Practical neurology — 3

Back pain and leg weakness

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Hilary's story

Hilary, who is 67 years old, presented to her general practitioner with increasing low back pain over several months, on a background of similar but intermittent pain that had responded to simple analgesics over the past 20 years. Two years earlier, she had been knocked over by a slow moving car, twisting her torso and landing on her buttocks. Although she had had no new back pain immediately after the injury, her pre-existing back pain intensified and spread to involve her right leg over the ensuing weeks.

She described a continuous dull ache in her right lumbar region, which radiated across her hip and down to the lateral side of her right ankle. She experienced exacerbations with "stabbing, cramping spasms" of several minutes' duration in the same distribution, triggered by forward flexion, lifting, twisting, sneezing or standing in one position for a prolonged period. The dull ache was eased in certain sitting positions and with simple analgesic medication. She felt that her right leg had become weaker than it used to be, she recalled her foot occasionally catching on the ground while walking, and described the dorsum of her right foot as being intermittently numb. She did not report any disturbance of sphincter function.

The pain interfered with sleep, self-care tasks such as showering and dressing, and household and leisure activities such as gardening. Hilary could not sit for long periods in a car and reported that massage therapy had been unhelpful. While admitting that the back pain would "get her down", she denied feeling depressed or tearful about her situation. The precipitating accident was not subject to a compensation claim. Hilary's previous occupation as a shop assistant involved some heavy lifting. She had no history of diabetes and did not smoke.

Results of neurological examination above Hilary's waist were normal, as was the morphology of her back. Mild lower lumbar tenderness was elicited on palpation, and was worse on the right side. She had no leg muscle wasting or muscle fasciculations, and her muscle tone was normal. On the right side, there was moderate weakness of dorsiflexion of her great toe, and mild weakness of dorsiflexion, inversion and eversion of her foot, and of abduction and internal rotation at her hip. Plantar flexion was preserved. There was ill defined alteration in light tough sensation over the dorsum of her foot and great toe, but without a clear sensory deficit. Ankle reflexes were present, plantar responses were flexor, and saddle sensation was preserved. Straight-leg raising with her right leg above 45 degrees elicited pain in her right calf, but raising her right leg from the prone position (femoral stretch test) did not elicit pain. She walked with a subtle right footdrop. She could rise one-legged from a chair and could stand on the heel or toes of her left foot, but could only stand on the toes and not the heel of her right foot.

Summary

- Back pain is very common; it has a point prevalence of 25% and is the third most common reason for consultation in Australian general practice.
- A thorough history and examination can identify the minority of patients who require urgent neuroimaging or other targeted investigations.
- Careful correlation of clinical and radiological findings is required when abnormal neurological findings are detected. Radiological investigations may detect abnormalities at multiple levels but cannot confirm which level is primarily responsible for a patient's symptoms.
- A trial of conservative treatment is appropriate, even in cases of radiculopathy. Most patients with an acute episode of back pain recover within 6–12 weeks, but at least a third go on to have a recurrent episode within 1 year.
- Various invasive treatments, such as transforaminal steroid injection and discectomy, may speed up recovery from radiculopathy, but the long-term benefits of invasive treatment are uncertain.

APPROACH TO THE PROBLEM

Background

Back pain is one of the most common reasons for medical consultation.[1] It is the third most common reason for presentation to Australian general practice.[2] Men and women are equally affected, mostly between the ages of 30 and 50 years. It is the most costly cause of work-related disability.[3] With a point prevalence of 25% and with half the people with back pain likely to seek care, the economic burden is considerable, estimated at $1 billion in direct costs and a further $8 billion in indirect costs in 2001.[4]

Back pain is typically a recurrent condition. Although the prognosis for patients with an acute episode is favourable, with many recovering within 6–12 weeks, at least a third of such patients go on to have a recurrent episode within 1 year of the initial episode.[5] Recurrent episodes contribute to much of the burden of back pain.

The major causes of back pain are shown in Box 1. The most common cause (> 85% of patients) is presumed musculo-ligamentous injury or "strain", for which there is no clear pathophysiological basis and no identifying features on currently available clinical tests. It is most usefully classified as non-specific or idiopathic.[3,4] Less common causes include radiculopathy (eg, disc herniation or spinal stenosis), referred visceral pain (eg, penetrating peptic ulcer), and other serious spinal conditions (eg, vertebral fracture, malignancy or osteomyelitis).

1 Major causes of back pain and proportions of patients affected[1,3]

Non-specific causes (80%–90%)
- The presumed origin of pain is one of the many pain-sensitive structures in the spine such as the disc annulus, ligaments or zygapophyseal joints

Radiculopathy (5%–15%)
- Disc herniation and compressive or irritative radiculopathy
- Spinal stenosis
- Osteophytic nerve root compression
- Neoplastic nerve root compression or infiltration
- Inflammatory radiculopathy
- Failed back surgery syndrome
- Infection (eg, herpes zoster)

Referred visceral pain (1%–2%)
- Aortic aneurysm
- Gastrointestinal disease (eg, penetrating ulcer)
- Pelvic disease (eg, prostatitis)
- Renal disease (eg, pyelonephritis)

Other serious spinal conditions (1%–2%)
- Compression fracture
- Traumatic fracture
- Neoplasia (primary or metastatic)
- Infection (eg, osteomyelitis, epidural abscess)
- Spondyloarthropathies (eg, ankylosing spondylitis)

Other (2%–4%)
- Fibromyalgia
- Somatisation
- Malingering ◆

Interpretation of history and examination

As non-specific back pain commonly resolves spontaneously, and given that serious aetiologies are rare, undertaking extensive investigations routinely is wasteful and potentially counterproductive. Conversely, history taking and examination should be done carefully to disclose any red flags (Box 2), which raise the potential of more serious underlying causes and demand a more intensive approach.[1] In broad terms, these red flags relate to four diagnostic groups: serious neurological illness (eg, myelopathy, cauda equina syndrome), systemic illnesses (eg, malignancy, infection), vertebral fractures, and abnormal illness behaviour (eg, somatisation, psychogenic pain, malingering) (Box 2).

Information should be obtained about precipitating, exacerbating and relieving factors, and the location and radiation of the pain. Although pain may be described in mechanical or neuropathic terms, such terminology does not necessarily predict the underlying pathological process or the response to treatment. However, as illustrated in Hilary's case, radiation of pain below the knee ("sciatica"), especially with accompanying numbness or paraesthesia, strongly suggests irritation of a lumbosacral nerve root. In these patients, leg pain that is exacerbated by twisting the back, sneezing or a Valsalva manoeuvre implicates herniation of a disc (Box 3), whereas exertional pain in the calf which is eased by forward flexion (eg, walking uphill or pushing a shopping trolley) may suggest spinal canal stenosis. If none of these classic histories is obtained, other potential causes of lumbosacral radiculopathy, plexopathy or sciatic neuropathy should be considered.

2 Red flags that should raise index of suspicion of a serious underlying condition[1,3]

Serious neurological illness
- Leg weakness
- Saddle anaesthesia
- Sphincteric disturbance
- Gait disturbance
- Hyporeflexia
- Decreased anal tone
- Babinski sign

Systemic illness
- Age < 20 or > 50 years
- Immunosuppression
- Intravenous drug misuse
- Unrelenting pain (especially nocturnal)
- Constitutional symptoms

Vertebral fractures
- Trauma
- Age > 50 years
- Immunosuppression

Abnormal illness behaviour
- Unrelenting pain
- Depression
- Compensation dispute
- Poor sleep and/or appetite
- Psychogenic clinical signs
- Drug and/or alcohol misuse
- Multiple other somatic complaints

◆

Localising the lesion

It is important to attempt to localise the neuroanatomical cause of the clinical signs, as multilevel disc herniations are common in older adults and it can be unclear radiologically which level is primarily responsible for a patient's symptoms. Clinical and radiological correlation can confirm an L4/L5 disc protrusion — most commonly in the posterolateral direction, which can compress the L5 nerve root in the lateral recess of the spinal canal (Box 3), and less commonly in the far lateral direction, which can compress the descending L4 nerve root.

When the onset of back pain is historically remote from presentation with a neurological deficit, other neurological causes warrant exclusion, particularly in older patients who have multiple comorbidities (eg, disc herniation and motor neurone disease). Discomfort may limit a patient's ability to cooperate with examination, but encouragement must be given to fully attempt motor tasks. It is often helpful to get the patient to stand one-legged from a chair (L3/L4), or stand on the heel (L5) or toes (S1) of one foot, to unmask subtle weakness that is not readily detectable on the examination couch.

The sciatic (L5/S1) and femoral (L3/L4) stretch tests are reasonably sensitive, but not specific for the presence of radiculopathy.

Sciatic stretch test: This test is commonly called the "straight-leg raising test" or Lasègue sign, and is performed with the patient in the supine position with the pelvis stabilised; the result is positive if calf pain is elicited when the leg is raised to less than 70 degrees (although lower limits are used in some studies).[7] Straight-leg raising is a highly sensitive test for L5/S1 radiculopathies (positive in 90% of cases),[7] but it is non-specific and may be positive in many other conditions (eg, hip osteoarthritis, trochanteric bursitis). Conversely, the "crossed straight-leg raising test" (where straight leg raising induces contralateral pain) is insensitive (positive in about 30% of cases) but highly specific for radiculopathy (about 90%).[7]

Femoral stretch test: This test is performed lying on the unaffected side, with the pelvis stabilised and the affected leg pulled posteriorly.

Trying to localise the site of a radiculopathy based on the motor signs is challenging, as most muscles are innervated by more than one nerve root and this varies between individuals. The overall pattern of weakness is more critical than the function of any single muscle being tested (Box 4). Consideration should be given to the pretest probability of

finding an abnormality and the knowledge that 95% of all symptomatic disc herniations affect the L5 and S1 roots. Sensory symptoms are usually quite accurate in terms of localising the lesion, but sensory deficits may be ill defined or absent due to overlap of dermatomes (Box 4).

DIFFERENTIAL DIAGNOSES

The differential diagnoses of a footdrop (from caudal to cranial) include: common peroneal and sciatic neuropathies, lower lumbosacral plexopathy, radiculopathy, hemi-conus or hemi-cord lesions, motor neurone disease, and parasagittal cortical or subcortical lesions. These can usually be distinguished by history taking and examination alone.

> Hilary's hip weakness ruled out both common peroneal and sciatic neuropathies, and there were no upper motor neurone features or crossed sensory disturbance to suggest a pathological process involving the cerebrum or spinal cord. Her prominent sensory symptoms and lack of upper motor neurone features exclude motor neurone disease.

A spontaneous-onset lumbosacral trunk plexopathy, while distinctly unusual, can be clinically identical to an L5 radiculopathy, and may require nerve conduction studies for differentiation, if clinical suspicion of plexopathy is high.

APPROPRIATE USE OF INVESTIGATIONS

Urgent neuroimaging is indicated in situations where important neurological symptoms or signs are present in a patient with low back pain. This includes patients with extensive deficits, which are usually due to compression of multiple nerve roots in the cauda equina, and produce symptoms of peri-anal ("saddle") sensory loss, bowel and urogenital dysfunction and variable leg numbness and weakness.[9] Prolapse of a lower lumbar central disc is the commonest cause of such a disturbance, but malignancy must be excluded. Although computed tomography (CT) is widely available and provides good delineation of bony architecture, magnetic resonance imaging (MRI) is the modality of choice as it provides greater contrast between soft tissue structures and does not use ionising radiation. In Australia, this is best facilitated by urgent referral to a specialist (neurologist, neurosurgeon or spinal surgeon).

FINAL DIAGNOSIS

Right L5 radiculopathy; multilevel disc prolapse

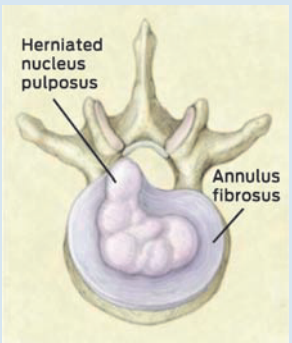
Expert opinion is unanimous in recommending that imaging not be performed in the absence of red flags (Box 2).[1,3,10] As well as the high cost, routine early imaging has not been shown to improve a patient's level of pain, function or satisfaction, in the short or long term (Grade A evidence).[11] As disc herniations are commonly found in asymptomatic individuals, the demonstration of possibly incidental abnormalities may reinforce or prompt abnormal illness behaviour. In cases of mild radicular features and a low suspicion of malignancy or infection, a trial of conservative therapy is recommended before undertaking neuroimaging (usually a CT scan before referral to a specialist), since many patients' symptoms will resolve spontaneously over several weeks. Neuroimaging is required if surgery is being considered for the treatment of persistent symptoms or neurological deficits.

> Hilary's symptoms did not resolve with conservative management and she was referred to a neurosurgeon. MRI of her lumbar spine was performed, which showed broad-based posterolateral L4/L5 disc herniation with compression of the right L5 nerve root (Box 5). The scan also showed fatty replacement of the right paraspinal muscles, suggesting denervation atrophy and indicating chronicity. This also excluded a plexopathy (Box 5) and indicated that no further investigation was necessary.

Nerve conduction studies are only indicated when the diagnosis is uncertain — for instance, when differentiating a low-lumbar plexopathy from an L5 or S1 radiculopathy. Sensory responses should be preserved in radiculopathy, as compression usually preserves the dorsal root ganglion and its distal (afferent) arm.

In other clinical settings, diagnostic studies that can justifiably be ordered in patients with back pain include plain x-rays and/or bone densitometry (for suspected vertebral compression fractures), tests for inflammatory markers (for possible infection), and investigations for referred causes of back pain (eg, ultrasound for suspected abdominal aneurysm).

3 Herniated disc



Degeneration or trauma affecting the annulus fibrosus allows the nucleus pulposis to escape, usually posterolaterally, and compress the descending nerve root in its lateral recess (eg, L5 nerve root at L4/L5). A far lateral disc prolapse may compress the exiting nerve root at the neural foramen (eg, L4 nerve root at L4/L5).

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4 Patterns of clinical signs associated with specific nerve root lesions[6,8]

Nerve root	Distribution of potential weakness	Hypo-reflexia	Sensory symptoms
L3	Hip flexion; hip adduction; knee extension	Adductor ± knee jerk	Low anteromedial thigh to knee
L4	Hip abduction and internal rotation; knee extension; foot dorsiflexion and inversion	Knee jerk	Knee and medial shin
L5	Foot and toe dorsiflexion; foot inversion and eversion; knee flexion; hip extension, abduction and internal rotation	Hamstring jerk	Anterolateral leg to hallux
S1	Ankle and toe plantar flexion; toe dorsiflexion; ankle eversion; knee flexion; hip extension	Ankle jerk	Posterior thigh and calf; lateral foot and sole

Approximate dermatomes for nerve roots in the leg are shown in the figure (bold lines indicate non-consecutive dermatomal boundaries, where overlap is less marked). Because of dermatomal overlap, radiation of sensory symptoms is a more reliable guide than sensory loss for localising the site of a radiculopathy.

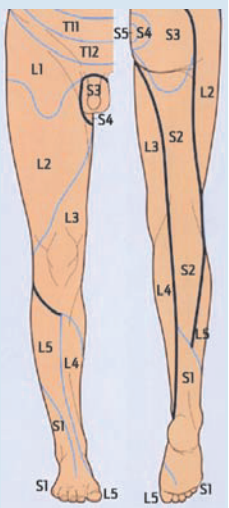


Figure (dermatome diagram only) reproduced with permission from Elsevier, copyright 2010.[6] ◆

MANAGEMENT

Management dilemmas

1. What type of symptomatic analgesia should be used?
2. Should the patient receive conservative treatment or interventional treatment?

Conservative treatment

In cases of acute-onset back pain and radicular symptoms, it is important to educate the patient. Non-interventional treatment is usually indicated as more than three-quarters of patients recover spontaneously within several weeks (Grade B evidence).[12] Despite widespread use, there is inconsistent evidence supporting treatment of sciatica with non-steroidal anti-inflammatory drugs (NSAIDs), traction, physiotherapy or immunosuppressive agents (Grade B evidence). As bed rest does not appear to hasten recovery or improve symptoms,[13] patients should generally be encouraged to mobilise. Although there is a paucity of data guiding the choice of specific analgesic medication (Grade C evidence),[12] it seems reasonable to extrapolate from back pain guidelines[14] and to commence with paracetamol (1 g four times a day) before escalating to an NSAID, then opioid analgesia (slow-release formulations of oxycodone are generally used), and (if pain lasts more than 3–4 weeks) a tricyclic antidepressant (Grade B evidence). The speed of escalation in this treatment algorithm should be determined by the degree of patient discomfort. As a general rule, conservative treatments should be encouraged for several weeks.

Interventional treatment

The approach to interventional treatment is highly individualised. It depends greatly on the response to conservative measures, the presence of radicular signs, and patient preferences. Some evidence indicates that transforaminal corticosteroid injection improves short- but not long-term outcomes,[15] but this is a controversial treatment (Grade B evidence)[12] that is best reserved for patients wishing to delay or avoid surgery (and should therefore be ordered by specialists). Although there is good evidence that early surgery (6–12 weeks) hastens recovery (Grade A evidence), it may not improve long-term (> 1 year) outcomes.[12] Various surgical approaches are available (open discectomy, micro discectomy and micro-endoscopic discectomy and transforaminal discectomy), but as none has proven superiority, patient preferences play a role in choosing between them (Grade B evidence). Fusion procedures should rarely be performed. Structured rehabilitation hastens recovery after surgery (Grade B evidence).[16]

FACT OR FICTION?

FACT: It is *true* that surgical treatment of patients with disc herniation and radiculopathy may speed up recovery, although it may not lead to better long-term outcomes.

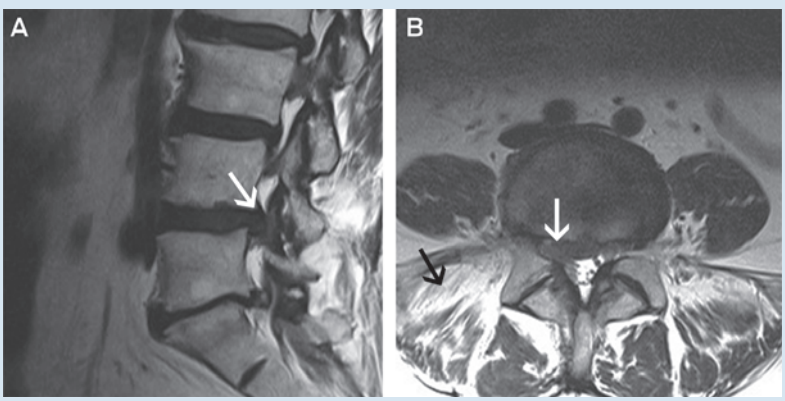
FICTION: It is *not true* that urgent neuroimaging is warranted in all patients with neurological signs. ◆

6 Treatment options for radiculopathy

- Advice (nature of condition, simple safe treatments for pain, graded activity resumption, avoidance of bed rest)
- Graded analgesia (paracetamol, non-steroidal anti-inflammatory drug, opioid, tricyclic antidepressant) (Grade B evidence)[14]
- Transforaminal corticosteroid injection (Grade B evidence)[12,15]
- Surgery if symptoms do not resolve (Grade A evidence)[12] ◆

Treatment options (Box 6) were discussed with Hilary and she was encouraged to remain active. A stepwise approach to analgesia was suggested (paracetamol, then NSAIDs, then codeine plus paracetamol, and nocturnal amitriptyline). After 5 weeks, a reasonable balance of analgesia and side effects was obtained with moderate doses of oxycodone (5–10 mg four times a day). A transforaminal steroid injection is planned, with subsequent elective micro discectomy if the steroid injection is not effective.

5 Magnetic resonance imaging scans of Hilary's lumbar spine



A: *T2 sagittal image showing a right posterolateral L4/L5 disc herniation, which compresses and angulates the L5 nerve root (arrow). Multiple other disc herniations are visible, most notably at L5/S1.* ***B:*** *T2 axial image showing loss of normal high signal around the right L5 nerve root adjacent to the herniation (white arrow) and probable paraspinal denervation atrophy (fatty replacement) (black arrow).* ◆

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Provenance: Commissioned; externally peer reviewed.

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