Spondylosis, Degeneration of Intervertebral Disc

Diagnosis/Condition:	Spondylosis and allied disorders, Degeneration of cervical intervertebral disc, Degeneration of thoracic or lumbar intervertebral disc
Discipline:	DC
ICD-10 Codes:	M47.812; M47.814; M47.817; M50.30
Origination Date:	1996
Review/Revised Date:	07/2025
Next Review Date:	07/2027

The focus of this clinical pathway is spondylosis. This is a chronic, noninflammatory disease caused by degeneration of the disc and/or facet joints. The etiology of spondylosis is multifactorial. It occurs primarily in the Lumbar and Cervical regions and can occur with or without myelopathy or radiculopathy. The degenerative changes are associated most with the natural consequences of aging (primary) but also occasionally with post traumatic degeneration (secondary). While the popular press and some medical sources refer to spondylosis as a product of "wear and tear", most studies suggest no causative relationship between the development of spondylosis and factors such as lifestyle, height, weight, body mass, physical activity, cigarette and alcohol consumption, or reproductive history.[1] The effects of heavy physical activity are controversial in a purported relationship to disc degeneration. Rates are higher in patients with Diabetes Mellitus.[2] A 2024 study (n=4109) highlights a significant association between intervertebral disc degeneration (IDD) and low-grade bacterial infections, predominantly Propionibacterium acnes (P. acnes) being the most common causative agent.[3]

Spondylosis is extremely common. In the US, more than 80% of individuals older than 40 years have lumbar spondylosis, increasing from 3% of individuals aged 20-29 years. Approximately 95% of people age 65 have cervical spondylosis to some degree.[4] Degenerative cervical myelopathy is most caused by cervical spondylosis, with a predominant elderly population, and is the most common cause of spinal cord impairment.[5]

As the intervertebral disc ages, its hydrophilic properties decline with resultant loss of disc height and increased mechanical instability. This results in greater tensile forces at the junction between the vertebral body margin and the outer margins of the disc and intervertebral ligaments. This in turn causes the formation of new bone excrescences seen on x-ray examination and are described radiographically as osteophytes.

Often referred to as degenerative disc disease and spinal osteoarthritis, it is neither uniquely pathological nor inflammatory. By itself, spondylosis is most likely not a cause for clinical concern. With few exceptions, most of the evidence indicates there is no reliable association between degenerative changes of the disc and facet joints and pain.[6,7,8,9] For example, lumbar spondylosis is present in 27-37% of the asymptomatic population and there is no greater frequency of signs or symptoms among individuals with osteophytes than among those without them. However, spondylosis can become significant with more morbid conditions such as acute and chronic spine pain, myelopathy and radiculopathy.

It has been long observed that a diagnosis of "spondylosis, disc degeneration or arthritis" is not without consequence for patients. "The patient hears that there is a long-term structural difficulty with their spine or, even worse, is given a diagnosis of arthritis. Consequently, the patient takes care of his or her back and limits physical activity, which is a natural response to damage to an organ. This response runs directly contrary to current advice on the management of back pain, which emphasizes active rehabilitation to prevent chronic disability, even in the face of continuing pain."[10] In similar fashion, referring to spondylosis as a product of "wear and tear" may lead to the unhelpful tendency to avoid physical activity in an effort to reduce wear and tear.[11]

Surgery is often recommended for patients with spondylosis in association with symptoms of disabling back or neck pain, radiculopathy, lateral or central stenosis. The evidence supporting surgery for degenerative lumbar stenosis is of variable quality and shows conflicting results regarding a preferred surgical technique and patient centered outcomes.[12,13] Given the uncertainty of invasive procedures, consideration of non-surgical alternatives is warranted.

Subjective Findings and History

- Past medical history with documentation of diagnosis.
- Age, family history.
- Acute, subacute, or chronic spine pain. Often local, uni- or bilateral.
- Radicular or referred limb pain and paresthesia.
- Symptoms of myelopathy (spinal cord compression).
- Tingling, numbness, or weakness.
- Difficulty with fine motor skills, such as writing or buttoning a shirt.
- Difficulty walking.
- Loss of urinary or bowel control.
- Issues with balance and coordination.
- Proprioception can be impaired.[14]

Objective Findings

- Loss of intervertebral disc integrity that is due to degenerative change associated with normal aging, immobilization, or post-traumatic conditions.

- Screen for “red flags”.
- Radiographic examination: Evidence of degenerative changes on imaging.
- Loss of normal spinal ROM.
- Palpation for tenderness.
- Segmental joint dysfunction/subluxation.
- Spasm of paravertebral muscle.
- Orthopedic and neurological examination directed at differentiating scleratogenous from neurogenic pain.
- Discogenic stress maneuvers often reproduce the patient's lumbar and referred symptoms.
- Provocative orthopedic tests may reproduce the pain (e.g., straight leg raise and other tests that cause spinal motion may increase back pain).

Assessment

Disc degeneration should be considered a secondary diagnosis. The clinical impression should indicate the specific anatomical structures (spinal level) involved and clinically correlate them with the mechanism of injury, history, subjective complaints, and objective findings.

Kirkaldy-Willis described a pathophysiologic model that occurs in 3 phases of a continuum with gradual transition from one to the next. However, great variation of phases can be expected in different discs in any given individual and individuals of similar ages vary greatly.

- Phase I, the dysfunctional phase, is characterized histologically by circumferential tears or fissures in the outer annulus. Changes to the zygapophyseal joints during the dysfunctional phase may include synovitis and hypomobility. The facet joint may serve as a pain generator.
- Phase II may result from progressive loss of mechanical integrity of the three-joint complex. Disc-related changes include multiple annular tears (e.g. radial, circumferential), internal disc disruption and resorption, or loss of disc-space height. Changes in the zygapophyseal joints include cartilage degeneration, capsular laxity, and subluxation. The biomechanical result of these alterations leads to segmental instability and associated symptoms.
- Phase III, stabilization, is characterized by further disc resorption, disc-space narrowing, endplate destruction, disc fibrosis, and osteophyte formation. Discogenic pain from such discs may have a higher incidence than that of the pain from the discs in phases I and II.

Plan

Spondylosis by itself probably does not require treatment and should not be used to justify a treatment plan. Spondylosis, however, is often associated with symptomatic conditions, for example, axial spine pain, referred pain, intersegmental dysfunction or radicular syndromes,

all of which may require therapeutic intervention. There is a growing body of evidence based treatment recommendations for conditions such as pain that is associated with spondylosis.[15] An appropriate treatment plan should address the pathomechanics and clinical sequelae that attend each phase of disc degeneration as well as any associated disorder (e.g., pain, dysfunction, radicular syndrome).

Diet:

- Weight loss when indicated.

Supplements and Nutrients:

- Supplementation, e.g. glucosamine, chondroitin, MSM.

Pharmaceuticals (OTC):

- Medications: OTC NSAIDS, mild analgesics, herbal medicines.

Physical Modalities (Western):

- Physical therapy modalities.
- Percutaneous neuromuscular electrical stimulation.[16]
- Low level laser therapy.[17]

Movement and Exercise:

- Active exercise for mobility and strength.
- Posture training.

Manual Adjustments/Manipulation:

- Spinal manipulation and/or mobilization with exercises.[18,19]

Injection Therapies:

- Epidural administration of PRP offers comparable benefit as epidural steroid injection (ESI) in the management of radiculopathy due to LDD. The safety profile of the epidural PRP is also similar to ESI.[20]

Lifestyle Counseling:

- Education is one of the most important components of any back-care program and should include an explanation of the natural history of acute, subacute, and chronic disc problems.
- Smoking cessation.[21]
- Activity/work restriction, if appropriate.

Length of Treatment

- Conservative therapy: 3-6 months.

Referral Criteria

- Referral to an appropriate non-surgical spine specialist if no improvement noted after 6-8 weeks.
- Consultation with a spine surgeon may be appropriate for patients with intractable severe function-limiting symptoms, for those with symptoms lasting longer than 6 months who have had no relief from nonsurgical approaches, and for people with abnormal neurologic findings.

Outcome Assessment Tools

There is no OAT specific to spondylosis outcomes apart from signs and symptoms such as pain, radiculopathy, or myelopathy that may be associated with spondylosis. The commonly used OAT's (VAS, PFS, PROs, etc.) may be useful to track patient progress.

Resources for Clinicians

eMedicine from WebMD. Lumbar spondylosis. Bruce M Rothschild, MD
http://www.emedicine.com/med/topic2901.htm

eMedicine from WebMD. Lumbar degenerative disc disease. Rajeev K Patel, MD.
http://www.emedicine.com/PMR/topic67.htm

Resources for Patients

Spine Universe. https://www.spineuniverse.com/conditions/spondylosis

KP.org. https://healthy.kaiserpermanente.org/oregon-washington/health-wellness/health-encyclopedia/he.lumbar-spondylosis.abr8401?kpSearch=spondylosis

Calliet R: Neck pain and disability.

Kirkaldy-Willis W: Managing low back pain. Churchill-Livingstone, New York, 1992.

Bogduk N, Twomey LT. Clinical Anatomy of the Lumbar Spine. 6th *ed*. London: Churchill Livingstone;July 30, 2022.

Clinical Pathway Feedback

Heraya desires to keep our clinical pathways customarily updated. If you wish to provide additional input, please use the e-mail address listed below and identify which clinical pathway you are referencing. Thank you for taking the time to give us your comments.

Clinical Services Department: cs@herayahealth.com

Disclaimer Notice

Heraya clinical pathways are a resource to assist clinicians and are not intended to be nor should they be construed/used as medical advice. The pathways contain information that may be helpful for clinicians and their patients to make informed clinical decisions, but they cannot account for all clinical circumstances. Each patient presents with specific clinical circumstances and values requiring individualized care which may warrant adaptation from the pathway. Treatment decisions are made collaboratively by patients and their practitioner after an assessment of the clinical condition, consideration of options for treatment, any material risk, and an opportunity for the patient to ask any questions.

Heraya makes no representation and accepts no liability with respect to the content of any external information cited or relied upon in the pathways. The presence of a particular procedure or treatment modality in a clinical pathway does not constitute a representation or warranty that this service is covered by a patient's benefit plan. The patient's benefit plan determines coverage.

---

[1] Battie, M.C.; Videman, T.; Parent, E.; Lumbar Disc Degeneration: Epidemiology and Genetic Influences. SPINE. 2004 DEC Vol. 29(23) Pgs. 2679-90.

[2] Gao Z, Chen K, Huang C, Li Y, Diabetes Mellitus and Intervertebral Disc Degeneration: A Meta-Analysis. World Neurosurg. 2024 Aug:188:e81-e92. doi: 10.1016/j.wneu.2024.05.043. Epub 2024 May 14.

[3] Zhang M, Jia J, Deng L Cao Z, Hu X, Lei S, Zhang G, Zhu D, Duan Y, Kang X, Risk factors associated with low-grade virulent infection in intervertebral disc degeneration: a systematic review and meta-analysis. Spine J. 2024 Jun;24(6):1034-1045. doi: 10.1016/j.spinee.2024.02.001. Epub 2024 Feb 15.

[4] Takagi MD, Javed Khader Eliyas MD, Noam Stadlan. Cervical Spondylosis: An Update on Pathophysiology, Clinical Manifestation, and Management Strategies MDDis Mon . 2011 Oct;57(10):583-91. doi: 10.1016.

[5] Williams J, D'Amore P, Redlich N, Darlow M, Suwak P, Sarkovich S, Bhandutia A. Degenerative Cervical Myelopathy: Evaluation and Management, Orthop Clin North Am. 2022 Oct;53(4):509-521. doi: 10.1016/j.ocl.2022.05.007.

[6] Raastad J1, Reiman M2, Coeytaux R3, Ledbetter L4, Goode AP5. The association between lumbar spine radiographic features and low back pain: a systematic review and meta-analysis. Semin Arthritis Rheum. 2015 Apr;44(5):571-85. doi: 10.1016/j.semarthrit.2014.10.006. Epub 2014 Dec 8.

[7] Rudy IS1, Poulos A1, Owen L1, Batters A1, Kieliszek K1, Willox J1, Jenkins H1. The correlation of radiographic findings and patient symptomatology in cervical degenerative joint disease: a cross-sectional study. Chiropr Man Therap. 2015 Feb 9;23:9. doi: 10.1186/s12998-015-0052-0. eCollection 2015.

[8] Maataoui A1, Vogl TJ1, Middendorp M1, Kafchitsas K1, Khan MF1. Association between facet joint osteoarthritis and the Oswestry Disability Index. World J Radiol. 2014 Nov 28;6(11):881-5.

[9] Wilkens P1, Scheel IB, Grundnes O, Hellum C, Storheim K. Prognostic factors of prolonged disability in patients with chronic low back pain and lumbar degeneration in primary care: a cohort study. Spine (Phila Pa 1976). 2013 Jan 1;38(1):65-74. doi: 10.1097/BRS.0b013e318263bb7b.

[10] Roland M, van Tulder M.Should radiologists change the way they report plain radiography of the spine? The Lancet, Volume 352, Issue 9123, Pages 229 - 230, 18 July 1998

[11] Stokes, I.A.; Iatrindis, J.C.; Mechanical Conditions That Accelerate Intervertebral Disc Degeneration: Overload Versus Immobilization. SPINE. 2004 DEC Vol. 29(23) Pgs. 2724-32

[12] Gibson JA, Waddell G. Surgery for degenerative lumbar spondylosis. Cochrane Database of Systematic Reviews 2005, Issue 4. Art. No.: CD001352. DOI: 10.1002/14651858.CD001352.pub3

[13] Nikolaidis I, Fouyas IP, Sandercock PAG, Statham PF. Surgery for cervical radiculopathy or myelopathy. Cochrane Database of Systematic Reviews 2010, Issue 1. Art. No.: CD001466. DOI: 10.1002/14651858.CD001466.pub3

[14] Shankar Reddy R, Shanker Tedla J, Dixit S, Abohashrh M. Cervical proprioception and its relationship with neck pain intensity in subjects with cervical spondylosis, Musculoskelet Disord, 2019 Oct 15;20(1):447. doi: 10.1186/s12891-019-2846-z.

[15] Hurwitz E, et al. Treatment of Neck Pain: Noninvasive Interventions: Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders JMPT Volume 32, Issue 2, Supplement, Pages S141-S175 (February 2009)

[16] Qiang Miao 1, Jian-Hong Qiang, Yan-Li Jin. Effectiveness of percutaneous neuromuscular electrical stimulation for neck pain relief in patients with cervical spondylosis. Randomized Controlled Trial Medicine (Baltimore) . 2018 Jun;97(26):e11080.

[17] Chow R, et al. Efficacy of low-level laser therapy in the management of neck pain: a systematic review and meta-analysis of randomised placebo or active-treatment controlled trials
The Lancet, Volume 374, Issue 9705, Pages 1897 - 1908, 5 December 2009

[18] Vieira-Pellenz F1, Oliva-Pascual-Vaca A2, Rodriguez-Blanco C2, Heredia-Rizo AM3, Ricard F4, Almazán-Campos G4. **Short-term effect of spinal manipulation on pain perception, spinal mobility, and full height recovery in male subjects with degenerative disk disease: a randomized controlled trial.** Arch Phys Med Rehabil. 2014 Sep;95(9):1613-9. doi: 10.1016/j.apmr.2014.05.002. Epub 2014 May 24.

[19] Zhu LG, Wei X, Wang SQ. Does cervical spine manipulation reduce pain in people with degenerative cervical radiculopathy? A systematic review of the evidence, and a meta-analysis. Clin Rehabilit 2016;30:145–55. [PubMed]

[20] Muthu S, Viswanathan VK, Gangadaran P, Is platelet-rich plasma better than steroids as epidural drug of choice in lumbar disc disease with radiculopathy? Meta-analysis of randomized controlled trials. Exp Biol Med (Maywood). 2025 Feb 4:250:10390. doi: 10.3389/ebm.2025.10390. eCollection 2025.

[21] Akmal, M.; Kesani, A.; Anand, B.; Singh, A.; Wiseman, M.; Goodship, A.; Effect of Nicotine on Spinal Disc Cells: A Cellular Mechanism for Disc Degeneration. SPINE. 2004 MAR Vol. 29(5) Pgs. 568-75.