Introduction

Thoracic spondylotic myelopathy is a relatively rare disease,\(^1\,2\) in which thoracic cord compression causes spinal symptoms, such as spastic paresis and numbness of the lower limbs and bladder and bowel irregularities.\(^3\) The causes of thoracic spondylotic myelopathy include ossification of the posterior longitudinal ligament (OPLL), ossification of the yellow ligament (OYL), degenerative thoracic spondylotic myelopathy, and disk herniation.\(^4\) Thoracic spinal cord compression due to OPLL often occurs in the upper thoracic spine, whereas that due to herniation and OYL often occurs in the lower thoracic spine. In addition, it is reported that patients with diffuse idiopathic skeletal hyperostosis (DISH), in which ossification of the anterior longitudinal ligament causes osseous bridging of vertebral bodies resulting in ankylosis,\(^5\) are likely to develop thoracic spondylotic myelopathy because of degeneration due to mechanical stress between the adjacent vertebral bone bridges.\(^6\)

Thoracic spondylotic myelopathy is treated by decompression of the compressed spinal cord or a combination of decompression and fusion. Because the surgical outcome of this condition is poorer than that of cervical myelopathy,\(^7\) early detection and treatment are important. We herein present a case of degenerative thoracic spondylotic myelopathy between vertebrae fused by anterior fixation in the lower cervical spine and vertebrae fused by DISH in the upper thoracic spine. The patient subsequently developed lower limb paresis and underwent decompression and percutaneous pedicle screw fixation.
pedicle screw (PPS) fixation, which produced favorable therapeutic outcomes.

Case

A 74-year-old man presented with the chief complaints of gait disturbance due to bilateral lower limb paresis and sensory disturbances of the trunk and both lower limbs. He had a history of undergoing anterior fixation for cervical disk herniation approximately 20 years earlier. For around 2 months before surgery, the area from the right side of the lower back to the posterior side of the right lower leg was numb without any triggers, and walking was progressively difficult. Approximately 2 weeks before surgery, the patient had a fall and was unable to walk. He was diagnosed with cauda equina syndrome due to lumbar spinal stenosis at a nearby hospital. Subsequently, he was referred and admitted to our hospital.

On admission, tactile sensation and thermal nociception were lost in the right lower limb, and hypoesthesia was noted in the left foot. Patellar tendon reflex (PTR) and Achilles tendon reflex (ATR) were enhanced and associated with severe spasticity. No muscle weakness was observed in the lower limbs. Computed tomography (CT) revealed C5-C7 anterior fixation performed 10 years earlier and interbody fusion of C5-C7 (Figure 1(a)) and T2-T7 (Figure 1(b)) due to DISH. Osteophyte formation and facet joint degeneration were observed at the T1-T2 level (Figure 1(c)). Magnetic resonance imaging revealed stenosis compressing the spinal cord at the T1-T2 level (Figure 1(d) and (e)). The patient was diagnosed with degenerative thoracic spondylotic myelopathy between the vertebral bodies fused by anterior fixation and DISH. He was prepared for surgery along with bed rest.

Decompression was performed by T1-T2 laminectomy and C7-T4 PPS fixation (two screws fixed superiorly and three inferiorly; Figure 2(a)–(d)). A midline longitudinal incision was placed at the C4-T3 level to expose the subcutaneous area. With minimal detachment of muscles from the spinous processes and vertebral arches of the T1 and T2 vertebrae, these vertebral arches were resected up to the center of the right and left facet joints to decompress the spinal cord. Under X-ray fluorescence guidance, PPSs were inserted into the right and left pedicles of the C7-T4 vertebral bodies, and the screws were connected with rods. The connectors were placed through the site exposed for decompression. On the day after surgery, lower limb strength training and range of motion training were started, and the patient was encouraged for ambulation. Numbness and spasticity in the lower limbs were gradually alleviated. For the daily activities, he could use a wheelchair 2 weeks after surgery and a walker 1 month after surgery. Six months after surgery, although numbness and mild spasticity persisted in the lower limbs, he could walk using a single cane. CT performed 6 months after surgery revealed T1-T2 bridging that was presumably caused by DISH. Interbody fusion was found to have been achieved (Figure 2(e)). Because the patient could not stand up before surgery, standing whole-spine lateral plain radiography was performed after surgery, which revealed that the T1-T2 level was the inflection point between the lordotic cervical spine and the kyphotic thoracic spine. We assumed that greater mechanical stress was more likely to be generated at this level while the patient looked ahead (Figure 2(f)).

Discussion

In this report, we presented a case of degenerative thoracic spondylotic myelopathy that was presumably caused by mechanical stress generated between fused vertebrae C5-T1 due to DISH after anterior fixation and fused vertebrae T2-T7 due to DISH. We performed decompression and PPS fixation and achieved stabilization via interbody bridging and relief of clinical symptoms.

DISH is a pathological condition in which ossification of the ligaments in the entire body progresses over time. In the spine, ossification of the anterior longitudinal ligament causes ankylosis of multiple vertebral bodies. Patients with DISH are prone to vertebral fracture and are likely to develop pseudarthrosis after lumbar spinal fusion and have poor outcomes after decompression surgery for lumbar spinal stenosis. DISH is associated with the diagnosis and therapeutic outcomes of various spinal diseases. In addition, DISH is a risk factor for the development of thoracic spondylotic myelopathy, hence, there are sporadic reports of thoracic spondylotic myelopathy associated with DISH. Because thoracic spondylotic myelopathy does not present with any upper limb symptoms, patients with coexisting lumbar spinal stenosis often have no enhanced tendon reflex of the lower limbs (PTR and ATR), and some patients have only symptoms caused by lumbar spinal stenosis alone, such as numbness and muscular weakness of the lower limbs. Thus, caution should be exercised not to overlook thoracic spondylotic myelopathy. Our patient was misdiagnosed with lumbar spinal stenosis in the first hospital. As for the treatment of thoracic spondylotic myelopathy, decompression alone is often performed when the disease is caused by compression of OYL or other ligaments. In our case, the coexistence of DISH suggested that degeneration was caused by stress concentrated between many fused vertebral bodies. When we realized that only decompression could result in instability or pain, we decided to perform the fixation.

For spinal fusion, it is necessary to open the posterior aspect of the area to be fixed, insert pedicle screws, fix them with a rod, and place a bone graft. Without bone grafting, screws would eventually loosen from the bone.

Although PPS fixation is less invasive than open surgery, it has a disadvantage of not allowing bone grafting. However, in patients with coexisting DISH, bone grafting can be omitted because the stability is likely to be achieved by bridging ossification of the anterior longitudinal ligament. In our
case, PPS fixation was selected, and the bridging between the fused vertebral bodies was achieved without bone grafting.

Decompression and fixation is not a new technique for the treatment of thoracic myelopathy. However, we indicated that even in the relatively rare case of thoracic myelopathy caused by DISH, decompression and PPS fixation without bone grafting may be a treatment option.

The limitation of this study is that because it is a case report, it is unclear whether interbody bridging can be occurred in all DISH patients who undergo PPS fixation. In the future, more cases will need to be studied.

Thus, to conclude, thoracic spondylotic myelopathy occurring at the site between interbody fusion caused by DISH, decompression and PPS fixation in a less invasive manner.
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