL. Satisfactory neurological recovery was achieved after surgery.

Case Reports

A 50-year-old man presented with a 3-month history of paresthesia and weakness of both lower extremities. He described that his neurological symptoms developed after hyperflexion of his trunk forcefully 6 weeks ago during exercise. The neurological examination revealed spastic paraparesis, an absence of abdominal reflexes and hypoesthesia below T10. The bladder and bowel functions were normal. The Japanese orthopedic association (JOA) score for the assessment of myelopathy was 9.

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A plain radiograph of the thoracic and lumbar spine showed spondylotic changes with multi-level disc space narrowing (Fig. 1). Computerized tomography (CT) revealed multiple ossification of the yellow ligament including the T2-3, T3-4, T9-10 and T10-11. A broken and displaced fragment of the OYL at the T10-11 level was also revealed (Fig. 2). Magnetic resonance imaging (MRI) of the thoracic spine showed a round extradural mass at the T10-11 level that compressed the spinal cord posteriorly. The lesions showed inhomogenously hypointense signal on the T1-weighted images and more hypointense signal in the T2-weighted images. In addition, a signal change in the spinal cord, which located at the T10-11 level and meant spinal cord damage, was revealed in the T2-weighted images (Fig. 3).

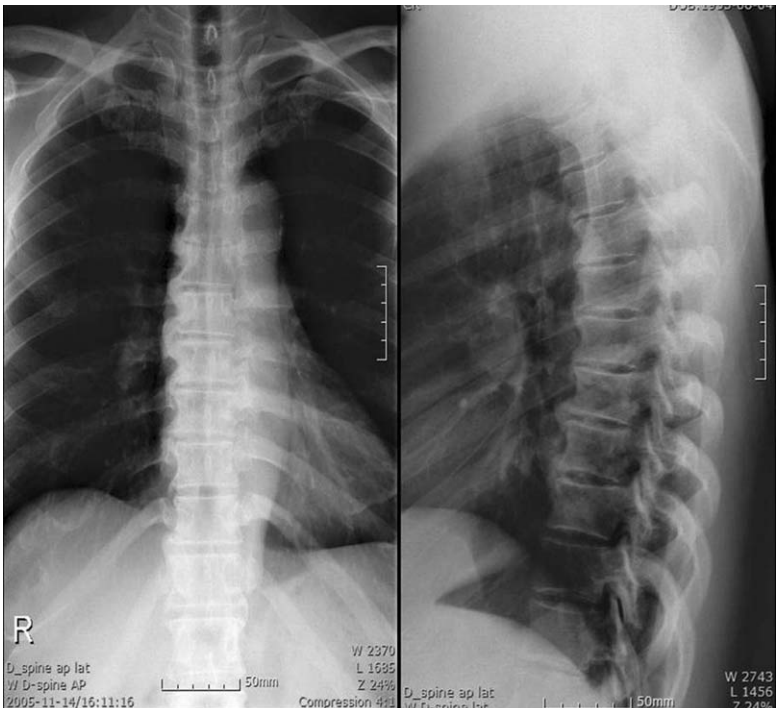

Fig. 1. A plain radiograph of the thoracic spine shows multiple spurs formation with narrowed disc spaces predominantly on the right side.

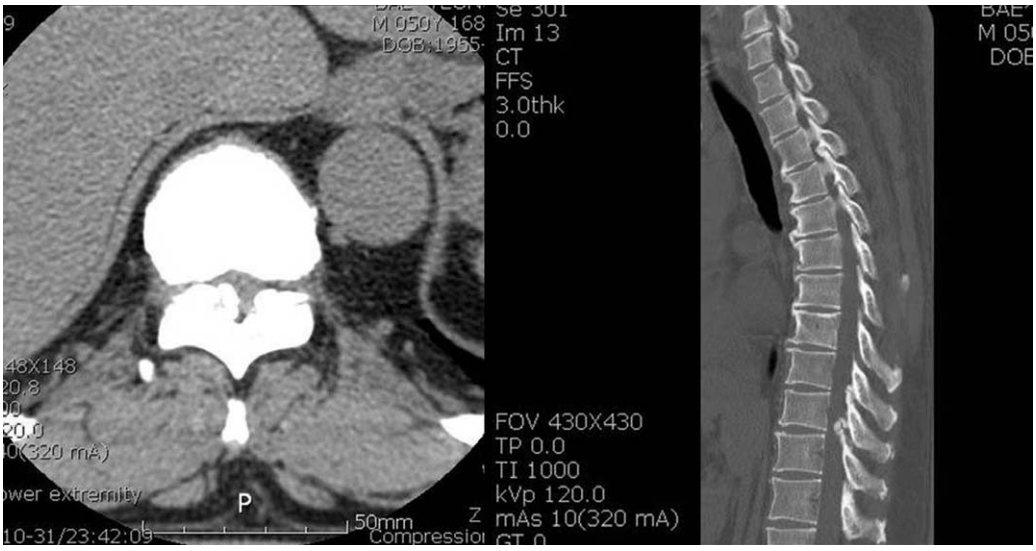

Fig. 2. CT scan shows multiple ossification of the yellow ligament at T10-11. The axial scan shows a broken and displaced fragment of OYL at the same level.

A T10-11 decompressive lamincetomy using a high-speed burr was performed to remove the ossified ligamentum flavum. After removing the OYL, a 0.6×0.5 cm-sized fragment that was compressing the thecal sac was removed. The mass was easily removed with a pituitary forcep without the severe adhesion on the dural sac. The gross findings revealed it to be an irregular shaped hard mass. The histological examination showed lamellated bone, woven bone and enchondral ossification (Fig. 4).

After surgery, the patient's neurological symptoms were improved rapidly. The JOA score was 15 at the final follow-up in June 2006.

Discussion

OYL at the lower thoracic level is often asymptomatic, and the underlying etiology and pathogenesis of OYL are

Fig. 3. Magnetic resonances imaging of the thoracic spine shows a round extradural mass at the T10~11 level. The lesions are inhomogenously hypointense signal on the T1-weighted image (**A**) and more hypointense signal on the T2-weighted image (**B, C**). In addition, signal change in the spinal cord was revealed in the T2-weighted image.

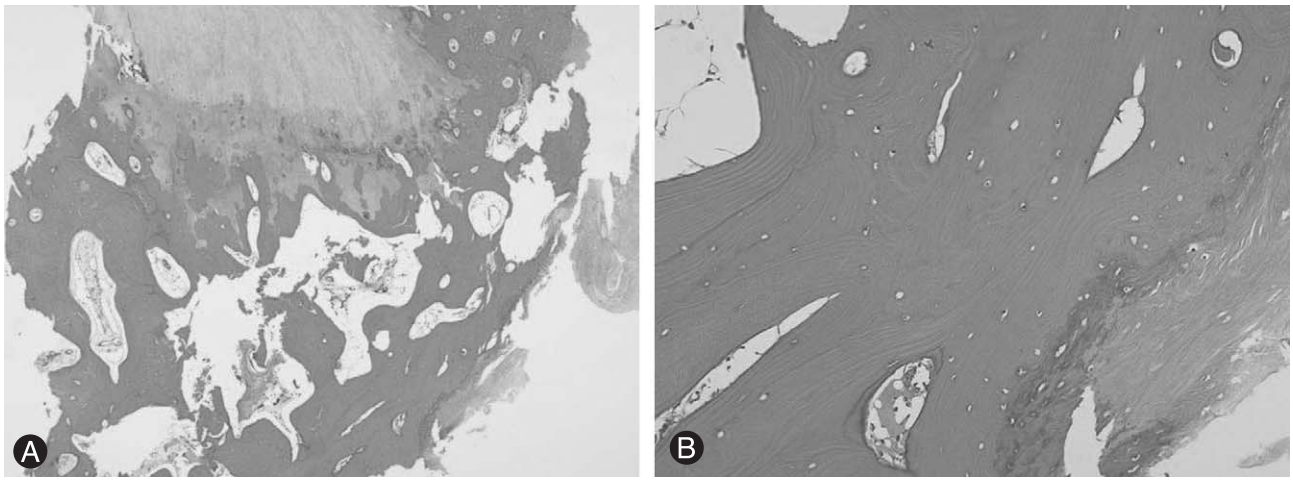

Fig. 4 Histopathologic examination of the excised fragment shows lamellated and woven bone. The enchondral ossification was also identified. (Hematoxylin-Eosin, ×40)

mostly unknown. Repeated mechanical stress of the lower thoracic and the thoracolumbar spine has been reported to promote hypertrophy and ossification of the yellow ligament[4]. The most symptomatic OYL is likely to occur at the lower thoracic levels[5]. However, the upper and mid-thoracic levels are not exempt. A sense of tightness of the trunk, pain on the thoracic area and a stiff back are common complaints. Neurological symptoms likely to develop progressively include gait disturbances, spasticity of the lower extremities, bladder involvement and sexual dysfunctions. In some cases, the sudden onset neurological symptoms were related to minor trauma, particularly hyperextension. Omoyola et al[6] suggested that hyperextension of the spine could cause the forward displacement of the calcified ligament and aggravate the neurological symptoms. In contrast, the neurological symptoms in our case developed after hyperflexion of his back during exercise. The mechanism of the fracture of OYL in the present case was unclear, but the site of the lesion would be responsible for the OYL fracture. The T11 and T12 level allows more motion compared with the higher thoracic spine which is fixed to the ribs[7]. As we could observe a disruption of the posterior ligament complex of the thoracolumbar spine at the flexion- distraction injury, hyperflexion of the thoracolumbar spine might lead to distraction forces on the yellow ligament and even a fracture of the OYL in the stiff spine.

The involvement of the dura by the OYL is an important factor to consider during surgery. In the present case, the fragment had not adhered to the dura and was easily removed with pituitary forceps. Decompressive lamincetomy and excision of the OYL may still be the best surgical option available. Nishiura et al[5] recommended early surgical intervention by a partial lamincetomy and foraminotomy because the OYL is usually already quite large at the time of detection in the patients with motor weakness. Laminoplasty, a limited foraminotomy and an extensive laminectomy have also been remommended[3,5,8]. The outcomes after surgery have been reported to be different in the literature and known to be related to several factors such as the duration of symptoms, age, gender and cord changes on the imaging studies[9,10]. In the present case, a decompressive laminectomy was performed using a high-speed burr to decompress and excise the OYL and a round fragment was removed.

We report a very rare case of a middle-aged male with a fracture of the OYL presenting with neurological deficits and hypothesize the possible mechanism for the fracture of the OYL resulting in myelopathy at the thoracolumbar spine.

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국 문 초 록

흉추의 황색인대 골화증은 드문 질환으로 척수 신경증의 증세를 유발할 수 있는 질환이다. 방사선 검사의 발달로 진단이 어렵지 않으나 신경학적인 증세가 합병되는 경우에는 골화된 황색인대의 제거를 통한 감압이 치료의 선택이 된다. 저자들은 50세 남자에서 발생한 제 10~11 흉추 황색인대 골화증 및 골편의 골절에 의한 척수증을 경험하였기에 황색인대 골화증의 골절의 기전을 제시하며 문헌고찰과 함께 보고한다.

색인단어: 흉추 척수증, 황색인대, 골화증, 골절

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