Original Article

Evaluation of characteristics and surgical outcomes in cervical spondylotic amyotrophy

Hong-Li Wang, Heng-Chao Li, Jian-Yuan Jiang, Fei-Zhou Lū, Wen-Jun Chen, Xiao-Sheng Ma

Abstract

Background: Cervical spondylotic amyotrophy (CSA) is a rare clinical syndrome resulting from cervical spondylosis. Surgical treatment includes anterior cervical decompression and fusion (ACDF), and laminoplasty with or without foraminotomy. Some studies indicate that ACDF is an effective method for treating CSA because anterior decompression with or without medial foraminotomy can completely eliminate anterior and/or anterolateral lesions. We retrospectively evaluated outcome of surgical outcome by anterior cervical decompression and fusion (ACDF).

Materials and Methods: 28 CSA patients, among whom 12 had proximal type CSA and 16 had distal type CSA, treated by ACDF, were evaluated clinicoradiologically. The improvement in atrophic muscle power was assessed by manual muscle testing (MMT) and the recovery rate of the patients was determined on the basis of the Japanese Orthopedic Association (JOA) scores. Patient satisfaction was also examined.

Results: The percentage of patients, who gained 1 or more grades of muscle power improvement, as determined by MMT, was 91.7% for those with proximal type CSA and 37.5% for those with distal type CSA ($P < 0.01$). The JOA score-based recovery rates of patients with proximal type and distal type CSA were 60.8% and 41.8%, respectively ($P < 0.05$). Patient satisfaction was 8.2 for those with proximal type CSA and 6.9 for those with distal type CSA ($P < 0.01$). A correlation was observed among the levels of improvement in muscle power, JOA score based recovery rate, patient satisfaction and course of disease ($P < 0.05$).

Conclusion: ACDF can effectively improve the clinical function of patients with CSA and result in good patient satisfaction despite the surgical outcomes for distal type CSA being inferior to those for proximal type CSA. Course of disease is the fundamental factor that affects the surgical outcomes for CSA. We recommend that patients with CSA undergo surgical intervention as early as possible.

Key words: Amyotrophy, anterior cervical decompression and fusion, cervical spondylosis
MeSH terms: Spine, cervical vertebrae, spondylitis, decompression, amyotrophy

Introduction

Cervical spondylotic amyotrophy (CSA) is a rare clinical syndrome resulting from cervical spondylosis. CSA is characterized by severe muscle atrophy in the upper extremities, with or without significant sensory disturbance and lower extremity symptoms. Reports suggest that <7% of patients with cervical spondylotic myelopathy presented with minimal sensation loss.[1,2] CSA has always been described as unilateral but can occasionally present as bilateral disease.[3]

CSA is classified as either of proximal or distal type, depending on which muscle groups are most predominantly affected. Proximal type CSA is characterized by weakness in deltoids and biceps while the distal type CSA is characterized by weakness in forearms and hand muscles.[1] Although some studies suggested that CSA is caused by selective impingement against the anterior horn (AH) or the ventral nerve root (VNR) of the spinal cord, the pathomechanism of this syndrome remains controversial.[1]

The treatments for CSA include conservative and operative intervention. Surgical treatment includes anterior cervical decompression and fusion (ACDF), and laminoplasty with or without foraminotomy. Some studies indicate that ACDF is an effective method for treating CSA because anterior decompression with or without medial foraminotomy can completely eliminate anterior and/or anterolateral lesions.[4,5]

Department of Orthopedics, Huashan Hospital, Fudan University, Shanghai 200040, China

Address for correspondence: Dr. Jian-Yuan Jiang,
Department of Orthopedics, Huashan Hospital, Fudan University, No. 12 Urumqi Zhong Road, Shanghai 200040, China.
E-mail: jianyuanjiang@163.com

Access this article online	
Quick Response Code:	Website: www.ijoonline.com
	DOI: 10.4103/0019-5413.139875

By contrast, surgical intervention has only been applied to a few cases because differentiating CSA from motor neuron diseases is difficult.[6] In addition, surgical outcomes for this course of treatment have not been fully investigated.

We collected data from a cohort of patients with CSA for retrospective analysis of characteristics and surgical outcomes for the disease.

Materials and Methods

We retrospectively reviewed our institutional database to identify patients who underwent surgical treatment for CSA at the Department of Orthopedics Surgery in our institute between June 2006 and February 2012. 28 patients (18 men and 10 women) were enrolled in this study. We diagnosed the disease as CSA when the following criteria met: (1) presence of cervical spondylosis, (2) presence of unilateral or bilateral severe muscle atrophy of the upper extremities, (3) mild or no sensory deficit in the upper and lower extremities, (4) absence of gait disturbance and (5) either presence or absence of radicular pain of the upper limbs. Hirayama's disease and amyotrophic lateral sclerosis were excluded from the criteria.

Patients were classified into two groups according to the preoperative severity of muscle atrophy in the upper extremities. 12 patients had proximal type CSA (impairment of scapular, deltoid and biceps muscles) and 16 had distal type CSA (impairment of triceps, forearms, and hand muscles); 26 patients had CSA of the unilateral upper extremities (13 left and 13 right) and two patients had CSA of the bilateral upper extremities. Six patients exhibited shoulder girdle pain before motor loss but showed no sensory loss.

Results were assessed based on (1) electromyographic examination (2) radiographic examination (3) manual muscle testing (4) recovery rate based on Japanese Orthopedic Association (JOA) scores and (5) patient satisfaction.

1. Denervation potentials and decreased motor unit potentials were observed in the atrophic muscles by standard needle electromyography (EMG). The thoracic paraspinal muscles or lower limb muscles were normal. No abnormal findings were obtained by the sensory nerve conduction velocity tests for the bilateral median and ulnar nerves. The results imply that lesions are present in the AH or VNR[6]
2. The radiological examination included plain radiographs, two dimensional computed tomography (CT) and high resolution magnetic resonance imaging (MRI). Cervical canal stenosis (<14 mm or disappearance of the subarachnoid space on the sagittal T2-weighted MRI) was identified on the CT and MRI scans.[5,7,8] The compression of the spinal cord from a force acting on the front part was classified on the T2-weighted MRI and intraoperatively confirmed as of medial, paramedial or foraminal type.[5] The high signal intensity zone within the spinal cord parenchyma was identified on the sagittal and axial T2-weighted MRI scans.[7,9] The impingement against the AH and/or VNR at the corresponding level for the most atrophic muscle was determined on the transverse T2-weighted MRI scan.[7] The impingement was comprehensively evaluated in each case by studying the radiographic and electromyographic findings[7,10,11]
3. The pre and postoperative power of the most severely atrophic muscle were assessed by manual muscle testing (MMT). The improvement in muscle power was classified into four grades: "Excellent" or at least two grades of recovery, "good" or at least one grade of recovery, "fair" or less than one grade of recovery, as determined by MMT; and "poor" or no recovery or worsening condition, as revealed by MMT
4. The recovery rate based on JOA scores was used to evaluate the recovery effect of clinical function after surgical treatment. The recovery rate was calculated using the following formula: (Postoperative JOA scores -preoperative JOA scores)/(17 -preoperative JOA scores) × 100%
5. Patient satisfaction was used to subjectively evaluate how satisfied the patients were with the surgical treatment. The score ranged from 0 to 10, representing the worst and best levels of satisfaction, respectively.

These patients were followed up at 1 month, 3 months, 6 months, 9 months and 12 months after surgery. Then, we recorded the muscle power, JOA score and patients satisfaction at the last followup and estimate these indexes. MRI was performed to assess the presence of adjacent segment degeneration.

All patients underwent standard ACDF treatment, which is believed to provide patients with an optimal chance of neurological recovery with fewer complications given the complete elimination of cord-compressing lesions.[5] To expose the dural sac throughout the length of the discectomy and corpectomy, as well as the compressed VNR, we performed extensive decompression, which covered the removal of the disc, vertebral end plate, medial uncinate process, posterior osteophytes, herniated nucleus pulposus and posterior longitudinal ligaments. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Hospital. Written informed consent was obtained from all participants.

Statistical analysis

Statistical analysis was performed using the unpaired *t*-test, Chi-square test, one-factor ANOVA, Pearson's

correlation, and Spearman's rho. $P < 0.05$ was considered as statistically significant. All statistical analyses were conducted using SPSS version 18.0 software (IBM SPSS, Armonk, NY, USA).

Results

The mean age of the cohort was 55.9 years (range 38-69 years). The mean followup period was 26.6 months (range 8-75 months). The mean course of the disease was 12.3 months (range 1-60 months). The bone graft incorporated at final followup in all cases. There was no loosening of graft or dislocation. When CSA presented as proximal type (impairment of scapular, deltoid, and bicep muscles), the biceps reflex could be decreased or normal; and when it presented as distal type (impairment of triceps, forearms, and hand muscles), the triceps reflex could be decreased or normal. All patients were performed cervical MRI in following up after surgery. Only one patient had been seen adjacent segment degeneration without related symptoms.

Table 1 summarizes the differences in the characteristics and surgical outcomes for proximal and distal type CSA, from which we deduced the following CSA characteristics:

Table 1: Difference between the proximal type and distal type

Parameters	Group 1 (proximal type)	Group 2 (distal type)	*P* value
No. of cases	12	16	
Mean age (years)	56.3±10.6	55.6±7.7	NS
Mean course of disease (months)	10.9±10.2	13.3±16.3	NS
Mean followup (months)	18.0±10.3	33.1±17.3	0.013*
Recovery rate of JOAs	0.608±0.175	0.418±0.259	0.029*
Patients' satisfaction	8.2±1.1	6.9±1.0	0.003*
Improvement of muscle power			0.006[&]
Excellent and good (%)	11 (91.7)	6 (37.5)	-
Fair and poor (%)	1 (8.3)	10 (62.5)	-
Responsible levels			0.008[&]
C3-C4+C4-C5 (%)	2 (16.7)	0 (0)	-
C3-C4+C4-C5+C5-C6 (%)	0 (0)	1 (6.3)	-
C4-C5 (%)	3 (25)	0 (0)	-
C4-C5+C5-C6 (%)	6 (50)	2 (12.5)	-
C4-C5+C5-C6+C6-C7 (%)	0 (0)	2 (12.5)	-
C5-C6 (%)	1 (8.3)	2 (12.5)	-
C5-C6+C6-C7 (%)	0 (0)	7 (43.8)	-
C6-C7 (%)	0 (0)	2 (12.5)	-
No. of impingement lesion			NS
AH (%)	6 (50)	11 (68.8)	-
VNR (%)	4 (33.3)	5 (31.3)	-
AH+VNR (%)	2 (16.7)	0 (0)	-
HIZ on T2-weighted MRI (%)	3/12 (25)	6/16 (37.5)	NS
No. of canal stenosis (%)	8/12 (66.7)	11/16 (68.8)	NS

*Unpaired *t*-test, [&]Chi-square test. NS=Not significant, AH=Anterior horn, VNR=Ventral nerve root, HIZ=High intensity zone, MRI=Magnetic resonance imaging, JOAs=Japanese orthopedics association score

The proximal type CSA, the most commonly affected level was C4-5, followed by C5-C6 and C3-C4. For distal type CSA, the most commonly affected level was C5-C6, followed by C6-C7, C4-C5, and C3-C4. The levels frequently responsible for the most severely atrophic muscles were involved at two levels namely; C4-C5 and C5-C6 for proximal type CSA and C5-C6 and C6-C7 for distal type CSA ($P < 0.01$).

The CSA can be caused by impingement against the AH and/or the VNR [Figure 1A]. Impingement against the AH and the VNR was observed in 17 and 9 cases, respectively; impingement against both the AH and the VNR was observed in 2 cases. There were no statistically significant differences in the course of the disease ($P = 0.176$), excellent/good improvement of muscle power ($P = 0.517$), recovery rate based on JOA scores ($P = 0.068$), and patients' satisfaction ($P = 0.214$) between the AH group and VNR group.

A high intensity zone on the T2-weighted MRI in the spinal cord and canal stenosis was observed for both proximal and distal type CSA. However, no statistically significant differences in high intensity zone were found between the two types.

The recovery rates based on JOA scores for proximal and distal type CSA were 60.8% and 41.8%, respectively ($P < 0.05$). Patient satisfaction levels with the treatments for proximal and distal type CSA were 8.2 and 6.9, respectively ($P < 0.01$). The percentage of patients, who gained one or more grades of muscle power improvement, as determined by MMT, was 91.7% for those with proximal type CSA and 37.5% for those with distal type CSA ($P < 0.01$). Therefore, the recovery rate based on JOA scores, patient satisfaction, and improvement in muscle power of patients with proximal type CSA were superior to those of patients with distal type CSA [Figure 1B] despite the mean followup months for the former being less than that for patients with distal type CSA ($P < 0.05$) [Table 1].

Muscle power improvement

The muscle power was rated as "excellent" in six cases (21.4%), "good" in 11 cases (39.3%), "fair" in nine cases (32.1%), and "poor" in two cases (7.1%). There were statistically significant differences in the course of disease, recovery rate based on JOA scores and patient satisfaction among the four grades ($P < 0.01$) [Table 2]. In addition, no statistically significant differences in the high intensity zone on the T2-weighted MRI and in impingement lesions were observed among the four grades. The results indicate that the improvement in atrophic muscle power correlates with the disease course, but not with the high intensity zone on the T2-weighted MRI. Shorter the course of the disease for

Table 2: Difference in characteristics and surgical outcomes according to the improvement of muscle power

Parameters	Excellent (%)	Good (%)	Fair (%)	Poor (%)	*P* value
No. of cases	6 (21.4)	11 (39.3)	9 (32.1)	2 (7.1)	
Mean age (years)	59.5±10.5	57.4±8.2	52.2±8.7	53.5±7.8	NS
Mean course of disease (months)	6.6±4.5	6.8±6.9	16.2±13.8	42.0±25.5	0.001*
Mean followup (years)	19.0±11.4	28.3±19.1	32.0±15.9	16.0±4.2	NS
Recovery rate of JOAs	0.689±0.191	0.582±0.129	0.383±0.205	0	0.000*
Patients' satisfaction	8.3±1.4	7.8±0.8	6.9±1.1	5.5±0.7	0.007*
HIZ on T2-weighted MRI	2 (33.3)	2 (18.2)	4 (44.4)	1 (50)	NS
No. of impingement lesion					NS
AH	3 (50)	7 (63.6)	5 (55.6)	2 (100)	-
VNR	2 (33.3)	4 (36.4)	3 (33.3)	0 (0)	-
AH+VNR	1 (16.7)	0 (0)	1 (11.1)	0 (0)	-

*ANOVA. NS=Not significant, AH=Anterior horn, VNR=Ventral nerve root, HIZ=High intensity zone, MRI=Magnetic resonance imaging, JOAs=Japanese Orthopedics Association score

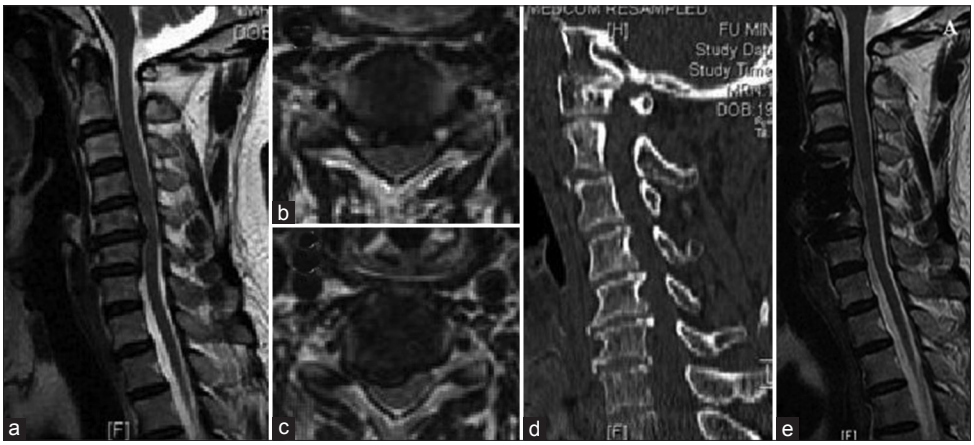

Figure 1A: A 41 year old male patient who presented as proximal type and undergone anterior cervical decompression and fusion (ACDF). (a) The sagittal T2 weighted MRI showed that the spinal cord was compressed at C4-C5 and C5-C6 vertebral levels. (b) Axial T2 weighted MRI demonstrated that the cord was centrally compressed at C4-C5 disc level. (c) Axial T2 weighted MRI demonstrated that impingement against anterior horn at C5-C6 vertebral level. (d) Two dimension CT showed that the presence of cervical canal stenosis (e) Postoperative sagittal T2 weighted magnetic resonance image was obtained 1 year after surgery. It showed that adequate decompression of the cord behind the corpectomy levels with no compression at any other levels

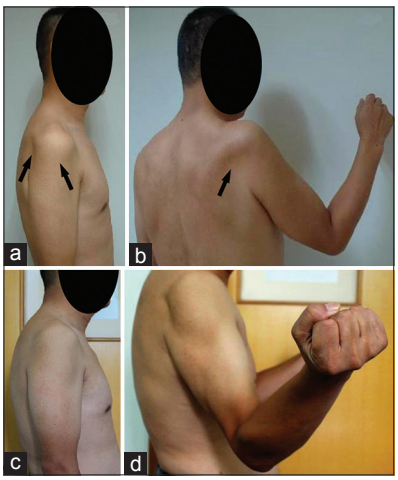

Figure 1B: Clinical photographs of same patient showing (a) the infraspinatus and deltoid atrophy in right upper extremity (arrow). (b) The patient could not fully external rotate and abduct his right shoulder against gravity preoperatively. (c) The appearance of atrophic infraspinatus and deltoid become plump 1 year after ACDF. (d) His right shoulder could be fully abducted and external rotated 1 year after ACDF. Informed consent by the patient for the following photos and pictures

a patient, the better the improvements in atrophic muscle power, recovery rate based on JOA scores and patient satisfaction [Figure 2].

Multiple factor analysis

We found that the high intensity zone on the T2-weighted MRI was associated with the impingement lesions but not with the improvement in muscle power, recovery rate based on JOA scores, or patient satisfaction. Meanwhile, we found a correlation among the improvement in muscle power, recovery rate based on JOA scores patient satisfaction and course of disease ($P < 0.05$). This finding indicates that course of disease is the fundamental factor that affects the prognosis of CSA [Table 3].

Discussion

CSA is a rare clinical syndrome resulting from cervical spondylosis; it is characterized by severe muscle atrophy and weakness in the upper extremities without significant sensory deficit or myelopathy.[2] The affected upper limb

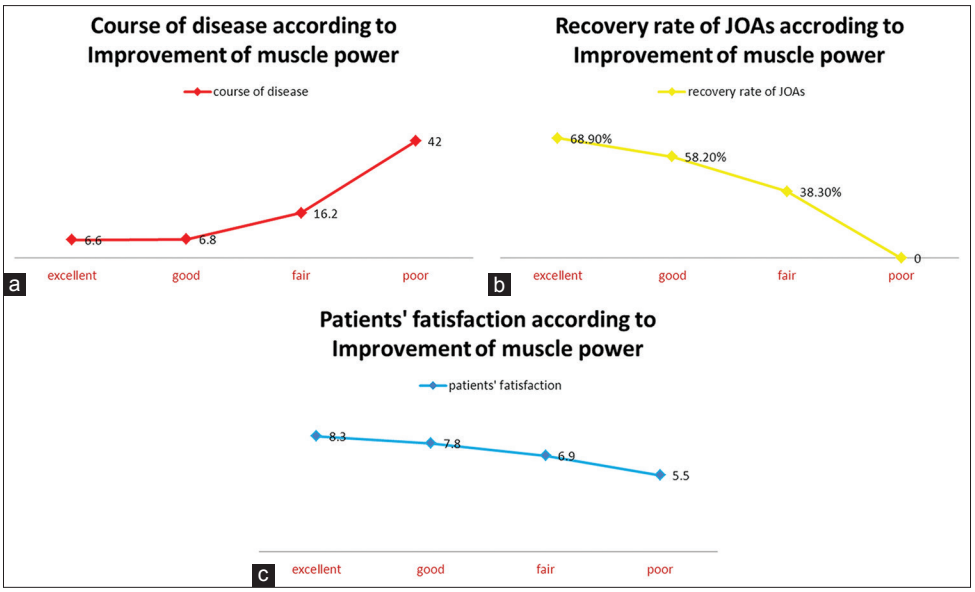

Figure 2: A graph showing the improvement of power muscle was closely correlated with course of disease (a), recovery rate of Japanese Orthopedic Association score (b), and patients' satisfaction (c)

Table 3: Multiple factors correlated with the surgical outcomes

Parameters	HIZ on T2-weighted MRI	Recovery rate of JOAs	Patients' satisfaction	Improvement of muscle power
Impingement lesion	r_s=−0.543 P=0.003[&]	NS	NS	NS
Course of disease	NS	r=−0.662 P=0.000*	r=−0.416 P=0.028*	r_s=−0.391 P=0.040[&]
Patients' satisfaction	NS	r=0.585 P=0.001*	NS	r_s=0.399 P=0.036[&]
Improvement of muscle power	NS	r_s=0.443 P=0.018[&]	NS	NS

*Pearson's correlation, [&]Spearman's rho. NS=Not significant, HIZ=High intensity zone, MRI=Magnetic resonance imaging, JOAs=Japanese Orthopedics Association score

is usually unilateral but can occasionally be bilateral.[3] In this study, two patients presented with bilateral muscle atrophy and six patients presented with shoulder girdle pain, which disappeared after surgery. We assume that the pain originated from the dura mater or the dural sleeve and not from the posterior nerve root.[11]

Debate is ongoing regarding whether the pathomechanism of CSA is impingement against the AH or the VNR. Some researchers attribute the syndrome to the selective intradural compression of the ventral motor roots by posterolateral osteophytes.[1] Others attribute it to a circulatory insufficiency in the territories of the spinal central arteries, as well as to selective damage to the AHs.[1] Fujiwara *et al.*[7] have reported that distal type CSA is regarded as caused by impingement against the AH and not by the impingement against the VNR alone. By contrast, Imajo *et al.*[2,12] proposed that the pathophysiology of CSA is a combination of lesions in the AHs and VNRs. Based on the EMG and T2-weighted MRI results in the present study, we propose that CSA is caused by lesions in either the AH or the VNR.

Several studies reported that the commonly affected vertebral segment in proximal type CSA is C4-C5, whereas that in distal type CSA is C5-C6.[1,5,13] Meanwhile, several researchers demonstrated that CSA may be characterized by multi segmental damage resulting from the loss of AH cells, which is possibly caused by dynamic cord compression through circulatory insufficiency.[7,9,12,14,15] Given that the primary mechanism of compression is more ischemic than compressive, we can assume that minimal spondylotic changes in regions adjacent to the anterior spinal or feeder arteries can result in segmental ischemic events in the AHs.[1,15] In our work, we also demonstrated that the most commonly affected levels were C4-C5 and C5-C6 for proximal and distal type CSA, respectively. Nevertheless, we found that the levels frequently responsible for the most severely atrophic muscles were bi-levels; i.e. C4-C5 and C5-C6 for proximal type CSA and C5-C6 and C6-C7 for distal type CSA. Cervical canal stenosis was observed in 8 out of the 12 (66.7%) patients with proximal type CSA and 11 out of the 16 (68.8%) patients with distal type CSA. Therefore, we attribute the pathophysiology of CSA to the dynamic multi segmental compression of the spinal cord caused by lesions in either the AH or the VNR.

Previous studies demonstrated that anterior decompression and fusion is an effective surgical intervention for CSA. However, the influencing factors for surgical outcomes have not been fully examined. Shinomiya *et al.*[11] revealed that all patients with proximal type CSA improved with anterior

decompression and fusion after conservative treatment without any significant recovery. However, patients with root lesions enjoyed superior outcomes compared with those who had AH lesions. Uchida *et al*.[5] described a similar surgical outcome and indicated that even in patients with proximal type atrophy, a long preoperative period and medial compression of the spinal cord are factors which correlate with poor muscle power improvement.

We demonstrated that the surgical outcomes for proximal type CSA (improvement in muscle power, recovery rate based on JOA scores, and patient satisfaction) are superior to that for distal type CSA. In the study of Fujiwara *et al*.,[7] muscle power improved in 92% of patients with proximal type CSA, whereas the proportion of distal type CSA patients showing muscle power improvement was only 38% after surgery. However, we found no significant differences in the course of disease and high intensity zone on the T2-weighted MRI between the two types. Similarly, Inui *et al*.[16] found no statistical difference in surgical outcomes between disease types and sites of compression. Fujiwara *et al*. attribute this phenomenon to the fact that distal type CSA is characterized by impingement against the AH because the spinal cord, including the AH, is less likely to regenerate compared with the VNR. We found no statistically significant differences in the extent of impingement against the AH between the two CSA types, although that against the AH in distal type CSA was greater than that in proximal type CSA. A possible explanation for this finding is the longer distance from the spinal cord to muscle in distal type CSA than in proximal type CSA. Therefore, muscle power in patients with proximal type CSA considerably improved, leading to improved clinical function and high patient satisfaction.[7]

Despite the findings of previous studies on risk factors related to surgical outcomes for CSA, the subject remains controversial. Researchers have shown that edema, myelomalacia and gliosis involve high signal intensity on T2-weighted MRI and low signal intensity on T1 weighted MRI, suggesting irreversible changes in the spinal cord. This change in signal intensity is closely associated with poor postoperative outcomes for cervical spondylotic myelopathy.[17] Fujiwara *et al*.[7] reported that the surgical outcomes for distal type CSA are inferior to those for proximal type CSA; they are also characterized by fewer number of cases, longer preoperative periods, greater number of stenotic canal levels, and more cases with a T2 high intensity zone. In the current work, we found no correlation between the high intensity zone on the T2 weighted MRI and poor surgical outcomes. Uchida *et al*.[5] also reported no correlation between high intensity signals on MR images before decompressive surgery and poor motor recovery after decompressive surgery. The study confirmed that the high signal intensity changes on MRI scans do not predict surgical outcomes or prognoses. In general, a lengthy period of cord compression is believed to be caused by high intensity signals in the spinal cord.[18] Tauchi *et al*.[19] have reported that changes in intramedullary signal intensity is usually consistent with damage in the central portion of the spinal cord among cervical spondylotic myelopathy patients. However, CSA results from the compression and damage of the AH or the VNR of the spinal cord. This characteristic is perhaps the reason why no correlation was found between signal intensity changes on MRI and poor surgical outcomes.

Furthermore, we found that the high intensity zone on the T2-weighted MRI correlated with the impingement lesion of the AH or the VNR. This finding can be attributes to the AHs located in the terminal territory of the sulcal (central) arteries, which are considered the most vulnerable to the effects of circulatory insufficiency.[1] Wada *et al*.[20] demonstrated that multi segmental (linear) high intensity areas on T2 weighted MRI scans are associated with clinical evidence of extensive AH cell damage and radiographic evidence of gray matter cavitation.

In studies by Uchida, Inui *et al*. and Tauchi *et al*.[5,16,19] it was found that the preoperative period is closely associated with the recovery of muscle power, but they did not describe the relationship in detail. In our study, we not only demonstrated the correlation between course of disease and recovery of muscle power, but also found that is former is the only risk factor correlating with the surgical outcomes for both types of CSA. The shorter the course of diseases, the better the atrophic muscle power and recovery rate based on JOA scores and patient satisfaction. Therefore, we do not recommend conservative therapy for long periods because this approach prevents patients from receiving the best therapeutic opportunities. We suggest that surgical intervention be carried out as early as possible for patients with CSA.

Several limitations of our study must be acknowledged. First, our patient sample was small and therefore insufficient for statistically evaluating the differences in risks of surgical outcomes. Second, the study design was retrospective and no surgical therapeutic method was selected to serve as the control. Nonetheless, we added the JOA scores and patient satisfaction rates to assess the surgical outcomes, including objective and subjective evaluations that were not adopted in previous studies.

Conclusion

ACDF can effectively improve the clinical function of patients with CSA and result in good patient satisfaction despite the surgical outcomes for distal type CSA being

inferior to those for proximal type CSA. Course of disease is the fundamental factor that affects the surgical outcomes for CSA. We recommend that patients with CSA undergo surgical intervention as early as possible.

References

1. Jiang SD, Jiang LS, Dai LY. Cervical spondylotic amyotrophy. Eur Spine J 2011;20:351-7.
2. Imajo Y, Kato Y, Kanchiku T, Suzuki H, Taguchi T. Pathology and prognosis of proximal-type cervical spondylotic amyotrophy: New assessment using compound muscle action potentials of deltoid and biceps brachii muscles. Spine (Phila Pa 1976) 2011;36:E476-81.
3. Gebere-Michael SG, Johnston JC, Metaferia GZ, Wuhib MZ, Fernandez HH. Bilaterally symmetric cervical spondylotic amyotrophy: A novel presentation and review of the literature. J Neurol Sci 2010;290:142-5.
4. Saringer W, Nöbauer I, Reddy M, Tschabitscher M, Horaczek A. Microsurgical anterior cervical foraminotomy (uncoforaminotomy) for unilateral radiculopathy: Clinical results of a new technique. Acta Neurochir (Wien) 2002;144:685-94.
5. Uchida K, Nakajima H, Yayama T, Sato R, Kobayashi S, Kokubo Y, *et al.* Anterior and posterior decompressive surgery for progressive amyotrophy associated with cervical spondylosis: A retrospective study of 51 patients. J Neurosurg Spine 2009;11:330-7.
6. Kaneko K, Taguchi T, Toyoda K, Kato Y, Azuma Y, Kawai S. Distal-type cervical spondylotic amyotrophy: Assessment of pathophysiology from radiological findings on magnetic resonance imaging and epidurally recorded spinal cord responses. Spine (Phila Pa 1976) 2004;29:E185-8.
7. Fujiwara Y, Tanaka N, Fujimoto Y, Nakanishi K, Kamei N, Ochi M. Surgical outcome of posterior decompression for cervical spondylosis with unilateral upper extremity amyotrophy. Spine (Phila Pa 1976) 2006;31:E728-32.
8. Tanaka N, Fujimoto Y, Yasunaga Y, Ochi M. Functional diagnosis using multimodal spinal cord evoked potentials in cervical myelopathy. J Orthop Sci 2005;10:3-7.
9. Shibuya R, Yonenobu K, Yamamoto K, Kuratsu S, Kanazawa M, Onoue K, *et al.* Acute arm paresis with cervical spondylosis: Three case reports. Surg Neurol 2005;63:220-8.
10. Tanaka N, Fujimoto Y, An HS, Ikuta Y, Yasuda M. The anatomic relation among the nerve roots, intervertebral foramina, and intervertebral discs of the cervical spine. Spine (Phila Pa 1976) 2000;25:286-91.
11. Shinomiya K, Komori H, Matsuoka T, Mutoh N, Furuya K. Neuroradiologic and electrophysiologic assessment of cervical spondylotic amyotrophy. Spine (Phila Pa 1976) 1994;19:21-5.
12. Imajo Y, Kato Y, Kanchiku T, Suzuki H, Yoshida Y, Funaba M, *et al.* Prediction of surgical outcome for proximal-type cervical spondylotic amyotrophy novel mode of assessment using compound action potentials of deltoid and biceps brachii and central motor conduction time. Spine (Phila Pa 1976) 2012;37:E1444-9.
13. Zhang JT, Yang da L, Shen Y, Zhang YZ, Wang LF, Ding WY. Anterior decompression in the management of unilateral cervical spondylotic amyotrophy. Orthopedics 2012;35:e1792-7.
14. Srinivasa Rao NV, Rajshekhar V. Distal-type cervical spondylotic amyotrophy: Incidence and outcome after central corpectomy. J Neurosurg Spine 2009;10:374-9.
15. Ahdab R, Créange A, Benaderette S, Lefaucheur JP. Cervical spondylotic amyotrophy presenting as dropped head syndrome. Clin Neurol Neurosurg 2009;111:874-6.
16. Inui Y, Miyamoto H, Sumi M, Uno K. Clinical outcomes and predictive factors relating to prognosis of conservative and surgical treatments for cervical spondylotic amyotrophy. Spine (Phila Pa 1976) 2011;36:794-9.
17. Yagi M, Ninomiya K, Kihara M, Horiuchi Y. Long term surgical outcome and risk factors in patients with cervical myelopathy and a change in signal intensity of intramedullary spinal cord on Magnetic Resonance imaging. J Neurosurg Spine 2010;12:59-65.
18. Uchida K, Nakajima H, Sato R, Kokubo Y, Yayama T, Kobayashi S, *et al.* Multivariate analysis of the neurological outcome of surgery for cervical compressive myelopathy. J Orthop Sci 2005;10:564-73.
19. Tauchi R, Imagama S, Inoh H, Yukawa Y, Kanemura T, Sato K, *et al.* Risk factors for a poor outcome following surgical treatment of cervical spondylotic amyotrophy: A multicenter study. Eur Spine J 2013;22:156-61.
20. Wada E, Ohmura M, Yonenobu K. Intramedullary changes of the spinal cord in cervical spondylotic myelopathy. Spine (Phila Pa 1976) 1995;20:2226-32.

How to cite this article: Wang H, Li H, Jiang J, Lû F, Chen W, Ma X. Evaluation of characteristics and surgical outcomes in cervical spondylotic amyotrophy. Indian J Orthop 2014;48:511-7.

Source of Support: This work was funded by National Natural Science Foundation of China (No. 81071438), **Conflict of Interest:** None.