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Extended Cord Injury in Acute Traumatic Central Cord Syndrome after Operation

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Central cord syndrome is the most common incomplete spinal cord injury, and it often occurs in patients suffering from a hyperextension injury. Early surgery has been advocated to improve the outcome and neurological function. However, extended spinal cord injury following anterior cervical discectomy and fusion for central cord syndrome rarely occurs and is seldom reported in the literature. We report a 61-year-old man who suffered from a hyperextension injury, which was diagnosed as traumatic central cord syndrome. Because of the ensuing instability and cord compression, he underwent anterior cervical discectomy and fusion. However, more severe myelopathy was observed postoperatively and extended cord injury was diagnosed based on the expansion of intramedullary high-intensity signal on T2-weighted magnetic resonance images. Patients and surgeons need to be informed of this uncommon but now recognized complication after anterior cervical discectomy and fusion for traumatic central cord syndrome.

Key words: central cord syndrome, anterior cervical discectomy and fusion, magnetic resonance imaging, myelopathy

INTRODUCTION

Central cord syndrome is the most common type of incomplete spinal cord injury, and it is characterized by disproportionate motor weakness in upper limbs compared with the lower limbs, as well as bladder dysfunction and variable sensory deficits below the level of injury[1-3]. The syndrome frequently occurs in patients who have suffered a hyperextension injury of cervical spine and those who may harbor cervical spondylosis or stenosis[2-4]. Early surgical intervention for acute traumatic central cord syndrome has been shown to improve motor function and prevent delayed neurological deterioration[5]. Extended spinal cord injury following cervical discectomy and fusion for acute traumatic central cord syndrome has not previously occurred in our experience. Therefore, we report this case of extended cord injury following surgical intervention for acute traumatic central cord syndrome and discuss the possible causes.

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ASE REPORT

A 61-year-old right-handed man was walking on a downhill path while climbing a mountain. He fell from a height of 2 m, causing a blow to his forehead. Initially, he developed shooting pain and weakness in all extremities. Some of these symptoms subsided after several minutes, but the shooting pain and weakness in his bilateral upper forearms, hands, and fingers continued. He also complained of neck soreness but denied loss of bowel or bladder control. The patient had no history of neck or upper extremity injuries.

The results of the physical examination were unremarkable, except for the multiple abrasion wounds over his face. Neurological examination showed dysesthesia (tingling-like sensation) in bilateral upper forearm, hands, and fingers. The patient had diminished grip strength of grade 1/5, biceps grade 4/5, and triceps grade 4/5 bilaterally. The muscle power in his lower limbs was grade 5/5. Hyperreflexia in the bilateral upper and lower extremities and the presence of Hoffmann reflex were observed. The Babinski sign was also present bilaterally. Bulbocavernosus reflex was present. Urinary retention was observed but anal tone was intact.

Radiography of cervical spine showed a narrowing

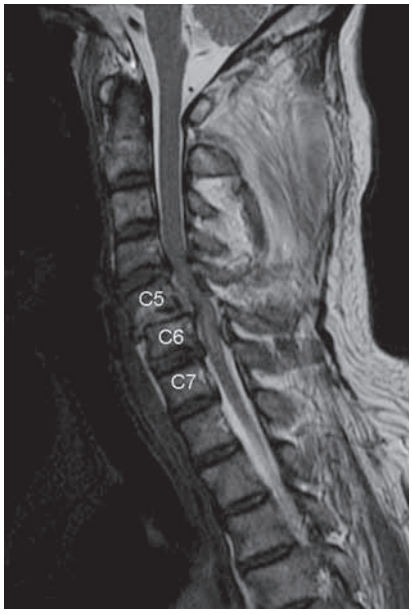

Fig.1. The preoperative T2-weighted magnetic resonance images of cervical spine showed subluxation and cord compression at the C5-6 level. The protruding discs and hypertrophy of ligamentum flavum at the C4-5 and C6-7 level was also observed. The high intensity signal was showed in the prevertebral region from C6 to C7 and in the intramedullary region at the C6 level.

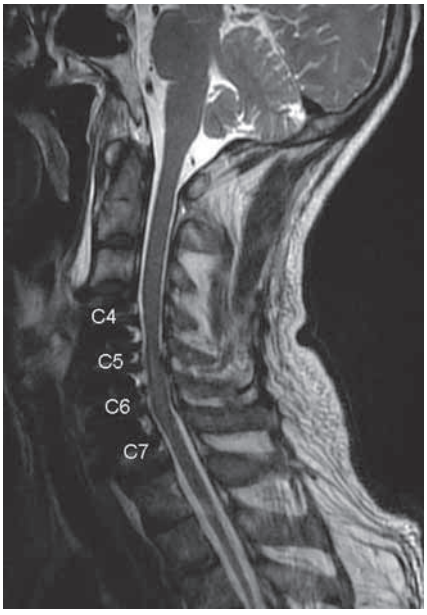

Fig. 2. The postoperative T2-weighted magnetic resonance images of cervical spine showed restoration of the alignment of cervical spine. The high intensity signal of intramedullary region was extended to C5 level.

disc space and subluxation at the C5-6 level. Magnetic resonance imaging (MRI) of the cervical spine revealed a fracture through the C5/6 disc space with anterior subluxation between C5 and C6. Herniated discs between C4 and C5, C5 and C6, and C6 and C7 compressed the spinal cord, which appeared hyperintense at the C5-6 level in T2-weighted images (Fig. 1). High-dose methylprednisolone was administered based on the protocol of the second National Acute Spinal Cord Injury Study, which was given as a bolus dose of 30 mg/kg over 15 min followed by a continuous infusion of 5.4 mg/kg/h for 47 h. The dysesthesia improved and was observed only in the bilateral hands and fingers. The Babinski and Hoffmann signs disappeared. Muscle strength of the upper extremities did not recover however. Because of the instability of the cervical spine, the patient underwent anterior cervical decompression and fusion surgery from C4 to C7 5 days after admission.

During the operation, no obvious manipulation of the cord or mobilization of spine was noted. Postoperatively, rigidity of lower extremities was observed. Neurological examination showed dysesthesia (especially numbness) below the level of C4. Muscle strength was grade 3/5 in the bilateral lower extremities, grip grade 0/5, biceps grade 3/5, and triceps grade 3/5 bilaterally. Hyperreflexia was found in the bilateral upper and lower extremities. Reappearance of the Babinski sign was noted bilaterally. Radiography of the cervical spine showed no displacement of cages or plate. The follow-up T2-weighted MRI of spine revealed extended cord edema to the C5 level (Fig. 2). Because of the diagnosis of extended spinal cord injury high-dose methylprednisolone was administered again as a bolus dose of 30 mg/kg over 15 min followed by a continuous infusion of 5.4 mg/kg/h for 23 h.

One month after surgery, his muscle strength increased in the four extremities (grip grade 2/5, biceps grade 4/5, triceps grade 4/5, and grade 4/5 in lower extremities, bilaterally) and he could walk with assistance. Numbness below the level of C4 and urinary incontinence were still present.

DISCUSSION

Traumatic central cord syndrome is the most common type of spinal cord injury and patients with this syndrome usually have a better outcome than

those with other incomplete cord injuries[4]. Because of injury to the corticospinal tract, the typical clinical presentation of central cord syndrome is characterized by disproportionately more weakness of the upper limbs than of the lower extremities, bladder dysfunction, and varying sensory loss below the injured level[2]. The syndrome commonly develops during a hyperextension injury in patients with cervical spondylosis, spinal canal stenosis, anterior osteophytes, or hypertrophy of ligamentum flavum1. For diagnosis of central cord syndrome, MRI of cervical spine is superior to computed tomography scanning because it provides noninvasive, accurate imaging of soft tissue including muscle, disc, ligament, and spinal cord[4]. The intramedullary hyperintensity seen on T2-weighted images is a classic presentation in patients with traumatic central cord syndrome[6].

Many factors have been shown to affect the neurological and functional outcomes of patients with traumatic central cord syndrome, including the timing of the injury, anteroposterior diameter of spinal canal on computed tomography, age older than 65 years, instability of the cervical spine, prevertebral hyperintensity and intramedullary high signal intensity on T2-weighted images, and cord compression[3,4,6,7]. Because most patients improve spontaneously, conservative treatment is currently considered the standard strategy in the management of these kinds of patients[4,5]. However, the longer-duration sequelae including neurological deficits in upper extremities or urinary and bowel incontinence resulting in the poor quality of daily life, and the natural history of traumatic central cord syndrome, which often proceeds with an initial improvement of neurological function followed by a plateau in recovery and then by late deterioration, were also observed in patients who were conservatively managed[8]. Early surgical intervention, within 2 weeks of injury, for decompression and stabilization of the cervical spine has been advocated to improve motor and sensory functions and shorten the hospitalization and rehabilitation period[2-4,8]. Although the presence of prevertebral hyperintensity and intramedullary high signal intensity on T2-weighted images, instability, and cord compression have been shown to influence the neurological outcome[6], extended spinal cord injury following anterior cervical discectomy and fusion for traumatic central cord syndrome has rarely been reported in the literature.

In general, extended cord injuries have been demonstrated in either anterior (discectomy) or posterior decompression (laminoplasty or laminectomy) for cervical disease[9,12]. The change of signal intensity on MRI is considered by many authors to be correlated with clinical outcome[10,13]. Hyperintensity on T2-weighted images indicates edema, inflammation, ischemia, gliosis, or myelomalacia and is associated with better prognosis. In contrast, hypointensity on T1-weighted images indicates irreversible spinal cord damage and poor prognosis and portends cystic necrosis or secondary syrinx. In a cohort study of 114 patients treated by laminoplasty, Seichi et al. reported that only 7 patients (6.1%) showed postoperative expansion of intramedullary high intensity areas on T2-weighted images[12]. Only 4 of these 7 patients developed a motor-dominant type of paresis of the unilateral upper extremity, and the other 3 patients did not develop any postoperative paresis. None of these patients showed a similar postoperative presentation to our patient. Although the mechanism and cause of expanded cord edema have not been precisely defined[5], it is considered a devastating complication resulting from direct iatrogenic injury related to the operative technique, anatomical alterations, cellular mediators, or biochemical mediators[12]. Other authors have hypothesized that the expansion of the intramedullary lesion may be attributable to reperfusion injury rather than to iatrogenic cord injury[11,12].

In our case, conservative treatment with a high dose of methylprednisolone was the initial course of action in the acute stage. In our experience, no patient with traumatic central cord syndrome or cervical spondylotic myelopathy developed this similar presentation after surgery. Although the spinal cord was not injured directly during operation, iatrogenic cord injury could not be excluded because no intraoperative neurophysiological monitoring was performed[14]. Based on our clinical experience, reperfusion injury was highly likely; however, other potential mediators such as manipulation or compression of spinal cord, ischemia, or vasospasm may have contributed to this devastating complication. The mechanism of injury should be determined in the further studies.

In conclusion, although early surgical intervention improves the functional outcome of patients with traumatic central cord syndrome, this devastating complication remaina a challenge for surgeons. Intraoperative neurophysiological monitoring may be helpful in detecting intraoperative iatrogenic cord injury. Patients and surgeons need to be informed about this uncommon but recognized complication after anterior cervical discectomy and fusion for traumatic central cord syndrome.

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