Lumbar disc herniation:
Evidence-based guidelines–a review

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More than 100 years have passed since the pioneering work of the German surgeon Fedor Krause, who in 1908 performed the first successful lumbar disc herniation surgery in cooperation with the neurologist Hermann Oppenheim.[1,2] After the Second World War researchers and physicians have produced a huge body of evidence on lumbar disc herniation, the use of which can improve its diagnosis and treatment.

In recent years numerous national and institutional guidelines for the diagnosis and treatment of low back pain and radicular pain syndromes were published by several organizations such as the:

- North American Spine Society (USA)[3]
- COST B13 Working Groups of the European Commission Research Directorate General (Europe)[4,5]
- UK Royal College of General Practitioners (UK)[6]
- National Institute for Health and Clinical Excellence (UK)[7]
- British Pain Society [8]
- Dutch College of General Practice (Netherlands)[9]
- American College of Physicians and American Pain Society (USA)[10,11]
- German Societies of Neurosurgery and Neurology (Germany)[12,13]
- Australian Acute Musculoskeletal Pain Group (Australia)[14]

Since most of the patients with low back pain due to a lumbar disc herniation are treated in a primary care setting, the majority of guidelines refer to the diagnosis and treatment of nonspecific back pain, rather than sciatica due to intervertebral disc herniation. In fact, just a few guidelines regarding the specific diagnosis and treatment of lumbar disc herniation have been established in recent years (see Table 1).

The purpose of this review of guidelines regarding the specific diagnosis and treatment of lumbar disc herniation is to support practitioners and neurologists in their clinical efforts to provide the best possible care for patients suffering from low back pain and sciatica resulting from lumbar disc herniation.

Table 1

Title of Guideline	Country	Society	Year
Low back and radicular pain: a pathway for care[8]	UK	British Pain Society	2013
Diagnosis and Treatment of Lumbar Disc Herniation with Radiculopathy[3]	USA	North American Spine Society (NASS)	2012
Lumbale Radikulopathie[13]	Germany	Deutsche Gesellschaft für Neurologie	2012
Leitlinie: Lumbaler Bandscheibenvorfall[12]	Germany	Deutsche Gesellschaft für Neurochirurgie	2005
NHG-Standaard Lumbosacraal radiculair syndroom[9]	Dutch	The Dutch College of General Practitioners	2005

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Level of Evidence (LoEv – According to the guideline of the NASS[3])

A High degree of clinical certainty regarding the recommendation or rejection of diagnostic or therapeutic procedures based on multiple Level I studies demonstrating consistent findings.

B Good to fair evidence regarding the recommendation or rejection of diagnostic or therapeutic procedures based on a single Level I study of multiple Level II and III studies showing consistent findings.

C Moderate to poor quality evidence regarding the recommendation or rejection of diagnostic or therapeutic procedures based on Level IV and Level V studies.

D Unclear clinical certainty regarding the recommendation or rejection of diagnostic or therapeutic procedures due to insufficient or conflicting evidence.

Definition

Lumbar disc herniation (LDH) is a localized displacement of intervertebral disc tissue beyond the physiological margins of the intervertebral disc space that can result in low back pain, radicular pain, motor weakness, numbness, and/or tingling in a myotomal and dermatomal distribution.[15]

Epidemiology and pathology

LDH typically occurs in patients with preexisting degenerative alterations of the intervertebral disc (IVD). Traumatic LDH is rare. The degeneration of the IVD is a common process of aging and is usually asymptomatic. However, structural damage to the IVD due to flexion/extension, lateral bending, and axial loading may lead to the annular fissuring and fragmentation of nuclear tissue, which can result in altered biomechanical properties of the IVD with the loss of its structural integrity. The IVD is no longer able to transmit the nuclear loading forces into annular tensile forces. The fragmented tissue of the *nucleus pulposus* follows the fissures of the annulus *fibrosus*, resulting in a displacement of the intervertebral disc tissue beyond the physiological margins - the lumbar disc herniation.

The prevalence of LDH is about 2% in Western industrial nations, peaking in patients between 30 and 50 years of age. Population studies indicate that males are affected twice as often as females are. Almost 95% of all LDHs occur at levels L4/5 and L5/S1 of the lower lumbar spine.[16]

Clinical presentation

Typical symptoms of a LDH are low back pain and radicular pain, as well as sensorimotor deficits. In addition, in severe cases a complete transverse section – *caudaequina* syndrome (CES) -may be observed including bladder, bowel, and/or sexual dysfunction.

Low back pain (lumbago)

LDH patients typically but not consistently complain of localized low back pain that increases under strain and axial loading. Thus prolonged sitting and upright standing are typically more debilitating than remaining in a recumbent position. Medially herniated discs frequently result in predominant lumbago without any radicular pain. Lateral disc herniation, however, can lead to radicular pain without the low back pain.

Radicular pain

The typical presentation of radicular pain is radiation of (low back) pain along the dermatome of a peripheral nerve. The most common form of radicular pain is sciatica, which refers to a pain that radiates from the lower spine along the gluteal region and the back of the upper thigh and calf, and into the foot. Coughing and sneezing typically lead to an increased pain sensation. Lasegue's sign is positive in about 95% of all patients suffering from LDH.[17,18]

Neurological deficits

With a progressive severity of nerve root compression, impaired reflexes, sensory disturbances (hypesthesia, hyperalgesia), and motor weakness can occur. Sensory abnormalities and motor deficits are found in more than half of all LDH patients. The locations of the sensory or motor loss of these neurological deficits are not sensitive enough to determine the exact level of the LDH.[19]

Cauda equina syndrome (CES)

The CES is a term identical with the *conus medullaris* syndrome. It refers to a functional loss of fibres of the cauda medullaris usually involving saddle anesthesia, motor weakness, urinary and bowel incontinence. A transverse section of all fibres of the cauda equina results in complete paraparalysis of the legs and loss of sensory and bladder and bowel function.

Diagnosis of lumbar disc herniation

The medical history and physical examination of the patient, including muscle testing, sensory testing, supine straight leg raise, and Lasegue's sign, are crucial for the diagnosis of an LDH with radiculopathy.[20,21](LoEv: A)

Regarding additional clinical tests, such as the cough impulse test, hyperextension, femoral nerve stretch, slump test, lumbar range of motion, or absence of reflexes, insufficient evidence exists.[22,23](LoEv: D)

Table 2 shows typical clinical presentations of LDH dependent on the level of the LDH and compressed nerve root.

Spinal imaging

When history and physical examinations are consistent with a LDH with radiculopathy spinal imaging is justified to confirm or exclude the suspected diagnosis.

Magnetic resonance imaging (MRI)

The MRI of the lumbar spine including the medullary cone and the lumbosacral junction currently provides the

Table 2- Clinical presentations of lumbar disc herniation[24]

	Level of lumbar disc herniation		
	L3/4	**L4/5**	**L5/S1**
Nerve root (usually)	L4	L5	S1
Rate of LDH	5%	40-45%	45-50%
Reflex diminished	Knee jerk (patellar reflex)	Medial hamstring	Ankle jerk (Achilles reflex)
Motor weakness	Knee extension (M. quadriceps femoris)	Foot dorsiflexion resulting in foot drop (M. tibialis anterior)	Plantarflexion (M. gastrocnemius)
Sensory disturbances	Medial malleolus and medial foot	Large toe and dorsum of foot	Lateral malleolus and lateral foot
Pain distribution	Anterior thigh	Dorsolateral thigh and calf	Posterior thigh and calf

highest sensitivity and specificity in the diagnosis of LDH and is therefore the imaging study of choice.[25-28] MRI is non-invasive and does not use ionizing radiation. The imaging protocol should comprise sequences with different weightings (T_1, T_2, proton density, and MRI myelography) and orientations (at least axial and sagittal). When the recurrence of an LDH is suspected, acquiring additional T1-weighted images with the administration of contrast agents is recommended.[29](LoEv:A)



Figure 1 – Spinal imaging of lumbar disc herniation using MRI

Computed tomography (CT), myelography, and post-myelographic CT

Spinal MRI is superior to all other imaging techniques. When MRI is contraindicated (e.g., patients with electronic devices like a pacemaker, metallic foreign bodies, or older aneurysm clips) or inconclusive (e.g., in which there are artifacts due to pedicle screws, anterior inter body cages, or artificial disc replacement), CT scan, myelography, and post-myelographic CT are appropriate imaging techniques to identify a LDH.[27,30] A CT scan may even provide additional information about adjacent bony structures. CT and myelography should be avoided if possible because of the exposure to radiation. (LoEv: A)

X-Ray and discography

In the era of cross-sectional imaging modalities such as the MRI and the CT scan, native x-rays and discography have lost their relevance in the diagnosis of LDH and are reserved for special issues only (e.g., spondylolysis and spondylolisthesis).

Electrophysiological test

Somatosensory-evoked potentials (SEP), electromyelography, nerve conducting studies, and motor-evoked potentials (MEP), may have limited value for the diagnosis of an LDH. These tests are of high sensitivity but low specificity and therefore less useful.[31-33] Thus, electrophysiological testing is helpful in differentiating LDH from comorbid conditions and differential diagnoses (e.g., myelopathia or polyneuropathy). (LoEv: B)

Differential diagnoses

- Acute aortic dissection
- Aortoiliac occlusive disease
- Coxarthrosis
- Epidural and subdural infections
- Herpes zoster
- Mechanical back pain
- Metabolic neuropathy
- Pelvic fracture
- Rheumatoid arthritis
- Spinal bleeding

- Spinal cord injuries, infections, and neoplasms
- Spinal fractures (traumatic, osteoporotic)
- Spinal stenosis (including myelopathy)
- Spondylodiscitis
- Spondylolisthesis
- Syringomyelia

Treatment of lumbar disc herniation

Complete spontaneous resolution of LDH is frequently seen. Usually, improvement occurs within several weeks with limited or no medical treatment at all. Only about 5% of patients complain of insufficient pain relief after 6-12 weeks.[16] Spinal MRI studies have shown that LDH has a high potential for resolving completely or shrinking in size over time.[34]

Primary operative treatment is indicated in cases of acute or progressive motor deficits or loss of bladder or bowel dysfunction (emergency surgery). Urgent surgery may be justified for patients whose pain remains intolerable despite sufficient analgesic therapy. A paresis of unknown duration does not justify a primary surgical intervention, since no study has shown an improvement of the paresis after surgery in these cases.[35] (LoEv: C)

Nonsurgical treatment should be considered for all patients who demonstrate no neurological deficits.

Nonsurgical treatment

More than 90% of all lumbar disc herniation can be controlled within 6-8 weeks without surgical intervention. If the patient's pain remains intolerable, a surgical intervention should be considered in order to avoid pain chronification as well as side effects of administered drugs and painkillers. Because of the numerous conservative approaches currently available, no treatment of choice has been decided on. Nevertheless, pain relief can be achieved by the following means:

Modification of activity

Long-term immobilization or abstention from any physical activity is not uniformly recommended, since bed rest lasting longer than two to four days seems to produce weakness, stiffness, and increased pain in patients with LDH. Rather, a gradual return to normal activities should be pursued. The aim is to continue normal daily activities at a tolerable level of pain or discomfort, without increasing strain.[36-38] (LoEv: A)

Physical exercise and spinal manipulation

It is generally thought that supervised moderate aerobic exercise (through physical therapy) during the first month of symptoms may be beneficial and at the same time minimize debility that can occur due to inactivity. Low-stress exercises such as swimming, walking, and bicycling are also recommended. However, due to the lack of high-quality studies, no reliable evidence-based recommendations can be made.[39] (LoEv: C)

Some studies have revealed that spinal manipulation by professional physiotherapists may be effective for pain relief in patients with back pain and radiculopathy and therefore a therapeutic option in the treatment of LDH.[40,41] (LoEv: B)

Analgesics

Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen (Parcetamol) are recommended during the first short-term period of pain. NSAIDs do not, however, appear to have better pain relief than acetaminophen.[42,43] Stronger analgesics such as opioids are reserved for patients with severe pain and should not be prescribed for longer than a few weeks.[44] Unfortunately, most studies refer only to patients with low back pain. With respect to sciatica, no current conclusive evidence has been presented. (LoEv: B)

Steroids

In daily clinical practice, intravenous administration of glucocorticoids in patients with sciatica is very common. Nevertheless, their transient benefit of up to only three days' relief of pain, and the absence of any functional recovery in patients with LDH, argue against such wide clinical use.[45](LoEv: B)

The role of epidural steroid injection (ESI) in LDH treatment is very controversial. While some studies have shown beneficial effects of ESI in patients with LDH,[46,47] several other well-regarded studies have produced contrary results.[48-50] Specifically, ESI does not reduce the need for surgery. The response to ESI of patients in acute pain seems to be better than the response of patients in chronic pain. Additionally, it has been reported that epidural injections have often been accompanied by severe complications, including spinal infection, nerve damage, and hemorrhaging. Patients have in fact died from such complications. Thus ESI should only be an option for short-term pain relief, or for patients whose comorbidities do not allow for them to undergo surgery. (LoEv: D)

Surgical treatment

Since more than 85% of patients with acute sciatica will improve within six weeks without surgical treatment, most neurosurgeons advocate elective surgery for patients suffering LDH without neurological deficits only if non-surgical treatment has not led to sustained pain relief after a period of five to eight weeks and if the symptoms of the LDH are severe enough to warrant surgical intervention.[51,52] In these cases, it has been shown that surgery provides substantial benefit, and that, compared to non-surgical treatment, it results in earlier and greater short- and medium-term improvement of symptoms.[53-55] Long-term observations, however, have shown comparable results in both groups.[56-58] (LoEv: B)

The most common surgical procedure is a microdiscectomy by a posterior approach to the spine. The use of an intraoperative microscope provides similar results to the performance of open discectomy, but because of its better illumination and less traumatic procedure, its use seems to be more favored.[59,60] (LoEv: A)

In terms of reherniation rates, no differences have been observed between aggressive discectomy and sequestrectomy. The less traumatic sequestrectomy, however, has exhibited better long-term results, particularly in younger patients.[61,62] (LoEv: B)

Several other options for surgical therapy of an LDH, such as chemonucleolysis, automated percutaneous lumbar discectomy, endoscopic, or laser disc decompression, are available. Whether any of these procedures are superior to microdiscectomy has not yet been demonstrated (LoEv: D).

Postoperative care

There is no evidence for the efficacy of prolonged postoperative rest. Instead, a more active postoperative regimen under the supervision of professional physiotherapists lead to less short-term pain and disability as compared to no treatment.[63] For the first few days, however, patients should be instructed to avoid bending at the waist, lifting, twisting, and prolonged sitting and standing. Patients are generally not required or even advised to wear a back brace after microdiscectomy.[64-66] (LoEv: A).

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