Recognising the Silent Squeeze: Why Physiotherapists Should Pay Attention to Degenerative Cervical Myelopathy

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INTRODUCTION

Degenerative cervical myelopathy (DCM) is the most prevalent cause of spinal cord impairment in adults, yet remains under-recognised in primary care (Davies et al., 2018). DCM affects approximately 2.3% of adults, rising to a striking 5% in those over the age of 40 years of age (i.e., one in 20) (Davies et al., 2018). Despite this, diagnosis is frequently delayed (average of 2.5 years), by which time individuals with DCM face irreversible neurological impairment and very low quality of life (Davies et al., 2018).

With an ageing population, physiotherapists are increasingly likely to encounter individuals with undiagnosed DCM and are well-positioned as first-contact clinicians to provide early recognition and appropriate referral. However, our recent survey data indicates very low levels of condition awareness and confidence in recognising the clinical features of DCM among primary care clinicians, including physiotherapists (Chauhan, Kennedy et al., 2025). This editorial provides a brief overview of DCM and highlights key considerations for physiotherapists to support timely recognition and referral.

CONDITION OVERVIEW

DCM arises from chronic compression of the cervical spinal cord due to commonly occurring age-related changes including intervertebral disc degeneration, osteophyte formation, ligamentous hypertrophy, and congenital central canal narrowing (Davies et al., 2018). Chronic cord compression results in spinal cord ischaemia, neuronal inflammation, and cellular apoptosis (programmed cell death) (Davies et al., 2018). Unlike acute spinal cord injury, DCM typically has an insidious onset of symptoms that progressively evolve over months or years (Nouri et al., 2022).

Clinically, individuals with DCM may present with progressive gait and balance impairment, fine motor hand impairment, hand clumsiness, and hand/upper limb numbness and/or paraesthesia (Table 1) (Jiang et al., 2023). Due to delays in diagnosis and intervention, many individuals experience a stepwise pattern of deterioration characterised by periods of relative stability followed by episodes of sudden decline, which may occur spontaneously or following seemingly minor trauma (Nouri et al., 2022).

Importantly, neurological deficits associated with DCM are largely irreversible (Nouri et al., 2022). Delayed recognition and intervention are associated with significant long-term consequences; up to 95% live with chronic disability (Fehlings et al., 2013), 37% are unable to maintain employment, and 42% are unable to maintain day-to-day independence (Pope et al., 2020). Surgical decompression is the primary intervention for DCM and aims to halt further neurological decline rather than reversing established impairment (Fehlings et al., 2017). Non-operative management is seldom recommended, except in selected cases of mild or stable DCM, given the established progressive natural history (Chauhan, Demetriades et al., 2025).

The clinical examination findings in DCM differ from the lower motor neuron signs typically associated with unilateral cervical radiculopathy with nerve root compression (diminished or absent reflexes). Instead, spinal cord compression results in upper motor neuron dysfunction (Table 1) due to disruption of descending inhibitory pathways.

A diagnosis of DCM requires concordance between clinical signs and/or symptoms and spinal cord compression on T2-weighted cervical magnetic resonance imaging (MRI) (Figure

Table 1

Characteristic Clinical Features of DCM

Characteristic	Features associated with DCM
Higher risk clinical populations	Aged over 40 years Established diagnosis of lumbar spinal stenosis History of head/neck trauma Ethnic groups: New Zealand Māori, Pacific peoples, Korean, Japanese
Characteristic symptoms	Gait disturbance (wide, unsteady, spastic-like gait) Balance decline (unexpected trips/stumbles/falls) Hand fine motor impairment (difficulty with tasks requiring dexterity) Hand clumsiness (dropping objects) Hand and/or upper limb numbness/paraesthesia (often bilateral)
Clinical examination signs	Romberg's sign Tandem gait (heel-toe walking – caution if high falls risk) Deep tendon hyper-reflexia Hoffmann's sign Inverted supinator sign/brachioradialis reflex Ankle clonus sign Babinski sign

1). Isolated MRI-evidence of high-grade central canal stenosis or asymptomatic spinal cord compression, albeit still clinically relevant for patient counselling and ongoing monitoring, do not in isolation constitute a diagnosis of DCM (Martin et al., 2022). Plain radiographs and computed tomography are non-diagnostic but often aid surgical planning, while myelography can provide diagnostic insight where MRI is contraindicated (Martin et al., 2022).

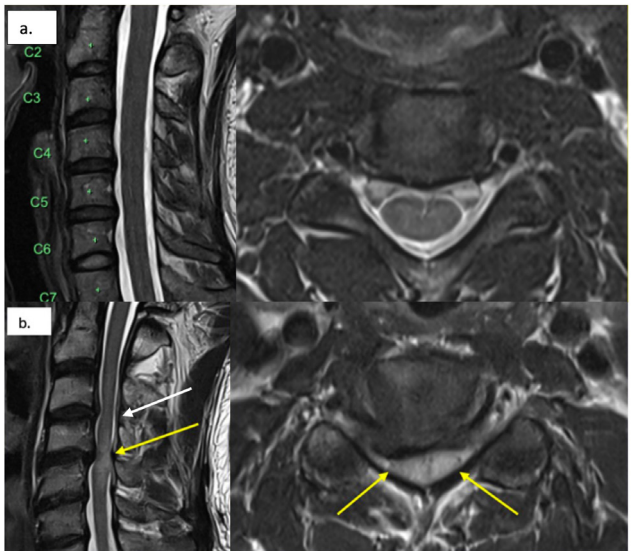
WHERE DOES EVALUATION OF DCM FIT INTO USUAL CLINICAL ASSESSMENT?

Several clinical groups are at an elevated risk of DCM including individuals over the age of 40 years, including those with an established diagnosis of lumbar spinal stenosis or with a history of head/neck trauma (Cervellini et al., 2025). The prevalence of DCM is greater among individuals of East Asian ethnicity, particularly Korean and Japanese, given the genetic predisposition for ossification of the posterior longitudinal ligament and ligamentum flavum, which causes canal stenosis and cord compression (Nouri et al., 2015). In the Aotearoa New Zealand context, Māori and Pacific peoples are disproportionately affected, which has been attributed, in part, to congenitally narrower cervical anteroposterior canal dimensions compared with New Zealand Europeans (1.5 mm narrower in Māori; 2.5 mm in Pacific peoples), conferring a greater vulnerability to canal and cord compromise (Goddard-Hodge et al., 2024).

The clinical presentation of DCM is subtle in its early stages and frequently misdiagnosed as peripheral neuropathies such as carpal tunnel syndrome or cervical radiculopathy. Other misdiagnoses include age-related deconditioning (particularly in older adults with falls risk) or chronic widespread pain syndromes (Cervellini et al., 2025). Non-specific sensorimotor limb dysfunction and balance decline, as seen with DCM, are difficult for patients to attribute to cervical pathology.

Figure 1

Cervical Spinal Magnetic Resonance Imaging With and Without Cord Compression



Note. a. Largely unremarkable T2-weighted sagittal (left) and axial (right) sequences with a shallow C5–6 disc protrusion, but without contact or compression of the spinal cord. b. C4–5 spinal cord compression and cord oedema (yellow arrows), with moderate central canal stenosis, without cord compression, at C3–4 (white arrow). Reproduced and adapted with permission from the *New Zealand Medical Journal* (Chauhan, Harvey et al., 2025).

Additionally, neck pain is not a consistent feature of DCM and may be mild or absent (Jiang et al., 2023). As such, proactive and targeted questioning for the above at-risk clinical groups is required in the evaluation of DCM. The absence of neck pain should not be falsely reassuring where neurological symptoms or functional decline are present.

Physiotherapists should incorporate the assessment of upper motor neuron examination signs into their usual clinical neurological examination, for the aforementioned at risk groups (Table 1). Deep tendon hyper-reflexia in this age group (> 40 years of age) may be an early sign of DCM and should prompt clinicians to enquire further regarding balance and hand function (Jiang et al., 2024; Chauhan, Segar et al., 2025). The presence of multiple upper motor neuron signs increases the likelihood of DCM and should prompt referral for orthopaedic spinal or neurosurgical consultation to confirm the diagnosis and guide management (Cervellini et al., 2025; Chauhan, Segar et al., 2025).

While these examination features raise suspicion for DCM, they are not pathognomonic and may be observed across a range of central neurological diseases. In the absence of characteristic DCM symptomatology or recognised risk factors (Table 1), routine clinical screening may yield false positives, resulting in potential over-investigation and undue patient anxiety – particularly when isolated examination findings are interpreted outside appropriate clinical context (Jiang et al., 2024).

PHYSIOTHERAPY MANAGEMENT

Physiotherapists in Aotearoa New Zealand increasingly practise as first-contact practitioners, and are therefore likely to encounter undiagnosed DCM, which may present "silently" alongside musculoskeletal pain or vague neurological symptoms.

While non-operative management of DCM is not well-established at present, physiotherapists can play a broader role in the care of suspected or confirmed cases. First, where balance or gait impairment is identified, physiotherapists should counsel patients regarding the use of mobility aids and fall-risk reduction strategies, and implement appropriate lower limb and balance rehabilitation. This is particularly important given the broader consequences of falls in this age group and risk of further spinal cord compromise (Chauhan, Demetriades et al., 2025). Further, patient education regarding key symptoms of DCM progression and the need for re-evaluation is emphasised (Table 1) for individuals with incidentally identified high-grade central canal stenosis or asymptomatic cord compression on imaging (Chauhan, Demetriades et al., 2025). Finally, individuals with suspected or confirmed DCM may seek physiotherapy care for the management of concurrent spinal-related pain or mobility impairments. Physiotherapists should consider the presence of clinical DCM features (Table 1) in this decision-making process. While gait, balance and limb strength, coordination, and functional exercises can be safely prescribed, physiotherapists should exercise caution when considering cervical traction, high-grade rotatory mobilisation, and high-velocity thrust techniques (Chauhan, Demetriades et al., 2025).

CONCLUSION

With a greater awareness and understanding of DCM as a progressive and often "silent" condition, physiotherapists can support earlier recognition and referral to allow for timely intervention, all of which are key determinants of long-term functional outcomes and quality of life.

DISCLOSURES

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CONTRIBUTIONS OF AUTHORS

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