Cervical myelopathy due to single level prolapsed disc and spondylosis: a comparative study on outcome between two groups

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Abstract This article describes a retrospective study on myelopathy, induced by monosegmental prolapsed disc and spondylosis. To assess pre- and postoperative clinical and radiological findings related to myelopathy, and factors influencing the outcome, 20 disc herniation (group A) and 11 spondylosis patients (group B) were studied. Average duration of myelopathy in groups A and B were 3 and 8.7 months, respectively. Anterior decompression and fusion were performed. Pre- and postoperative clinical and radiological findings and outcomes were assessed. Average preoperative disc heights were 85.9% of normal in group A and 72.7% in group B. Average anteroposterior canal diameter and Pavlov ratio at diseased level were 13.9 mm and 0.81 in group A, respectively, and 12.1 mm and 0.78 in group B. Five group A (25.0%) and four group B cases (36.4%) had radiculopathy. Cord compressions among 20 group A patients were median in seven and paramedian in 13. In the 11 group B patients, nine were median and two were paramedian. High signal intensity was observed in 19 group A and ten group B patients. Postoperative regression of T₂-weighted high signal intensity in 14 group A (73.7%) and two group B patients (20.0%) was observed. Preoperative JOA scores in groups A and B were 10.3 and 12.8, respectively, which became 66.2

and 22.5 postoperatively. Neurological recovery was poorer in group B than in group A. Outcome was influenced by chronicity of myelopathy.

Introduction

Cervical myelopathy is caused by various diseases [1–6]. If ossification of posterior longitudinal ligament and ligamentum flavum is excluded, it is mostly caused by degenerative disc diseases such as pure disc herniation and spondylosis [1–6]. When the disc is degenerated, the disc loses its height, which is subsequently followed by a loss of lordosis, segmental instability, spur formation due to arthrosis of Lusk's and facet joints and thickening of ligamentum flavum. The stenosis secondary to those degenerative changes produces myelopathy. At the early stage of disc degeneration, myelopathy develops more frequently due to soft disc herniation, while in its late stage, myelopathy develops due to transverse spondylotic bar, a thickened ligamentum flavum and segmental instability [2–4].

The authors in this series classified patients with monosegmental myelopathy into two groups: pure soft disc herniation (group A) and spondylosis (group B) groups. They studied the pre- and postoperative clinical and radiological findings, neurological status, and overall outcome by Japanese Orthopedic Association (JOA) scores.

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Material and methods

Subject materials

Among the patients with cervical myelopathy, 31 patients with monosegmental cervical diseases were subjects of this

study. Patients were treated at the Spine Center between August 2002 and June 2006, and were followed up for one year postoperatively. The 31 patients were placed into two groups: so-called soft disc herniation in 20 patients as group A, and hard disc lesion (spondylotic bar) in 11 patients as group B (Figs. 1 and 2).

The patients' average age was 53.3 years (ranging from 28 to 65 years). The average ages of group A and B patients were 47.7 (range 28–53) and 55 (range 43–65) years, respectively. There was a total of 22 males and nine females. In the group A patients, 15 were males and five females, while in the group B patients, seven were males and four were females.

The diseased segments were localised to C₃₋₄, C₄₋₅, C₅₋₆ and C₆₋₇ as follows: two patients (two soft disc / no hard disc) at C₃₋₄, ten patients (seven soft disc / three hard disc) at C₄₋₅, 18 patients (11 soft disc / seven hard disc) at C₅₋₆, and one (one soft disc / no hard disc) at C₆₋₇.

The duration of preoperative clinical symptoms in groups A and B were 3 and 8.7 months, respectively ($P=0.026$).

Methods

All patients were examined preoperatively and assessed by plain cervical radiographs, CT, MRI, and JOA scores. On plain radiographs, disc height of the involved segment, anteroposterior canal diameter and Pavlov ratio were measured by CT and MRI spondylotic bar, central canal and foraminal stenosis, and cord signals were examined. The average follow-up time was 24.2 months (12–38 months).

To assess the neurological outcome, pre- and postoperative JOA scores were compared. For statistical analysis, Student's *t*-test and one-way analysis of variance (ANOVA) were carried out by using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA).

Results

There was no significant difference in gender ratio between the two groups ($P=0.521$). Group A patients were significantly younger than the group B ($P=0.031$) patients. The durations of clinical symptoms in group A and B patients were three and 8.7 months ($P=0.026$), respectively.

Four group A patients and one of the group B patients had a history of neck trauma ($P=0.429$). Five group A patients had radiculomyelopathy (25.0%), while four group B patients (36.4%) had radiculomyelopathy ($P=0.521$).

The levels of the lesion were at C₃₋₄ in two patients (A/B: 2/0), C₄₋₅ in ten patients (A/B: 7/3), C₅₋₆ in 18 patients (A/B: 11/7) and C₆₋₇ in one patient (A/B: 1/0). There were no statistically significant differences in the levels of the lesions in the two groups; though the case numbers in group B were half of group A.

The preoperative average disc height on plain radiographs was 81.2% of the normal disc height: 85.9% in group A and 72.7% in group B ($P=0.008$). Canal anteroposterior diagonal diameters of groups A and B were 13.9 mm and 12.1 mm ($P=0.022$), and Pavlov ratio in groups A and B were 0.81 and 0.78, respectively.

The sites of cord compression on MRI were central in seven group A and nine group B patients, and paracentral in 13 group A and two group B patients. Paracentral lesions were significantly higher in group A ($P=0.011$) (Figs. 1 and 2).

Increased cord signal intensity (ISI) on T₂-weighted MR images was found preoperatively in 19 out of 20 group A patients, which disappeared postoperatively in 14 patients (73.7%). ISI in only two (20.0%) out of the ten group B patients disappeared ($P=0.009$) (Fig. 3) despite the similar size of the signal intensity in the two groups.

Preoperative JOA scores in groups A and B were 10.3 and 12.8 ($P=0.043$), respectively, which became 66.2 and 22.5 at final assessment ($P=0.000$).

Fig. 1 Cervical myelopathy due to soft disc on axial view of T2 WI MRI and CT

Fig. 2 Cervical myelopathy due to spondylotic bar on view of T2 WI MRI and CT

Discussion

Cervical compression myelopathy is known to arise from various causes [1–6]. The common causes are cervical disc herniation, spondylotic bar and ossification of posterior longitudinal ligament. However, most of these were more frequently found in patients with congenital narrow spinal canal. Soft cervical disc herniation is found more often in younger patients, with minimal disc degeneration [1–5], while spondylotic myelopathy is found more often in older individuals, with advanced degenerative changes in disc, facet and Lusk's joints and thickened ligamentum flavum, and also in those with or without segmental instability [3].

Group A patients had higher rates of a history of trauma on the neck than group B patients. However, it was not clear whether the trauma was the primary cause of disc

herniation or whether it worsened the clinical symptoms of a previously herniated disc. Statistically, in the two groups in this study there was no significant difference in causation of myelopathy from trauma. Radiculopathy was associated more frequently with the spondylotic myelopathy group than the soft disc herniation myelopathy group [5], because the spondylotic myelopathy patients had secondary degenerative foraminal stenosis more often than the soft disc herniation group, and also infrequently associated retrolisthesis, which made the neural canal and foramen narrower [7–9].

In our series, the patients' age imposed no direct effect on the surgical outcome. The lack of a posterior surgery group as a control in this series can be listed as a limitation.

Increased signal intensity (ISI) of the spinal cord on T₂-weighted MRI and decreased signal intensity on T₁-weighted MRI are well-known changes considered to

Fig. 3 Cervical myelopathy due to soft disc on sagittal view of T₂ WI preoperative and postoperative MRI

reflect various intramedullary lesions [10, 11]. ISI is often seen in patients with cervical compressive myelopathy. As yet, a relationship between the degree of ISI in the cord, clinical symptoms and surgical outcome has not been well studied, though the ISI in the cord has been known to be one predictor of poor prognosis.

Some authors have developed grading scales for ISI into four grades and others into three grades. They looked for a relationship between ISI, clinical symptoms, patients' age, duration of disease, and surgical outcome [10].

In our series, discovery rates of preoperative ISI in two groups were similarly high: 19 (95%) in 20 group A patients and ten (90.1%) in 11 group B patients. However, intense (bright) cord signal postoperatively disappeared in 14 (73.7%) out of 19 group A patients, while in 11 group B patients ISI disappeared only in two (20.0%) out of ten patients. Thus, it is speculated that the regression of increased cord signals after decompression might be mild ISI, suggesting cord oedema. Where the non-reversible increased signal was intense it was an indication that the scarred cord lesion could not recuperate. Thereby, it is interpreted that an irreversible cord signal is a predictor of poor neural outcome. However, there continues to be disagreement as some authors do not support these views.

Unfortunately, as a limitation in this series, patients had no repeated MRI at regular intervals from the early stage of disease until the time of surgery in order to estimate the relationship of duration of disease and presence of high cord signals and to assess the density changes of cord signals from light to bright ISI. Consequently, it is difficult to conclude that ISI is the key predictor for neural prognosis.

Yukawa et al. reported that patients with ISI were significantly older, had a longer duration of disease, and showed less neurological improvement after surgery than patients without ISI. They also concluded that light ISI reflects mild neuropathological alteration in the cord, which reflects a greater recuperative potential, while intense ISI reflects severe alteration with less recuperative potential [11].

Therefore, in the management of compressive myelopathy with or without ISI on MRI, early decompression surgery is thought to be the key factor leading to a better neurological recovery through induction of the recuperative process compared to delayed decompression surgery [12, 13]. Through this series, it is concluded that the final neurological recovery rates depended on the duration of cord compression rather than on the density grade of ISI.

However, Chung et al. reported that the presence of high signal intensity on MRI proved to be of no prognostic importance [14], while Morishita et al. reported that evaluation of the median nerve SSEP was useful for prediction of the prognosis in the surgically treated patients [15].

Preoperative JOA scores in group A were lower than in group B. However, neurological recovery rates based on

JOA score were significantly higher in group A than group B. Some authors have recommended a more detailed neurological recovery assessment, a new flip coin test for upper limb function, and a six-minute walking test to monitor the walking ability of patients. The authors in this case did not carry out these assessments, but assessed the outcome by JOA score only [16, 17].

Hirabayashi et al. assumed that the degree of function of the upper limbs in patients with T2-high intensity lesion revealed more about a segment than about the long tract [18].

It is speculated on the basis of clinical outcome that cord compression by soft and hard tissues presents different clinical and histopathological pictures, which are reflected on MRI as different grades of ISI. Intense ISI is more frequently found in patients with spondylotic myelopathy (CSM) than patients with prolapsed intervertebral discs (PID). Further speculation is that the acute prolapsed disc compresses the cord more widely in front compared with the spondylotic bar, and causes initially more serious clinical symptoms associated with cord oedema. In cases of spondylotic myelopathy, the cord compression is rather gradual. Its presentation of clinical symptoms is mild initially, which becomes progressively worse when the cord is compressed over a longer period of time. Thus, chronic cord compression leaves irreversible cord lesion which is presented on MRI as intense ISI, and consequently, neurological recovery in spondylotic myelopathy patients is poorer than the herniated soft disc-induced myelopathy.

The average canal size in our series was narrower than the normal Koreans' cervical anteroposterior canal diameter of 16–17 mm [7, 19, 20]. Spondylotic myelopathy patients had a slightly narrower canal than the prolapsed disc patients. In these cases, the compressed cord had little or no room posteriorly to shift. Patients with normal, wide spinal canals develop mostly radiculopathy and rarely myelopathy, even in the presence of spondylosis.

As regards to the treatment of cervical myelopathy, there are two surgical options for decompression: anterior and posterior [2–4, 7, 9]. Anterior surgery consists of decompression and/or fusion with or without anterior plate fixation, while posterior surgery consists of decompressive laminectomy and expansive laminoplasty.

In cases with myeloradiculopathy, excision of Lusk's joints and foraminotomy were combined before fusion. In our series, anterior decompression and plate fixation was chosen because all patients had monosegmental lesions.

As a disadvantage of anterior decompression surgery for patients with developmental stenosis, enlargement of the spinal canal by anterior surgery is limited to the surgically repaired segments [7]. Thus, monosegmental anterior cervical decompression and fusion is not indicated for cervical myelopathy with developmental stenosis, particularly in patients with degenerative discs at the level adjacent to fusion.

In selecting surgical approaches, previous authors depended on the nature of the disease and the number of involved levels. In our series, the authors adopted anterior surgery because the lesions were confined to only one segment with intact adjacent discs [7, 13].

Generally, posterior expansive laminoplasty is more widely accepted for multi-level spondylotic myelopathy patients with developmental stenosis. Posterior surgery frequently results in postsurgical axial pain in the neck and shoulders as a disadvantage [7]. Some disadvantages of anterior surgery include higher incidence of restenosis at the adjacent joints, postsurgical swallowing difficulty, respiratory difficulty due to prevertebral haematoma, and recurrent laryngeal nerve and oesophagus injuries. In our series, no patients had hoarseness complications. However, five patients complained of dysphagia of a postpharyngeal nature, possibly due to surgery-related abnormal oesophagus movement.

In all patients, congenital narrow spinal canal was the primary contributing factor inducing the myelopathy, in addition to prolapsed disc and spondylotic bar. Myelopathy due to prolapsed discs had better prognosis than spondylosis in spite of the preoperative presence of a similar degree of ISI in the cord. Surgical outcome was influenced by the chronicity of cord compression. According to Cheung et al. the neurological recovery reached a plateau at six months after decompression surgery. The upper limb function had the best recovery followed by lower limb and sphincter functions [21]. In our series neurological recovery showed a similar trend. Postoperative regression of the high signal intensity in T₂-weighted images was thought to be a predictor of postsurgical prognosis, though there are differing opinions.

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