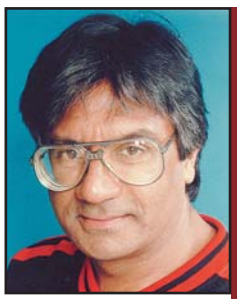
Lumbar radicular pain

BACKGROUND Radicular pain is caused by irritation of the sensory root or dorsal root ganglion of a spinal nerve. The irritation causes ectopic nerve impulses perceived as pain in the distribution of the axon.
The pathophysiology is more than just mass effect: it is a combination of compression sensitising the nerve root to mechanical stimulation, stretching, and a chemically mediated noncellular inflammatory reaction.

OBJECTIVE This article discusses the clinical features, assessment and management of lumbar radicular pain (LRP).

DISCUSSION Lumbar radicular pain is sharp, shooting or lancinating, and is typically felt as a narrow band of pain down the length of the leg, both superficially and deep. It may be associated with radiculopathy (objective sensory and/or motor dysfunction as a result of conduction block) and may coexist with spinal or somatic referred pain. In more than 50% of cases, LRP settles with simple analgesics. Significant and lasting pain relief can be achieved with transforaminal epidural steroid injection. Surgery is indicated for those patients with progressive neurological deficits or severe LRP refractory to conservative measures.



Jay Govind,
MBChB, DPH (OH), MMed, FFOM (RACP), is VMO, Royal Newcastle Hospital, and research officer, Department of Clinical Research, Bone and Joint Institute, Royal Newcastle Hospital, University of Newcastle, New South Wales.

Although commonly referred to as 'sciatica', the term lumbar radicular pain (LRP) is anatomically more correct. Lumbar radicular pain is a form of neuralgia due to an irritation of the sensory root or the dorsal root ganglion (DRG) of a spinal nerve. In contrast, sciatic neuralgia specifically refers to pain in the distribution of the sciatic nerve due to pathology of the nerve itself.[1]

By definition, radicular pain involves a region beyond the spine. In individuals presenting both with spinal pain and LRP, it is paramount that the characteristics and distribution of each pain should be defined and diagnosed separately, as it is likely they arise from different anatomical structures and are caused by different pathomechanisms. In LRP, ectopic impulses generated in the DRG are perceived as pain arising in the territory innervated by the affected axon. Somatic pain (nociception) is evoked by noxious stimulation of nerve endings; somatic referred pain is a function of interneuronal convergence within the spinal cord. Neuropathic pain is evoked by ectopic impulses generated in the axons of a peripheral nerve.[1]

Radicular pain should not be confused with radiculopathy. Radiculopathy is objective loss of sensory and/or motor function as a result of conduction block; the features of which might include numbness, motor loss, wasting, weakness, and loss of reflexes. Each can occur simultaneously or independent of each other.[2] Any lesion that affects the integrity of the lumbosacral nerve root can cause LRP, radiculopathy or both (*Table 1*).

Pathophysiology

Lumbar radicular pain is caused by more than a simple mass effect. With the advent of computerised tomography (CT) and magnetic resonance imaging (MRI), studies have confirmed that patients whose symptoms of sciatica had resolved still showed the same mass effect.[3,4] Additionally, disc herniations or protrusions evident on CT or MRI may not be associated with either low back pain or LRP.[5]

Ectopic impulses, and hence perception of pain,

Table 1. Causes of radicular pain[2]

- Disc herniation (commonest cause)
- Spinal stenosis
- Synovial cysts
- Infection
- Infestation
- Tumour
- Vascular abnormalities

Table 2. Distinguishing features of LRP and somatic referred pain[2]

Feature	Radicular pain	Somatic referred pain
Distribution	Entire length of lower limb BUT below knee > above knee	Anywhere in lower limb BUT proximal > distal
Pattern	Narrow band Travelling Quasi segmental but not dermatomal Not distinguishable by segment	Wide area Relatively fixed in location Quasi segmental but not dermatomal Not distinguishable by segment Boundaries difficult to define
Quality	Shooting, lancinating, like an electric shock	Dull, aching, like an expanding pressure
Depth	Deep as well as superficial	Deep only, lacks any cutaneous quality

Table 3. Conditions mimicking radicular pain[2]

- Spinal cord tumours
- Diabetic neuropathy
- The prodromal phase of herpes zoster
- Tabes dorsalis
- Direct contusion of the sciatic nerve
- Polyarthritis nodosa
- Gluteal injections
- Prolonged sitting
- Penetrating wounds
- Methyl methacrylate neuropathy following hip replacement

may be generated as a result of:

- mechanical deformation of DRG
- mechanical stimulation of previously damaged nerve roots
- inflammation of DRG, and/or
- possible ischaemic damage to DRG.

There are two distinctive, but not mutually exclusive pathomechanisms for LRP.

Nerve roots subjected to sustained compression for protracted periods may become sensitised to mechanical stimulation. The resulting patho-anatomical changes including focal demyelination, intraneural oedema, impaired microcirculation, Wallerian degeneration, partial axonal damage with or without neuroma in continuity, have the potential to generate ectopic impulses from the affected nerve.[2]

Studies have also confirmed that while pain may correlate with the size of disc herniation, and limitation of straight leg raising may correlate with pain, straight leg raising does not correlate with the size of disc herniation.[6] This implies that tension in a sensitised nerve root is most likely cardinal mechanism of pain rather than the effects of simple compression.

A complementary explanation implicates a chemically mediated noncellular inflammatory reaction – 'chemical radiculitis' – implying irritation of the nerve root by perineural spread of nucleus pulposus, which might occur through a disc rupture. Nucleus pulposus is inflammatogenic and leukotactic.[7,8]

Clinical features

Distinguishing between radicular pain and somatic pain (local or referred) is essential to the diagnosis. Radicular pain is often perceived in the territory innervated by the affected nerve root, eg. in the lower extremity when L4/5/S1 nerve roots are involved, and in the anterior thigh in the case of L2/3. Implicitly, pain does not follow the corresponding dermatomes and it is the sensory loss that indicates the affected segment. Lumbar radicular pain travels through the lower limb along a narrow band usually not more than 5–8 cm wide, and when experimentally reproduced, the perceived pain is qualitatively sharp, shooting or lancinating.[9] It can be experienced superficially and deeply.

In contrast, somatic referred pain is often felt deeply as a dull aching pain. Radicular pain and somatic referred pain are not mutually exclusive. They can co-exist. Radicular pain may be superimposed on a background of somatic referred pain. Hence, careful scrutiny is essential to distinguish whether the patient is describing somatic referred pain, radicular pain or a combination of both.

Although not absolute, certain features may assist in distinguishing between somatic pain and radicular pain (*Table 2*). Conditions that can mimic radicular pain

without affecting the nerve roots are shown in *Table 3*.

Clinical examination

Clinical examination does not diagnose the cause of LRP, but can establish the presence or absence of radiculopathy.

Although lumbar disc herniation is the commonest cause of LRP, there are no distinctive features either in the history or physical examination that would implicate the intervertebral disc as the cause of pain. Definitive diagnosis can only be made by imaging studies. The straight leg raise clinical test has the best sensitivity, but a low specificity with an average likelihood ratio of 1.5. Complementary testing procedures including dorsiflexion of the foot, impaired ankle reflex, sensory deficit and muscle atrophy, have modest to poor sensitivities and specificities. In younger and middle aged patients, the pretest probability of disc herniation is high, whereas in the elderly, foraminal stenosis or spinal stenosis is more likely.

The natural history

On average, patients with LRP can expect a dramatic reduction in the severity of pain with treatment limited to simple analgesics. At 12 months, at least 50% of patients can expect to be free of leg pain, but at least 60–70% will continue to experience low back pain.[10]

Investigations

Imaging

Given the favourable natural history, authorities recommend that in the absence of other indications, imaging is not required for 4–6 weeks after the onset of LRP.[11] Imaging is best reserved for patients who do not respond to conservative treatment, and for whom surgery is contemplated. In patients with a history of sciatica or in whom a 'red flag' condition seems likely, appropriate imaging should be requested. Generally, MRI is the investigation of choice. Not only does it provide a comprehensive survey of all possible causes, MRI is radiation free.

Electrophysiological studies

In the absence of clinically proven peripheral neuropathy, electrophysiological studies in patients presenting with acute LRP are generally not indicated. These tests cannot accurately determine the precise spinal nerve level associated with disc herniations and radicular pain cannot be explained by neurophysiological testing. Furthermore, electromyogram studies correlate poorly with the anatomical level of a disc herniation. Studies may be indicated to exclude a more distal nerve damage, verify suspected muscle weakness by needle electromyogram, and to assess pre-operative baseline muscle status in cases of recurrent disc operation.[11]

Treatment

Generally bed rest is no more effective than watchful waiting. Depending on the severity and response to medication, the early resumption of daily activities should be encouraged.

Clinical studies have shown that neither piroxicam,[10] indomethacin,[12] nor oral dexamethasone,[13] offer greater analgesia than placebo. For severe pain, opioids can be used judiciously.

The efficacy of physical modalities including manipulation and traction remains controversial,[11] and there is no compelling evidence to encourage their utilisation.

Injection techniques such as botulinum toxin, prolotherapy, or facet joint injections are irrational and illogical.

Epidural steroid injection

Epidural steroids are not indicated for the treatment of low back pain. The efficacy of steroid injection for the treatment of LRP may be due to its anti-inflammatory effect on inflamed nerve roots, inherent local anaesthetic properties,[14] or as a membrane stabiliser suppressing ectopic impulses.[15] Steroids injected transforaminally may offer substantial pain relief for a protracted period.[6,17] The results are less impressive if given by the caudal or interlaminar route.

Chemonucleolysis

In carefully selected patients, chemonucleolysis has been demonstrated to be effective with chronic symptoms, but residual back and leg pain are troublesome features. At least 20% of patients will proceed to surgery.[18]

Surgery

For the management of LRP alone, surgery may be considered where:

- severe unrelenting and disabling pain remains refractory to conservative measures after at least 6 weeks, but not more than 3–4 months
- neurological signs are present in the radicular distribution, and
- leg pain follows a radicular distribution and is the dominant complaint when compared with back pain.[11]

Conclusion

Back pain is not synonymous with LRP. These are two separate entities. In more than 50% of patients, LRP settles with simple analgesics and patients are encouraged to resume daily activities. Where feasible and practical, a transforaminal epidural injection of long acting local anaesthetic and steroid should be considered – given the potential for significant and lasting relief of pain. Regular review assists in identifying any progressive neurological deficit. In such instances, early referral for surgical review is necessary.

Summary of important points

- LRP is caused by irritation of the sensory root or DRG.
- Disc prolapse is the commonest cause of LRP, but foraminal or spinal stenosis is more likely in the elderly.
- LRP needs to be distinguished from spinal pain and somatic referred pain.
- Clinical examination will not identify the cause of LRP but can establish the presence or absence of radiculopathy (sensory or motor deficit).
- Investigation is only indicated for those who do not respond to conservative measures, as a preparation for surgery, or to exclude 'red flag' conditions.
- If required, MRI is the investigation of choice.
- Steroids injected transforaminally can give significant and lasting pain relief.
- Surgery is indicated for severe leg pain associated with neurological deficit not responding to conservative measures.

Conflict of interest: none declared.

References

1. Merskey H, Bogduk N, eds. Classifications of chronic pain. Description of chronic pain syndromes and definitions of pain terms. 2nd edn. Seattle: IASP Press, 1994.
2. Bogduk N, Govind J. Medical management of acute lumbar radicular pain. An evidence based approach. Newcastle Bone and Joint Institute, University of Newcastle, 1999.
3. Maigne JY, Rime B, Delinge B. Computed tomographic follow up study of forty eight cases of nonoperatively treated lumbar intervertebral disc herniation. Spine 1992;17:1071–1074.
4. Delauce-Cavallier MC, Budet C, Lardeo JD, Debie B, Wybier M, Dorfmann H, Ballner I. Lumbar disc herniation: computed tomography scan changes after conservative treatment of nerve root compression. Spine 1992;17:927–933.
5. Jensen MC, Bran-Zawadzki MN, Obucjowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994;331:69–73.
6. Thelander U, Fagerlund M, Friberg S, Larsson S. Straight leg raising test versus radiological size, shape and position of lumbar disc hernias. Spine 1992;17:395–399.
7. McCarron RF, Wimpee MW, Hudkins PG, Laros GS. The inflammatory effect of nucleus pulposus: a possible element in the pathogenesis of low back pain. Spine 1987;12:760–764.
8. Olmarker K, Blomquist J, Stromberg J, Nanmark U, Thomsen P, Rydevik B. Inflammatogenic properties of nucleus pulposus. Spine 1995;20:665–669.
9. Smyth MJ, Wright V. Sciatica and the intervertebral disc. An experimental study. J Bone Joint Surg 1959;40A:1401–1418.
10. Weber H, Holme I, Amlie E. The natural course of acute sciatica with nerve root symptoms in a double blind placebo controlled trial evaluating the effect of piroxicam. Spine 1993;18:1433–1438.
11. Anderson GBJ, Brown MD, Dvorak J, et al. Consensus summary on the diagnosis and treatment of lumbar disc herniation. Spine 1996;21(Suppl 24S):75S–78S.
12. Goldie I. A clinical trial with indomethacin (indomee) in low back pain and sciatica. Rheumatol Rehab 1975;14:222–225.
13. Haimovic IC, Beresford HR. Dexamethazone is not superior to placebo for treating lumbosacral radicular pain. Neurology 1986;36:1593–1594.
14. Johanson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive fibres. Acta Anaesthesiol Scand 1990;34:335–338.
15. Devor M, Govrin-Lippmann R, Raper P. Corticosteroids suppress ectopic neural discharge originating in experimental neuromas. Pain 1985;22:127–137.
16. Weiner BK, Fraser RD. Foraminal injection for lateral lumbar disc herniation. J Bone Joint Surg 1997;79B:804–807.
17. Lutz E, Vad VB, Wisneski RJ. Fluoroscopic transforaminal lumbar epidural steroids: an outcome study. Arch Phys Rehabil 1998;79:1362–1366.
18. Gogan WJ, Fraser RD. Chymopapain: a 10 year double blind study. Spine 1992;17:338–394.

Email: jaygovind@bigpond.com.au