ReGeo ISSN: 2177-3246

DEGENERATIVE CERVICAL MYELOPATHY AND CLINICAL PREDICTORS OF NEUROLOGICAL DETERIORATION: A STRUCTURED NARRATIVE REVIEW

MIELOPATIA CERVICAL DEGENERATIVA E PREDITORES CLÍNICOS DE DETERIORAÇÃO NEUROLÓGICA: UMA REVISÃO NARRATIVA ESTRUTURADA

MIELOPATÍA CERVICAL DEGENERATIVA Y PREDICTORES CLÍNICOS DE DETERIORO NEUROLÓGICO: UNA REVISIÓN NARRATIVA ESTRUCTURADA

https://doi.org/10.56238/revgeov17n11-022

Data de submissão: 04/10/2026 **Data de publicação:** 04/11/2026

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ABSTRACT

Background: Degenerative cervical myelopathy (DCM) has a heterogeneous course, ranging from prolonged stability to progressive loss of hand function, gait, and independence. Risk estimates are complicated by differences between incident myelopathy, deterioration of established disease, and postoperative recovery. Objective: To critically evaluate clinical predictors of neurological deterioration and their interaction with imaging, electrophysiology, and longitudinal assessment. Methods: A targeted PubMed-based narrative review examined guidelines, nonoperative natural-history cohorts, prospective studies of nonmyelopathic cord compression, and prognostic investigations available through October 24, 2026. Endpoints and populations were analyzed separately; no new meta-analysis or prediction model was developed. Results: Worsening gait, hand dexterity, objective weakness, and sphincter function are important indicators of clinical progression, but few baseline features reliably predict an individual trajectory. Symptomatic radiculopathy and electrophysiological abnormalities predict incident myelopathy in selected nonmyelopathic compression cohorts. Their effect sizes should not be transferred directly to established DCM. Historical reviews report deterioration in 20%–62% of nonoperatively treated symptomatic patients over several years, whereas a recent mild-DCM cohort reported 13.3% deterioration at one year among evaluable patients. These estimates differ in severity, follow-up, and outcome definition. Cord signal change, instability, and quantitative MRI can refine concern, but no imaging-only threshold reliably determines deterioration or surgical timing. Conclusions: Serial clinical change is more actionable than an isolated baseline risk marker. Moderate or severe DCM and neurological deterioration warrant surgical assessment; selected mild stable disease may be observed with structured follow-up and explicit escalation criteria.

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Keywords: Degenerative Cervical Myelopathy. Cervical Spondylosis. Prognosis. Neurological Deterioration. Gait. Spinal Cord Compression.

RESUMO
Introdução: A mielopatia cervical degenerativa (MCD) apresenta uma evolução heterogênea, variando de uma estabilidade prolongada a uma perda progressiva da função das mãos, da marcha e da independência. As estimativas de risco são complicadas por diferenças entre mielopatia incidente, deterioração da doença estabelecida e recuperação pós-operatória. Objetivo: Avaliar criticamente os preditores clínicos de deterioração neurológica e sua interação com características de imagem, eletrofisiologia e avaliação longitudinal. Métodos: Uma revisão narrativa direcionada baseada em PubMed examinou diretrizes, coortes de história natural não-operatória, estudos prospectivos de compressão medular não-mielopática e investigações prognósticas disponíveis até 24 de outubro de 2026. Desfechos e populações foram analisados separadamente; nenhuma nova metanálise ou modelo de predição foi desenvolvido. Resultados: A piora da marcha, da destreza manual, da fraqueza objetiva e da função esfincteriana são indicadores importantes da progressão clínica, mas poucas características basais predizem de forma confiável uma trajetória individual. A radiculopatia sintomática e as anomalias eletrofisiológicas predizem mielopatia incidente em coortes selecionadas de compressão não-mielopática. Seus tamanhos de efeito não devem ser transferidos diretamente para MCD estabelecida. Revisões históricas relatam deterioração em 20%–62% de pacientes sintomáticos tratados não-operatoriamente ao longo de vários anos, enquanto uma coorte recente de MCD leve relatou deterioração de 13,3% em um ano entre pacientes avaliáveis. Essas estimativas diferem em gravidade, acompanhamento e definição de desfecho. Mudança do sinal medular, instabilidade e ressonância magnética quantitativa podem refiná-la, mas nenhum limite apenas de imagem prediz de forma confiável deterioração ou momento cirúrgico. Conclusões: A mudança clínica serial é mais acionável do que um marcador de risco basal isolado. MCD moderada ou grave e deterioração neurológica justificam avaliação cirúrgica; doença leve estável selecionada pode ser observada com acompanhamento estruturado e critérios de escalação explícitos.

Palavras-chave: Mielopatia Cervical Degenerativa. Espondilose Cervical. Prognóstico. Deterioração Neurológica. Marcha. Compressão Medular.

RESUMEN
Antecedentes: La mielopatía cervical degenerativa (MCD) tiene un curso heterogéneo, que va desde una estabilidad prolongada hasta la pérdida progresiva de la función de las manos, la marcha y la independencia. Las estimaciones de riesgo se complican por las diferencias entre la mielopatía incidente, el deterioro de la enfermedad establecida y la recuperación posoperatoria. Objetivo: Evaluar críticamente los predictores clínicos del deterioro neurológico y su interacción con la imagen, la neurofisiología y la evaluación longitudinal. Métodos: Una revisión narrativa dirigida basada en PubMed examinó directrices, cohortes de historia natural no operatoria, estudios prospectivos de compresión medular no mielopática e investigaciones pronósticas disponibles hasta el 24 de octubre de 2026. Los puntos finales y las poblaciones se analizaron por separado; no se desarrolló un nuevo metanálisis ni modelo de predicción. Resultados: El empeoramiento de la marcha, la destreza manual, la debilidad objetiva y la función esfinteriana son indicadores importantes de la progresión clínica, pero pocas características basales predicen de forma confiable una trayectoria individual. La radiculopatía sintomática y las anomalías neurofisiológicas predicen mielopatía incidente en cohortes seleccionadas de compresión no mielopática. Sus tamaños

de efectos no deben transferirse directamente a la MCD establecida. Las revisiones históricas informan deterioro en el 20%–62% de los pacientes sintomáticos tratados de forma no operatoria durante varios años, mientras que una cohorte reciente de MCD leve informó un deterioro del 13,3% al año entre los pacientes evaluables. Estas estimaciones difieren en severidad, seguimiento y definición de resultado. El cambio de señal medular, la inestabilidad y la resonancia magnética cuantitativa pueden refinarla, pero ningún umbral de imagen únicamente predice de forma confiable el deterioro o el momento quirúrgico. Conclusiones: El cambio clínico seriado es más procesable que un marcador de riesgo basal aislado. La MCD moderada o grave y el deterioro neurológico justifican una evaluación quirúrgica; la enfermedad leve estable seleccionada puede observarse con seguimiento estructurado y criterios de escalación explícitos.

Palabras clave: Mielopatía Cervical Degenerativa. Espondilopatía Cervical. Pronóstico. Deterioro Neurológico. Marcha. Compresión Medular.

1 INTRODUCTION

Degenerative cervical myelopathy is spinal cord dysfunction caused by degenerative changes in the cervical spine. Hand clumsiness, altered sensation, gait imbalance, weakness, hyperreflexia, and loss of independence may develop gradually and can initially be attributed to peripheral neuropathy, joint disease, or ageing. The clinical question is not simply whether cord compression exists on MRI, but whether neurological dysfunction is present and whether it is progressing.[1,2]

Patients and clinicians often ask which baseline findings predict deterioration. This question has several meanings: development of first myelopathic symptoms in a person with radiographic compression, worsening of established mild DCM during observation, conversion to surgery, or failure to regain function after surgery. The populations and endpoints differ, and a marker validated for one should not be presented as a universal predictor for the others.[3,4,5]

This review evaluates clinical predictors with those distinctions preserved. Imaging and electrophysiology are discussed as modifiers of clinical assessment, while particular emphasis is placed on repeated measurement and the practical recognition of change. The aim is to support counseling and timely referral without assigning false certainty to a single symptom, MRI measurement, or prognostic score.

2 METHODS

A targeted PubMed search combined degenerative cervical myelopathy or cervical spondylotic myelopathy with deterioration, progression, natural history, prognosis, and management. Supplementary searches retrieved nonmyelopathic cord-compression cohorts and contemporary recommendations. Sources available through October 24, 2026 were considered, and every reference was verified in PubMed. Guidelines and longitudinal primary studies were prioritized; postoperative cohorts were included only to distinguish recovery prediction from untreated progression.

The review is narrative and selective. No registered protocol, independent duplicate screening, formal study-level risk-of-bias scoring, or new quantitative synthesis was performed. Findings were assessed according to baseline severity, treatment selection, follow-up duration, and the definition of deterioration. Study-derived thresholds and machine-learning results are presented as investigational when external validation or calibration is insufficient.

2.1 CLINICAL DEFINITION AND BASELINE CHARACTERIZATION

DCM requires a compatible clinical syndrome and evidence of degenerative cervical cord compression. Radiographic narrowing alone is common and does not establish myelopathy. The modified Japanese Orthopaedic Association (mJOA) scale is widely used to classify mild disease as 15–17, moderate disease as 12–14, and severe disease as 11 or less. These categories organize management but do not replace a detailed examination or describe every dimension of disability.[1],[6]

Baseline assessment should document upper- and lower-limb motor function, dexterity, sensory abnormalities, reflexes, balance, walking capacity, and urinary symptoms. Relevant daily activities include fastening buttons, handwriting, utensil use, handling keys, climbing stairs, and walking in dim light or on uneven ground. An apparently mild total score can conceal a functionally important deficit for a person whose occupation depends on fine motor control.[2]

Hoffmann signs, Babinski responses, clonus, increased tone, and hyperreflexia support upper motor neuron dysfunction, but their absence does not exclude early DCM. Diabetes or peripheral neuropathy can modify reflex findings, and radiculopathy may produce segmental weakness or reduced reflexes alongside cord disease. Bilateral hand symptoms, gait dysfunction, or a combination of upper- and lower-limb findings should prevent premature attribution to a single peripheral nerve lesion.[2],[7]

2.2 WHAT NATURAL HISTORY STUDIES SHOW

An AO Spine natural-history review reported neurological deterioration in approximately 20%–62% of nonoperatively treated symptomatic patients over three to six years. This broad range reflects heterogeneous cohorts rather than a stable annual hazard. Disease severity, recruitment, definitions of decline, and follow-up differ, and some patients undergo surgery before further untreated deterioration can be observed.[3],[8]

A more recent Canadian multicentre cohort enrolled 140 patients with mild DCM initially treated nonoperatively. A decline of at least two mJOA points was reported in 13.3% of those evaluated at one year. Seventeen patients, 12.1% of the initial cohort, crossed over to surgery at an average of 3.4 years. Baseline mJOA was lower in crossover patients, but no clear, broadly useful deterioration predictor emerged.[9]

These findings are compatible with many mildly affected patients remaining stable over a shorter period while a clinically important minority worsens. They do not prove that waiting

is equally safe for every patient or that surgery should be deferred until a fixed score decrement occurs. The choice of treatment is influenced by current disability, progression, operative risk, patient preference, and access to reliable reassessment.[1,10]

Conversion to surgery is an imperfect surrogate for biological progression. Pain, disability, treatment preference, clinician judgment, and health-system access can all affect that decision. Conversely, a patient may deteriorate without undergoing surgery because of comorbidity or preference. Studies should therefore report neurological change and treatment conversion separately.

3 CLINICAL FEATURES ASSOCIATED WITH GREATER CONCERN

3.1 GAIT IMPAIRMENT AND FALLS

Gait imbalance, reduced walking endurance, difficulty with tandem gait, and new falls are clinically important because they affect independence and may indicate cord dysfunction extending beyond sensory complaints. At baseline, these features characterize disease burden. During follow-up, a reproducible decline is evidence of progression rather than merely a forecast. Timed walking and a consistent balance assessment can make subtle change more visible than an unstructured question about whether symptoms are better or worse.[1,2]

Falls are both a consequence of neurological impairment and a possible source of additional injury. In a retrospective multicentre surgical cohort of 360 patients, 177 recalled at least one preoperative fall and 105 reported fall-related symptom deterioration. The study supports fall assessment and prevention, but surgical selection, recall, and subjective symptom reporting prevent use of these proportions as the prospective injury risk for all patients with mild DCM.[11]

3.2 HAND DYSFUNCTION AND OBJECTIVE WEAKNESS

Progressive difficulty with buttons, handwriting, grip release, or object manipulation can precede a conspicuous change in strength testing. Serial examination should use the same tasks where feasible and distinguish pain-limited performance from neurological loss. New intrinsic hand weakness, loss of dexterity, or increasing bilateral dysfunction deserves prompt reassessment, especially when accompanied by lower-limb signs.[2]

Motor-dominant symptoms may identify a more persistent impairment phenotype. A 2026 secondary analysis of 90 nonoperatively managed mild-to-moderate cases described

more fluctuation in sensory states and greater persistence in lower-limb motor deficits. Its one-year state probabilities were modeled from scheduled follow-up extending to six months, and patients who underwent surgery were excluded from that analysis. These findings are hypothesis-generating; they do not establish an independent surgical threshold or prove that sensory-only disease is safe to ignore.[12]

3.3 SENSORY SYMPTOMS AND SPHINCTER CHANGE

Paresthesia is common and can be disabling, but fluctuating subjective sensation alone is less specific than a corroborated functional decline. The distribution, associated dexterity loss, proprioceptive impairment, and examination findings help separate cord dysfunction from peripheral causes. Worsening sensation should not be dismissed, but neither should every variation be counted as irreversible neurological deterioration.[2,12]

New urinary urgency, impaired emptying, or incontinence can occur in advanced cord disease, but common urological disorders provide alternative explanations. New sphincter dysfunction accompanied by worsening gait, weakness, or widespread neurological signs warrants urgent assessment. Clinicians should not wait for bladder involvement before recommending evaluation of progressive DCM; it is a late and potentially consequential development rather than a necessary diagnostic feature.[1]

3.4 BASELINE SEVERITY AND SYMPTOM TRAJECTORY

Lower baseline mJOA indicates greater functional impairment and can influence treatment decisions. In the recent mild-DCM cohort, patients who crossed over to surgery had slightly worse baseline mJOA. However, this association did not yield a robust individual deterioration rule. A score of 17 does not guarantee stability, and a score of 15 should not automatically be described as a quantified high-risk state without considering the actual deficits and trajectory.[9]

A history of progressive symptoms is especially relevant because the clinician may already be observing the adverse outcome that a prediction model seeks to forecast. Accumulating falls, loss of walking distance, new weakness, or documented dexterity decline should therefore carry greater immediate weight than the absence of a particular MRI signal abnormality. Stable pain or an unchanged global score should not obscure deterioration within a clinically important domain.[1]

3.5 PREDICTORS OF INCIDENT MYELOPATHY IN NONMYELOPATHIC COMPRESSION

Evidence for baseline clinical prediction is stronger in certain nonmyelopathic compression cohorts than in established mild DCM. Kadanka and colleagues followed 112 individuals with degenerative cord compression but no clinical myelopathy for a median of three years. Fifteen developed first signs or symptoms of DCM. Radiculopathy, cord cross-sectional area of 70.1 mm² or less, and compression ratio of 0.4 or less were independent predictors in that cohort. Gait/run time and electrophysiological abnormalities were also associated in broader analyses.[4]

Bednarik and colleagues followed 199 presymptomatic patients, of whom 45 developed symptomatic myelopathy. Symptomatic radiculopathy and abnormal somatosensory or motor evoked potentials were associated with earlier progression, while T2 hyperintensity was associated with later progression. The cohort was not equivalent to a random sample of entirely asymptomatic adults: some participants had pain or radiculopathy and were already under specialist evaluation.[13],[14]

These data support closer observation and counseling when radiculopathy or subclinical cord dysfunction accompanies compression. They should not be used to claim that the same imaging thresholds predict a two-point mJOA decline in someone who already has DCM. Consistent with this distinction, guidelines advise against routine prophylactic surgery for nonmyelopathic compression without radiculopathy, while recognizing a higher-risk group when radiculopathy is present.[1]

3.6 IMAGING AND ELECTROPHYSIOLOGY AS CLINICAL MODIFIERS

Conventional MRI confirms the anatomical substrate and identifies cord signal change, compression pattern, and potential alternative pathology. T2 hyperintensity may reflect several processes and is not a direct measurement of irreversible tissue loss. In established disease, more severe compression on a single image does not consistently translate into more rapid decline. The absence of signal change similarly cannot guarantee a benign course.[3],[15]

A selected cohort of 45 mildly affected patients with T2 signal change illustrates the contribution of mechanics. Sixteen gradually deteriorated and underwent surgery, while two additional patients required surgery after minor-trauma spinal cord injury. Greater cervical range of motion, segmental kyphosis, and local slip were associated with deterioration-related conversion. Because all participants had T2 hyperintensity, the study cannot

determine how its presence changes risk compared with otherwise similar signal-negative patients.[16]

Quantitative MRI may offer biological information beyond focal compression. In a study of 49 patients with mild DCM contributing 110 six-month observations, combined clinical and quantitative MRI models achieved an area under the curve of 0.87 for six-month deterioration. Reduced magnetization transfer metrics and selected sensory findings contributed to prediction. The small sample, repeated observations, and need for independent validation limit transfer to routine practice.[17]

A separate 51-patient imaging study suggested that canal size and multilevel compression patterns matter in combination, rather than as isolated values. Contemporary AO Spine recommendations do not endorse routine MRI-only prediction of deterioration or surgical timing in mild DCM on the available very-low-quality evidence. Quantitative techniques should complement careful clinical surveillance rather than displace it.[6],[15]

Electrophysiology can clarify peripheral mimics and detect subclinical central conduction abnormalities. Its strongest prognostic evidence comes from nonmyelopathic compression cohorts. It may be useful when symptoms, examination, and imaging are discordant, but it is not necessary to delay a surgical assessment in a patient with clear clinical progression.[4],[13]

Table 1

Prognostic evidence and the population to which it applies

Study or feature	Population and endpoint	Finding	Clinical interpretation
Bednarik et al. [13]	199 presymptomatic patients with cervical cord compression; incident myelopathy.	45 developed clinical myelopathy; radiculopathy and conduction abnormalities related to earlier progression.	Predicts first clinical disease in a selected compression cohort, not deterioration of established DCM.
Kadanka et al. [4]	112 nonmyelopathic compression subjects; median follow-up three years.	15/112 developed DCM. Independent predictors included radiculopathy, CSA ≤70.1 mm², and CR ≤0.4.	Thresholds are study-specific. They do not independently mandate prophylactic surgery.
Oshima et al. [16]	45 mild cases with T2 signal change, initially observed.	16 gradually deteriorated and underwent surgery; two additional trauma-related injuries. Motion, kyphosis, and slip were adverse factors.	Selected signal-positive cohort; conversion combines disease course and treatment decisions.
Canadian mild-DCM cohort [9]	140 patients initially observed; neurological decline and surgical crossover assessed separately.	Reported 13.3% one-year decline among evaluable patients; 17/140 crossed over at a mean 3.4 years.	Do not calculate the one-year deterioration percentage using all enrolled patients. No robust individual predictor emerged.
Clinical plus quantitative MRI model [17]	49 patients, 110 six-month observations; short-term deterioration.	Best combined model AUC 0.87; magnetization-transfer and selected clinical findings contributed.	Small repeated-measures dataset. Requires external validation and calibration before routine clinical use.
Serial gait or hand decline [1]	Established DCM under clinical observation.	New weakness, falls, dexterity loss, or walking deterioration demonstrate progression.	Observed neurological change supports prompt surgical reassessment even if a global score or scan appears unchanged.
Postoperative prediction model [5]	757 surgically treated patients; postoperative functional outcome.	Age, severity, duration, gait, comorbidity, and smoking contributed to prediction.	Recovery prediction should not be presented as a validated untreated-deterioration model.

AUC, area under the receiver operating characteristic curve; CR, compression ratio; CSA, cord cross-sectional area; DCM, degenerative cervical myelopathy; MRI, magnetic resonance imaging. Evidence for incident disease, observed progression, surgical crossover, and postoperative recovery is separated deliberately.
Source: Prepared by the authors.

3.7 PREDICTORS OF RECOVERY ARE A DIFFERENT QUESTION

Age, symptom duration, smoking, comorbidity, and baseline neurological severity repeatedly appear in surgical outcome studies. In an international prospective surgical dataset of 757 patients, these variables and gait impairment contributed to a model of

postoperative functional outcome. Such findings inform expectations after decompression, but do not establish the probability that an untreated person will deteriorate over the next year.[5]

This distinction prevents two common errors. Older age or diabetes should not be described as a validated stand-alone marker of imminent untreated decline merely because it predicts less complete recovery. Conversely, reduced expected recovery does not mean surgery offers no benefit; preventing further loss can be valuable even when normalization is unlikely. Clinical decisions should weigh achievable function, current burden, operative risks, and patient priorities.[1,5]

3.8 STRUCTURED SURVEILLANCE AND ESCALATION

Observation of selected mild stable DCM should be an active plan. Baseline documentation should include mJOA domains, specific hand tasks, walking and balance measures, falls, neurological findings, pain, and disability. Follow-up should repeat comparable measures and explicitly ask about new functional limitations. A total score can remain similar while a meaningful domain worsens, especially near the upper end of the scale.[1]

No single interval is validated for every patient. Timing should reflect symptoms, recent trajectory, imaging context, and the ability to obtain urgent reassessment. Patients should know which changes require earlier contact: increasing falls, new weakness, reduced hand function, deterioration in walking, or sphincter changes. Repeat MRI is useful when the clinical course changes or the initial study is inadequate, rather than as a substitute for examination in an otherwise stable patient.

Guidelines recommend surgery for moderate and severe DCM. For mild disease, surgery or a supervised trial of structured rehabilitation are accepted options, with operative intervention recommended if neurological deterioration occurs and considered if improvement fails to occur. Rehabilitation may address function and safety but should not be represented as proven reversal of the compressive disease process. Access to a spine specialist remains important even when an initial nonoperative approach is chosen.[1,10]

Rapid neurological loss requires urgent evaluation for acute injury or an alternative neurological process. Escalation should follow the observed deficit rather than await a minimum score change or additional MRI compression.

3.9 LIMITATIONS AND RESEARCH PRIORITIES

The prediction literature is constrained by small numbers of deterioration events, variable definitions, incomplete follow-up, and treatment-related censoring. Mild-DCM studies often enroll patients already selected for observation, while surgical cohorts overrepresent severe or progressive disease. Studies of incident myelopathy, symptom persistence, conversion to surgery, and postoperative recovery should therefore be synthesized separately.

A 2026 critical appraisal of twelve prediction-model studies found high analysis-domain risk of bias across the included models, with inadequate sample-event matching, inconsistent missing-data approaches, and limited calibration reporting. Discrimination alone is insufficient: an impressive area under the curve does not show that predicted individual probabilities are accurate or that model use improves decisions. External validation and clinically meaningful endpoints are essential before adopting a bedside deterioration calculator.[18]

Future studies should combine serial function with imaging and electrophysiology, distinguish fluctuation from persistent loss, and record crossover decisions and competing diagnoses. Useful models should improve intervention timing and remain accessible in routine practice.

4 CONCLUSION

Clinical deterioration in DCM is heterogeneous and incompletely predictable at baseline. Radiculopathy and electrophysiological abnormalities identify increased risk of incident myelopathy in selected nonmyelopathic compression cohorts, while progressive gait, hand, motor, or sphincter dysfunction is directly actionable in established disease. Imaging refines the assessment but does not provide a universal threshold for surgical timing. Structured surveillance and prompt response to documented neurological change remain more reliable than overconfident prediction from a single baseline characteristic.

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