The Philippine Journal of Orthopaedics

SYSTEMATIC REVIEW

Clinical and Radiographic Predictors of Deterioration in Mild Cervical Spondylotic Myelopathy: A Systematic Review

Jose Kreisher Rae B. Foscablo, MD,[1] John David B. Mata, MD, FAFN,[1,2] Immanuel V. Silverio, MD,[3] Ronald P. Tangente, MD, MBA, FPOA, FPSS[1,2]

[1]*Department of Neuroscience, Davao Doctors Hospital, Davao City, Philippines*
[2]*Spine and Scoliosis Center, Davao Doctors Hospital, Davao City, Philippines*
[3]*Department of Neuroscience, Western Visayas Medical Center, Iloilo City, Philippines*

ABSTRACT

Background and Objectives. The "best" approach for mild cervical spondylotic myelopathy (CSM) is still unclear, and the decision to perform outright surgery remains a topic of debate. Therefore, identifying clinical and imaging predictors of deterioration is crucial.

Methodology. This study followed the PRISMA Guidelines and reviewed published articles from 2000 to 2023 that involved adult patients with asymptomatic spondylotic cord compression and/or mild CSM who underwent conservative management. The search was conducted in MEDLINE via Pubmed, Cochrane Central Register of Controlled Trials, Herding Plus, Embase, and Google Scholar. Patient demographics, neurologic outcome, and clinical and imaging predictors were examined. We assessed study quality using the Newcastle-Ottawa Scale (NOS) for observational studies. We reported statistical data as presented and calculated RRs or ORs if not provided. Evidence quality was evaluated using the GRADE approach.

Results. Twelve studies were included, consisting of 1,046 patients. Cervical radiculopathy, electrophysiological abnormalities (EMG, SEP, MEP), decreased Torg ratio <0.80, cervical range of motion of >50°, and cervical instability (slippage >2 mm or segmental kyphosis) were significantly associated with myelopathy progression. MRI T2 hyperintensity of the spinal cord was associated with poor outcomes and delayed development of myelopathy. Furthermore, CSF column diameter, circumferential cord compression, cord T1 angular deformity, cross-sectional area (CSA) <70.1 mm^2, and cord compression ratio <0.4 were independent predictors of developing myelopathy. Progression was associated more with focal than with diffuse disc herniation.

Conclusion. Early recognition of clinical features and imaging predictors of deterioration may help clinicians decide when to do early surgery in patients with mild CSM. Consensus is still needed on the role of surgery in patients with mild CSM. Patients may exhibit improvement, stability, or deterioration following conservative measures.

Keywords. cervical, myelopathy, deterioration, systematic review, surgery

KEY POINTS

- The natural progression of mild CSM has not been clear. There are no definitive recommendations regarding the need for surgery for its management.
- Identifying clinical and imaging predictors of deterioration can help clinicians anticipate symptom progression. Early recognition of these predictors can also assist clinicians in deciding on initiating early surgery, avoiding neurologic deterioration.
- Consensus is still needed on the role of surgery in patients with mild CSM. Patients may exhibit improvement, stability, or deterioration following conservative measures.

eISSN 2012-3264 (Online)
Printed in the Philippines.

Received: February 27, 2027.
Accepted: April 15, 2024.
Published Online: May 3, 2024.
https://doi.org/10.69472/poai.2024.10

Corresponding author:
Jose Kreisher Rae B. Foscablo, MD
Department of Neurosurgery, Davao Doctors Hospital
118 Elpidio Quirino Ave, Poblacion District,
Davao City, 8000 Davao del Sur, Philippines
Tel. No.: (082) 222 8000
E-mail: josekreisherraebfoscablo@gmail.com

INTRODUCTION

Cervical spondylotic myelopathy (CSM) is a condition that develops from the degenerative processes of the spine. These processes lead to facet hypertrophy, disc degeneration, vertebral subluxation, and ossification of ligamentous structures. Narrowing of the spinal canal can cause spinal cord compression, leading to vascular compromise and potentially causing irreversible damage to the involved area.[1-3] Surgery is recommended for moderate to severe CSM, while its role is unclear in mild CSM. Moreover, studies showed that rigorous conservative management can be as effective as surgery in this population.[4-6]

The decision to proceed with surgery remains a dilemma. Doing aggressive surgery may result in unnecessary risks and complications. Conversely, patients undergoing conservative management may experience neurologic deterioration.[6] Therefore, early detection of those at risk of progression and those who will benefit from early surgery is paramount. Likewise, it is crucial to identify the clinical and imaging predictors of deterioration.

This study was conducted to determine the clinical and imaging predictors of neurologic deterioration in patients with mild CSM. Moreover, this will also guide clinicians in identifying patients at risk of deterioration and those who might benefit from early surgery. The results should contribute to clinicians' decision-making when treating this patient population.

Table 1. Nurick grade[24]

Nurick Grade	Clinical Presentation
0	Root signs or symptoms. No evidence of cord involvement
1	Signs of cord involvement. Normal gait.
2	Mild gait abnormality. Able to be employed.
3	Gait abnormality prevents employment.
4	Able to ambulate only with assistance.
5	Chair bound or bedridden.

METHODOLOGY

This systematic review used the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) Guidelines.

Eligibility criteria

This study reviewed published articles from 2000 to 2023 that involved adults with asymptomatic spondylotic cord compression and mild CSM (Nurick I-II, mJOA 15-17, JOA>13) (Tables 1 to 3) who underwent conservative management. The following were excluded: 1) manuscripts involving moderate to severe CSM; 2) case series/case reports; 3) conference or poster abstracts; and 4) studies published in languages other than English.

Table 2. Modified Japanese Orthopedic Association (mJOA) Score[25]

Category	Score	Description
Upper Extremity Motor Subscore (/5)	0	Unable to move hands
	1	Unable to eat spoon nut able to move hands
	2	Unable to button a shirt but able to eat with a spoon
	3	Able to button a shirt with great difficulty
	4	Able to button a shirt with mild difficulty OR other mild fine motor dysfunction (marked handwriting change, frequent dropping of objects, difficulty clasping jewelry, etc.)
	5	Normal hand coordination
Lower Extremity Motor Subscore (/7)	0	Complete loss of movement and sensation
	1	Complete loss of movement, some sensation present
	2	Inability to walk but some movement
	3	Able to walk on flat ground with walking aid
	4	Able to walk without walking aid, but must hold a handrail on stairs
	5	Moderate to severe walking imbalance but able to perform stairs without handrail
	6	Mild imbalance when standing OR walking
	7	Normal walking
Upper Extremity Sensory Subscore (/3)	0	Complete loss of hand sensation
	1	Severe loss of hand sensation OR pain
	2	Mild loss of hand sensation
	3	Normal hand sensation
Urinary Function Subscore (/3)	0	Inability to urinate voluntarily (requiring catheterization)
	1	Frequent urinary incontinence (more than once per month)
	2	Urinary urgency OR occasional stress incontinence (less than once per month)
	3	Normal urinary function
Mild	15-17	
Moderate	12-14	
Severe	<14	

Table 3. Japanese Orthopaedic Association Score[26]

Motor function	
Upper Extremity	
0	Unable to feed oneself with any tableware including chopsticks, spoon, or fork, and/or unable to fasten buttons of any size
1	Possible to eat with spoon but not with chopsticks
2	Possible to eat with chopstick, but inadequate
3	Possible to eat with chopstick, but awkward
4	Normal
Lower extremity	
0	Unable to stand up and walk by any means
1	Need cane or aid on flat ground
2	Need cane or aid only on stairs
3	Possible to walk without cane or aid, but slow
4	Normal
Sensory function	
Upper extremity	
0	Apparent sensory loss
1	Minimal sensory loss
2	Normal
Trunk	
0	Apparent sensory loss
1	Minimal sensory loss
2	Normal
Lower extremity	
0	Apparent sensory loss
1	Minimal sensory loss
2	Normal
Bladder function	
0	Complete uninary retention
1	Severe disturbance
2	Mild disturbance
3	Normal
Mild	>13
Moderate	9-13
Severe	<9

Search strategy

An electronic search in MEDLINE via Pubmed, Cochrane Central Register of Controlled Trials, Herdin Plus, Embase, and Google Scholar was done, with the following search terms: (Asymptomatic spondylotic compression, mild cervical spondylotic myelopathy, degenerative cervical myelopathy, clinical predictors, imaging predictors, predictive factors of deterioration) AND (mild cervical spondylotic myelopathy management, deterioration, progression). The search used Boolean operators such as AND and OR to combine these terms effectively. Database-specific limiters for publication dates were used. A bibliography search was done within the references of included studies. Hand-searching of printed journals was not done.

Selection of studies

Three authors (JF, JM, IS) independently screened the titles and abstracts. Eligible full-text articles were retrieved. Full-text copies of potentially relevant papers selected by at least one author were also reviewed. Articles that met the inclusion criteria were assessed independently, with any inconsistencies resolved through consensus.

Data extraction and management

The primary outcome for analysis was neurologic status on the initial and follow-up period (graded by mJOA, JOA, or Nurick grading), and clinical and imaging predictors of deterioration in mild CSM patients. Study title, author, date of publication, country where the study was conducted, study design, patient demographics, factors predicting deterioration, follow-up duration, and summary of results were extracted using a standardized extraction form. Data was independently retrieved and assessed by the three authors. Data was cross-checked, and discrepancies were resolved by a fourth author (RT).

Assessment of risk of bias

For the methodological quality of the individual studies, the Newcastle Ottawa Scale (NOS) for observational studies was used to score representativeness, sample size, ascertainment of exposure, and outcome. A score of 8–9 represents a low risk of bias; 6–7, a medium risk of bias; and 5 or lower, a high risk of bias. A score of at least 6 was considered acceptable. The scoring was done by three authors (JF, JM, IM). If a consensus was not reached, the fourth author (RT) settled the issue (Table 4).

Data synthesis

Studies that fulfilled the inclusion criteria were eligible for synthesis. Descriptive statistics for all studies were reported as is because there was variability in the reporting of prognostic factors and frequency of outcomes. For prognostic factors that were reported as proportions (e.g., percentage of the sample with a Pavlov ratio <0.8), we reported the calculated relative risks (RRs) or odds ratio (OR) and their 95% CIs if not provided by the authors. For continuous prognostic factors (e.g., neck range of motion [ROM]), we reported analytical statistics as detailed by the authors. We recorded comparative statistics if included in the reviewed articles. All calculations were performed using the Statistical Packages for the Social Sciences (SPSS version 29). In case of missing data or if raw data was unavailable, we tried contacting the corresponding authors through the email address listed in their article. We used narrative synthesis to summarize the results for data that could not be retrieved. Due to the heterogeneity of the included articles, meta-analysis was not conducted. Therefore, a qualitative synthesis was done (Table 5).

Strength of evidence

We used the GRADE (Grades of Recommendation, Assessment, Development, and Evaluation) framework to assess the overall quality of evidence. Five essential factors

Table 4. Newcastle-Ottawa scale for risk-of-bias

Studies	Selection				Comparability		Outcome			Score
Bednarik (2004)[2]	*	*	*	*	*	*	*	*	*	9
Bednarik (2008)[8]	*	*	*	*	*	*	*	*	*	9
Matsumoto (2000)[22]	*	*	*	*	*	*	*	*	–	8
Cao (2017)[11]	*	*	*	*	*	*	*	–	–	7
Shimomura (2007)[13]	*	*	*	*	*	*	*	*	*	9
Matsumoto (2001)[14]	*	*	*	*	*	+	*	*	*	9
Yoshimatsu (2001)[5]	*	*	*	*	*	–	*	*	*	8
Oshima (2012)[6]	*	*	*	*	*	*	*	*	*	9
Kong (2013)[1]	*	*	*	*	*	*	*	*	*	9
Sumi (2012)[12]	*	*	*	*	*	*	*	*	*	9
Kadanka (2017)[15]	*	*	*	*	*	*	*	*	*	9
Feng (2020)[9]	*	*	*	*	*	*	*	*	–	8

could decrease the quality of evidence: (1) Limitations in study design and/or execution; (2) inconsistency of results; (3) indirectness of evidence; (4) imprecision of results; and (5) publication bias. There were also three factors for upgrading. Three independent reviewers (JF, JM, and IS) assessed the quality of the evidence. We downgraded the quality of evidence by one level if one of the factors discussed was met.

The following grading of quality of evidence and definitions were used: (1) High quality: Further research is very unlikely to change our confidence in the estimate of effect; (2) Moderate quality: Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate; (3) Low quality: Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate; and (4) Very low quality: Any estimate of effect is very uncertain (Table 6).

RESULTS

Retrieval of studies

Our literature search resulted in 563 articles. We excluded 26 duplicate studies. Another 537 studies were excluded because of irrelevant titles. We reviewed 23 full-text articles for eligibility. Out of 23 articles, 12 were included in the review based on inclusion criteria (Figure 1). Specifically, the study

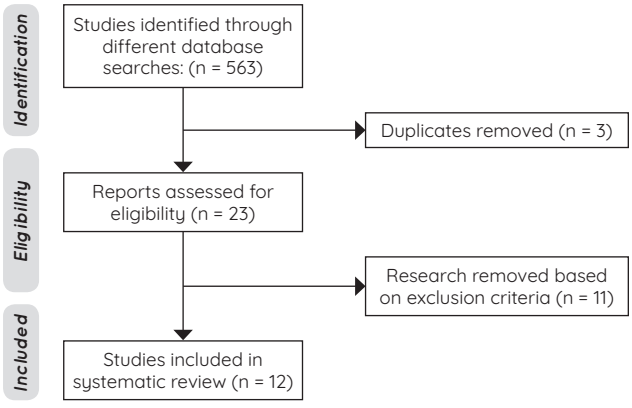

Figure 1. Flow of study selection.

of Koyanagi et al., was excluded because it involved only a surgical group of mild CMS patients.[7]

Study characteristics

The included studies were published from 2000 through 2023. The major publishing countries were Japan (n = 6), the Czech Republic (n = 3), and China (n = 3). The studies contained a total of 1,046 patients. The primary outcomes were initial and follow-up neurologic scores (mJOA, JOA, or Nurick), and clinical and imaging predictors of deterioration. The study characteristics are summarized in Table 5.

Risk of bias assessment

Almost all studies were observational. Based on the Newcastle-Ottawa score, all were at low risk for bias. One study did not discuss the follow-up period and adequacy of follow-up (Table 4).

Radiculopathy, SEP, MEP, EMG and progression

In the study by Bednarik et al., involving 66 patients presenting with radiculopathy or axial pain, results from univariate logistic regression analysis indicated a significant correlation between elevated risk of myelopathy and the presence of radiculopathy (OR = 36.92, 95% Cl = 4.19–325.50, p = 0.001), anterior horn cell abnormality through EMG (OR = 3.51, 95% CI = 0.89–13.87, p = 0.001), and Sensory Evoked Potential (SEP) abnormality (OR = 12.5, 95% CI = 2.97–52.41, p = 0.016). Other factors were not associated with CSM development. Male gender was noted to be significantly associated with myelopathy based on univariate analysis (OR = 4.02, 95% CI = 1.06–16.8, p = 0.038), but this was significantly associated with radiculopathy and thus was not considered as an independent factor.[2]

Their follow-up study (2008), involving 199 subjects presenting with radiculopathy or axial pain, showed that radiculopathy (RR = 3.68, CI = 2.03–6.69, p = 0.001), SEP (RR = 3.21, CI = 1.75–5.87, p = 0.001) and Motor Evoked Potential (MEP) abnormalities (RR = 2.91, CI = 1.60–5.29,

Table 5. Study characteristics and results

Author, year, type of study	Locale	Demographics	Age (years)	Follow-up	Factors predictive of neurologic deterioration	Results	Summary
***Yoshimatsu*, *2001*[5]** Retrospective Observational	Japan	N = 69 Conservative = 47 Surgical = 22 Male 35 Female 34	Mean age = 67 (42-87)	Mean: 38-39 months	• Duration of symptoms • Presence of intensive conservative treatment	Multivariate analysis: improvement correlated with shorter disease duration (p = 0.0141) Progression was associated with longer disease duration (p = 0.001)	Multivariate analysis showed significant correlations between clinical outcome, disease duration, and rigorous conservative treatment. Conservative treatment for CSM is considered to be effective if it is performed intensively on selected patients.
***Bednarik*, *2004*[2]** Prospective Observational	Czech	N = 66 Male 34 Female 32	Median age = 50 (32-75)	Minimum: 2 years	• Abnormal motor evoked potential (MEP) • Abnormal EMG • Clinical signs of radiculopathy	Univariate logistic regression analysis Radiculopathy and myelopathy (OR = 36.92, 95% CI = 4.19-325.50, p = 0.001) Anterior horn cell abnormality through EMG (OR = 3.51, 95% CI = 0.89-13.87, p = 0.001) SEP abnormality (OR = 12.5, 95% CI = 2.97-52.41, P = 0.016)	Electrophysiological abnormalities together with clinical signs of cervical radiculopathy could predict clinical manifestation of preclinical spondylotic cervical cord compression.
***Bednarik*, *2008*[8]** Prospective Observational	Czech	N = 199 Male 104 Female 94	Median = 51 (28-82)	Median: 44 months Minimum: 2 years	• Abnormal motor evoked potential (MEP) • Abnormal EMG • MRI hyperintensity (>12 months)	Radiculopathy and Myelopathy radiculopathy (RR = 3.68, CI = 2.03-6.69, p = 0.001) SEP and myelopathy (RR = 3.21, CI = 1.75-5.87, p = 0.001) MEP abnormalities and myelopathy (RR=2.91, CI=1.60-5.29, p = 0.001) T2 hyperintensity is associated with later (>12 months) manifestation of myelopathy (P = 0.001)	Electrophysiological abnormalities of cervical cord dysfunction together with clinical signs of cervical radiculopathy and MRI hyperintensity are useful predictors of early progression into symptomatic SCM in patients with P-SCCC.
***Kadanka*, *2017*[15]** Prospective Observational	Czech	N = 112 Male 57 Female 55	Median age = 59 (40-79)	Minimum: 2 years (mean 3 years)	• Radiculopathy • Electrophysiological (SEP, MEP, EMG) • Cross-sectional area • Compression ratio	Cross-sectional area (CSA) of less than or equal to 70.1 mm^2 (OD = 6.16, 95% CI = 1.61-23.72, p = 0.008) as well as compression ratio of less than or equal to 4.0 (OD = 5.61, 95% CI = 1.45-21.7, p = 0.012) were independent predictors of developing myelopathy	In addition to previously described independent predictors of DCM development (radiculopathy and electrophysiological dysfunction of cervical cord), MRI parameters, namely CSA and CR, should also be considered as significant predictors for the development of DCM.
***Matsumoto*, *2000*[11]** Retrospective Observational	Japan	N = 52 Male: 39 Female: 13	Mean age = 55 (30-80)	Mean: 3 years	• Increased Signal Intensity • Diameter of spinal canal	T2 signal hyperintensity and transverse area of the spinal cord do not correlate with poor outcome	Increased signal intensity was not related to a poor outcome of conservative treatment or severity of myelopathy in the patients with mild cervical myelopathy.
***Matsumoto*, *2001*[14]** Retrospective Observational	Japan	N = 27 Male: 20 Female: 7	Mean age = 44.4 (27-69)	Mean: 3.9 years	• Type of herniation • Level of herniation	Although not statistically significant (p = 0.15), diffuse-type herniation spontaneously regressed more frequently than focal-type	Conservative treatment is an effective treatment option for mild cervical myelopathy caused by cervical soft disc herniation. A good outcome can be expected in patients with a median-type and/or diffuse-type herniation on magnetic resonance imaging.
***Oshima*, *2012*[6]** Retrospective Observational	Japan	N = 45 Male: 27 Female: 18	Mean age =59(35-76)	Mean: 78 months	• Cervical range of motion • Segmental kyphosis • Local slip	Segmental lordotic angle at the maximum segment was significantly higher in nonsurgical patients (cox HR = 4.51, CI = 1.59-12.8, p = 0.001) Local slip of >2 mm at the involved segment was also significantly higher in the surgical group (cox HR = 4.67, CI = 1.67-13.0, p = 0.03)	Fifty-six percent of patients with clinically mild CSM with increased signal intensity (ISI) had not deteriorated or undergone surgery at 10 years. Large range of motion, segmental kyphosis, and instability at the narrowest canal were adverse prognostic factors.

Table 5. Study characteristics and results *(continued)*

Author, year, type of study	Locale	Demographics	Age (years)	Follow-up	Factors predictive of neurologic deterioration	Results	Summary
Cao, 2017[10] Retrospective Observational	China	N = 68 Male: 37 Female: 31	Mean age = 52.6 (38-72)	–	• Torg ratio • Cervical spine instability • MRI T2 weighted high intensity	>3.5 mm listhesis, was noted to be associated with symptomatic cases (*P* <0.05) myelopathy was dependently associated with cervical instability (OR 5.898, *P* = 0.037) range of motion >50 degrees at the maximum segment involved was also noted to be significantly higher in the surgical group (cox HR: 3.25, CI = 0.42-3.17, *p* = 0.04 symptomatic cases were significantly associated with T2 hyperintensity, showing a significant difference from asymptomatic cases (*p* <0.05) Further analysis showed that myelopathy was associated with T2 hyperintensity (OR = 9.718, *P* = 0.020) Torg-Pavlov Ratio was significantly associated with asymptomatic cases compared to those who present with symptoms (*P* <0.05) Myelopathy was dependently associated with a decreased Torg-Pavlov ratio (OR = 0.155, *P* = 0.006)	Cervical segmental instability, a high intramedullary signal on T2-weighted MRI, and the Torg ratio had the greatest capacity to distinguish between asymptomatic and symptomatic patients with CSM with mild to moderate cervical spinal cord compression.
Shimomura, 2007[13] Prospective Observational	Japan	N = 70 Male: 49 Female: 21	Mean age = 55.1 ± 11.8	Mean: 35.6 months	• Circumferential spinal cord compression	The only noted factor associated with deterioration in mild CSM was circumferential spinal cord compression on axial MRI (OR = 26.624, CI = 1.682-421.541, *P* <0.05)	Outcomes of mild forms of CSM during nonsurgical treatment were generally good as shown by average JOA scores. The only prognostic factor for mild forms of CSM was circumferential spinal cord compression in the maximum compression segment on axial MRI. Surgical treatment can be considered for patients with this prognostic factor.
Sumi, 2012[12] Prospective Observational	Japan	N = 60 Male: 42 Female: 18	Mean age = 56.1 ± 11.8	Mean: 94.3 months	• Spinal cord shape (angular) • MRI imaging findings	Ovoid deformity and T2 hyperintensity were noted to have no significant statistical relationship with myelopathy deterioration angular deformity of the cord on T1-weighted axial scans was significantly correlated with myelopathy deterioration (OR = 8.22, *P* = 0.006)	The tolerance rate of mild CSM was 70% in this study, which proved that the prognosis of mild CSM without surgical treatment was relatively good. However, the tolerance rate of the cases with angular-edged deformity was 58%. Therefore, surgical treatment should be considered when mild CSM cases show angular-edged deformity on axial MR imaging, even if patients lack significant symptoms.
Kong, 2013[1] Prospective Observational	China	N = 78 Male: 45 Female: 33	Mean age = 57.8 (37-71)	Mean: 40 months	• Segmental instability • Cervical spinal stenosis	segmental instability (>2 mm slippage) was significantly associated with the surgical group (*P* = 0.01) diameter of the CSF column was also correlated with deterioration (10.7 ± 1.8 vs. 12.1 ± 1.2 mm, *P* = 0.02)	Conservative treatment is effective in MCSM patients. Patients with segmental instability and cervical spinal stenosis tend to deteriorate, but conservative treatment remains the recommendation for the first action. If the myelopathy deteriorates during conservative treatment, timely surgical intervention is effective.
Feng, 2020[9] Prospective Observational	China	N = 200 Male: 92 Female: 108	Mean age = 55.8 ± 8.7 (28-80)	1-year follow-up	• Sensory Evoke Potential (SEP) waveform	The incidence of progressive myelopathy was significantly correlated with upper SEP (r = 0.94, *p* <0.01) and the combined SEPs (r = 0.95, *p* <0.01)	The incidence of progressive degenerative myelopathy increased with the upper and combination SEP classifications. Thus, classification of SEPs could predict the clinical decline in mJOA in CSM, reflecting the probability of worsening of myelopathy.

Table 6. GRADE certainty of evidence

Outcomes	Number of studies	Impact	Certainty of Evidence	Comments
Myelopahy progression and Radiculopathy, electrophysiologic abnormalities	3 non-randomized studies	• Radiculopathy and electrophysiologic abnormalities are associated with the progression of myelopathy in two studies	⨁◯◯◯ Very low	• Purely observational studies • Two studies are similar, the latest study is the updated one
Myelopathy and segmental instability	3 non-randomized studies	• Segmental instability was associated with progression of myelopathy in three studies	⨁◯◯◯ Very low	• Purely observational studies • Heterogeneity exists among studies • No data on follow-up in one study
Myelopathy and T2 hyperintensity	4 non-randomized studies	• T2 hyperintensity was associated with myelopathy in two studies, no correlation was established in the other two studies	⨁◯◯◯ Very low	• Purely observational studies • Heterogeneity exists among studies
Myelopathy and Torg-Pavlov Ratio	1 non-randomized studies	• Torg ratio had the greatest capacity to distinguish between asymptomatic and symptomatic patients with CSM with mild to moderate cervical spinal cord compression	⨁◯◯◯ Very low	• Purely observational studies • Lack of follow-up data
Myelopathy and T1 Cord Deformity, Circumferential Cord Compression, CSA <70 mm, Compression Ratio	4 non-randomized studies	• Circumferential cord compression was associated with progression in one study • Angular deformity in t1 was also associated with deterioration • CSF column diameter was correlated with deterioration • CSA and CR were correlated with myelopathy development	⨁◯◯◯ Very low	• Purely observational studies • Heterogeneity exists among studies
Myelopathy and Disc herniation	1 non-randomized study	• Conservative treatment is an effective treatment option for mild cervical myelopathy caused by cervical soft disc herniation. A good outcome can be expected in patients with a median-type and/or diffuse-type herniation on magnetic resonance imaging	⨁◯◯◯ Very low	• Purely observational studies • The sample size is limited

Table 7. Sensory Evoked Potentials (SEP) waveforms by Feng et al.[9]

SEP waveforms	
Class I	Normal amplitude and latency
Class II	with normal latency but abnormal amplitude
Class III	with normal amplitude but abnormal latency
Class IV	abnormal latency and abnormal amplitude
Class V	with unmeasurable or absent waveforms

p = 0.001) contributed independently to the development of myelopathy and constituted a very significant prediction model for this endpoint. Furthermore, the risk of the development of early myelopathy was seen contributed by radiculopathy (OR = 4.69, CI = 1.61–13.7, p = 0.004), SEP (OR = 3.97, CI = 1.36–11.6, p = 0.011) and MEP (OR = 2.94, CI = 1.04–8.75, p = 0.001).[8]

Feng et al., conducted a study involving 200 patients, classifying 5 types of SEP waveforms (Table 7). They reported that waveforms can predict the deterioration of CSM and correlate with disease progression. According to them, the incidence of progressive myelopathy was 2.6%, 27.7%, 23.8%, 86.7%, and 100% in Class I, II, III, IV, and V of upper SEPs, respectively, and 18.8%, 39.4%, 42.3%, and 83.3% in Class I, II, III, and IV of lower SEPs, respectively. The incidence of myelopathy was 0%, 13.7%, 24.3%, 91.1%, and 100% in Class I, II, III, IV, and V respectively for the combined SEPs. The incidence of progressive myelopathy was significantly correlated with upper SEP (r = 0.94, p <0.01) and the combined SEPs (r = 0.95, p <0.01).[9]

Imaging features and progression

Segmental instability

Oshima et al., revealed that the segmental lordotic angle at the maximum segment was significantly higher in nonsurgical patients (cox HR = 4.51, CI = 1.59–12.8, p = 0.001). Association with a local slip of more than 2 mm at the involved segment was also significantly higher in the surgical group (cox HR = 4.67, CI = 1.67–13.0, p = 0.03).[6] A retrospective study by Cao et al., of 68 patients found that cervical instability and a listhesis greater than 3.5 mm were noted to be associated with symptomatic cases (p <0.05). Further analysis showed that myelopathy was dependently associated with cervical instability (OR 5.898, p = 0.037).[10] In addition, Kong et al., noted that segmental instability (>2 mm slippage) was significantly associated with the surgical group (p = 0.01).[1] Oshima et al., found that a range of motion >50° at the maximum segment involved was also noted to be significantly higher in the surgical group (cox HR: 3.25, CI = 0.42–3.17, p = 0.04). According to this study, segmental kyphosis, local slip, and ROM of >50° were adverse prognostic factors in mild CSM patients.[6]

T2 Hyperintensity

In the study of Bednarik et al., MRI hyperintensity was associated with later (>12 months) manifestation of myelopathy (p = 0.001).[8] Cao et al. found that symptomatic cases were significantly associated with T2 hyperintensity, showing a significant difference from asymptomatic cases (p <0.05). Further analysis showed that myelopathy was associated with T2 hyperintensity (OR = 9.718, p = 0.020).[10] On the other hand, the study of Matsumoto et al. concluded that T2 signal hyperintensity and transverse area of the spinal cord do not

correlate with poor outcomes.[11] Furthermore, Sumi et al. showed that T2 hyperintensity was noted to have no significant statistical relationship with myelopathy deterioration.[12]

Torg-Pavlov ratio

Cao et al. studied 68 patients, finding that an increased Torg-Pavlov Ratio was significantly associated with asymptomatic cases compared to those who present with symptoms (p <0.05). However, the range of motion of the affected segment did not correlate with symptomatology. Further analysis showed that myelopathy was dependently associated with a decreased Torg-Pavlov ratio (OR = 0.155, p = 0.006).[10]

T1 Cord deformity and circumferential cord compression

Shimomura et al. showed that mild CSM can be conservatively managed with good results. The only factor associated with deterioration in mild CSM was circumferential spinal cord compression on axial MRI (OR = 26.62, CI = 1.68–421.54, p <0.05).[13] Sumi et al. investigated 55 patients with mild CSM and found that angular deformity of the cord on T1-weighted axial scans was significantly correlated with myelopathy deterioration (OR = 8.22, p = 0.006). Ovoid deformity and T2 hyperintensity were noted to have no significant statistical relationship with myelopathy deterioration. The overall tolerance rate of mild CSM is 70%, and the tolerance rate of those subgroups having angular deformity of the cord was decreased to 58%.[12]

Other findings

According to Kong et al., the diameter of the CSF column was also correlated with deterioration (10.7 ± 1.8 vs. 12.1 ± 1.2 mm, p = 0.02) resulting in surgery. No significant differences were identified between the two groups in relation to age, gender, duration of disease, C2-C7 angle, MRI ISI and levels and degree of SC compression.[1]

Matsumoto et al. noted that diffuse-type disc herniation may be conservatively treated in patients with mild CSM. Although not statistically significant (p = 0.15), diffuse-type herniation spontaneously regressed more frequently than focal type. More aggressive measures should be done in patients with a focal type of disc herniation in patients with mild CSM.[14]

A study by Kadanka et al. consisting of 112 subjects found that aside from radiculopathy, cross-sectional area (CSA) of less than or equal to 70.1 mm^2 (OD = 6.16, 95% CI = 1.61–23.72, p = 0.008) as well as compression ratio of less than or equal to 4.0 (OD = 5.61, 95% CI = 1.45–21.7, p = 0.012) were independent predictors of developing myelopathy in asymptomatic patients with cervical cord compression.[15]

DISCUSSION

The composite analysis of data derived from various studies consistently revealed that the following factors have a substantial correlation with the progression of myelopathy: cervical radiculopathy, somatosensory evoked potentials, motor evoked potentials, EMG alterations indicating anterior horn involvement, cervical instability (slippage >2 mm or segmental kyphosis), circumferential compression of the cord, CSF column diameter, and angular deformity at T1-weighted axial scan. T2-weighted signal hyperintensity and transverse spinal cord area have been associated with poor outcomes in some studies of mild CSM patients undergoing conservative treatment. However, most studies found no correlation between MRI hyperintensity and early deterioration. Nonetheless, MRI hyperintensity was identified as a determinant of delayed development of myelopathy.

Cervical radiculopathy is a strong predictor of myelopathy development and is one of the symptomatology in patients with degenerative spine disease.[8] A comprehensive set of assessments, including EMG, SEP, and MEP, should be included in the initial examination for individuals with mild CSM to identify those at risk for disease progression. Electrophysiological findings reflect the long tract involved and are a strong predictor of myelopathy progression. Degenerative changes causing canal narrowing that leads to cord compression can manifest as changes in electrophysiologic reading. The study by Kanchiku et al., noted that ¼ lateral cord compression was associated with long tract abnormalities.[16]

Segmental instability was a significant predictor of deterioration. It can lead to the progressive degeneration of the affected segments. Instability, such as vertebral slippage and segmental kyphosis, can cause an imbalance in the vector forces within each spinal unit. Abnormal soft tissue mechanics can lead to further instability and degenerative changes. The buckling of intervertebral ligaments (including the ligamentum flavum and posterior longitudinal ligament) accelerated osteophyte formation, reduction in disc space, and bulging of the disc into the spinal canal all contribute to a decrease in spinal or root canal size.[17,18] These events can further accelerate myelopathy development.

The Torg-Pavlov Ratio refers to the sagittal spinal canal-to-vertebral body ratio, which is directly correlated with spinal canal diameter by eliminating the effect of radiographic magnification.[19] A smaller value indicates a smaller canal diameter and volume. In degenerative changes in the spine, small ossification of the posterior longitudinal ligament or ligamentum flavum hypertrophy can result in significant volume change, compressing the cord and further worsening myelopathy.

MRI T2 signal hyperintensity reflects parenchymal injury to the spinal cord due to compression. These signal changes can vary from reversible (edema, inflammation) to irreversible damage (gliosis, osteomalacia, necrosis) of the cord substance.[11] According to Ramanauskas et al., there are three stages of signal hyperintensity in the cord. The early stages involve reversible edema, followed by cystic necrosis of the grey matter in the intermediate stage, and ultimately progressing into irreparable cavity and syrinx formation at later stages.[20] The irreversible

injury occurs during the intermediate and late stages; it is challenging to distinguish between reversible and irreversible stages based solely on MRI intensity. Consequently, correlating signal hyperintensity with myelopathy progression presents difficulties.

According to Shimomura et al., the sole significant prognostic factor linked with deterioration was circumferential cord compression. The study observed that circumferential compression and severe cord distortion indicate irreversible degeneration.[13] The CSF column diameter reflects the degree of canal stenosis since CSF displacement indicates narrowing before complete compression of the cord. Therefore, clinicians need to recognize that patients with more significant circumferential compression are at a higher risk of progression than those with only partial compression.

Angular T1 deformity correlates with adverse prognosis in patients with mild CSM.[12] Kemayama et al. noted that a triangular deformity was associated with more damage to the white and gray matter than an ovoid deformity; it is a critical sign of cord damage. The angular shape of the cord at the lateral recess indicates a poorer prognosis.[21] Another factor of interest is the CSA and Compression Ratio. These factors were useful in predicting symptomatic patients with cervical cord compression. In the study of Kadanka et al., the severity of compression on MRI could stratify patients in terms of further management.[15]

In patients with myelopathy secondary to disc herniation, Matsumoto et al., found that spontaneous regression was more common in patients with the diffuse type of disc herniation. In contrast, the focal type had a lesser incidence of spontaneous regression. Therefore, the latter type should be addressed aggressively.[14]

Most of the identified risk factors for CSM progression are poorly understood and further studies are needed to better understand and strengthen the knowledge about this condition.[22] Likewise, consensus is still needed on the role of surgery in patients with mild CSM. Patients may exhibit improvement, stability, or deterioration following conservative measures. Predictors can aid in identifying candidates for early surgical intervention among patients with mild CSM. Detecting such patients prone to rapid deterioration could help avert devastating neurological consequences.

LIMITATIONS

The impact of a systematic review depends on the data's quality and homogeneity. The study is limited by several factors affecting the ability to make a firm conclusion about the result. First, the study only included English articles, which may lead to an incomplete literature search, leading to publication bias. Second, the review included heterogeneous studies, and we could not conduct a meta-analysis. Thus, combining the results to make a solid basis to support the conclusion is difficult. Similar factors were discussed in different studies, however, with conflicting results. Assessing the quality of each outcome was difficult due to inconsistencies in risk factors and comparator variables. More studies are needed to establish a strong relationship. Third, some of the studies were conducted more than ten years ago, which may affect the consistency of the results.

CONCLUSION

Early recognition of clinical features such as myelopathy with radiculopathy and electrophysiologic abnormality is crucial in catching patients at risk of deterioration. Moreover, immediate identification of imaging predictors of deterioration such as segmental instability (segmental slippage > 2mm and segmental kyphosis), decreased Torg-Pavlov ratio, circumferential cord compression, CSF column diameter, and angular deformity on T1, cross-sectional area (CSA) of less than or equal to 70.1 mm^2 as well as compression ratio of less than or equal to 4.0 may compel us to do early surgery in these patients. The role of MRI T2 hyperintensity is still vague in predicting early deterioration, although it is associated with the development of myelopathy. Early surgery should be considered in patients with myelopathy and focal disc herniation. Diffuse-type herniation spontaneously regressed more frequently than the focal type. Early identification of these predictors can assist clinicians in determining the optimal timing for early surgical intervention.

RECOMMENDATIONS

The findings from this study hold significant implications for the management of mild Cervical Spondylotic Myelopathy (CSM) patients. Fehling et al. outlined guidelines indicating that patients with radiculopathy and electrophysiologic abnormalities are prone to deteriorate.[23] By including electrophysiological tests in the initial work-up, we can identify subpopulations susceptible to early deterioration, enabling us to offer timely surgical intervention to these patients. Understanding the imaging predictors of myelopathy progression can help us anticipate patients who are prone to deteriorate and determine when early surgery should be considered. However, conflicting results exist in some predictors, and further studies are necessary. By identifying these predictors, we can significantly improve patient outcomes. In addition, this study will serve as a backbone for further studies to give us robust data on the different predictors of deterioration in mild CSM that can help in decision-making. Few data involving conservative management were collected, so it is essential to conduct further studies.

STATEMENT OF AUTHORSHIP

All authors certified fulfillment of ICMJE authorship criteria.

AUTHORS DISCLOSURE

The authors declared no conflict of interest.

FUNDING SOURCE

None.

REFERENCES

1. Kong LD, Meng LC, Wang LF, Shen Y, Wang P, Shang ZK. Evaluation of conservative treatment and timing of surgical intervention for mild forms of cervical spondylotic myelopathy. Exp Ther Med. 2013;6(3):852–6. PMID: 24137278 PMCID: PMC3786935 DOI: 10.3892/etm.2013.1224
2. Bednarik J, Kadanka Z, Dusek L, et al. Presymptomatic spondylotic cervical cord compression. Spine (Phila Pa 1976). 2004;29(20):2260–9. PMID: 15480138 DOI: 10.1097/01.brs.0000142434.02579.84
3. Tetreault L, Kalsi-Ryan S, Davies B, et al. Degenerative cervical myelopathy: a practical approach to diagnosis. Global Spine J. 2022;12(8):1881–93. PMID: 35043715 PMCID: PMC9609530 DOI: 10.1177/21925682211072847
4. Kadaňka Z, Bednařík J, Novotný O, Urbánek I, Dušek L. Cervical spondylotic myelopathy: conservative versus surgical treatment after 10 years. Eur Spine J. 2011;20(9):1533–8. PMID: 21519928 PMCID: PMC3175900 DOI: 10.1007/s00586-011-1811-9
5. Yoshimatsu H, Nagata K, Goto H, et al. Conservative treatment for cervical spondylotic myelopathy. prediction of treatment effects by multivariate analysis. Spine J. 2001;1(4):269–73. PMID: 14588331 DOI: 10.1016/s1529-9430(01)00082-1
6. Oshima Y, Seichi 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 (Phila Pa 1976). 2012;37(22):1909–13. PMID: 22511231 DOI: 10.1097/BRS.0b013e318259a65b
7. Koyanagi I. Options of management of the patient with mild degenerative cervical myelopathy. Neurosurg Clin N Am. 2018;29(1):139-44. PMID: 29173426 DOI: 10.1016/j.nec.2017.09.009
8. Bednarik J, Kadaňka Z, Dušek L, et al. Presymptomatic spondylotic cervical myelopathy: an updated predictive model. Eur Spine J. 2008;17(3):421–31. PMID: 18193301 PMCID: PMC2270386 DOI: 10.1007/s00586-008-0585-1
9. Feng X, Hu Y, Ma X. Progression prediction of mild cervical spondylotic myelopathy by somatosensory-evoked potentials. Spine (Phila Pa 1976). 2020;45(10):E560–7. PMID: 31770314 DOI: 10.1097/BRS.0000000000003348
10. Cao JM, Zhang JT, Yang DL, et al. Imaging factors that distinguish between patients with asymptomatic and symptomatic cervical spondylotic myelopathy with mild to moderate cervical spinal cord compression. Med Sci Monit. 2017;23:4901–8. PMID: 29028790 PMCID: PMC5652139 DOI: 10.12659/msm.906937
11. Matsumoto M, Toyama Y, Ishikawa M, et al. Increased signal intensity of the spinal cord on magnetic resonance images in cervical compressive myelopathy. Spine (Phila Pa 1976). 2000;25(6):677–82. PMID: 10752098 DOI: 10.1097/00007632-200003150-00005
12. Sumi M, Miyamoto H, Suzuki T, Kaneyama S, Kanatani T, Uno K. Prospective cohort study of mild cervical spondylotic myelopathy without surgical treatment. JNS Spine. 2012;16(1):8–14. PMID: 21981274 DOI: 10.3171/2011.8.SPINE11395
13. Shimomura T, Sumi M, Nishida K, et al. Prognostic factors for deterioration of patients with cervical spondylotic myelopathy after nonsurgical treatment. Spine (Phila Pa 1976). 2007;32(22):2474–9. PMID: 18090088 DOI: 10.1097/BRS.0b013e3181573aee
14. Matsumoto M, Chiba K, Ishikawa M, et al. Relationships between outcomes of conservative treatment and magnetic resonance imaging findings in patients with mild cervical myelopathy caused by soft disc herniations. Spine (Phila Pa 1976). 2001;26(14):1592–8. PMID: 11462093 DOI: 10.1097/00007632-200107150-00021
15. Kadanka Z Jr, Adamova B, Kerkovsky M, et al. Predictors of symptomatic myelopathy in degenerative cervical spinal cord compression. Brain Behav. 2017;7(9):e00797. PMID: 28948090 PMCID: PMC5607559 DOI: 10.1002/brb3.797
16. Kanchiku T, Taguchi T, Kaneko K, Fuchigami Y, Yonemura H, Kawai S. A correlation between magnetic resonance imaging and electrophysiological findings in cervical spondylotic myelopathy. Spine (Phila Pa 1976). 2001;26:294–9. PMID: 11458169 DOI: 10.1097/00007632-200107010-00014
17. Izzo R, Guarnieri G, Guglielmi, G, Muto M. Biomechanics of the spine. Part II: spinal instability. Eur J Radiol. 2013;82(1):127–38. PMID: 23088878 DOI: 10.1016/j.ejrad.2012.07.023
18. Goel A, Dandpat S, Shah A, Rai S, Vutha R. Muscle weakness-related spinal instability is the cause of cervical spinal degeneration and spinal stabilization is the treatment: an experience with 215 cases surgically treated over 7 years. World Neurosurg. 2020;140:614–21. PMID: 32797990 DOI: 10.1016/j.wneu.2020.03.104
19. Aebli N, Wicki AG, Rüegg TB, Petrou N, Eisenlohr H, Krebs J. The Torg-Pavlov ratio for the prediction of acute spinal cord injury after a minor trauma to the cervical spine. Spine J. 2013;13(6):605–12. PMID: 23318107 DOI: 10.1016/j.spinee.2012.10.039
20. Ramanauskas WL, Wilner HI, Metes JJ, Lazo A, Kelly JK. MR imaging of compressive myelomalacia. J Comput Assist Tomogr. 1989;13(3):399–404. PMID: 2723169 DOI: 10.1097/00004728-198905000-00005
21. Kameyama T, Hashizume Y, Ando T, Takahashi A, Yanagi T, Mizuno J. Spinal cord morphology and pathology in ossification of the posterior longitudinal ligament. Brain. 1995;118 (Pt 1):263–78. PMID: 7895010 DOI: 10.1093/brain/118.1.263
22. Baucher G, Taskovic J, Troude L, Molliqaj G, Nouri A, Tessitore E. Risk factors for the development of degenerative cervical myelopathy: a review of the literature. Neurosurg Rev. 2022;45(2):1675–89. PMID: 34845577 DOI: 10.1007/s10143-021-01698-9
23. Fehlings MG, Tetreault LA, Riew KD, et al. A clinical practice guideline for the management of patients with degenerative cervical myelopathy: recommendations for patients with mild, moderate, and severe disease and nonmyelopathic patients with evidence of cord compression. Global Spine J. 2017;7(3 Suppl):70S–83S. PMID: 29164035 PMCID: PMC5684840 DOI: 10.1177/2192568217701914
24. Nurick S. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. Brain. 1972;95(1):87-100. PMID: 5023093. DOI: 10.1093/brain/95.1.87
25. Martin AR, De Leener B, Cohen-Adad J, et al. Monitoring for myelopathic progression with multiparametric quantitative MRI. PLoS One. 2018;13(4):e0195733. PMID: 29664964 PMCID: PMC5903654 DOI: 10.1371/journal.pone.0195733
26. Song GC, Cho KS, Yoo DS, Huh PW, Lee SB. Surgical treatment of craniovertebral junction instability: clinical outcomes and effectiveness in personal experience. J Korean Neurosurg Soc. 2010 Jul;48(1):37-45. PMID: 20717510 PMCID: PMC2916146 DOI: 10.3340/jkns.2010.48.1.37