

Spinal stenosis, radicular pain, and compression fractures: causes of back pain in elderly patients

Back pain is a common symptom that can indicate serious disease, particularly in elderly patients. Separation of the serious pathologies such as malignancy, infection, and cauda equina syndrome from benign axial back pain is a challenge. The red flags have clinical use for assisting in obtaining key features in the history and examination that may relate to sinister pathology. Knowing what and when to refer to a spinal specialist, such as an orthopaedic surgeon or neurosurgeon, is important. Spinal stenosis, radicular pain, and vertebral compression fractures of the spine may require specialist skills in discectomy, decompression, and reconstruction. This article deals with the assessment and treatment of spinal stenosis, radicular pain, and vertebral compression fractures.

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Back pain is a common symptom that can be a harbinger of serious disease, particularly in elderly patients. The clinical dilemma is to separate the serious spinal disorders presenting as back pain or neurological symptoms from the benign axial back pain that affects a substantial proportion of the population at some point in their lives.¹ The prevalence of back pain varies with age, with a point prevalence of 17–30%, a 1-month prevalence of 19–43%, and lifetime prevalence of 60–80%.²

In a survey, 80% of people older than 60 years of age recalled an experience of back pain in their lifetime.² Degenerative changes occur in almost all patients as part of normal ageing. At age 20 years, degenerative changes are noted on X-ray and MRI in less than 10% of the population.³ By age 40 years, such changes are seen in 50% of the asymptomatic population and by the age of 60, this number reaches 90%. In a set of three articles, we describe common life-threatening disorders of the spine. This second article covers spinal stenosis, radicular pain, and compression fractures. The key points are shown in the box.

Assessing back pain

A flag system for assessment of back pain is useful for identifying factors specific to individual patients, and to assist in the day-to-day management of spinal disorders. This tool highlights obstacles to recovery from back pain (table 1). Red flags identify risk factors for serious spinal

disorders. Yellow flags define psychosocial obstacles to recovery. Blue flags are related to occupational factors. Black flags may be administrative obstacles to returning people to employment. Orange flags identify psychiatric disorders. In elderly patients, who are more likely to be retired, doctors should be aware of the red and yellow flags because serious spine disorders are an important threat to life or to quality of life.

Box: Key points

1. Any patient reporting saddle anaesthesia has cauda equina syndrome until proven otherwise.
2. Red flags, such as previous history of cancer, weight loss, neurological deficit, new onset of spinal pain, immunosuppression, or simply an unwell patient should result in a low threshold for specialist investigations.
3. Mild or moderate spinal stenosis is best treated conservatively with a combination of pain-relieving agents and exercise. Surgical management is the best option for severe stenosis.
4. Surgical discectomy provides effective clinical relief for carefully selected patients with sciatica due to lumbar disc prolapses that do not resolve with conservative treatment.
5. Addressing factors such as falls, supplementation of calcium and vitamin D, and giving bisphosphonates is important in the conservative management of vertebral fractures.

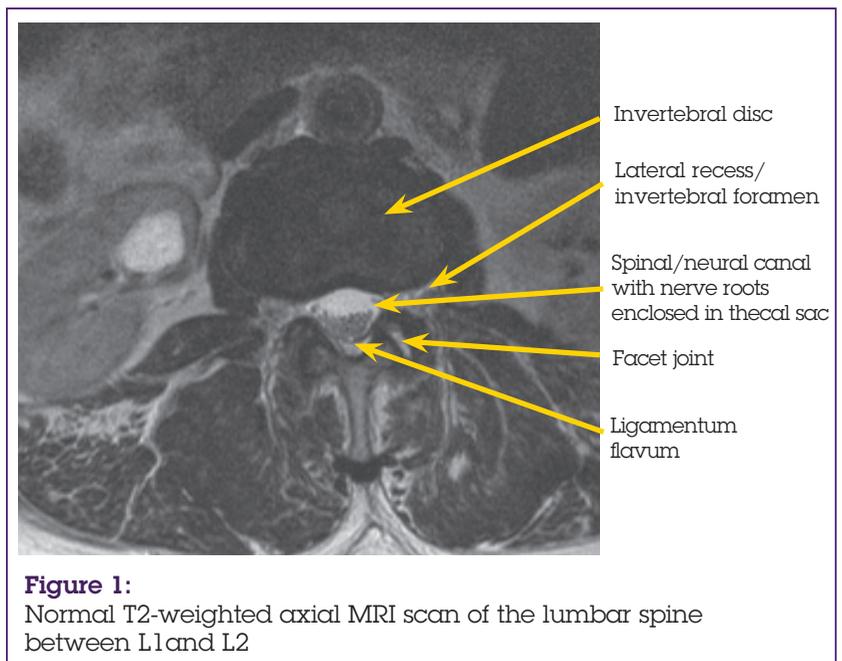
Spinal stenosis

Expansion of the facet joints as a result of degenerative changes can encroach on the central spinal canal and the lateral intervertebral foramina. This encroachment in the presence of large osteophytes from the vertebral bodies (especially in the lumbar spine) can become quite marked and result in substantial stenosis.¹ These changes can also cause intermittent disruption of function in the spinal cord and nerve roots. Such disruption can be permanent, leading to neurological deficits as a result of encroachment on the spinal cord—cauda equina syndrome.⁶

Symptoms of this condition are diverse; including leg pain and classic neurogenic claudication—characterised by pain during walking, numbness, tingling, weakness, and radiating pain down to ankles that is provoked by standing and relieved by sitting. Taking a flexed position reduces symptoms because the available space in the spinal canal increases.⁷

Lumbar spinal stenosis in elderly people is mainly due to degenerative disorders. In younger patients, developmental and congenital causes should be considered (eg, excessive scoliosis).^{8,9} In stenosis of the central canal, nerve roots in the cauda equina may be compressed. Stenosis of the lateral recess or foramina may cause compression of the nerve roots leaving the spine.¹⁰

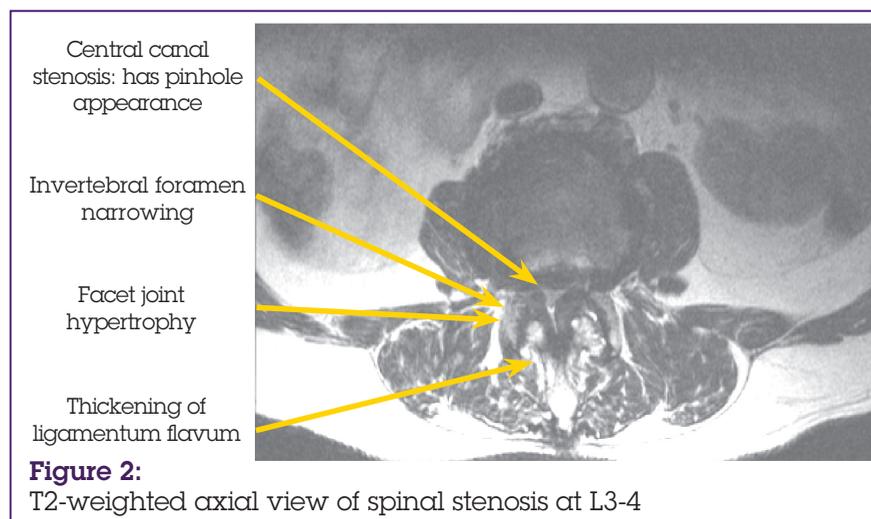
Discrepancies exist between clinical and radiological diagnosis for spinal stenosis. In a study by Boden and co-workers,³ lumbar stenosis is seen on MRI in 21% of asymptomatic individuals aged 60 or older, implying that a combination of clinical and imaging findings is best for diagnosis.



Symptoms are categorised as mild, moderate, or severe, on the basis of the extent of leg pain and pain-related disability.¹¹ Patients with severe symptoms have exercise intolerance and greatly restricted walking capacity, and may have bladder dysfunction (ie, urinary incontinence).¹² Conservative treatment with pain-relieving agents seems to be the natural choice when symptoms are mild.¹¹ Patients with moderate symptoms fall into a grey zone, in which appropriate treatment is not as obvious.¹³ Analgesia for mild and moderate symptoms of stenosis includes non-steroidal anti-inflammatory drugs; paracetamol; neuropathic agents such as gabapentin and amitriptyline, opiates; and muscle relaxants.

Studies suggest that surgical management is the best treatment option for severe stenosis.¹¹ Many patients require a posterior arthrodesis with instrumentation to treat the instability associated with spinal stenosis.¹⁴ Surgical treatment most commonly involves laminectomy, in which the nerve roots are decompressed by opening the spinal canal. Sometimes, when bony instability or severe back pain are particular problems, laminectomy can be supplemented with stabilisation.

Generally, data for the effect of conservative treatment are lacking in this group of patients because they often receive surgery shortly after diagnosis. In patients with neurogenic claudication, surgery tends to increase mobility. Average walking distance



among 30 patients increased from 100 yards to 1320 yards,¹⁵ and walking tolerance increased from less than 15 minutes in 50 of 51 patients to more than 30 minutes in 39 of 48 patients (19 with unlimited tolerance).¹²

NICE advocates using interspinous distraction devices for neurogenic claudication caused by lumbar spinal stenosis because no major safety concerns have been identified, but evidence of efficacy is limited and confined to short and medium terms.¹⁶ Implants are placed between adjacent spinous processes to act as physical blocks to extension (and thus lumbar canal narrowing) on standing or walking, relieving pressure on the nerves. Such procedures may have a role between conservative management of mild symptoms and the more invasive procedure of laminectomy for severe symptoms.

In elderly patients undergoing lumbar decompressive surgery, a greater number of fused levels is associated with greater intraoperative blood loss, longer operating time, and a higher prevalence of major complications such as wound infection, pneumonia, renal failure, respiratory distress, and myocardial infarction.¹⁴

Radicular pain

When compressive or rotational forces are applied to the spine, the fibres of the annulus fibrosis can be stretched beyond their elastic capacity and tear. If these tears are oriented radially, the nucleus pulposus may migrate through the tear, causing a protrusion of the disc beyond its natural borders. This can occur as an acute process in healthy discs with sufficient force. Degenerative discs that already have some degree of annular tearing, usually in a circumferential pattern, have less elastic proteoglycans and are less able to withstand these forces.¹

Loss of mechanical competence and flattening of the disc may create diffuse bulging, which should be differentiated from focal bulges or true herniations, characterised macroscopically by nuclear migration

	Type of barrier	Examples
Red flags	Risk factors for serious disorders	New onset of spinal pain at age <20 or >50 years Fever or night sweats Weight loss Pain that is non-variable and getting worse Immunosuppression Saddle anaesthesia Urinary or fecal incontinence
Yellow flags	Psychosocial obstacles to recovery	Fears and beliefs Catastrophising Compensation claims Illness behaviour (the sick role) Depression and anxiety Failure to accept medical reassurance
Blue flags	Occupational factors	Repetitive, low-paid employment Conflict in the workplace
Black flags	Administrative obstacles to work	No availability of therapeutic return to work
Orange flags	Psychiatric factors	Alcohol or drug abuse Fixed beliefs about cause of pain
Green flag	Normal	Patient has none of the above risk factors: reassurance and discharge

Table 1: The flag system for assessment of back pain^{4,5}

through radial fissures of the disc.¹⁷ Disc herniation requires pre-existing age-related degenerative changes. If the posterior longitudinal ligament is disrupted, nuclear material can extrude through the annulus, narrowing the diameter of the neural canal. If the disc herniation protrudes posteriorly in the midline to narrow the central canal of the spine, compression of the cauda equina or spinal cord can occur. If the disc protrudes laterally, it can extend into the lateral foramina, encroaching on the nerve root.

The diagnosis of disc prolapse is based on the history of sharp shooting pain which is dermatomal in distribution (radiculopathy) and associated with paraesthesia and or numbness. Ruling out all red flag symptoms (table 1) is important, especially problems with urinary and bowel incontinence, and saddle anaesthesia. A thorough neurological examination including a straight-leg raise is necessary because it helps to establish the pathogenesis. The most sensitive clinical sign is that leg pain is induced (in the leg that normally has pain) during a crossed straight-leg test in the contralateral leg. MRI of the lumbar spine, in which prolapses occur most often, is the gold-standard investigation. Conservative treatment options if red flags are absent include optimum pain management, injection therapy using local anaesthetics, physiotherapy, and reassurance. As for stenosis, analgesia

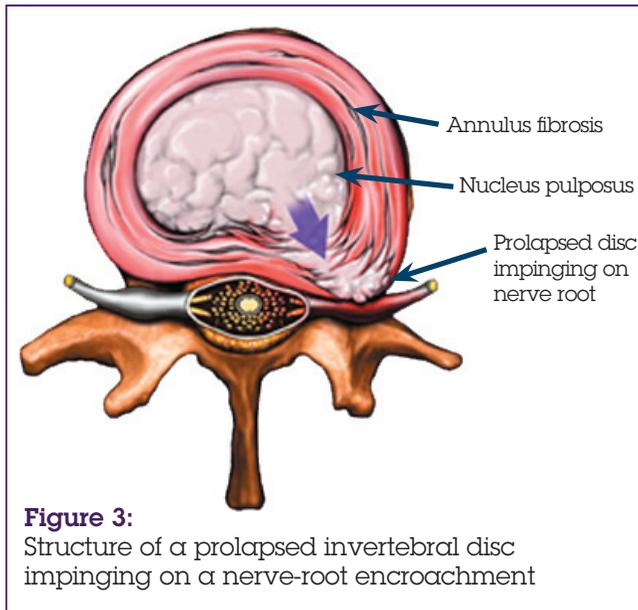


Figure 3:
Structure of a prolapsed intervertebral disc impinging on a nerve-root encroachment

for mild and moderate radicular pain includes non-steroidal anti-inflammatory drugs; paracetamol; neuropathic agents such as gabapentin and amitriptyline, opiates; and muscle relaxants. The primary rationale of any form of surgery for disc prolapse is to relieve the nerve-root irritation or compression due to herniated disc material.

Open discectomy, done with (micro-discectomy), or without an operating microscope, is the most common procedure, but some other less invasive surgical techniques are available. One clinical trial only has compared the surgical treatment of lumbar disc prolapse with conservative treatment. This trial was not masked and considerable crossover of treatments took place. Both patient's and observer's ratings showed that discectomy was significantly better than conservative therapy at 1 year, but no significant differences in outcomes were seen at 4 and

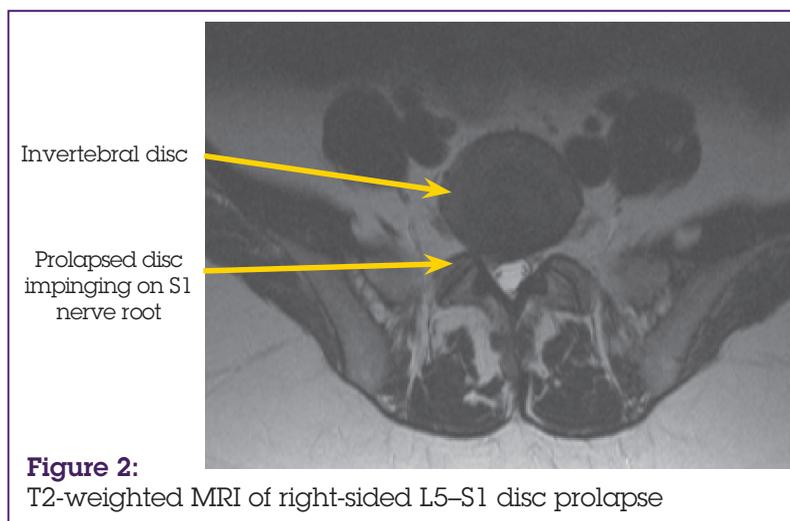


Figure 2:
T2-weighted MRI of right-sided L5–S1 disc prolapse

10 years.¹⁸ Surgical discectomy provides effective clinical relief for carefully selected patients with sciatica due to lumbar disc prolapses that fail to resolve with conservative management. No evidence is available to influence the choice of micro-discectomy or standard discectomy on the basis of favourable clinical outcomes.¹⁹

Compression fracture

A direct axial force applied to the spine, especially in flexion, can result in a collapse of the vertebral body. The intrinsic bone structure is disrupted, which is followed by oedema and healing of the bone.²⁰ If severe, these compression fractures can force spicules of bone or the entire vertebral body to move posteriorly, encroaching on the neural canal, or laterally, encroaching on the neuroforamen.¹ As a result of compression forces, the endplate of the vertebral body may collapse, allowing herniation of the nucleus pulposus into the vertebral body, which is known as a Schmorl's node.²¹ Epidemiological studies indicate a sharp increase in the incidence of osteoporotic vertebral fractures, and population prevalence as high as 52% in post-menopausal women has been noted in age-cohort studies.^{22,23} Vertebral fractures are associated with severe morbidity and a mortality of 30% over 5 years.²⁴

In 2000, the number of osteoporotic fractures in Europe was estimated at 3.79 million, of which 0.4 million were vertebral fractures.²⁵ The total direct costs were estimated at €31.7 billion, which is expected to increase to €76.7 billion in 2050, on the basis of expected changes in the demographics of Europe.²⁶ The incidence of new vertebral deformities has also been calculated by taking two sequential X-rays of the spine. In the European Perspective Osteoporosis Study (EPOS),²⁷ 1% of women at the age of 65 years and 2.9% between the ages of 75 and 79 years had new vertebral fractures every year. For men, the corresponding figure was 0.6% at age 65.

The diagnosis of spinal fractures is made by taking an X-ray of the spine and measuring the size of the vertebrae. Without treatment, postmenopausal women have a 19.2% risk of a second fracture within a year. If two or more fractures have already occurred, the risk further increases to 24%.²⁵ The reduced thickness and increased porosity of the cortical bone and the rarefaction of the trabecular network dramatically affect the fixation strength of implants. The impaired sensibility of osteoblasts to cyclic strain might be

responsible for the delay in fracture healing and remodelling in osteoporosis,²⁵ which lead secondarily to impaired implant stability and loosening.

These fractures are frequently not caused by acute trauma, and often result in progressive spinal deformity, and chronic back pain. The differentiation of acute from older osteoporotic fractures is not always easy and is mostly guided by the clinical symptoms (ie, acute pain). In unclear situations, the age of the fracture may be judged by MRI by the occurrence of bone bruise (acute fractures appear dark in T1-weighted scans).²⁵

The indication for new percutaneous treatment strategies such as stabilisation (eg, vertebroplasty), reduction (eg, kyphoplasty), or conservative treatment, is, however, dependent on the age of the fracture. The short-term results for invasive techniques are favourable, but at present, whether the internal application of bone cement to the vertebral body is effective in the long term, and outcome is better than conservative treatment, cannot be determined. Conservative modalities include analgesia, a reduction in risk factors, balanced diet and drugs, including sufficient amounts of calcium, vitamin D, and bisphosphonates, as well as physiotherapy, local

injection, and orthotic devices such as a thoracolumbar corset for support. Again, analgesia for mild and moderate symptoms of this condition includes non-steroidal anti-inflammatory drugs; paracetamol; neuropathic agents such as gabapentin and amitriptyline, opiates; and muscle relaxants.

We have no conflict of interest.

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