Treatment options and pathophysiology of degenerative spine disease

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Abstract

The adult spine is frequently the source of severe pain, deformity and disability. Axial spinal pain is very common, affecting adults of all ages. It can be caused by biomechanical factors, degenerative conditions, trauma, insufficiency fractures, infection or tumours. Associated nerve root compression and spinal instability may cause additional pain and provide a target for surgical intervention. An important socio-economic burden results, including: a high number of consultations (e.g. general practitioner, physiotherapist and specialists), investigations (e.g. MRI scan), loss of time from employment and disability. This article summarizes the degenerative processes that affect the adult spine and the treatment options available.

Keywords Back pain; degenerative disc disease; disc prolapse; microdiscectomy; spinal fusion; spinal stenosis

Introduction

The principal causes of neural compression attributable to spinal degenerative disease include prolapsed fragments of degenerate discs and sequelae of facet joint degeneration including facet joint hypertrophy and ligamentous thickening causing spinal canal stenosis. The lumbar and cervical spine regions are most commonly afflicted due to the high mobility in these areas.

Pathogenesis of disc degeneration

Disc degeneration is most commonly encountered in the most mobile parts of the spine (lumbar, sub-axial cervical and thoracolumbar junction) and those subject to axial loading (e.g. lower lumbar). Although biomechanical environmental factors are important, twin studies have indicated that genetic factors are of prime importance in causing degenerative disc disease.[1]

Disc degeneration is initiated by age-related dehydration and fragmentation of the nucleus pulposus.[2] This reduces tension in the nucleus pulposus and thereby reduces the radial forces exerted on the circumferential annulus. This creates a situation akin to letting air out of a pneumatic tyre. As a result of the reduction in 'splinting' of the annulus, increased mobility occurs, rendering the annulus vulnerable to tears, often in combination with dehiscence of the posterior longitudinal ligament. As a result, the nucleus can herniate into the extradural spinal canal. Rarely, disc herniation can be intradural. Disc herniation is usually posterolateral rather than central or extraforaminal (Figure 1) due to the relative weakness of the posterior longitudinal ligament in that area. In the lumbar spine, disc herniation (prolapse) causes compression of the transiting rather than exiting nerve root (i.e. an L4/5 disc prolapse causes L5 root compression). In the cervical spine a disc prolapse usually compresses the exiting root (e.g. a C5/6 disc prolapses causes C6 root compression).

Calcification of the cartilaginous endplate can contribute to the increased vulnerability to develop disc herniation with age. The endplate normally contributes to the nutrition of the disc, essential for the maintenance of the normal biochemical environment. With increasing age, calcification of the endplate impairs disc nutrition resulting in an almost avascular disc that calcifies and is more vulnerable to herniation, particularly in association with axial compressive and rotational forces.[2]

Patterns of disc herniation can vary: Schmorl's nodes represent herniation of the disc through the endplate into the adjacent vertebral body and are usually incidental. A limbus fracture can occur if such a herniation causes separation of a bone fragment from the bony endplate at the site of annular attachment.

Spinal stenosis is classified as central canal stenosis or lateral recess/foraminal stenosis. Central canal stenosis describes narrowing of the anterior–posterior dimensions of the spinal canal: it can be congenital or acquired as a result of degenerative disease or spondylolisthesis. The lateral recess is defined as the space between the vertebral body anteriorly, pedicle laterally and superior articular facet posteriorly. Hypertrophy of the superior articular facet, a common feature of facet joint degeneration, can contribute to compression of the exiting nerve root, along with hypertrophy and buckling of the ligamentum flavum causing lateral recess stenosis.

In the lumbar spine, spinal stenosis causes neurogenic claudication from nerve root dysfunction, while in cervical spine, myelopathy, due to spinal cord dysfunction, occurs. The precise pathogenesis of symptoms is uncertain but includes neuronal compression as well as impaired neural blood supply and/or venous drainage.

Lumbar spine degenerative disease

Lumbar disc herniation

Disc herniation is most common at L4/5 and L5/S1 vertebral levels. The symptoms produced are dependent upon the size and direction of disc herniation. Symptoms are usually attributable to compression of the transiting nerve root but can be due to severe and complete compression of the cauda equina.[3]

Symptoms: Patients usually report initial low back pain accompanied by radicular pain. The onset can be gradual or abrupt and can occur with or without obvious provocation. Sometimes pain can be alleviated by flexing the hip and exacerbated by coughing

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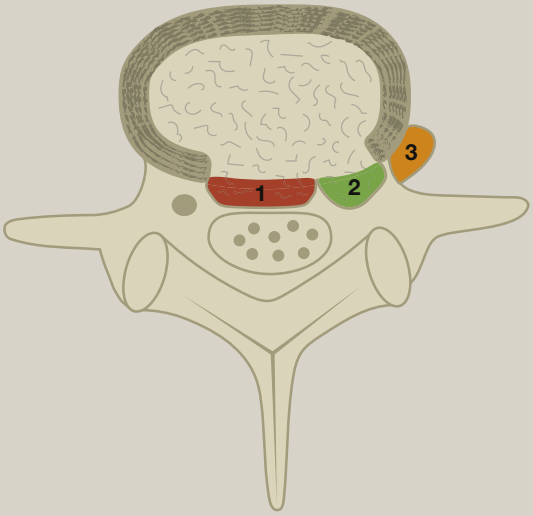
Common sites of disc herniation in the lumbar spine



1. A **central** disc prolapse may compress the cauda equina causing bilateral lower limb symptoms with or without an insensate bladder.
2. A **posterolateral** herniation may compress the transiting nerve root. If located more laterally the exiting root may be compressed in the foramen.
3. Only occasionally do **extra-foraminal** disc prolapses warrant any surgical intervention.

Figure 1

or sneezing. In around 70% of cases the disc fragment impinges the transiting nerve root that exits one level below the level of the disc. Compression of the nerve root causes back, buttock and lower limb pain in a dermatomal or myotomal distribution, myotomal weakness, sensory impairment and loss of innervated reflexes. Positive nerve root tension signs (e.g. Lasegue's manoeuvre) are usually present. Severe cauda equina compression causes acute onset insensate urinary retention/incontinence, faecal incontinence, decrease/loss of anal tone, saddle paraesthesia/anaesthesia, bilateral lower limb weakness and sexual dysfunction. Detection of red flag symptoms is crucial in the early assessment of lumbar disc pathology (Table 1). Yellow flag symptoms are indicative of a tendency to develop chronic pain.[4]

Neuroimaging: MRI is of prime importance in the investigation of spinal disease. T1- and T2-weighted images provide information concerning the extent of disc degeneration, herniation, thecal sac and nerve root compression (Figures 2 and 3). Modic described associated changes in adjacent vertebral bodies that may correlate with axial symptoms.[5] A CT myelogram can be performed if MRI is contraindicated, although imaging detail is inferior. If bone anatomy is required, CT is useful. The plain X-ray provides information regarding alignment, height of intervertebral space and it is used intraoperative for level check. Flexion/extension X-rays provide information regarding instability in cases with malalignment (i.e. spondylolisthesis).

Red and yellow flags

Red flags	Yellow flags
Thoracic pain	Negative attitude that back pain is harmful or potentially severely disabling
Fever and unexplained weight loss	Fear avoidance behaviour and reduced activity levels
Bladder or bowel dysfunction	An expectation that passive rather than active treatment will be beneficial
History of carcinoma	A tendency to depression, low morale and social withdrawal
Ill health or presence of other medical illness	Social or financial problems
Progressive neurological deficit	
Disturbed gait, saddle anaesthesia	
Age of onset <20 or >55 years	

Adapted from Ref 4.

Table 1

Management: Since 85% of patients improve with non-surgical treatment, an initial period (>6 weeks) of conservative management is recommended if there are no cauda equina compressive symptoms or profound neurological deficits. In around 30% of patients there is clinical improvement without resolution of the imaging appearance: chemical irritation of nerve roots, at the time of the prolapse has been postulated as

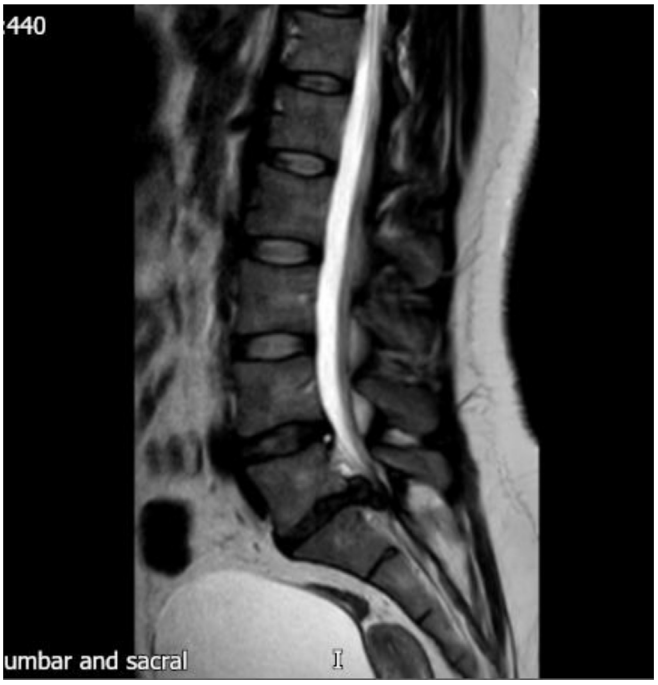


Figure 2 MRI T2 sequence sagittal view of the lumbar spine showing disc degeneration at L4/5 and L5/S1 level, with a very large disc prolapse at L5/S1 level.

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