Outcome and Predictive Factors in Rapid Progressive Cervical Spondylotic Myelopathy: A Retrospective Case-Control Study

Haimiti Abuduaini
Sichuan University West China Hospital
Hao Liu (liuhaospine@163.com)
Sichuan University West China Hospital
Beiyu Wang
Sichuan University West China Hospital
Yang Meng
Sichuan University West China Hospital
Yi Yang
Sichuan University West China Hospital
Chen Ding
Sichuan University West China Hospital
Tingkui Wu
Sichuan University West China Hospital
Chengyi Huang
Sichuan University West China Hospital

Research article

Keywords: Cervical Spondylotic Myelopathy, Rapid progression, Neurological dysfunction, MRI, Torg-Pavlov ratio, Increased signal intensity

Posted Date: June 25th, 2020

DOI: https://doi.org/10.21203/rs.3.rs-32994/v1

Version of Record: A version of this preprint was published at Clinical Neurology and Neurosurgery on November 1st, 2020. See the published version at https://doi.org/10.1016/j.clineuro.2020.106226.

Abstract

Background: To evaluate epidemiological, clinical and radiographic features in the development and prognosis of rapid progressive cervical spondylotic myelopathy (rp-CSM).

Methods: A retrospective study of 175 patient records was performed between March 2011 and January 2017. Patients were divided into rp-CSM group and chronic CSM (c-CSM) group according to the deterioration time and severity of preoperative neurological dysfunction. After selection, 25 rp-CSM patients were matched to a control group of 75 patients with c-CSM. The clinical outcomes were assessed by the Modified Japanese Orthopaedic Association (mJOA) score at six different follow-up time points. The imaging parameters including Torg-Pavlov Ratio (TPR) on conventional lateral x-ray and magnetic resonance images (MRI), intervertebral disc level compression ratio and increased signal intensity (ISI) on T2W1 were analyzed between the two groups, and predictors for rapid neurological dysfunction in CSM patients were evaluated using multivariate analysis.

Results: Twenty-five patients experienced rp-CSM (18 males; median age 59.04 ± 12.81 years) and were matched with Seventy-five control subjects that with CSM without rapid progression (54 males; median age 56.88 ± 12.31 years). The average time to develop severe neurological deterioration was 0.8 month in rp-CSM group and 24 month in c-CSM group (p=0.001), preoperative mJOA were 6 in rp-CSM patients and 12 in c-CSM patients (p=0.014) and rp-CSM patients demonstrated worse outcomes than the controls in one year after surgery (mJOA improvement rate 54.5% and 80%, p=0.021). There were no differences between the two groups except the history of diabetes and the long-term smoking in basic condition, radiographic measurements signified that TPR_{MRI}, intervertebral disc level compression ratio and increased signal intensity (ISI) on T2W1 were inferior in patients with rp-CSM than patients with c-CSM. Regression analysis verified that the history of diabetes, $TPR_{MRI} < 0.4$, compression ratio ≥50%, the sagittal diameter of ISI ≥50% of spinal canal diameter on T2W1 have significant correlations with the rapid progressive neurological dysfunction in patients with CSM.

Conclusion: The prognosis of rapid progressive CSM is worse than that of common chronic CSM. The rapid neurological deterioration can be identified by TPR_{MRI} (< 0.4), compression ratio (≥50%), sagittal diameter of ISI (≥50% of spinal canal diameter). Besides, a history of diabetes was also a negative factor for these patients.

Background

Cervical spondylotic myelopathy (CSM) is currently considered to be the most common and major cause of cervical spinal cord dysfunction in people over 55 years of age [1]. Most patients with CSM usually present chronic progressive and phased compression, the neurological dysfunction of these patients is gradually aggravated. Previous studies have shown that 82% and 56% of CSM patients will not experience severe neurological deterioration 5 years and 10 years after systematic conservative treatment [2]. However, neurological function of some CSM patients develop to severe dysfunction (such as unable

to button shirt or walk) in a very short time without any trauma, which called in previous studies as rapid progressive CSM (rp-CSM) [3-5].

Although the specific pathological mechanism of CSM is still unclear, it is closely related to spinal cord ischemia has widely recognized [6-9]. There have been studies about the natural course of CSM, and the chronic exacerbation of CCM and it has been reported that rapid neurological deterioration is associated with increased signal intensity (ISI), advanced age and anterior spondylolisthesis [4,5]. Acute exacerbation has yet to be studied clearly, it is necessary to carry out further study which combined more objective clinical scales and radiographic measurement.

In this case-control study, we aimed to investigate different epidemiological, clinical and radiographic features associated with the development and prognosis of rapid progressive CSM with the prospect of specific interventions in time for patients who are at high risk of rapid severe neurological dysfunction.

Methods

This retrospective study was approved by the Medical Ethical Committee of our hospital. All of the patients were recruited after providing informed consent for analysis of their clinical data.

The definition of rapid progressive cervical spondylotic myelopathy

The patients were divided into rp-CSM group and c-CSM group according to the rp-CSM defined by Morishita [3] and categorization of mJOA described by Fehlings [10]. In brief, rp-CSM means that CSM patients develop rapid and severe neurological dysfunction without history of trauma within the first month after diagnosis, and preoperative mJOA score was from 0-11. While c-CSM refers to the slow deterioration of neurological dysfunction, preoperative mJOA score was from 12-17 (**table 1**).

Table 1
Comparison of clinical features of rapid progressive and chronic CSM

Clinical features	rapid progressive CSM	chronic CSM
Time to severe ND	≤ 1 month	> 1 month
A history of trauma	none	possible
Conservative treatment	poor effect	effective in the early
Preoperative mJOA score	from 0 to 11	from 12 to 17
Upper limb symptoms	inability to button shirt	able to button shirt
Lower limb symptoms	unable to walk	able to walk
CSM: rapid progressive cervical spondylotic myelopathy; ND: neurological dysfunction.		

Inclusion and exclusion criteria

Patients diagnosed cervical spondylotic myelopathy through clinical symptoms, imaging data and clinical physical examination older than 18, and meet the definition of rp-CSM. Excluding the patients with vertebral nodules, tumors, severe osteoporosis, congenital fusion, mandatory spondylitis and other differential diagnoses, a history of cervical trauma and surgery. Patients who were required to change their surgical method, patients who lost contact after surgery, patients who suffered from other serious systemic diseases, and patients who died.

Participant

A total of 175 patients with CSM were treated in our department from March 2011 to January 2017. According to whether or not these patients conform to the definition of rp-CSM and c-CSM, 100 patients were selected into this study (**figure 1**). All 100 patients with CSM were treated with surgical decompression by the same experienced surgeon. Various clinical and imaging characteristics were compared between the two groups, and logistic regression analyses were performed to determine the independent risk factors for rapid neurological deterioration in patients with rp-CSM and to explore the prognosis of these patients.

Operative procedure

All patients underwent unilateral open-door cervical expansive laminoplasty plus centrepiece titanium plate fixation. The paravertebral muscles were stripped to remove the C7 spinous process and the interspinous ligament from C7-T1. The inner plate of the medial edge of the articular process of the lamina was removed with 1 mm grab-type bone biting forceps. The junction between the lamina and articular process of the open-door axis side was used as the hinge to lift the C3-C7 laminae gradually. The screw holes were made with the magic drill, and 4 screws were placed into each titanium plate. The spinous processes and laminae were removed to form granulated bone and placed on the left hinge. The wound was rinsed and sutured layer by layer, and a negative drainage tube was placed.

Clinical outcome Measures

The Modified Japanese Orthopaedic Association (mJOA) scoring system was measured at before operation,1 week, 1 month, 3 months, 6 months, 12 months after surgery to evaluate the neurological function.

Imaging measurements

On the preoperative MR images, the following parameters were measured: 1) the sagittal diameter of the vertebral body; 2) the sagittal outer diameter of the subarachnoid space at the midpoint of the vertebra (midvertebral canal diameter); 3) the sagittal outer diameter of the subarachnoid space at the level of the intervertebral disc (disc-level canal diameter), and 4) the sagittal diameter of the spinal cord at the levels C2 and T1 (**figure 2**), The median of the two values was used for spinal cord compression ratio at the

level of the intervertebral disc .The spinal canal to vertebral body ratio (Torg-Pavlov Ratio, TPR_{MRI}) was calculated by dividing the midvertebral canal diameter (ie, outer diameter of the subarachnoid space at the midpoint of the vertebra) by the diameter of the vertebral body [11]. The ratio of the sagittal diameter of ISI on spinal canal was calculated by using intramedullary high signal diameter and spinal canal diameter (diameter of ISI larger than 50% of spinal canal diameter was considered significant in this study).

On the lateral radiographs, the sagittal diameter of the vertebral body and the developmental sagittal diameter of the spinal canal[12]. The TPR_{CR} was calculated by dividing the spinal canal diameter by the vertebral body diameter [13] (**figure 3**).

Statistical Analysis

The statistical analysis software SPSS (version 20.0) was used for data analysis. The chi-square and Fisher exact tests were used to compare test was used to compare the enumeration data, and the results are expressed as cases (percentages). The Mann-Whitney U-test was conducted for ranked data, the measurement data were first tested for normality, and an independent sample t test was used when it conformed to a normal distribution. The results are expressed as the mean ± standard deviation. Variables that exhibited a significant difference in the univariate analysis were entered into a multivariate logistic regression analysis.. P-values of less than 0.05 were considered significant in this study.

Results

Twenty-five in the rp-CSM group and seventy-five in the c-CSM group. The proportion of smokers were significantly higher in rp-CSM group than that in c-CSM (p = **0.04**). There were no significant differences in other demographic data between two groups (p < 0.05, Table 2).

Table 2
Demographic and baseline data between two groups

Group	rp-CSM group	c-CSM group	P value
No. of patients	25(25%)	75(75%)	
Age (y)	59.04 ± 12.81	56.88 ± 12.31	0.772
Gender (F/M)	7/18	21/54	0.06
BMI index	23.70 ± 2.99	24.33 ± 2.50	0.334
Smoking	12 (48%)	20 (26.7%)	**0.04**
Alcohol	10 (40%)	24 (32%)	0.352

44% and 12% patients have a history of diabetes in rp-CSM group and c-CSM group, respectively, and the difference was significant (P = **0.001**). There were no significant differences between the two groups in

other comorbidities, including the history of hypertension and cardiovascular disease ($p > 0.05$, Table 3) .

Table 3
Comparison of comorbidities between two groups

Group	rp-CSM group	c-CSM group	P value
Diabetes (%)	44%	12%	**0.001**
Hypertension (%)	20%	34.7%	0.170
Cardiovascular disease(%)	20.8%	18.7%	1.103

The mJOA scores in rp-CSM group and c-CSM group were 6 and 12 before surgery, 8 and 14 at 1 week, 9 and 14 at 1 month, 10 and 15 at 3 month, 12 and 15 at 6 month and 12 and 15 at 1 year after surgery, respectively. The mJOA scores at each follow-up time points in rp-CSM were significantly lower than those in c-CSM group (**$p < 0.05$**, Table 4**)**.

Table 4
Comparison of mJOA score between two groups

	rp-CSM	c-CSM	p Value
mJOA score			
preoperative	6	12	**0.014**
1 week	8	14	**0.018**
1 month	9	14	**0.022**
3 month	10	15	**0.030**
6 month	12	15	**0.035**
1 year	12	15	**0.032**
Improvement rate	54.5%	80%	**0.021**

As presented in Table 5, the TPR_{MRI} averaged 0.21 in the rp-CSM group and 0.64 in the c-CSM group (P = **0.030**). The TPR_{CR} averaged 0.33 in the rp-CSM group and 0.54 in the c-CSM group (P = 0.061), and the compression ratio averaged 84% in the rp-CSM group and 32% in the c-CSM (p = **0.010**), and ISI ≥ 50% on T2W1 was 63% in the rp-CSM group and 17% in the c-CSM group.

Table 5
Radiological data

Group	rp-CSM group	c-CSM group	P value
TPR_{MRI}	0.21	0.64	**0.030**
TPR_{CR}	0.33	0.58	0.061
Compression ratio	84%	32%	**0.010**
ISI ≥ 50% on T2W1	60%	16%	**0.000**

Results from the univariate analysis revealed that a history of diabetes (p = **0.040**), TPR_{MRI} (p = **0.020**), compression ratio (p = **0.004**), ISI ≥ on T2W1 (p = **0.026**) were significantly associated with rapid progressive neurological dysfunction in patients with CSM. Furthermore, results of multivariate logistic regression also confirmed that CSM patients with diabetes, TPR_{MRI} <0.4, compression ratio ≥ 50% and ISI ≥ 50% on T2W1 were liable to have rapid neurological deterioration (Table 6).

Table 6
Correlations of various variables and rapid deterioration of neurological dysfunction

Independent variable	Response	Total patients (n = 100)	With rapid deterioration (n = 25)	P value
Age	≥ 50	76	18	0.073
	< 50	24	7	
Diabetes	Yes	20	11	**0.040**
	No	80	14	
Smoking	Yes	32	12	0.350
	No	68	13	
TPR_{MRI}	≥ 0.4	69	6	**0.020**
	< 0.4	31	19	
TPR_{CR}	≥ 0.4	72	11	0.352
	< 0.4	28	14	
Compression ratio ratio	≥ 50%	45	21	**0.004**
	< 50%	55	4	
ISI on T2W1	≥ 50%	27	15	**0.026**
	< 50%	73	10	

Table 7
Results of logistic regression analysis of confounding factors for rapid progressive neurological dysfunction

Group	Response	Wald	P	OR	95% CI
Diabetes	Yes	0.836	**0.041**	2.368	0.309– 1.750
	No				
TPR_{MRI}	≥ 0.4	4.201	**0.020**	0.705	2.317–7.574
	< 0.4				
Compression ratio	≥ 50%	3.827	**0.016**	9.100	1.486– 4.920
	< 50%				
ISI ≥ 50% on T2W1	≥ 50%	12.905	**0.025**	4.261	1.350– 7.603
	< 50%				

Discussion

In the study of Kadanka et al, 15% of the patients had deteriorated or remained unchanged at or below a mJOA score of 14 at the 1-year follow-up, 34% at 2 years, and 27% at 3 years [7]. Tachibana has been reported that 18.60% patients with CSM suffered rapid progressive clinical deterioration within 4 weeks of the onset of symptoms [5]. However, they mainly included patients with a clear history of trauma, the difference is that we mainly focused on the patients that rapid neurological dysfunction without any trauma. Besides some anatomical changes have not been discussed with objective assessment , such as vertebral body ratio and compression rate on MRI.

The prognosis of patients with rapid progressive CSM is quite different among previous studies, Morishita reported that the preoperative JOA score is 5.4 and 9.6 at 1 year after surgery, which were significantly lower than in chronic CSM which were 10.1 and 12.9, respectively, and they concluded that prognosis of rp-CSM group was inferior than those in c-CSM group [3]. However, in the study of Takasawa [4] , JOA score was 5.7 preoperative and 12.9 after one year surgery (improvement rate was 64.5%) in rp-CSM group and 10.1 preoperative and 12.8 after one year surgery (improvement rate was 40.7%) in c-CSM, they reached a conclusion that patients with rp-CSM can get better recovery than those in c-CSM. In present study, The mJOA scores in rp-CSM group and c-CSM group were 6 and 12 before surgery, 8 and 14 at 1 week, 9 and 14 at 1 month, 10 and 15 at 3 month, 12 and 15 at 6 month and 12 and 15 at 1 year after surgery, respectively ,the mJOA scores at each follow-up time points in rp-CSM were significantly lower than those in c-CSM group. Furthermore, the improvement rate was higher in c-CSM (80%) than those in rp-CSM (80%).

Although correlation between T2 hypersignal and acute spinal cord injury (SCI) have been reported in some previous articles, in the clinical practice, it is very common to observe patients with some degree of

T2 hyperintensity in the spinal cord and very mild (or even no) symptoms of CSM. Simply categorizing patients in a dichotomous fashion based on the presence or absence of T2WI hyperintensity in the spinal cord seems to be a quite rudimentary method of analyzing such an important radiological parameter. Therefore, the ratio of the sagittal diameter of ISI was calculated by using intramedullary high signal diameter and spinal canal diameter, and in order to decrease the effect of T2 high signals not associate with rapid neurological deterioration, we consider the diameter of ISI larger than 50% of spinal canal diameter was considered significant in this study.

The Torg-Pavlov Ratio (TPR) [13] is one of the most popular and commonly used method to measure the spinal stenosis, and previous studies have been proved that it can effectively evaluate the degree of spinal canal stenosis. However, poor predictive value caused by vertebral size and malformation may results false positive that affect the accuracy of the measurement [13-17]]. Furthermore, spinal canal stenosis caused by soft tissue cannot be observed on conventional X-ray radiographs. Previous studies have revealed that MR image parameters are reliable for predicting the occurrence and course of cervical spinal neurapraxia in athletes suffering from sports traumata [15,18]. However, the relevance of MR image parameters for predicting the course of rapid progressive cervical spondylotic myelopathy without a trauma has not been established.

Based on the present results, compared with other parameters, spinal cord compression ratio (at the level of the intervertebral disc) and TPR_{MRI} presented the highest correlation with the rapid neurological dysfunction, and CSM patients with spinal canal compression ratio $\geq$50% and TPR_{MRI}<0.4 are at risk of acute neurological dysfunction without a minor trauma. However, the clinical value of this finding should be interpreted cautiously, because when the two indicators are applied, false-positive and false-negative cases may be encountered, besides the sensitivity is still insufficient. In our study, 21 of 45 patients with spinal canal compression ratio $\geq$50% and 19 of 31 patients with TPR_{MRI}<0.4 presented rapid neurological dysfunction, while the other 24 and 12 patients did not experience rapid deterioration of neurological function, respectively. Whether these two indicators have critical predictive value in patients with rapid progressive CSM without trauma will need to be confirmed additional in other large, and high-quality studies.

Limitations of the study

The limitations of this study were the retrospective case-control study design and the small sample size. The definition of rp-CSM was mainly based on the clinical features like symptoms and mJOA scores, the imaging and biological proofs were not considered. However, the previously mentioned findings [3,9,19]. support the pathophysiology of rp-CSM defined in this study. In order to eliminate temporary spinal cord edema, we regard the change of MR T2- hyperintensity signal sagittal diameter larger than 50% as meaningful, this approach may bring a disadvantage that it may miss the permanent neuronal changes with a smaller diameter. Future studies should examine the characteristics and pattern of the MR signal changes to promote an understanding of the pathophysiology of rp-CSM. The optimal operation time and preoperative drug treatment after rapid neurological deterioration have not been fully discussed, Thus,

next, we will conduct larger population-based prospective study which will focus on the treatments in patients with rapid progressive CSM.

Conclusion

The prognosis of rapid progressive CSM is worse than that of common chronic CSM. The rapid neurological deterioration can be identified by TPR_{MRI} (< 0.4), compression ratio at intervertebral disc (≥ 50%), sagittal diameter of ISI (≥ 50% of spinal canal diameter). Besides, a history of diabetes was also a negative factor for these patients.

Declarations

Ethics approval and consent to participate

Ethical approval was granted by West China Hospital of Sichuan University Human Research Ethics Committee, Health and Medical Research Human Research Ethics Committee, and all participants provided written informed consent prior to commencement of the study.

Consent for publication

All authors have read and approved the content, and agree to submit it for consideration for publication in BMC Musculoskeletal Disorders. We deeply appreciate your consideration for this manuscript.

Competing interests

No conflict of interest exits in the submission of this manuscript.

Funding

This study was supported by Sichuan Province Science and Technology Support Program (grant no. 2019YFQ0002) and West China Hospital, Sichuan University (grant no. HX-H1812210).

Authors' contributions

HA and HL contributed to the conception of the study;

BYW and YM collected the clinical and radiographic data;

YY and CD contributed significantly to analysis and manuscript preparation;

HA and TKW performed the data analyses and wrote the manuscript;

CYH helped perform the analysis with constructive discussions.

Acknowledgements

None.

References

1. Fehlings M.G, Skaf G, et al. A review of the pathophysiology of cervical spondylotic myelopathy with insights for potential novel mechanisms drawn from traumatic spinal cord injury. Spine, 1998; 23(24):2730-2736.
2. Oshima Yeichi A, Takeshita K, et al. Natural course and prognostic factors in patients with mild cervical spondylotic myelopathy with increased signal intensity on T2-weighted magnetic resonance imaging. Spine, 2012, 37(22):1909-1913.
3. Morishita Y, Matsushita A, Maeda T, et al. Rapid progressive clinical deterioration of cervical spondylotic myelopathy. Spinal cord, 2015,53(5):408-410.
4. Takasawa E, Sorimachi Y, Iizuka Y, et al. Risk Factors for Rapidly Progressive Neurological Deterioration in Cervical Spondylotic Myelopathy.Spine, 2019, 4(12):E723-E730.
5. Tachibana T, Maruo K, Arizumi F, , et al. Predictive factors for acute exacerbation of cervical compression myelopathy. Journal of Clinical Neuroscience, 2018, 48:160-162.
6. Sumi M, Miyamoto H, Suzuki T, et al. Prospective cohort study of mild cervical spondylotic myelopathy without surgical treatment[J]. Journal of Neurosurgery. Spine, 2012, 16(1):8-14.
7. Kadanka Z, Mareš M, Bednar k.J, et al. Approaches to spondylotic cervical myelopathy: conservative versus surgical results in a 3-year follow-up study. Spine, 2002, 27(20):2205-2210.
8. Kubota K, Saiwai H, Kumamaru H, et al. Neurological recovery is impaired by concurrent but not by asymptomatic pre-existing spinal cord compression after traumatic spinal cord injury. Spine,2012, 37(17):1448-1455.
9. Baptiste D.C, Fehlings M.G, et al. Pathophysiology of cervical myelopathy. The Spine Journal, 2006, 6(6):190-S197.
10. Fehlings M. G, Wilson J.R, et al. Efficacy and Safety of Surgical Decompression in Patients with Cervical Spondylotic Myelopathy. The Journal of Bone & Joint Surgery, 2013, 95(18):1651–1658.
11. Aebli N, Rüegg T.B, et al. Predicting the risk and severity of acute spinal cord injury after a minor trauma to the cervical spine. The Spine Journal, 2013, 13(6):597–604.
12. EDWARDS W.C, LaROCCA H. (1983). The Developmental Segmental Sagittal Diameter of the Cervical Spinal Canal in Patients with Cervical Spondylosis. Spine, 8(1), 20–27.
13. Torg JS, Pavlov H, Genuario SE, et al. Neurapraxia of the cervical spinal cord with transient quadriplegia. J Bone Joint Surg Am 1986;68:1354–70.
14. Herzog RJ, Wiens JJ, Dillingham MF, Sontag MJ. Normal cervical spine morphometry and cervical spinal stenosis in asymptomatic professional football players. Plain film radiography, multiplanar com- puted tomography, and magnetic resonance imaging. Spine 1991;16:S178–86.
15. Presciutti SM, DeLuca P, Marchetto P, et al. Mean subaxial space available for the cord index as a novel method of measuring cervical spine geometry to predict the chronic stinger syndrome in

American football players. J Neurosurg Spine 2009;11:264–71.
16. Blackley HR, Plank LD, Robertson PA. Determining the sagittal dimensions of the canal of the cervical spine. The reliability of ratios of anatomical measurements. J Bone Joint Surg Br 1999;8:110-2
17. Hukuda S, Xiang LF, Imai S, et al. Large vertebral body, in addition to narrow spinal canal, are risk factors for cervical myelopathy. J Spinal Disord 1996;9:177–86.
18. Shin JJ, Jin BH, Kim KS, et al. Intramedullary high signal intensity and neurological status as prognostic factors in cervical spondylotic myelopathy. Acta Neurochir (Wien) 2010;152:1687–94.
19. Yue WM, Tan SB, Tan MH, et al. The Torg-Pavlov ratio in cervical spondylotic myelopathy: a comparative study between patients with cervical spondylotic myelopathy and a non-spondylotic, non- myelopathic population. Spine (Phila Pa 1976) 2001;26:1760 – 4.

Figures

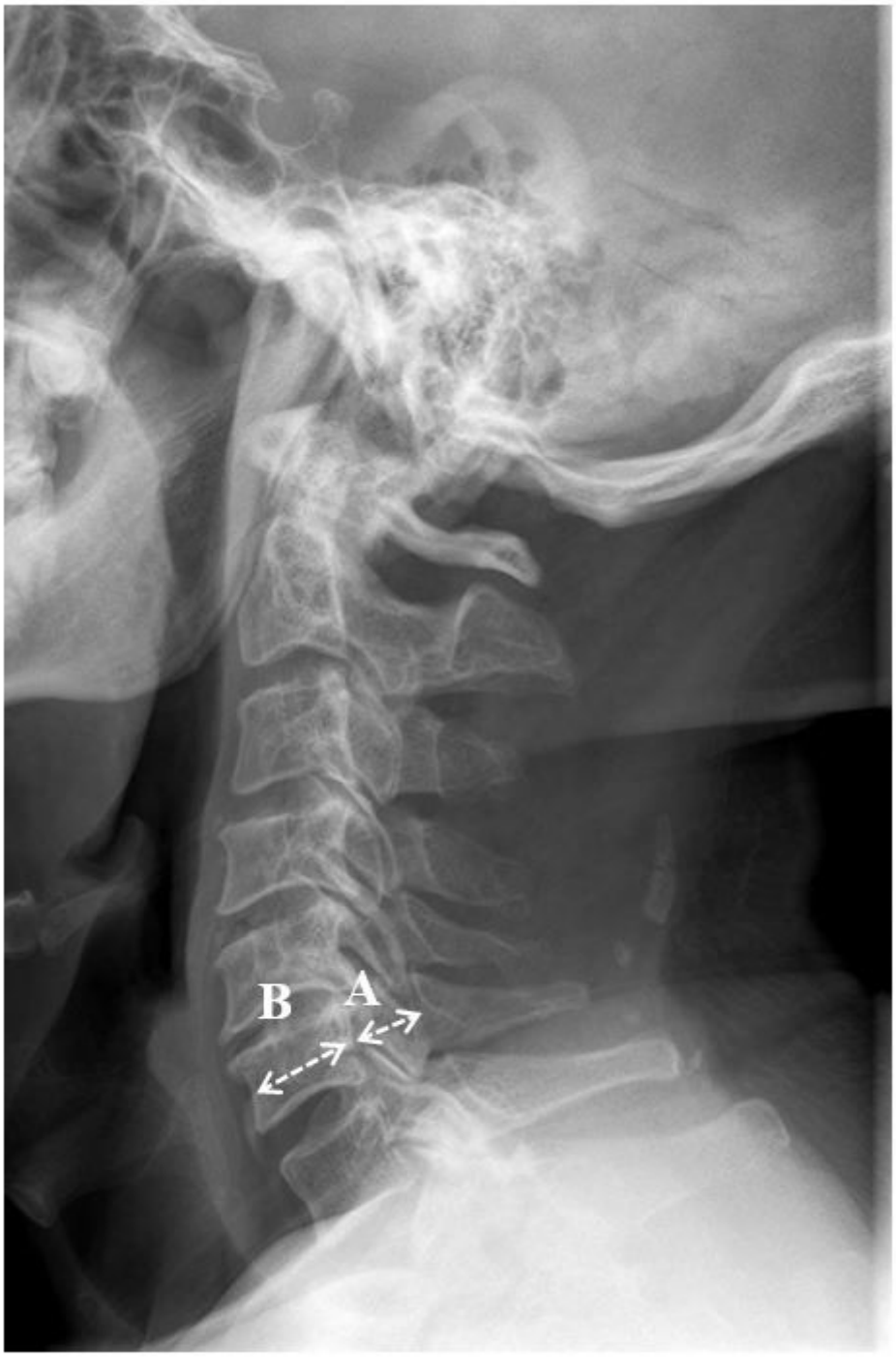

Figure 1

Measurement for the calculating the Torg-Pavlov ratio on lateral lateral radiograph of cervical spine in a patient with rapid progressive cervical spondylotic myelopathy . (a) Developmental sagittal diameter of the spinal canal as the shortest distance from the midpoint of the posterior surface of the vertebral body to the spinolaminar line [12]. (b) Sagittal diameter of the vertebral body measured between the midpoints of the anterior and posterior surfaces [13].

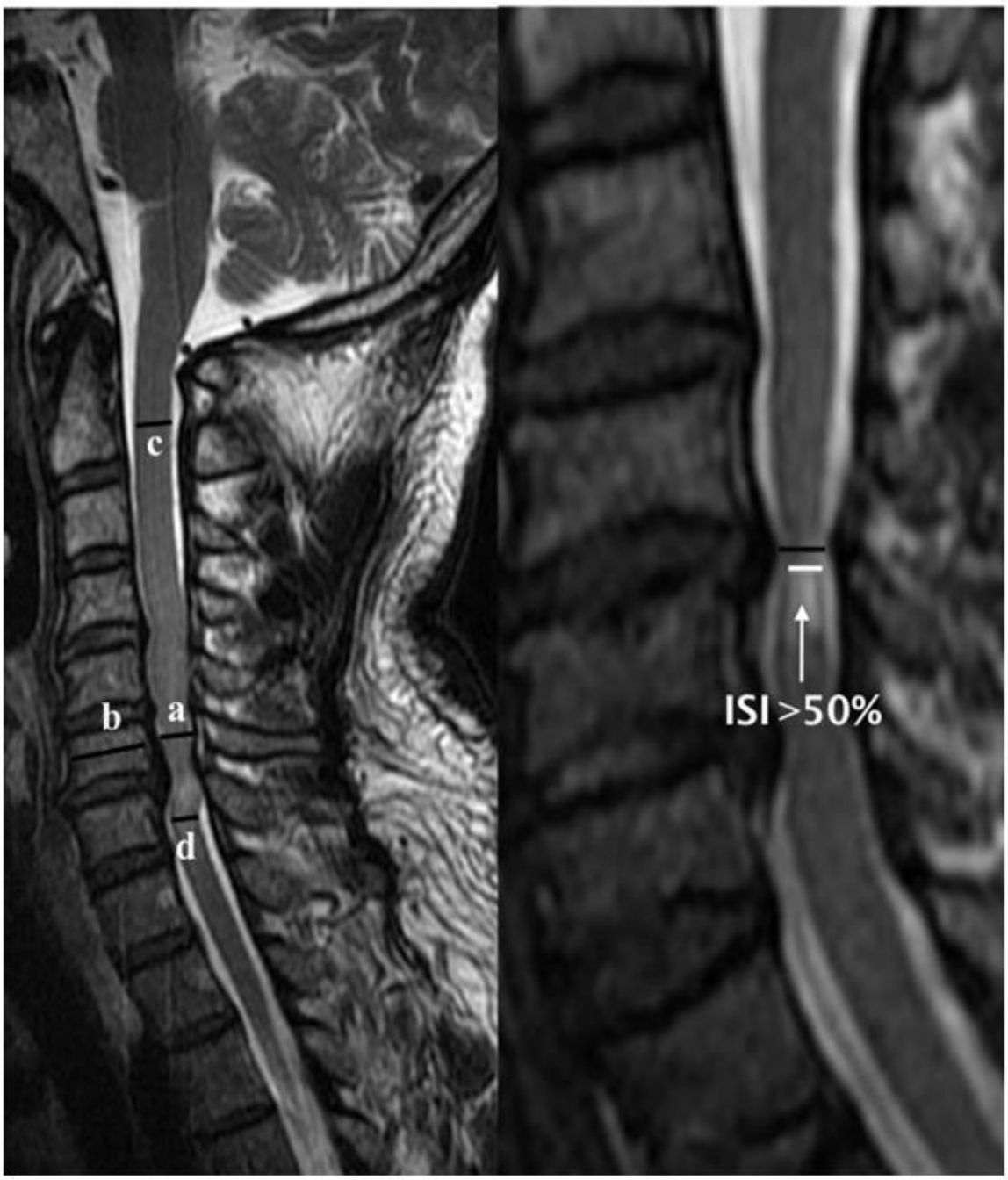

Figure 2

A. Sagittal view of the cervical spine with the parameters measured on T2-weighted magnetic resonance images of a patient who was diagnosed as rapid progressive cervical spondylotic myelopathy. (a) Sagittal outer diameter of the subarachnoid space at the midpoint of the vertebra (midvertebral canal diameter). (b) Sagittal diameter of the vertebral body measured between the midpoints of the anterior and the posterior surfaces. (c) Sagittal diameter of the spinal cord at C2. (d) sagittal diameter of the spinal cord at T1. B. Regional enlarged drawing of ISI on T2-MRI. (A) Sagittal diameter of the spinal canal at the midpoint of C6/7 intervertebral disc was 0.34 mm, and the sagittal diameter of intramedullary signal increased (ISI) was 0.26mm, the ratio of the sagittal diameter of ISI on spinal canal was 76.5%.

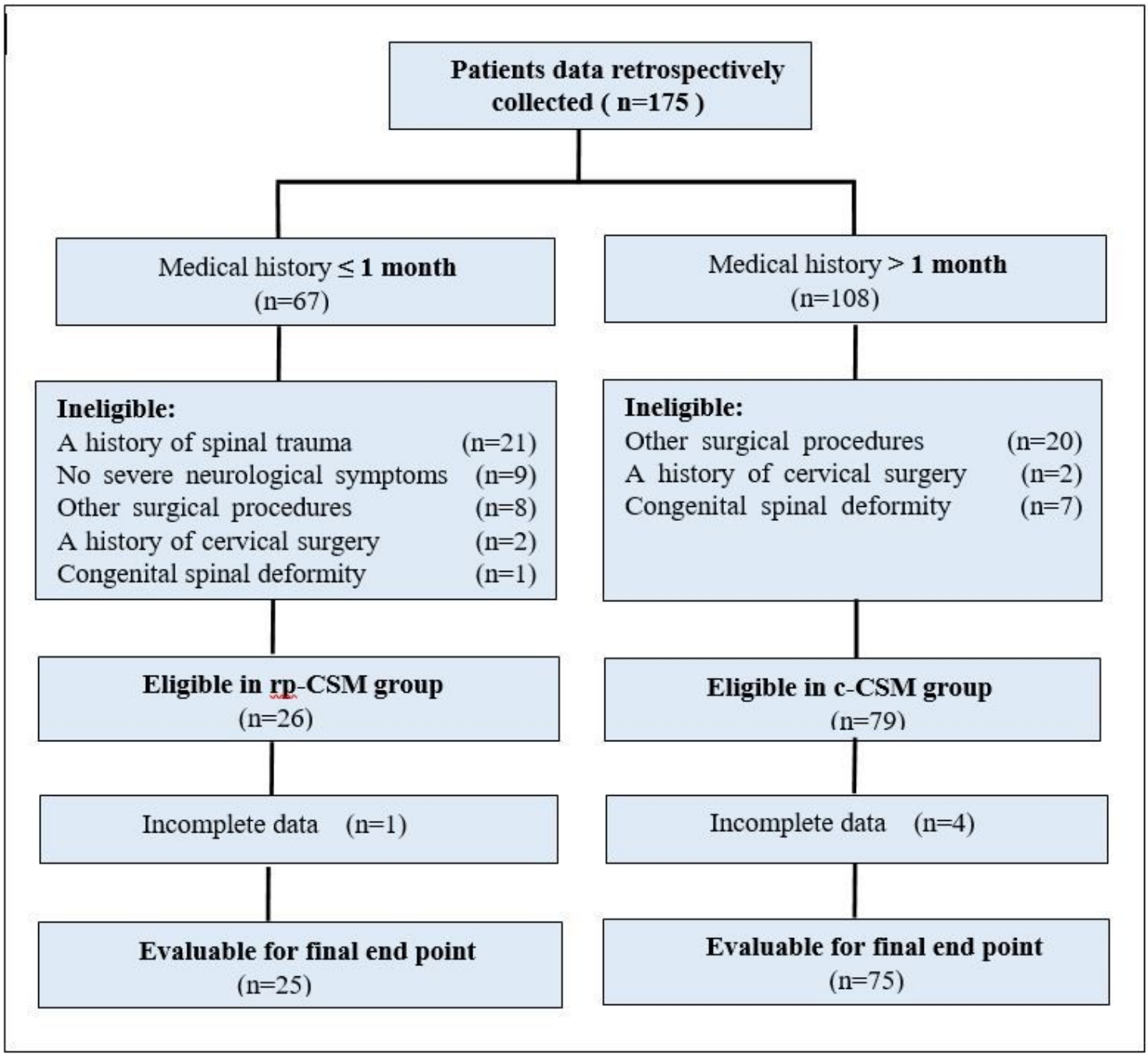

Figure 3

Consort diagram of patient flow.