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Low Lumbar Spondylotic Myelopathy in a Patient with Low Placed Conus Medullaris —A Case Report—

Kazuhiro HASEGAWA[1], Takao HOMMA[1], Jun SUMITA[2], Hiroyuki OHI[2] and Ikuya YAMAZAKI[2]

[1]Department of Orthopaedic Surgery, Niigata University School of Medicine, Niigata, and [2]Saku General Hospital, Nagano, Japan

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Summary. **A rare pathologic condition causing lumbar myelopathy in an elderly patient is described, and the onset mechanism is discussed based on operative findings. A 69-year-old female presented a slow onset of pain extending from the low back to the right lower extremity, followed by a vesico-rectal disturbance. The diagnosis of low lumbar myelopathy was established by magnetic resonance imaging (MRI) and computed tomography (CT) myelography. At operation the low placed conus medullaris was discolored and the epiconus was severely compressed by the narrowed spinal canal. However, the filum terminale was neither "taut" nor "thickened". Decompression of the narrowed portion improved the symptoms. We consider that the narrowed spinal canal due to spondylotic change played the most important part in the development of the symptom of this elderly patient with the low placed conus medullaris.**

Key words—low placed conus medullaris, lumbar spondylosis, myelopathy, surgical treatment.

INTRODUCTION

Low placed conus medullaris or tethered cord (conus) syndrome has been considered a developmental disease or anomaly of the neuroaxis, usually diagnosed in childhood. Tethered cord syndrome has been recognized as well as in adult or elderly patients.[1-4] In such late onset cases, specific circumstances such as the additional tugging of an already tight conus, narrowing of the spinal canal, or direct trauma to the back or buttocks often precipitates symptomatic onset.[2,4] These speculative causative factors are not based on direct observation. Here, we report a case that presented lumbar myelopathy brought about by a combination of a low placed conus medullaris and spondylosis, which were confirmed by intraoperative observation.

Correspondence: Takao Homma, M.D., Department of Orthopaedic Surgery, Niigata University School of Medicine, Asahimachi-dori 1-757, Niigata 951, Japan.

CASE REPORT

A 69-year-old woman was admitted to our hospital (Saku General Hospital) complaining of chronic pain of the lower back to the right lower extremity, increased urinary frequency and occasional incontinence. She had been symptomatic for about 3 months and did not recognize any causative factor. She had not suffered from low back pain before this episode. She had undergone a hysterectomy for a myoma uteri at 30 years of age and ovariectomy for an ovarian cyst at 34 years of age.

On presentation she had continuous lower back pain extending to the right lower extremity and could not walk more than 500 m without resting. It was difficult for her to maintain sitting and standing positions due to pain in the lower back and the right lower extremity. She did not have any lipoma, hypertrichosis, pigmented nevi, dermal dimples, or dermal sinuses of the buttock. No deformity of her lower extremities was recognized. The straight leg raising test was limited to less than 70° bilaterally. She showed bilateral normal patella reflexia and decreased ankle reflexia with bilateral ankle clonus and positive Babinski sign of the right foot. No apparent sensory deficit, weakness or muscle wasting of her lower extremities was seen. Urinary frequency, occasional incontinence and mild weakness of the anal sphincter were observed.

Plain lumbosacral radiograph showed mild lumbar spondylosis without any spina bifida, widened canal,

Fig. 1. Magnetic resonance imaging (MRI, T2 weight image) of the lumbosacral region shows that a low placed conus medullaris is compressed at the level of L4/5 due to a narrowed spinal canal *(arrow)*.

sacral defect, or other dysplastic findings. Magnetic resonance imaging (MRI) of the lumbar region disclosed the low placed conus medullaris and its compression at the level of L4/5 due to the narrowed spinal canal (Fig. 1). Subsequent myelography with computed tomography (CT) showed that the epiconus was compressed at the L4/5 level secondary to degenerative change of the spine (Fig. 2).

Surgery was performed nearly one year after the onset of her symptoms. The posterior aspect of the laminae was exposed from the level of L3 down to S3 without any finding of spinal dysraphic malformation. Marked osteoarthrotic change was shown at bilateral L4/5 facet joints. The dural tube was severely compressed, entirely due to a hypertrophic ligamentum flavum and osteocartilaginous proliferation especially at the L4/5 level (Fig. 3a). The hypertrophic flavum and bony spur were excised completely, decompressing the dural tube (Fig. 3b). Then the dura mater was split longitudinally at the level of the L5 and S1 vertebra to observe the conus medullaris and the filum terminale. The conus medullaris appeared discolored and hyperemic at the level of the L5 vertebra. The filum terminale looked as slack as the other nerve roots (Fig. 3c). The dura mater was repaired without severance of the filum terminale.

Decompression of the conus medullaris was confirmed by postoperative MRI (Fig. 4). The patient

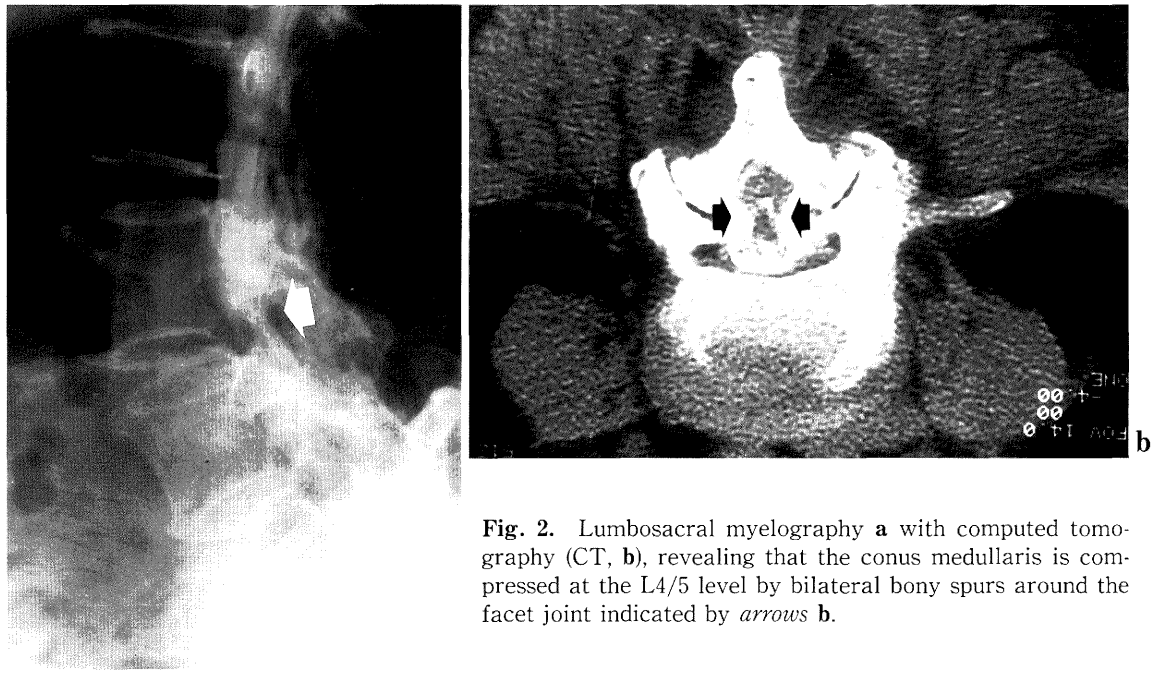

Fig. 2. Lumbosacral myelography **a** with computed tomography (CT, **b**), revealing that the conus medullaris is compressed at the L4/5 level by bilateral bony spurs around the facet joint indicated by *arrows* **b**.

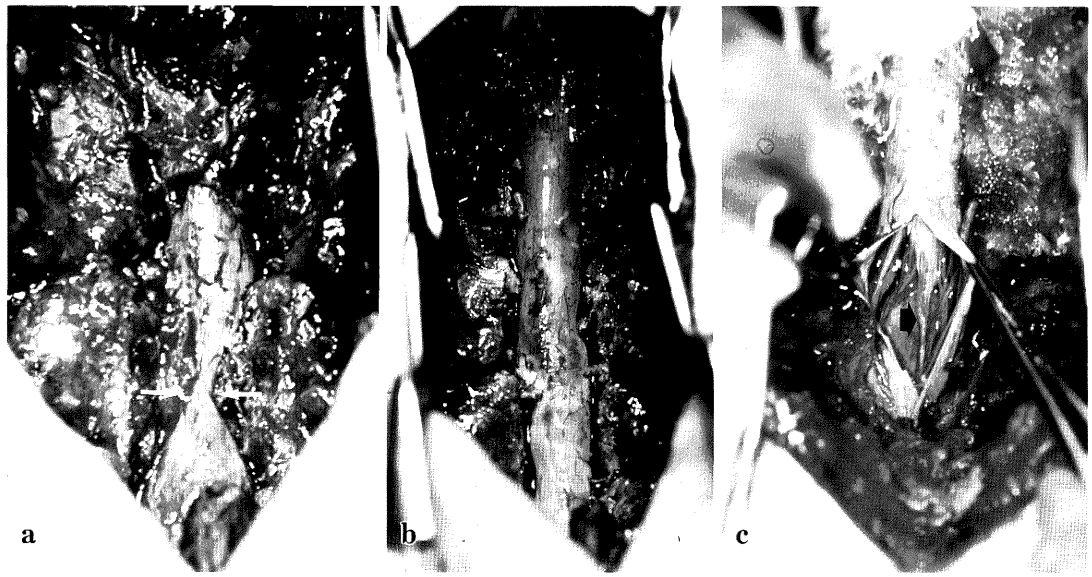

Fig. 3. Intraoperative photographs. **a.** The dural tube is severely compressed entirely due to a hypertrophic ligamentum flavum and bony spur *(arrow)*, especially at the L4/5 level. **b.** The hypertrophic flavum and bony spur were excised completely, decompressing the dural tube. **c.** Then the dura mater was split longitudinally at the level of the L5 and S1 vertebra. The conus medullaris appears discolored and hyperemic, and the filum terminale *(arrow)* looks as slack as the other nerve roots.

Fig. 4. Postoperative MRI (T2 weight image) shows complete decompression of the low placed conus medullaris.

improved steadily. Her activities of daily living ameliorated because of a reduction in pain of the lower back and the lower extremity. Vesicorectal symptoms also partly resolved during the follow-up period of one year after the operation.

DISCUSSION

Some characteristic clinical features were present in our late onset case of low placed conus medullaris. This case presented pain of the lower back and the right lower extremity, and vesicorectal disturbance without any sensory deficit. There was no triggering episode such as trauma to the spine, and also no cutaneous abnormality. All twelve adult cases with tethered conus syndrome described by Kaplan had some abnormal lumbosacral spine radiographic findings.[1] However, in our case, no plain radiographic abnormality was identified except for mild spondylotic change. MRI examination was able to clarify the low placed conus medullaris and its compression at the lower lumbar region. MRI would be the diagnostic imaging examination of second choice in the outpatient clinic in such a patient.

The vertebral level of termination of the immature human spinal cord rapidly ascends to the fourth lumbar vertebra by the 19th week of pregnancy,

followed by a slower ascent thereafter with the cord ending approximately opposite the lower border of the second lumbar vertebra at full term, attaining the "adult" level about 2 months postnatally.[5] The adult cord may terminate anywhere between the last thoracic and the third lumbar vertebra, though the great majority end opposite the first and second lumbar vertebrae.[6-9] From MRI and operative findings in our case, the vertebral level of termination of the spinal cord was opposite the fifth lumbar vertebra. Although a clear definition of low placed conus medullaris has not yet been established, the fifth lumbar vertebral level of termination is apparently abnormal, based on criteria after Nakamura.[10]

Previous authors have pointed out several factors contributing to late onset symptoms: traction or pressure upon the spinal cord and nerve roots by a growing lipoma; repetitive minor trauma; ischemia at the base of a taut filum terminale. A relatively small amount of traction might be sufficient to elongate the spinal cord during growth, although not enough to produce early symptoms.[11-15] Although trauma, traction on the spinal cord by adhesive bands, or expansion of an associated lipoma may play a role in precipitating the symptomatology, acquired spinal stenosis was the major factor responsible for this patient's symptoms in late adulthood.[4]

Although myelography has been advocated as a useful examination to delineate a tight filum terminale,[1,3,4] it is not sufficient to show the tension of the filum terminale in terms of mechanical condition only by means of imaging techniques. We confirmed intraoperatively that the filum terminale was neither "taut" nor "thickened", with compression of the epiconus by the canal stenosis representing the only positive finding in our case.

We conclude that a narrowed spinal canal due to spondylotic change can play the most important part in the development of symptoms in an elderly patient with a low placed conus medullaris.

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