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Risk Factors for Rapidly Progressive Neurological Deterioration in Cervical Spondylotic Myelopathy

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Study Design. A retrospective single-center study.

Objective. This study sought to clarify the risk factors and to evaluate the surgical outcome in patients with rapidly progressive cervical spondylotic myelopathy (rp-CSM).

Summary of Background Data. CSM is a degenerative spine disease presenting a slow development of myelopathy. Some patients, however, show rapidly progressive neurological deterioration (especially gait disturbances) without any trauma. At present, there is little information about this condition.

Methods. We studied 71 consecutive CSM patients (52 men, 19 women) with a mean age of 67.1 years, and the follow-up period was 1 year. Patients were divided into two groups: rp-CSM and chronic-CSM (c-CSM) groups. The Japanese Orthopaedic Association score and various clinical differences, including age, sex, comorbidity, the waiting period from symptomatic onset to surgery, cervical range of motion, and intramedullary MR T2-hyperintensity were analyzed, and independent risk factors were determined using a logistic regression analysis.

Results. Eighteen of 71 patients (25.4%) were diagnosed with rp-CSM. There were no significant differences between the two groups with regard to age, sex, or cervical range of motion. In the rp-CSM group, the preoperative upper/lower extremities and bladder functions were worse, and the waiting period for surgery was shorter (rp-CSM 1.2 mo, c-CSM 25.7 mo). Patients with rp-CSM had a history of cardiovascular event (CVE) (rp-CSM 44.4%, c-CSM 15.1%) and presented with MR T2-hyperintensity (rp-CSM 94.4%, c-CSM 58.5%), especially at the C4/5 disc level. Independent risk factors were a history of CVE (odds ratio = 4.7) and MR T2-hyperintensity (odds ratio =12.5). The rp-CSM group showed a better neurological recovery after decompression surgery (the Japanese Orthopaedic Association recovery rate: rp-CSM 64.5%, c-CSM 40.7%).

Conclusion. A history of CVE and MR T2-hyperintensity were risk factors for rp-CSM. Despite rapid neurological deterioration, rp-CSM patients showed a good neurological recovery after surgery, and thus indicating that rp-CSM is a reversible condition.

Key words: cardiovascular event, cervical spondylotic myelopathy, gait disturbance, intramedullary MR T2-hyperintensity, laminoplasty, magnetic resonance imaging, microvascular insufficiency, rapidly progressive neurological deterioration, spinal cord edema, spinal cord ischemia.

Level of Evidence: 4

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Cervical spondylotic myelopathy (CSM) is an age-related degenerative spine disease and is believed to be a slowly progressive disorder (chronic CSM [c-CSM]). In the natural course of activity of daily living, the condition gradually declines over the long term, and subjects with mild functional impairment can be treated conservatively.[1] However, some patients with CSM show rapidly progressive neurological deterioration, particularly with respect to their gait disturbance and difficulty in walking. Although numerous studies have reported the pathogenesis of CSM, there is little information available on the clinical characteristics of rapidly progressive CSM (rp-CSM), a subtype of the CSM spectrum.

The leading pathologies of CSM reportedly include spinal cord compression, dynamic factors, and subsequent ischemia of the spinal cord.[2] In detail, compression of the spinal cord within a narrow spinal canal can cause mechanical neural injury and poor blood flow, resulting in spinal cord ischemia and edema.[3–5] We therefore hypothesized that several factors affecting the spinal blood flow and ischemia might induce diversity in the pathophysiology of CSM, resulting in the CSM spectrum.

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The purpose of this study was to clarify the characteristics and factors associated with rp-CSM.

MATERIALS AND METHODS

Definition of Rapidly Progressive Cervical Spondylotic Myelopathy

In this study, rp-CSM was defined as previously reported by Morishita *et al*.[6] In brief, the patients with rp-CSM had difficulty maintaining a standing posture or walking without support within 4 weeks of the symptomatic onset due to rapidly progressive neurological deterioration.

Study Population

Our retrospective, single-center study consisted of 71 consecutive patients diagnosed with CSM who underwent surgery between January 2008 and December 2015. There were 52 men and 19 women with a mean age of 67.1 years (range, 38–89 yr). In this study, we focused on those who had no evident history of trauma. Patients who had a history of any spinal surgery were excluded from this study. All patients demonstrated long tract signs, such as clumsy hands, gait disturbance, and deep tendon hyperreflexia in the lower limbs on admission. According to the definition, the patients were divided into two groups: an rp-CSM group and a c-CSM group. Overall, 18 of 71 CSM patients (men, n = 16; women, n = 2; mean age, 69.2 yr) were diagnosed with rp-CSM. None of the patients with rp-CSM could maintain a standing posture and walk without the support of another person. They had no obvious episodes of any trauma before their clinical deterioration. Various clinical factors, such as age, sex, comorbidity (hypertension, diabetes mellitus, kidney disease, and a history of cardiovascular event [CVE]), and the waiting period from the symptomatic onset of myelopathy to surgery, were also evaluated (Table 1). In this study, CVE included myocardial infarction and ischemic stroke.

This study was approved by our institutional review board, and informed consent was obtained from all of the patients.

TABLE 1. Patient Characteristics and the Results of the Univariate Analysis

	rp-CSM	c-CSM	*P*
No (%)	18 (25.4)	53 (74.6)	
Age (yr)	69.2 ± 11.2	66.4 ± 11.5	0.592
Sex (male/female)	16/2	36/17	0.124
Comorbidity (%)	83.3	77.4	0.745
HT	38.9	41.5	1.000
DM	22.2	26.4	1.000
KD	11.1	3.8	0.265
CVE	44.4	15.1	0.019*
Waiting period for surgery (mo)	1.2 ± 0.8	25.7 ± 35.3	0.000†
ROMpreop (°)	31.5 ± 17.7	28.5 ± 11.9	0.455
Flex	−16.2 ± 9.1	−16.2 ± 14.2	0.736
Ext	24.6 ± 8.8	26.5 ± 11.3	0.463
MR T2-hyperintensity (%)	94.4	58.5	0.004*
C3/4	23.5	16.1	
C4/5	58.8	38.7	
C5/6	11.8	32.3	
C6/7	5.9	12.9	
Perioperative complication (%)	0.0	0.0	1.000
JOA baseline	5.7 ± 2.5	10.1 ± 2.1	0.000†
Motor			
Upper extremity	1.0 ± 1.2	2.4 ± 1.0	0.000†
Lower extremity	0.6 ± 0.5	1.8 ± 0.7	0.000†
Sensation			
Upper extremity	0.8 ± 0.4	1.1 ± 0.4	0.007†
Lower extremity	0.8 ± 0.5	1.3 ± 0.5	0.003†
Trunk	1.2 ± 0.7	1.8 ± 0.9	0.000†
Bladder function	1.1 ± 0.7	1.8 ± 0.9	0.011†
JOA at 12 mo	12.9 ± 2.3	12.8 ± 2.3	0.638
JOA-recovery rate (%)	64.5 ± 18.8	40.7 ± 28.1	0.001†

**Fisher's exact test.*

†Mann-Whitney U test.

c-CSM indicates chronic cervical spondylotic myelopathy; CVE, cardiovascular event; DM, diabetes mellitus; HT, hypertension; JOA, Japanese Orthopaedic Association; KD, kidney disease; MR, magnetic resonance; ROMpreop, preoperative range of motion; rp-CSM, rapidly progressive CSM.

Neurological Evaluations and Outcome Measures

The neurological status (the motor function of the upper and lower limbs and the bladder function) was evaluated preoperatively and at 12 months postoperatively based on the Japanese Orthopaedic Association (JOA) scoring system, which has been well validated as an accurate assessment of the severity of myelopathy.[7,8]

Imaging Evaluation

We assessed the affected segmental level by examining intramedullary hyperintensity lesions on magnetic resonance T2-weighted images using a 3.0-T magnetic resonance imaging (MRI) system (Ingenia, Philips Healthcare, Best, The Netherlands). The preoperative cervical range of motion (ROM) was also evaluated using flexion and extension sagittal radiographs.[9]

Operative Procedure

Open-door cervical laminoplasty was performed for all patients. The C4–6 laminae received open *en-bloc* with C-7 rostral side and C-3 caudal side laminotomy or C-3 laminectomy to preserve the insertion of the deep extensor musculature into the C-2 spinous process. The insertion of the nuchal ligament into the C-7 spinous process was also preserved to maintain the cervical lordotic alignment after surgery. The duration of cervical collar placement was 1 week after surgery.[10,11]

Statistical Analyses

Univariate analyses were performed to compare the postoperative outcomes of the two groups, as assessed by the JOA score, and various clinical and imaging findings, including age, sex, comorbidity (hypertension, diabetes mellitus, kidney disease and a history of CVE), the waiting period from the symptomatic onset of myelopathy to surgery, cervical ROM, intramedullary MR T2-hyperintensity, and the affected segmental level. Fisher's exact probability tests were performed to compare the sex, comorbidity, and MR T2-hyperintensity. Mann-Whitney *U* test was also used to compare the JOA score, age, waiting period, and cervical ROM. A multivariate logistic regression analysis was performed to determine the factors associated with the presence of rp-CSM. Variables that exhibited a significant difference in the univariate analysis were entered into a multivariate logistic regression analysis. The predefined significance for inclusion in the regression model was *P* values of 0.10 or lesser. All statistical analyses were conducted using the EZR software program (version 1.36, Saitama Medical Center, Jichi Medical University, Saitama, Japan).[12] *P* values of less than 0.05 were considered to indicate statistical significance.

RESULTS

Eighteen of 71 patients were diagnosed with rp-CSM (25.4%) in the present study. There were no significant differences between the two groups with regard to age, sex, or cervical ROM (Table 1). Regarding the details of the cervical sagittal radiographs in our study, there were no clinical instabilities, as defined by the previous reports[6,13] (>3.5 mm of dynamic translation or angulation that was 11° greater than that in the adjacent segment). In the rp-CSM group, the preoperative motor functions of the upper/lower extremities, and the bladder function according to the JOA score were worse ($P < 0.01$) and the waiting period from the onset of symptoms to surgery was shorter (rp-CSM 1.2 ± 0.8 mo *vs.* c-CSM 25.7 ± 25.3 mo; $P \leq 0.001$) than in the c-CSM group.

Univariate analyses demonstrated that rp-CSM patients tended to have a history of CVE (rp-CSM 38.9% *vs.* c-CSM 15.1%; $P = 0.019$) with MR T2-hyperintensity (rp-CSM 94.4% *vs.* c-CSM 58.5%; $P = 0.004$), especially at the C4/5-disc level (rp-CSM 58.8% *vs.* c-CSM 38.7%) (Table 1).

A multiple logistic regression analysis showed that a history of CVE (odds ratio [OR] = 4.7, 95% confidence interval = 1.3–17.5, $P = 0.021$) and MR T2-hyperintensity (OR = 12.5, 95% confidence interval = 1.5–106.0, $P = 0.020$) were independently associated with the presence of rp-CSM (Table 2).

All patients underwent open-door laminoplasty. The rp-CSM group showed a better neurological recovery after decompression surgery in comparison to the c-CSM group: in the rp-CSM group, the recovery rate of the JOA score at the 1-year follow-up examination was higher than that in the c-CSM group (rp-CSM 64.5% *vs.* c-CSM 40.7%; $P = 0.001$) (Table 1).

Regardless of anticoagulant therapy, there were no critical adverse events (*e.g.*, postoperative hematoma, stroke, or myocardial infarction) in patients with a history of CVE.

Case Presentation

A 75-year-old man had difficulty walking that was progressively worsening without any trauma. He ultimately became unable to walk without support after 2 weeks. He also complained of bilateral clumsiness of the hands and paresthesia in his left arm. He was diagnosed with cervical radiculopathy and treated conservatively for 1 year at a

TABLE 2. A Logistic Regression Analysis Evaluating the Association Between Various Clinical Factors and the Presence of Rapidly Progressive Cervical Spondylotic Myelopathy

Independent Variable	Odds	95% CI	*P*
A history of cardiovascular event	4.7	1.3–17.5	0.021
MR T2-hyperintensity	12.5	1.5–106.0	0.020

95%CI indicates 95% confidential interval; MR, magnetic resonance.

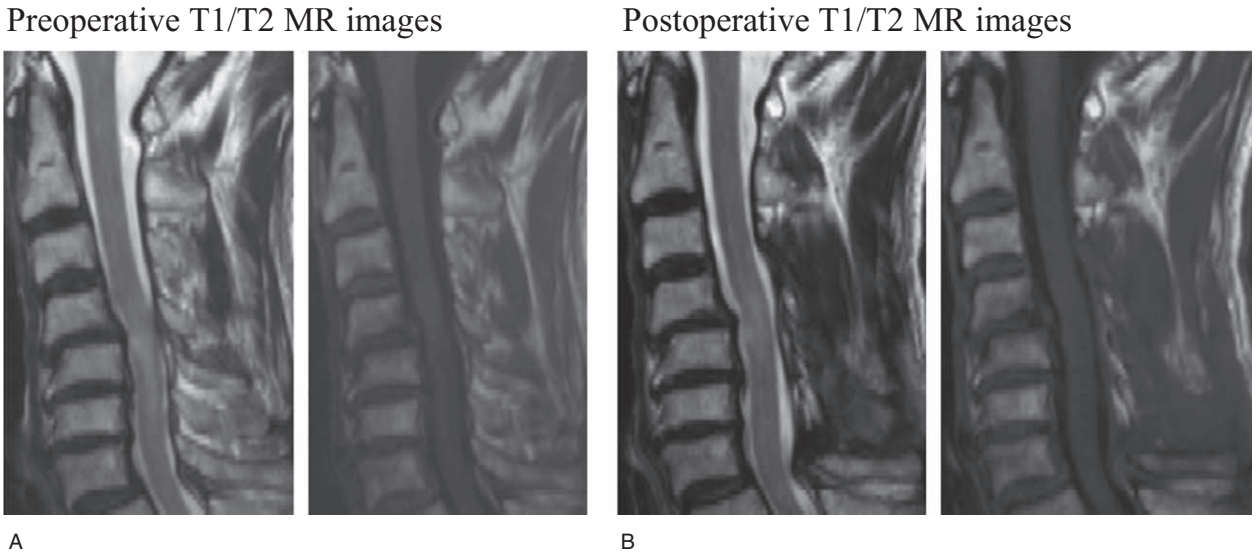

Figure 1. Case presentation. A 75-year-old man, who had a history of cardiovascular event, presented with walking difficulty that had persisted for 2 weeks. He was diagnosed with rapidly progressive CSM. **A**, Cervical magnetic resonance imaging (MRI) showed spinal compression and T2-hyperintensity at the C4/5 disc level with edema below the C4 level, probably due to spinal cord ischemia. **B**, Postoperative MRI revealed remission of the spinal edema and localization of the T2-hyperintensity at the C4/5 disc level without any T1-hypointensity lesions.

private clinic. He was also treated with aspirin for a history of cerebral infarction at another clinic.

On admission to our institution, cervical MRI showed spinal cord compression and T2-hyperintensity at the C4/5 disc level with edema below the C4 level, probably due to spinal cord ischemia (Figure 1A). The differential diagnoses of progressive neurological deterioration in older patients with CVE include spinal cord infarction and epidural hemorrhaging due to anticoagulant therapy. However, his clinical course and MR images seemed to be incompatible with these diseases. We diagnosed him with acute on chronic CSM, known as rp-CSM, and performed cervical laminoplasty after a 1-week washout period for aspirin. His neurological function remarkably improved, and he was able to walk again and use chopsticks at 1 week after surgery. There were no complications in the perioperative period. Postoperative MRI revealed remission of the spinal edema and localization of the T2-hyperintensity at the C4/5 disc level without any T1-hypointensity lesions (Figure 1B).

DISCUSSION

Our study had two main findings. First, we identified the independent risk factors for rp-CSM, including a history of CVE and MR T2-hyperintensity. Second, we showed that decompression surgery is effective and recommended for managing rapidly progressive neurological deterioration in CSM.

The Spectrum and Subtypes of Cervical Spondylotic Myelopathy

The natural history of CSM typically involves a stepwise deterioration in both the motor and sensory functions, with stationary plateau periods. In the previous study, 82% and 56% of patients with CSM were still conservatively followed without serious neurological deterioration at 5 and 10 years after the initial treatment, respectively.[1] In contrast, patients with CSM sometimes experience rapidly progressive neurological deterioration (particularly with gait disturbance).[6,14] However, not much attention has been paid to this subtype of the CSM spectrum, which may have limited our understanding and hampered our recognition of the clinical characteristics of rp-CSM.

Incidence of Rapidly Progressive Cervical Spondylotic Myelopathy

In our series, 25.4% of CSM patients demonstrated rapidly progressive neurological deterioration within 2 to 3 weeks of the onset of symptoms without any obvious trauma (*e.g.*, falling). Although little has been reported about this subtype of the CSM spectrum, Morishita *et al*[6] reported that 8 of 43 (18.6%) patients with CSM suffered rapid progressive clinical deterioration. In recent years, Tachibana *et al*[14] also reported on the acute exacerbation of cervical compression myelopathy; 19 of 59 (32.2%) patients received early surgery because of acute gait disturbance or acute paralysis (Table 3). The prevalence of rp-CSM has varied, because these have been single center and small sample-size studies. A further investigation is therefore required to yield robust data.

The Clinical Characteristics and Pathophysiology of Rapidly Progressive Cervical Spondylotic Myelopathy

In the line with prior studies,[6,14] we found that rp-CSM patients showed rapid progressive gait disturbance rather than degraded hand dexterity. We also noted that a great

TABLE 3. Clinical Characteristics of the Rapidly Progressive Cervical Spondylotic Myelopathy

Clinical Characteristics of the rp-CSM			
	The Present Study	**Morishita *et al*[6]**	**Tachibana *et al*[14]**
Proportion of rp-CSM	25.4% (18/71)	18.6% (8/48)	32.2% (19/59)
Clinical feature	Gait disturbance within 2–3 weeks of symptomatic onset	Gait disturbance within 4 weeks of symptomatic onset	Acute gait disturbance and acute paralysis
Age	NOT related	NOT related	Older patients (Odds 1.1)
Sex	NOT related	NOT related	—
Comorbidity associated with the onset of rp-CSM	Cardiovascular event (Odds 4.7)	—	—
Cervical instability	NOT related	Dynamic anterior slip (Weak evidence)	Spondylolisthesis (Odds 25.3)
MR T2-hyperintensity	94.1% (Odds 12.5)	100%	—
Preoperative JOA score	5.7 ± 2.5	5.4 ± 1.1	6.7 ± 3.6
Surgical outcome	Improved	Improved	—
Treatment strategy	Surgical decompression (Recommended)	Surgical decompression (Recommended)	Surgical decompression and fixation

JOA score indicates Japanese Orthopaedic Association score; MR, magnetic resonance; rp-CSM, rapidly progressive cervical spondylotic myelopathy.

majority of patients with rp-CSM presented with MR T2-hyperintensity lesions, as reported previously by Morishita *et al*.[6] MR T2-hyperintensity lesions are considered to reflect various intramedullary pathologies, such as edema, gliosis, demyelination, and myelomalacia.[15] The high frequency of MR T2-hyperintensity might be indicative of spinal edema followed by acute spinal ischemia, as described below (Table 3).

Although previous studies have reported an association between MR-T1 signal changes and the neurological outcome in CSM,[16,17] T1-hypointensity did not reflect a poor neurological prognosis in the patients with rp-CSM in our study. Recently, Nouri *et al* demonstrated that MR T1-hypointensity is a valuable guide for predicting a worse surgical outcome in CSM. However, the author also showed some limitation of this signal change: MR T1-hypointensity is associated with greater disease severity, and because it is present in only approximately 20% of patients, it is of lesser clinical utility. Moreover, only fair inter-rater agreement was observed.[16,17] In our study, the patients with rp-CSM maintained a walking ability before the onset of symptoms (*i.e.*, lower severity of disease), and were surgically treated in a time-sensitive manner (*i.e.*, restoring spinal cord blood flow and avoiding permanent cord injury). Thus, there was no relationship between the MR-T1 signal changes and the neurological prognosis of rp-CSM in this study.

Aging is a common factor in patients with degenerative spinal disease, and Tachibana *et al*[14] mentioned the association between the aged spinal cord and the acute exacerbation of CSM. Nevertheless, aging is associated with the comprehensive deterioration of not only the spinal structure and stability, but also the patient's physical status (*i.e.*, medical diseases) affecting the spinal cord blood flow and durability. Regarding the influence of comorbidities on CSM, Dokai *et al*[18] reported the clinical relationship between diabetes and a poor surgical outcome in patients with CSM. Karadimas *et al*[19] also mentioned that microvascular dysfunction is an important component of diabetes pathophysiology that affects the natural history of CSM. Although we agree with this view, the coexistence of diabetes and other comorbidities only suggests a potential risk of microvascular dysfunction, and cannot fully explain the microvasculature condition in patients with CSM.

In the present study, we noticed that the patients with rp-CSM tended to have a history of CVE, as well as other comorbidities. CVE can occur due to several factors, such as hypertension, diabetes, kidney disease, and smoking.[20] A history of CVE may therefore suggest the existence of arteriosclerosis and endothelial dysfunction, which can cause vasomotor dysfunction and insufficient blood flow in the cervical spinal cord. Such an unstable and friable blood stream may be less tolerant to blood pressure fluctuations and dynamic/static cord compression, ultimately resulting in the acute spinal cord ischemia observed as MR T2-hyperintensity.

As for the biomechanics, both static and dynamic factors are known to be related to the pathophysiology of the CSM spectrum. Bednarik *et al*[21] prospectively investigated the natural history of asymptomatic cervical spinal cord compression with discogenic or osteoligamentous spondylotic changes (*i.e.*, static factors), and coexisting risk factors, such as clinically symptomatic radiculopathy and MR T2-hyperintensity were associated with the development of myelopathy. The clinical course of an rp-CSM patient with cervical radiculopathy and MR T2-hyperintensity presented in our study, was consistent with this evidence. Moreover, Yue *et al*[22] reported the association between the development of myelopathy and a congenitally narrow cervical canal standardized by the vertebral body diameter (*i.e.*, the Torg-Pavlov ratio, which can reduce the influence of spondylotic

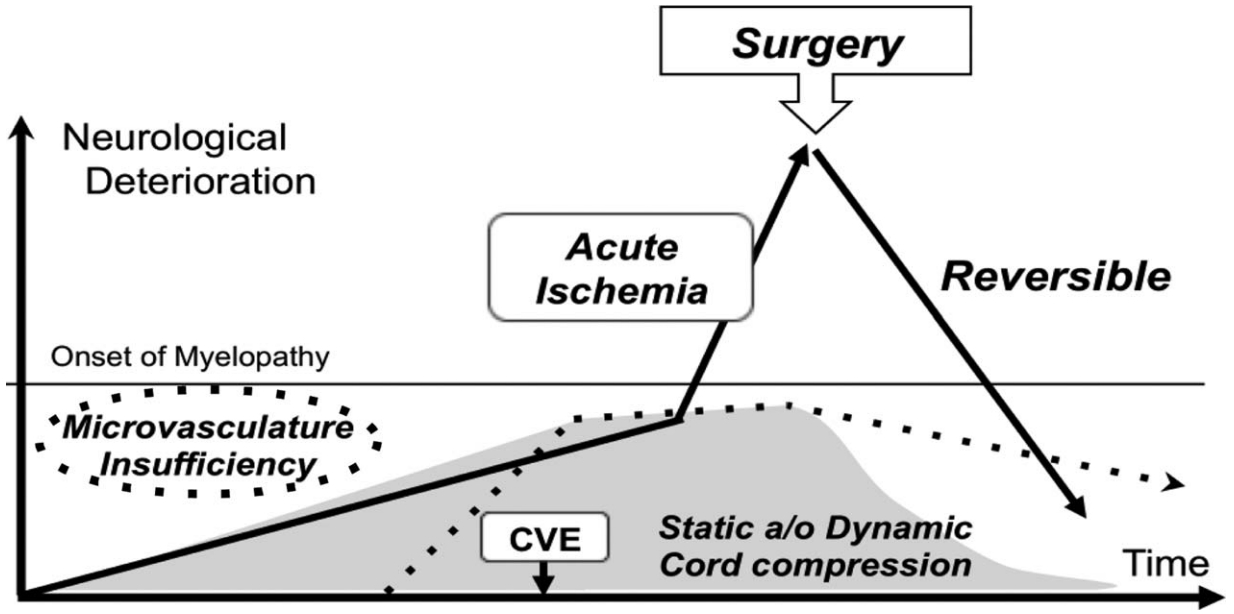

Figure 2. Schematic illustration of the pathophysiology of rapidly progressive cervical spondylotic myelopathy (rp-CSM). Acute spinal cord ischemia followed by rapidly progressive neurological deterioration in CSM may be caused by static/dynamic cord compression and microvascular insufficiency induced by a history of CVE. CVE indicates cardiovascular event.

changes and individual differences on the cervical canal diameter). In terms of dynamic factors, Oshima *et al*[1] found that a large cervical ROM ($\geq 50^{\circ}$) and segmental instability at the narrowest canal were related to the progression of myelopathy in c-CSM of mild severity. In particular, the role of cervical instability should be discussed when considering the pathophysiology of rp-CSM. Dynamic anterior slip[6] and spondylolisthesis[14] have been reported as factors associated with rp-CSM. Morishita *et al*[6] hypothesized that dynamic stress in addition to static cord compression may play an important role in the rapid progressive clinical deterioration of CSM. This issue remains controversial because static and dynamic factors might interact with each other in both c- and rp-CSM.

In the present report, we summarized the pathophysiology of rp-CSM as follows: acute spinal cord ischemia followed by rapidly progressive neurological deterioration in CSM may be caused by static/dynamic cord compression and microvascular insufficiency induced by a history of CVE (Figure 2).

The Surgical Outcome and Its Significance in Rapidly Progressive Cervical Spondylotic Myelopathy

In the patients with rp-CSM, the preoperative total score and each domain of the JOA score, including the motor/sensory functions of the limbs and the bladder function, were significantly lower than in the c-CSM patients. Nevertheless, the JOA score recovery rate of the patients with rp-CSM at the 1-year follow-up was significantly higher than that of the c-CSM patients.

A history of CVE may make spine surgeons hesitant to conduct decompression surgery out of concern about perioperative complications such as stroke/heart attack, postoperative hematoma, and subsequent paralysis due to anticoagulant therapy. In the present study, there were no such perioperative complications, and the surgical outcomes were satisfactory. Surgically treated rp-CSM subjects regained their hand dexterity and walking ability at the final follow-up examination.

Spinal ischemia must be treated in a time-sensitive manner, because evidence suggests that the rapid restoration of blood flow may improve patients' outcomes.[19,23,24] A recent study showed that delayed surgical decompression and prolonged cord ischemia causes an excessive increase in the spinal blood flow after surgery and exacerbates ischemia-reperfusion injury (IRI). The authors also found that IRI was associated with ongoing enhancement of the cytokine expression level, microglial activation, and astrogliosis and paralleled a poorer neurological recovery.[25] Thus, early surgical decompression can restore appropriate blood flow in spinal cord and avoid secondary spinal neuronal damage and death induced by inflammatory chemokines due to IRI.[25,26]

Surgical treatment should therefore be considered for subjects with rp-CSM and performed without missing the treatment opportunity window (Table 3). However, informed consent should be obtained, because it has been reported that comorbidities on admission are associated with an increased risk of perioperative stroke among patients undergoing spinal surgery.[27]

Limitations

The present study was associated with several limitations. First, the definition of rp-CSM was based on the clinical features, and biological evidence is still lacking. However, the previously mentioned findings[6,19,23–26] conceptually

support the pathophysiology of rp-CSM described in this study. Second, we could not confirm whether or not MR T2-hyperintensity indicates temporary spinal edema or permanent neuronal changes over the long term. In this study, MR T2-hyperintensity was associated with the presence of rp-CSM but not a poor surgical outcome. Future studies should investigate the characteristics and pattern of the MR signal changes in both T1 and T2 images to promote an understanding of the pathophysiology of rp-CSM. Third, the optimum timing of surgery for rp-CSM was difficult to determine. As previously mentioned, early decompression surgery may be recommended and efficient; however, we did not dichotomize and compare rp-CSM patients who received early or delayed surgery. The rp-CSM patients underwent early surgery as a priority because of their disease severity on admission and the rapid progression of their neurological deterioration. Thus, the shorter waiting period for surgery may not be linked to a better surgical outcome in this study. Further studies will be needed to clarify the optimum management of rp-CSM. Finally, the number of subjects investigated in this study was limited. Future studies with a larger cohort of patients are clearly needed to further confirm the results of our analyses.

CONCLUSION

In the present study, we identified the clinical characteristics of rp-CSM and noted that rp-CSM presents with rapidly progressive neurological deterioration over a period of a few weeks, and that a history of CVE and intramedullary MR T2-hyperintensity are independent risk factors for rp-CSM. Our results also suggested that microvascular dysfunction and subsequent spinal edema may be important pathophysiological factors in rp-CSM.

In managing patients with CSM, we should take care not to miss the existence of rp-CSM. Despite rapid neurological deterioration, patients with rp-CSM showed a good neurological recovery after surgery, which indicates that rp-CSM is a reversible condition. Decompression surgery should therefore be planned after appropriate neurological and differential diagnosis have been made.

➢ Key Points

- ❑ The patients with rp-CSM had difficulty maintaining a standing posture or walking without support within 4 weeks of the symptomatic onset due to rapidly progressive neurological deterioration.
- ❑ The independent risk factors for rp-CSM were a history of CVE (OR = 4.7) and intramedullary MR T2-hyperintensity (OR = 12.5).
- ❑ Decompression surgery is effective and recommended for managing rapidly progressive neurological deterioration in CSM, because this condition is reversible.

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