**SUPPLEMENTAL DIGITAL MATERIAL**

Demographics table

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Author** | **Study Design** | **Population** | **Baseline Inclusion Criteria**  | **Baseline Population Characteristics**  | **Disease Definition and Disease Course** |
| Development of myelopathy (A) versus no development of myelopathy (B) |
| Bednarik(2008)\*Bednarik (2011)\* | Prospective cohort | N = 199Sex: 52.8% (105/199) maleMedian age: 51 (28-82) yearsMedian F/U time: 44 (24-144) monthsF/U %: NR | * MRI signs of spondylogenic or discogenic compression of the cervical spinal cord with or without concomitant change in signal intensity from the cervical cord on T1/T2 images
* Axial pain or clinical signs and/or symptoms of radiculopathy that could be controlled by conservative treatment
* Absence of any current clinical signs and symptoms that could be possibly attributed to cervical cord involvement
 | *Baseline mJOA** 18 points: 70.9% (141/199)
* 16-17 points: 29.1% (58/199)
 | *Disease definition:* Development of clinical signs and symptoms of compressive cervical myelopathy corresponding with a decrease in mJOA of ≥ 1.*Disease course:* Subjects underwent at least a 2 year follow-up |
| In subjects diagnosed with OPLL, development of myelopathy (C) versus no development of myelopathy (D) |
| Fujiyoshi (2010) | Prospective cohort | N = 27Sex: 40.7% (11/27) maleMean age: 63.3 (37-78) yearsMean F/U time: 59 (12-95) monthsF/U%: NR | * Cervical OPLL diagnosed on MRI
* Space available for the spinal cord (SAC) at the cervical spine was ≤ 12 mm
* No signs or symptoms of myelopathy
 | *Baseline JOA** 17 points: 100% (27/27)

*OPLL Classification** Continuous: 63.0% (17/27)
* Mixed: 25.9% (7/27)
* Segmental: 11.1% (3/27)

*OPLL Occupation Ratio (%)*†* Continuous: 42.5 ± 11.1 (25-64)
* Mixed: 39.4 ± 7.6 (29-50)
* Segmental: 27.7 ± 3.3 (25-31)

*Segmental ROM (degrees)** Continuous: 2.4 ± 3.0 (0-9)
* Mixed: 4.9 ± 2.9 (1-10)
* Segmental: 9.7 ± 4.5 (5-14)
 | *Disease definition:* NR*Disease course:* The clinical disease course was assessed using the JOA scoring system for cervical myelopathy. |
| Matsunaga (2008) | Prospective cohort16 center study | N = 156Sex: 66.7% (104/156) maleMean age: 65.7 (41-86) yearsMean F/U time: 123.6 (60-276) monthsF/U%: NR | * Cervical OPLL diagnosed on CT/MRI
* No signs or symptoms of myelopathy during neurologic examination
* Minimum 5 years of follow-up evaluation
 | *OPLL Classification** Continuous: 35.3% (55/156)
* Mixed: 35.9% (56/156)
* Segmental: 28.8% (45/156)
 | *Disease definition:* NR*Disease course:* Subjects underwent plain radiographs, CT and MRI and were examined neurologically for the development of myelopathy. |
| Matsunaga (2004) | Prospective cohort | Total N = 450; N without myelopathy at first examination = 323Sex: 70.9% (319/450) maleMean age: 59.6 (54-78) yearsMean F/U time: 211.2 (120-360) monthsF/U%: 92.6% (450/486) | * Cervical OPLL on plain radiography
* No signs or symptoms of myelopathy
 |  | *Disease definition:* Development of myelopathy, which was estimated using the Nurick classification system and the JOA myelopathy scale.*Disease course:* Subjects were examined annually for progression of disease on radiography, clinical myelopathic features, and concomitant ability to perform activities of daily living. |

NR = not reported; MRI = magnetic resonance imaging; mJOA = modified Japanese orthopedic assessment; OPLL = Ossification of posterior longitudinal ligament; JOA = Japanese orthopedic assessment; ROM = range of motion; CT = computed tomography

\* Bednarik 2008 and 2011 reported different outcomes on the same subject population

† OPLL occupation ratio = (thickness of OPLL/anteroposterior diameter of the bony spinal canal) x 100

Outcomes

|  |  |  |  |
| --- | --- | --- | --- |
| **Author** | **Frequency and Timing of Myelopathy Development** | **Potential Prognostic Factors Evaluated\*** | **Significant Results**† |
| Development of myelopathy (A) versus no development of myelopathy (B) |
| Bednarik(2008)‡Bednarik (2011)‡ |  | A | B |  |
| Myelopathy development: 22.6% (45/199)Time until 25% of subjects developed myelopathy: 48.4 months | **Development of myelopathy***Demographic** Age > 50 years: 53.3% (24/45)
* Male sex: 64.4% (29/45)

*Clinical** Presence of symptomatic cervical radiculopathy: 60.0% (27/45)
* Minor traumatic event during follow-up period: 6.7% (3/45)

*Electrophysiological** Prolonged median and tibial SEPs: 37.8% (17/45)
* Prolonged abductor digiti minimi and abductor hallucis MEPs: 40.0% (18/45)
* EMG signs of anterior horn cell lesion: 42.2% (19/45)

*Radiographic*Type of compression* Osteophytes: 35.6% (16/45)
* Herniation: 15.6% (7/45)
* Osteophytes + herniation: 48.8% (22/45)

Number of stenotic levels* 1: 51.1% (23/45)
* 2: 26.7% (12/45)
* ≥ 3: 22.2% (10/45)
* Pavlov ratio < 0.8: 28.9% (13/45)
* Compression ratio ≤ 0.4: 33.3% (15/45)
* Cross-sectional cervical SCA ≤ 70mm2: 40.0% (18/45)
* Cervical cord MRI hyperintensity: 35.6% (16/45)
 | **No development of myelopathy***Demographic** Age > 50 years: 49.4% (76/154)
* Male sex: 49.4% (76/154)

*Clinical** Presence of symptomatic cervical radiculopathy: 20.1% (31/154)
* Minor traumatic event during follow-up period: 7.2% (11/154)

*Electrophysiological** Prolonged median and tibial SEPs: 12.9% (20/154)
* Prolonged abductor digiti minimi and abductor hallucis MEPs: 12.4% (19/154)
* EMG signs of anterior horn cell lesion: 17.5% (27/154)

*Radiographic*Type of compression* Osteophytes: 33.1% (51/154)
* Herniation: 27.9% (43/154)
* Osteophytes + herniation: 39.0% (60/154)

Number of stenotic levels* 1: 53.3% (82/154)
* 2: 33.8% (52/154)
* ≥ 3: 12.9% (20/154)
* Pavlov ratio < 0.8: 34.4% (53/154)
* Compression ratio ≤ 0.4: 22.3% (34/154)
* Cross-sectional cervical SCA ≤ 70mm2: 39.6% (61/154)
* Cervical cord MRI hyperintensity: 21.4% (33/154)
 | Associations with myelopathy development (compared to those who did not develop myelopathy):§ * Symptomatic radiculopathy *(P < .001*)
* EMG signs of anterior horn cell lesion (*P < .001*)
* Prolonged SEPs (*P < .001*)
* Prolonged MEPs (*P < .001*)
* MRI cervical cord hyperintensity (*P < .05*)

Not associated with myelopathy development:§ * Age > 50 years *(P = .808*)
* Male sex (*P = .072*)
* Type of compression (*P = .199*)
* Number of stenotic levels (*P = .302*)
* Pavlov ratio < 0.8 (*P = .485*)
* Compression ratio ≤ 0.4 (*P = .153*)
* Cross-sectional SCA ≤ 70mm2 (*P = .962*)
* Minor traumatic event during follow-up (*P = .921*)
 |
| Myelopathy development within 12 months: 8.0% (16/199) | **Development of myelopathy within 12 months***Demographic** Age > 50 years: 62.5% (10/16)
* Male sex: 43.8% (7/16)

*Clinical** Presence of symptomatic cervical radiculopathy: 62.5% (10/16)

*Electrophysiological** Prolonged median and tibial somatosensory-evoked potentials: 43.8% (7/16)
* Prolonged abductor digiti minimi and abductor hallucis motor-evoked potentials: 37.5% (6/16)
* EMG signs of anterior horn cell lesion: 43.8% (7/16)

*Radiographic*Type of compression* Osteophytes: 87.5% (14/16)
* Others: 12.5% (2/16)

Number of stenotic levels* 1: 37.5% (6/16)
* ≥ 2: 62.5% (10/16)
* Pavlov ratio < 0.8: 25.0% (4/16)
* Compression ratio ≤ 0.4: 31.3% (5/16)
* Cross-sectional cervical spinal cord area (CSA) ≤ 70mm2: 43.8% (7/16)
* Cervical cord MRI hyperintensity: 12.5% (2/16)
 | **No development of myelopathy within 12 months***Demographic** Age > 50 years: 53.0% (97/183)
* Male sex: 53.6% (98/183)

*Clinical** Presence of symptomatic cervical radiculopathy: 26.3% (48/183)

*Electrophysiological** Prolonged median and tibial somatosensory-evoked potentials: 16.4% (30/183)
* Prolonged abductor digiti minimi and abductor hallucis motor-evoked potentials: 16.9% (31/183)
* EMG signs of anterior horn cell lesion: 21.3% (39/183)

*Radiographic*Type of compression* Osteophytes: 73.8% (135/183)
* Others: 26.2% (48/183)

Number of stenotic levels* 1: 54.1% (99/183)
* ≥ 2: 45.9% (84/183)
* Pavlov ratio < 0.8: 33.9% (62/183)
* Compression ratio ≤ 0.4: 24.1% (44/183)
* Cross-sectional cervical spinal cord area ≤ 70mm2: 39.3% (72/183)
* Cervical cord MRI hyperintensity: 25.7% (47/183)
 | Associations with ≤ 12 month myelopathy development (compared to those who did not develop myelopathy within 12 months): \*\** Symptomatic radiculopathy (*P = .007*)
* Prolonged SEPs (*P = .007*)
* Prolonged MEPs (*P = .033*)
* Lack of MRI cervical cord hyperintensity (*P =.036*)

Not associated with ≤ 12 month myelopathy development:\*\** Age > 50 years
* Male sex
* EMG signs of anterior horn cell lesion
* Type of compression
* Number of stenotic levels
* Pavlov ratio < 0.8
* Compression ratio ≤ 0.4
* Cross-sectional CSA ≤ 70mm2
 |
| In subjects diagnosed with OPLL, development of myelopathy (C) versus no development of myelopathy (D) |
|  |  | C | D |  |
| Fujiyoshi (2010) | Myelopathy development: 0% (0/27) | **NR** | **NR** | NR |
| Matsunaga (2008) | Myelopathy development: 61.5% (96/156)Myelopathy development in subjects with ≥ 60% cervical spinal canal stenosis: 100% (39/39)Myelopathy development in subjects with < 60% cervical spinal canal stenosis: 48.7% (57/117) | **Development of myelopathy***Radiographic** Spinal canal stenosis ≥ 60%: 40.6% (39/96)
* Spinal canal stenosis < 60%: 59.4% (57/96)
* Cervical ROM: 50 ± 18

Axial OPLL type††* Central: 26.7% (12/45)
* Lateral deviated: 73.3% (33/45)
 | **No development of myelopathy***Radiographic** Spinal canal stenosis ≥ 60%: 0% (0/60)
* Spinal canal stenosis < 60%: 100% (60/60)
* Cervical ROM: 38 ± 10

Axial OPLL type††* Central: 65.0% (39/60)
* Lateral deviated: 35.0% (21/60)
 | Associations with myelopathy development (compared to those who did not develop myelopathy):§ * Spinal canal stenosis ≥ 60% *(P = NR*)
* Increased cervical ROM (*P = .03*)
* Lateral deviated (vs central) OPLL type in axial view (*P = .021*)
 |
| Matsunaga (2004)‡‡ | Myelopathy development: 17.0% (55/323)Myelopathy-free rate in patients without myelopathy at initial presentation: 71% at 30-year follow-up examination | **NR** | **NR** | NR |

NR = not reported; SEPs = somatosensory-evoked potentials; MEPs = motor-evoked potentials; EMG = electromyography, SCA = spinal cord area; MRI = magnetic resonance imaging; OPLL = Ossification of posterior longitudinal ligament; ROM = range of motion

\* Only reported outcome measures that related to study question

† p<.05

‡ Bednarik 2008 and 2011 reported different outcomes on the same subject population

§ P-values reported by authors in univariate analysis

\*\* Cox proportional multivariate regression analysis; p-values presented if reported by authors

P-values not reported

†† Total N = 105 for this category; subjects with ≥ 60% cervical canal stenosis or history of trauma-induced myelopathy were excluded

‡‡ Matsunaga 2004: The comparison groups in this article did not allow for assessment of risk factors that contribute to development of myelopathy.

**Web Appendix**

1. *Data Extraction*

Each retrieved citation was reviewed by two independently working reviewers. Most articles were excluded on the basis of information provided by the title or abstract. Citations that appeared to be appropriate or those that could not be excluded unequivocally from the title and abstract were identified, and the corresponding full text reports were reviewed by the two reviewers. Any disagreement between them was resolved by reviewer consensus. From the included articles, the following data were extracted: study design, patient demographics, inclusion and exclusion criteria, disease characteristics, treatment interventions, follow-up duration and the rate of follow-up for each treatment group (if reported or calculable), treatment outcomes, and complications.

2. *Study Quality*

Articles selected for inclusion were classified by class of evidence.  The method used for assessing the quality of evidence of individual studies as well as the overall quality of the body of evidence incorporates aspects of the rating scheme developed by the Oxford Centre for Evidence-based Medicine 1 and used with modification by *The Journal of Bone and Joint Surgery American Volume* (*J Bone Joint Surg Am*), 2 precepts outlined by the Grades of Recommendation Assessment, Development and Evaluation (GRADE) Working Group3 and recommendations made by the Agency for Healthcare Research and Quality (AHRQ)4.  Each individual study was rated by two different investigators against pre-set criteria that resulted in an evidence rating (Class of Evidence I, II, III, or IV).  Disagreements were resolved through discussion.

1. *Class of Evidence Tables*

*3a. Class of evidence (CoE) criteria for prognostic studies*

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Methodological principle** | **Bednarik****(2011)** | **Bednarik****(2008)** | **Fujiyoshi****(2010)** | **Matsunaga****(2008)** | **Matsunaga****(2004)** |
| Study design |  |  |  |  |  |
| Prospective cohort study | ✓ | ✓ | ✓ | ✓ | ✓ |
| Retrospective cohort study |  |  |  |  |  |
| Case-control study |  |  |  |  |  |
| Cross-sectional study |  |  |  |  |  |
| Case-series  |  |  |  |  |  |
| **COHORT STUDIES** |  |  |  |  |  |
| Patients at similar point in the course of their disease or treatment | ✓ | ✓ | ✓ | ✓ |  |
| Complete follow-up of > 80% |  |  |  |  | ✓ |
| Patients followed long enough for outcomes to occur | ✓ | ✓ | ✓ | ✓ | ✓ |
| Accounting for other prognostic factors\* |  | ✓ |  |  |  |
| **CASE-CONTROL STUDIES** |  |  |  |  |  |
| Incidence cases from defined population over a specified time period |  |  |  |  |  |
| Controls represent the population from which the cases come |  |  |  |  |  |
| Exposure precedes an outcome of interest |  |  |  |  |  |
| Accounting for other prognostic factors |  |  |  |  |  |
| **CROSS-SECTIONAL STUDIES** |  |  |  |  |  |
| A representative sample of the population of interest |  |  |  |  |  |
| Exposure that precedes an outcome of interest (e.g., sex, genetic factor)  |  |  |  |  |  |
| Accounting for other prognostic factors |  |  |  |  |  |
| For surveys, a return rate of > 80% |  |  |  |  |  |
| **Evidence class** | **III** | **II** | **III** | **III** | **III** |

\*Authors must consider other factors that might influence patient outcomes

*Blank cells indicate that the criterion was either not met or that it could not be determined*

*3b. Criteria for class of evidence (CoE) for prognostic studies*

|  |  |  |
| --- | --- | --- |
|  |  | **Studies of Prognosis** |
| **Class** | **Risk of bias** | **Study design** | **Criteria** |
| **I** | **Low risk;** Study adheres to commonly held tenets of high quality design, execution and avoidance of bias | Good quality cohort\* | * Prospective design
* Patients at similar point in the course of their disease or treatment
* F/U rate of ≥ 80%†
* Patients followed long enough for outcomes to occur
* Accounting for other prognostic factors‡
 |
| **II** | **Moderately low risk:** Study has potential for some bias; does not meet all criteria for class I but deficiencies not likely to invalidate results or introduce significant bias | Moderate quality cohort | * Prospective design, with violation of one of the other criteria for good quality cohort study
* Retrospective design, meeting all the rest of the criteria in class I
 |
| **III** | **Moderately high risk:** Study has flaws in design and/or execution that increase potential for bias that may invalidate study results | Poor quality cohortGood quality case-control or cross-sectional study | * Prospective design with violation of 2 or more criteria for good quality cohort, or
* Retrospective design with violation of 1 or more criteria for good quality cohort
* A good case-control study§
* A good cross-sectional study\*\*
 |
| **IV** | **High risk:** Study has significant potential for bias; does not include design features geared toward minimizing bias and/or does not have a comparison group | Poor quality case-control or cross-sectionalCase series§ | * Other than a good case-control study
* Other than a good cross-sectional study
* Any case series†† design
 |

\*Cohort studies follow individuals with the exposure of interest over time and monitor for occurrence of the outcome of interest.

†Applies to cohort studies only.

‡Authors must consider other factors that might influence patient outcomes and should control for them if appropriate.

§A good case-control study must have the all of the following: all incident cases from the defined population over a specified time period, controls that represent the population from which the cases come, exposure that precedes an outcome of interest, and accounting for other prognostic factors.

\*\*A good cross-sectional study must have all of the following: a representative sample of the population of interest, an exposure that precedes an outcome of interest (e.g., sex, genetic factor), an accounting for other prognostic factors, and for surveys, at least a 80% return rate.

††A case-series design for prognosis is one where all the patients in the study have the exposure of interest. Since all the patients have the exposure, risks of an outcome can be calculated only for those with the exposure, but cannot be compared with those who do not have the exposure. For example, a case-series evaluating the effect of smoking on spine fusion that only recruits patients who smoke can simply provide the risk of patients who smoke that result in pseudarthrosis but cannot compare this risk to those that do not smoke.

*4. Excluded articles.*

|  |  |  |
| --- | --- | --- |
| **Author** | **Year** | **Reason for exclusion** |
| Bednarik, J., Z. Kadanka, et al. (2004). "Presymptomatic spondylotic cervical cord compression." Spine (Phila Pa 1976) 29(20): 2260-9. | 2004 | Population overlap with Bednarik 2008 and 2011 articles; reported on a smaller subject population and did not report additional results/outcomes compared to later studies. |
| Bednarik, J., Z. Kadanka, et al. (1998). "The value of somatosensory and motor evoked evoked potentials in pre-clinical spondylotic cervical cord compression." Eur Spine J 7(6): 493-500. | 1998 | Population overlap with Bednarik 2008 and 2011 articles; reported on a smaller subject population and did not report additional results/outcomes compared to later studies. |
| Castro, F. P., Jr., J. Ricciardi, et al. (1997). "Stingers, the Torg ratio, and the cervical spine." Am J Sports Med 25(5): 603-8. | 1997 | Did not assess outcomes of interest (subjects were followed prospectively for development of transient neuropraxias) |
| Chang, H., K. J. Song, et al. (2012). "Factors related to the development of myelopathy in patients with cervical ossification of the posterior longitudinal ligament." J Bone Joint Surg Br 94(7): 946-9. | 2012 | Cross-sectional study; did not assess outcomes of interest |
| Chiba, K., I. Yamamoto, et al. (2005). "Multicenter study investigating the postoperative progression of ossification of the posterior longitudinal ligament in the cervical spine: a new computer-assisted measurement." J Neurosurg Spine 3(1): 17-23. | 2005 | Did not assess outcomes of interest (subjects with a history of surgical intervention were followed for progression of OPLL) |
| Foo, D. (1986). "Spinal cord injury in forty-four patients with cervical spondylosis." Paraplegia 24(5): 301-6. | 1986 | Did not meet inclusion criteria (subjects had history of spinal cord injury) |
| Ichihara, D., E. Okada, et al. (2009). "Longitudinal magnetic resonance imaging study on whiplash injury patients: minimum 10-year follow-up." J Orthop Sci 14(5): 602-10. | 2009 | Did not report outcomes of interest |
| Iwasaki, M., T. Yamamoto, et al. (2002). "Cervical kyphosis: predictive factors for progression of kyphosis and myelopathy." Spine (Phila Pa 1976) 27(13): 1419-25. | 2002 | Did not meet inclusion criteria |
| Kadanka, Z., M. Kerkovsky, et al. (2007). "Cross-sectional transverse area and hyperintensities on magnetic resonance imaging in relation to the clinical picture in cervical spondylotic myelopathy." Spine (Phila Pa 1976) 32(23): 2573-7. | 2007 | Cross-sectional study; did not assess outcomes of interest |
| Kerkovsky, M., J. Bednarik, et al. (2012). "Magnetic resonance diffusion tensor imaging in patients with cervical spondylotic spinal cord compression: correlations between clinical and electrophysiological findings." Spine (Phila Pa 1976) 37(1): 48-56. | 2012 | Cross-sectional study; did not assess outcomes of interest |
| Lee, S. H., K. T. Kim, et al. (2010). "Asymptomatic cervical cord compression in lumbar spinal stenosis patients: a whole spine magnetic resonance imaging study." Spine (Phila Pa 1976) 35(23): 2057-63. | 2010 | Did not assess outcomes of interest (assessed the presence of asymptomatic cervical cord compression in subjects with lumbar stenosis) |
| Lees, F. and J. W. Turner (1963). "Natural History and Prognosis of Cervical Spondylosis." Br Med J 2(5373): 1607-10. | 1963 | Did not meet inclusion criteria |
| Matsumoto, M., E. Okada, et al. (2010). "Prospective ten-year follow-up study comparing patients with whiplash-associated disorders and asymptomatic subjects using magnetic resonance imaging." Spine (Phila Pa 1976) 35(18): 1684-90. | 2010 | Did not report outcomes of interest |
| Matsunaga, S., M. Kukita, et al. (2002). "Pathogenesis of myelopathy in patients with ossification of the posterior longitudinal ligament." J Neurosurg 96(2 Suppl): 168-72. | 2002 | Population overlap with Matsunaga 2004 article; reported on a smaller subject population and did not report additional results/outcomes compared to later study. |
| Meyer, S. A., K. R. Schulte, et al. (1994). "Cervical spinal stenosis and stingers in collegiate football players." Am J Sports Med 22(2): 158-66. | 1994 | Did not assess outcomes of interest (subjects were followed prospectively for development of transient neuropraxias) |
| Miyanji, F., J. C. Furlan, et al. (2007). "Acute cervical traumatic spinal cord injury: MR imaging findings correlated with neurologic outcome--prospective study with 100 consecutive patients." Radiology 243(3): 820-7. | 2007 | Did not meet inclusion criteria (subjects had history of spinal cord injury) |
| Mochizuki, M., A. Aiba, et al. (2009). "Cervical myelopathy in patients with ossification of the posterior longitudinal ligament." J Neurosurg Spine 10(2): 122-8. | 2009 | Population overlap with Fujiyoshi 2010 article; N = 6 and did not report additional results/outcomes compared to Fujiyoshi article. |
| Okada, E., M. Matsumoto, et al. (2009). "Aging of the cervical spine in healthy volunteers: a 10-year longitudinal magnetic resonance imaging study." Spine (Phila Pa 1976) 34(7): 706-12. | 2009 | Did not meet inclusion criteria and did not report outcomes of interest |
| Okada, S., T. Maeda, et al. (2009). "Does ossification of the posterior longitudinal ligament affect the neurological outcome after traumatic cervical cord injury?" Spine (Phila Pa 1976) 34(11): 1148-52. | 2009 | Did not meet inclusion criteria and did not assess outcomes of interest (subjects were followed for duration of hospital stay). |
| Presciutti, S. M., P. DeLuca, et al. (2009). "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 11(3): 264-71. | 2009 | Did not meet inclusion criteria and did not assess outcomes of interest (retrospective case control study that assessed factors which may contribute to transient neuropraxia development). |
| Regenbogen, V. S., L. F. Rogers, et al. (1986). "Cervical spinal cord injuries in patients with cervical spondylosis." AJR Am J Roentgenol 146(2): 277-84. | 1986 | Cross-sectional study; did not assess outcomes of interest |
| Setzer, M., E. Hermann, et al. (2008). "Apolipoprotein E gene polymorphism and the risk of cervical myelopathy in patients with chronic spinal cord compression." Spine (Phila Pa 1976) 33(5): 497-502. | 2008 | Cross-sectional study; did not assess outcomes of interest |
| Shimomura, T., M. Sumi, et al. (2007). "Prognostic factors for deterioration of patients with cervical spondylotic myelopathy after nonsurgical treatment." Spine (Phila Pa 1976) 32(22): 2474-9. | 2007 | Did not meet inclusion criteria; all subjects had CSM at baseline |
| Simo, M., I. Szirmai, et al. (2004). "Superior sensitivity of motor over somatosensory evoked potentials in the diagnosis of cervical spondylotic myelopathy." Eur J Neurol 11(9): 621-6. | 2004 | Cross-sectional study; did not assess outcomes of interest |
| Takamiya, Y., K. Nagata, et al. (2006). "Cervical spine disorders in farm workers requiring neck extension actions." J Orthop Sci 11(3): 235-40. | 2006 | Retrospective cohort study; subjects did not meet inclusion criteria |
| Torg, J. S., R. J. Naranja, Jr., et al. (1996). "The relationship of developmental narrowing of the cervical spinal canal to reversible and irreversible injury of the cervical spinal cord in football players." J Bone Joint Surg Am 78(9): 1308-14. | 1996 | Did not assess outcomes of interest (One cohort of subjects was followed prospectively for neurologic injury following history of transient neuropraxias) |
| Voskuhl, R. R. and R. C. Hinton (1990). "Sensory impairment in the hands secondary to spondylotic compression of the cervical spinal cord." Arch Neurol 47(3): 309-11. | 1990 | Did not meet inclusion criteria; all subjects were suspicious for CSM |
| Yagi, M., K. Ninomiya, et al. (2010). "Long-term surgical outcome and risk factors in patients with cervical myelopathy and a change in signal intensity of intramedullary spinal cord on Magnetic Resonance imaging." J Neurosurg Spine 12(1): 59-65. | 2010 | Did not meet inclusion criteria; all subjects had CSM |
| Yukawa, Y., F. Kato, et al. (2007). "MR T2 image classification in cervical compression myelopathy: predictor of surgical outcomes." Spine (Phila Pa 1976) 32(15): 1675-8; discussion 1679. | 2007 | Did not meet inclusion criteria; all subjects had CSM |