Degenerative Disc Disease

14

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Key Points

- Degenerative disc disease represents a broad category of back pain resulting from the degeneration of intervertebral discs.
- Disc degeneration may result from a variety of causes, including age, trauma, or excessive or repeated stress.
- In addition to damage to intervertebral discs, surrounding axial structures may also be affected and serve as pain generators. These nearby structures may include the vertebral bodies, facet joints, spinal ligaments, and exiting nerve roots.
- First-line treatment for degenerative disc disease consists of physical therapy, which works to reduce stress on the degenerated discs. When analgesic medications are needed, start with nonsteroidal anti-inflammatory oral medications.
- Epidural steroid injections may relieve pain and enhance rehabilitation from degenerative disc disease by facilitating improved participation in physical therapy and core-strengthening exercises.
- If function remains impaired despite conservative therapy, surgical options may be considered, though disc herniation and spinal stenosis are the two patient groups with the best evidence for improvement following spine surgery.

Introduction

Degenerative disc disease is a pathologic process that can result in acute or chronic low back pain from the loss of structure or integrity of intervertebral discs. When discs become dehydrated, they can narrow in height and collapse, resulting in aberrant changes in anatomical alignment that may result in nerve compression and pain symptoms. Radiographic findings of degenerative disc disease include disc space narrowing, osteophyte formation, degeneration of vertebral bodies, end plate changes, and vacuum disc [1–3]. However, advanced imaging demonstrating degenerative pathology has a poor predictive value in identifying pain in older adults between the ages of 53 and 70 [3]. Many patients in this age group have degenerative changes on imaging studies yet do not report significant low back pain. Degenerative disc disease may result in pain from multiple specific pathologies including discogenic pain, facet arthropathy, vertebral degeneration, disc herniation, and spinal stenosis.

Epidemiology and Natural Course

Low back pain as a result of degenerative disc disease has a significant socioeconomic impact in the United States. Factoring in lost wages and medical treatment, the financial cost of low back pain in the United States is estimated to exceed $100 billion annually [4]. The economic impacts stem from decreased quality of life from pain and associated neurological deficits. However, most degenerative disc disease is asymptomatic, making a true understanding of its prevalence difficult to discern. While exact estimates may vary widely, degenerative discs remain increasingly prevalent as patients age, with rates of 71% in men and 77% in women aged <50 years increasing to >90% in both men and women aged >50 years [5]. However, the incidence of low back pain in the entire cohort was 43%, suggesting that imaging findings do not always correlate

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with clinical symptoms. Thus, a practitioner must consider patients' presenting symptoms in conjunction with imaging findings when investigating the cause of low back pain.

Relevant Anatomy

Intervertebral discs are located between each vertebral body of the spine. The fibrocartilage-based pad of these discs provides flexibility, support, and minor load-sharing for the spine. Often considered as "shock absorbers" for the spine, discs balance the weight-bearing function of the spine with mobility needed to permit movement including rotation and bending [6]. They are primarily composed of two layers: an inner, soft, pulpy nucleus pulposus and an outer firm structure known as the annulus fibrosus. The annulus fibrosus is made up of fibrocartilage from type I and II collagen. Type I collagen provides greater strength and support to the disc and can withstand significant compressive force. The annulus fibrosus helps to distribute stress and pressure evenly across the disc and prevents damage to the underlying vertebrae. It also encircles the nucleus pulposus. The nucleus pulposus is made of a mucoprotein gel containing loose fibers and keeps the vertebrae separated in addition to absorbing impact from the body's movements. Disruptions in the normal architecture of the intervertebral discs, such as from herniation or degeneration, may result in significant pain and morbidity [6].

Mechanisms

In degenerative disc disease, pathologic changes occur in the intervertebral discs and, very frequently, in the vertebral bodies as well. Within the discs, the nucleus pulposus may suffer a reduced concentration of proteoglycans in its matrix, resulting in disc dehydration, loss of disc height, and narrowing of the intervertebral disc space. This usually occurs as a result of abnormal, repetitive microtrauma to the disc or from normal aging. The loss of disc height is also responsible for the normal age-related decline in height in the elderly [7]. In addition, with age, the annulus fibrosus becomes weaker and has a higher risk of tearing. If the fibrosus tears, the nucleus pulposus can extrude out of the disc, introducing a cascade of inflammatory cytokines and other chemicals that cause low back pain with or without radiating symptoms. The resultant disc herniation can additionally impinge on exiting nerve roots on the left or right side, leading to unilateral radiculopathy. Central canal narrowing may also result, leading to bilateral radiculopathy.

When disc height is lost, there is additional stress on the vertebral bodies themselves. This can result in degradation of vertebral end plates with compensatory sclerotic changes of the subchondral bone of the end plate [8]. These sclerotic changes can lead to the formation of osteophytes, which can protrude out from the vertebral bodies and impinge on exiting nerve roots. This impingement applies pressure to the spinal cord or nerve roots, resulting in radiating pain and sometimes focal areas of weakness. The specific symptoms reported by the patient will depend on which nerve roots are impinged upon by the protruding osteophytes and can vary greatly from patient to patient.

Recent studies have revealed that a number of genetic alterations are linked with structural changes in the intervertebral discs, predisposing patients to degenerative disc disease [9]. Mice studies with specific knockouts for genes suspected to play a role in disc degeneration, along with twin studies, reinforce this association between genetic factors and degenerative disc disease. Specific genes that may be involved in degenerative disc disease include those that code for scaffolding responsible for the integrity of the discs, such as collagens I, IX, and XI, as well as other genes for interleukin 1 (IL-1), the vitamin D receptor, aggrecan, and matrix metalloproteinase 3 (MMP-3) [9]. Patients with defects in any of these genes may have an increased risk for developing degenerative disc disease. Although each individual gene likely plays a small role, the complex interaction among these genes and the environment leads to the development of degenerative disc disease. Repetitive micro-stress from various physical activities and the aging process itself are likely the primary environmental contributors to degenerative disc disease.

Pathophysiology

In addition to intervertebral disc dehydration predisposing to disc tears, osteophyte formation, and nerve compression, the discs themselves can also be a source of pain. When discs serve as the primary pain generator, this clinical entity is termed discogenic pain [8]. Nociceptors in the annulus fibrosis can be stimulated by disc degeneration and cause nociceptive pain. Reactive nerve fibers in the outer layer of the annulus fibrosus can release inflammatory mediators including substance P, vasoactive intestinal polypeptide (VIP), calcitonin gene-related peptide, and other cytokines in response to repeated mechanical loads [8]. Degeneration of discs can also lead to abnormal mechanical stimulation of disc nociceptors. When this occurs, disc nociceptors generate an amplified response known as peripheral sensitization, leading to significant pain and morbidity. Low pH with rising lactic acid levels can also stimulate peripheral sensitization, worsening discogenic pain as well.

In addition, mast cell infiltration into torn fissures of the annulus fibrosis can lead to significant disc tissue inflam-

mation and degradation, fibrosis formation, neovascularization, and release of signaling factors, which can contribute to back pain. For example, phospholipase A2 (PLA2) has been detected at high concentrations in degenerative discs, stimulating the nociceptors of the outer third of the annulus fibrosus. This results in the further release of inflammatory materials that trigger pain [8]. In summary, discogenic pain originates from a structural failure of the disc that, through a variety of methods, leads to nociceptive stimulation and pathologic neurovascular proliferation into the annulus fibrosus.

Clinical Findings

Signs and symptoms of degenerative disc disease can vary significantly depending on the specific pathology occurring in the presenting patient, such as discogenic dysfunction, spinal stenosis, or facet arthropathy. Symptoms can include low back pain with or without radiculopathy, lower extremity weakness, and paraspinal tenderness.

Discogenic pain is a result of disc degeneration and is usually worsened with spine flexion owing to additional compressive stress on the discs. The patient may report pain with the act of sitting down or while walking uphill, as both of these activities typically result in some degree of spinal flexion [6]. In addition, any increase in intra-abdominal pressure from coughing, sneezing, or other Valsalva-like maneuvers can worsen pain due to this pressure being transmitted to the disc. Discogenic pain can often be difficult to differentiate from other sources of low back pain as it can present in a variety of ways.

If the degenerative disc disease results in spinal canal stenosis, the patient may report low back pain that is worse with spinal extension, with or without radiculopathy [7]. If radiculopathy is present, it is usually bilateral, since circumferential narrowing of the spinal canal will often affect the exiting nerve roots of both sides. Pain may radiate to the buttocks, hips, knees, or down to the feet. Pain is usually worse when standing from a sitting position or walking downhill, since both activities usually require significant spinal extension. Pain is often relieved when moving from a standing to sitting position due to lumbar flexion. If cord compression is significant enough, the patient may report weakness down one or both legs. This should alert the provider that further imaging and surgical consultation may be needed.

Sclerotic changes in the vertebral end plates can result in osteophyte formation, which may cause radicular pain as well [8]. Osteophytes can compress nerve roots as they exit the neural foramina, causing radiculopathy with or without lower extremity weakness. The specific type of activities that cause pain will vary significantly among patients, as this depends on which nerves are being compressed. Patients may endorse worsened pain after sitting or standing or after walking short distances. Osteophyte protrusion leading to radicular pain may be difficult to differentiate clinically from a herniated disc, as symptoms are similar.

Disc degeneration can also result in increased stress on the facet joints, causing facet pain [10]. This pain is often non-radiating and is usually relieved with lying down. Pain may be increased by extension of the lower back but not necessarily by flexion. It can be difficult to clinically distinguish between facet arthropathy and other pathologic sequelae of degenerative disc disease.

When evaluating a patient with possible degenerative disc disease, it is important for the practitioner to rule out other important causes of back pain, including abdominal pathologies such as abdominal aortic aneurysm, renal calculi, and pancreatic pathology [6–8]. In addition, identification of "alarm symptoms" is imperative. These include night sweats, significant weight loss, loss of bowel or bladder function, lower extremity weakness, recent history of trauma or assault, and saddle anesthesia. These signs or symptoms could suggest significant pathology other than degenerative disc disease, such as cauda equina syndrome, infection, tumors, or previously unrecognized trauma. Efficient workup and potentially urgent or emergent surgical consultation are especially important in these circumstances.

Imaging Findings

Initial imaging workup for patients with suspected degenerative disc disease without alarm symptoms should include cervical, thoracic, or lumbar radiographs depending on the region of reported pain symptoms. Radiographic images should include posteroanterior (PA) and lateral films. Radiographic findings of late degenerative disc disease include loss of disc space height, osteophyte formation, bony endplate sclerosis, and vacuum phenomenon [1, 2, 11]. The graphic in Fig. 14.1 shows degeneration of L5–S1 intervertebral discs with loss of disc height and osteophyte formation, while Fig. 14.2 shows multilevel degeneration. Figure 14.3 shows a lateral radiograph with loss of disc height and posterior articulation arthrosis. Vacuum phenomenon refers to the accumulation of nitrogen within the crevices of intervertebral discs or vertebrae. Figure 14.4 shows vacuum phenomena on a sagittal non-enhanced CT scan. Although not routinely utilized in degenerative disc disease, a non-contrast CT of the back may also be useful in the assessment of bulges, focal disc herniations, osteophyte formation, facet arthropathy, and central stenosis.

If a patient's degenerative disc disease does not respond to conservative therapy (see section "Treatment"), further imaging with MRI of the affected spinal region is warranted. The MRI sequence chosen will determine which

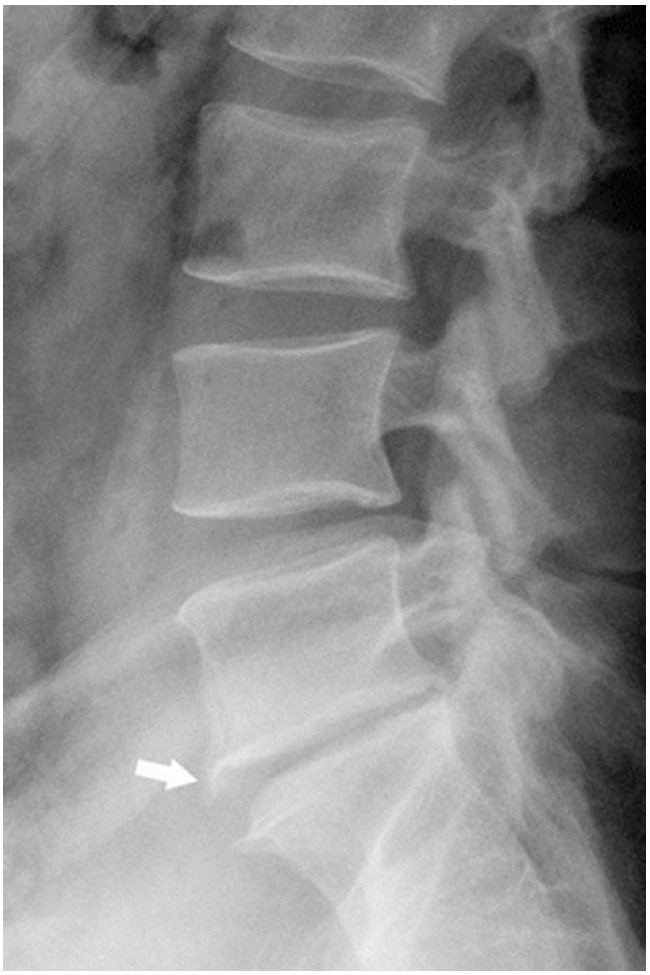

Fig. 14.1 Lateral radiograph of the lumbar spine shows reduction of disc height at L5–S1. Also pictured at L5–S1 are anterior osteophytes (arrow). (Reprinted by permission from Springer Nature: Imaging of Degenerative Disk Disease by Guillaume Bierry, Jean Louis Dietemann. Copyright 2016)

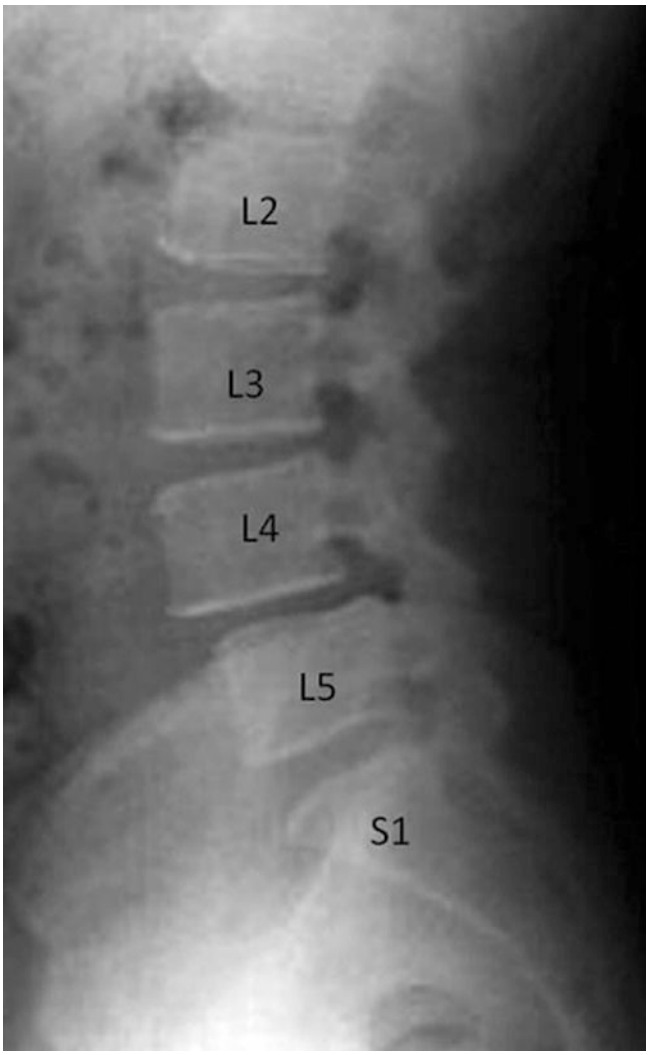

Fig. 14.2 Lateral radiograph of the lumbar spine shows reduction of disc height at multiple levels, most prominently at L4–L5. Also pictured at L4–L5 is spondylolisthesis, where the L5 vertebral body is located posteriorly to the L4 vertebral body. (Reprinted by permission from Springer Nature: Surgical Indications for Lumbar Degenerative Disease by Ravi R. Patel, Jeffrey A. Rihn, Ravi K. Ponnoppan et al. Copyright 2014)

findings of degenerative disc disease can be seen. In T1-weighted images, loss of disc space height, vacuum phenomenon (low signal within the disc), and degenerative end plate changes (graded I to III) can be identified. MRI with contrast and T1-weighted images can reveal discs with linear enhancement or enhancement within Schmorl's nodes. Schmorl's nodes are an upward or downward protrusion of a spinal disc's soft tissue into the bone of an adjacent vertebra and may also suggest the presence of degenerative disc disease on radiologic imaging [12]. In T2-weighted images, loss of signal from the nucleus pulposus can be seen, along with a loss of horizontal nuclear cleft. In addition, degenerative end plate changes can be found (graded I to III) [11, 12]. Figure 14.5 shows a sagittal MRI T2-weighted images with degenerated disc as low signal at L5–S1 compared to normal disc signal intensity at L3–L4 and L4–L5. MRI T2-weighted images may also reveal a hyperintense zone (HIZ) on the posterior annulus fibrosus of the disc, as seen in Fig. 14.6.

Although radiographs and MRI can identify disc pathology, they cannot reliably correlate pathology to clinical symptoms. Discography has historically been utilized as a means of localizing back pain to specific degenerated discs, though it has fallen out of favor due to concerns regarding the possibility of long-term damage that otherwise healthy discs incur during the procedure. This procedure involves the injection of contrast dye into the nucleus pulposus while simultaneously scanning the patient with CT (or fluoroscopic) imaging to identify extravasation of dye [13]. Positive extravasation suggests an annular tear. As the dye is injected, the patient's intra-

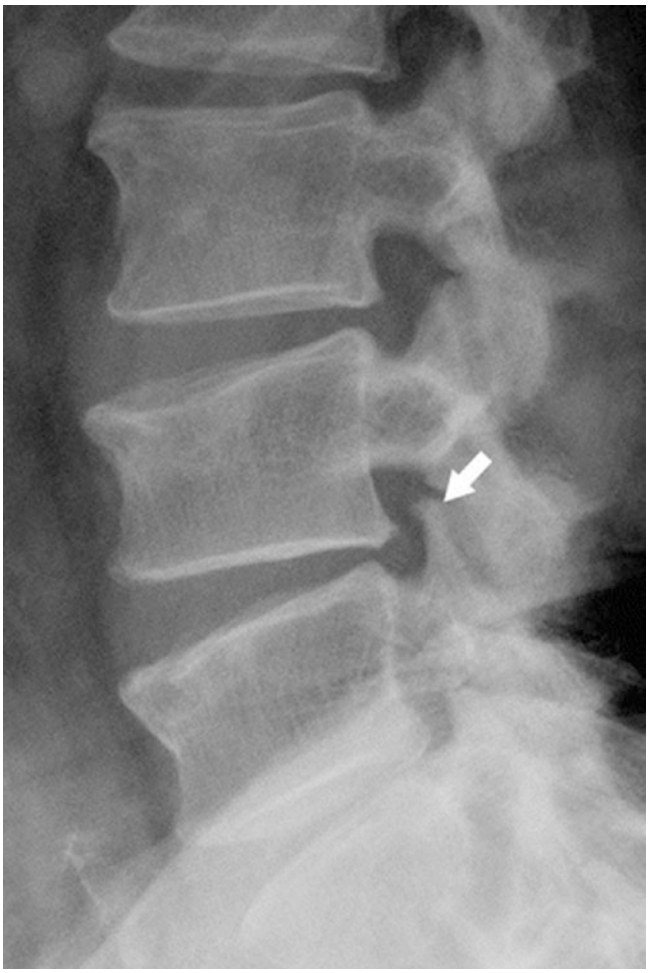

Fig. 14.3 Lateral radiograph of the lumbar spine shows narrowing of the intervertebral foramen in the setting of disc height loss and posterior articulation arthrosis (arrow). (Reprinted by permission from Springer Nature: Imaging of Degenerative Disk Disease by Guillaume Bierry, Jean Louis Dietemann. Copyright 2016)

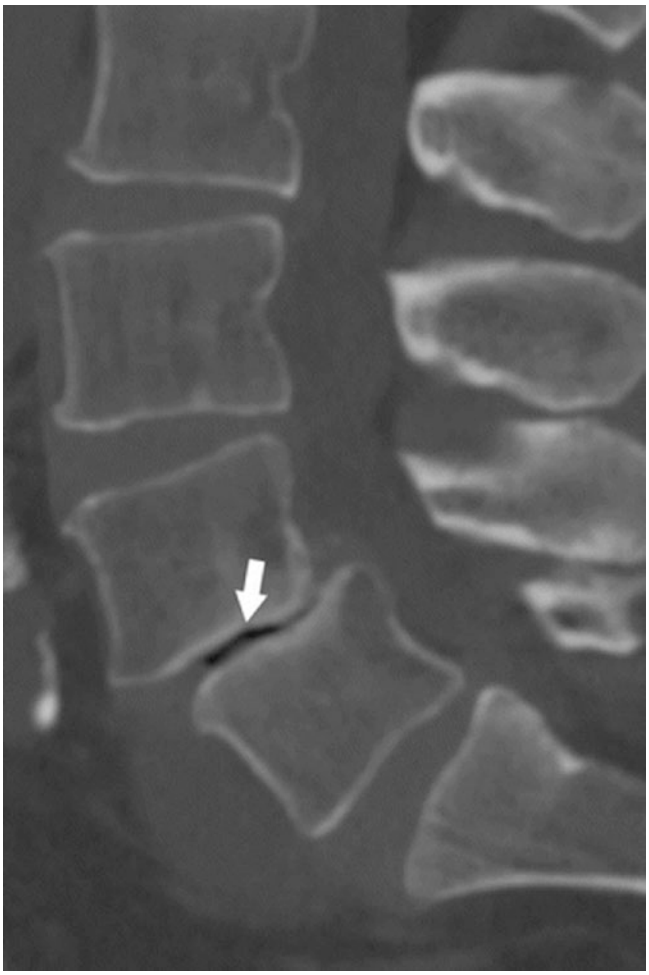

Fig. 14.4 Sagittal computed tomography (CT) of the lumbar spine showing degenerative changes with disc height loss at L4–L5. Also note intradiscal gas (arrow). (Reprinted by permission from Springer Nature: Imaging of Degenerative Disk Disease by Guillaume Bierry, Jean Louis Dietemann. Copyright 2016)

discal pressure is measured, and if pain occurs that is similar to the patient's usual back pain at a low injection pressure, the discogram is concordant [13]. If pain is produced with a high injection pressure or the pain on injection is different from the patient's usual back pain, then this is a discordant study and that particular disc is not likely the source of pain. However, discography has been found to have false-positive rates of up to 25% in asymptomatic individuals.

Treatment

Non-pharmacologic Therapy

Conservative management is the initial treatment strategy for a patient with suspected degenerative disc disease in the absence of alarm symptoms. Depending on the patient's functional status, a 6-week course of supervised physical therapy emphasizing strengthening of core muscles and stretching should be initiated [6, 7]. This would include physical therapy sessions one to two times a week, along with home exercises on all other days. Goals of therapy include improvement in core muscle strength, reduction in load-bearing on the intervertebral discs, and allowance for disc resorption/healing.

Pharmacologic Treatment

In addition, pharmacologic therapy can be initiated during the physical therapy course with nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen. One of the most common over-the-counter or prescribed NSAIDs, ibuprofen, has usual doses ranging from 400 to 800 mg every 6–8 h and a maximum total dose of 3200 mg within 24 h [6]. Caution

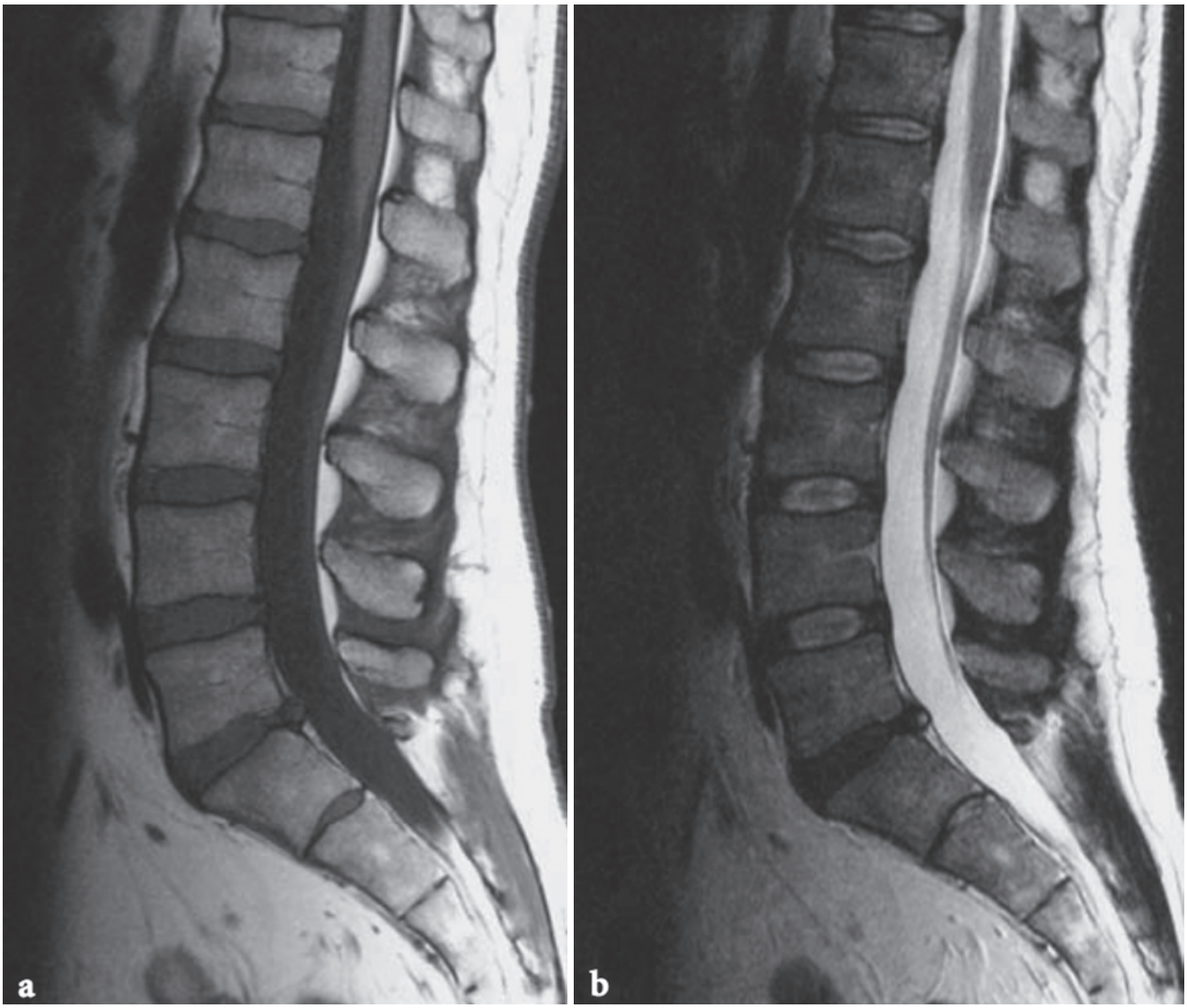

Fig. 14.5 Sagittal MRI images of lumbosacral spine of a 38-year-old man. T1-weighted images (**a**) show disc protrusion at L5–S1 disc. T2-weighted images (**b**) show decrease in signal intensity, suggestive of desiccation, and loss of disc height, which predispose to disc protrusion. Note retention of signal intensity in discs at higher levels such as L3–L4 and L4–L5 levels. (Reprinted by permission from Springer Nature: Degenerative Disc Disease by Paul M. Parizel, Johan W. M. Van Goethem, Luc Van den Hauwe . Copyright 2007)

should be taken when prescribing NSAIDs for patients with a history of renal insufficiency, GI ulcers, or bleeding disorders. Acetaminophen can also be prescribed during the conservative treatment period with a dose of 500–1000 mg every 4–6 h, with a recommended total dose of 3000 mg within 24 h [6]. Patients with mild liver disease should have their dose reduced, while those with moderate to severe liver disease should likely avoid acetaminophen.

Fortunately, many patients respond to conservative therapy, and their symptoms improve significantly. If this occurs, patients should continue home physical therapy exercises and try to reduce usage of NSAIDs and acetaminophen as tolerated, as long-term use can lead to kidney and liver dysfunction, respectively.

Injection Therapy

A conservative therapy modality that may be offered to patients with radicular pain as a result of their degenerative disc disease is an epidural steroid injection. The physician injects steroid medication directly into the epidural space to reduce inflammation of the exiting nerve roots. The steroid medication can be administered directly to the affected side with a transforaminal approach or bilaterally with an interlaminar approach [14, 15]. Literature suggests that epidural steroid injections may provide moderate, short-term pain relief from radicular pain resulting from disc herniation [14]. However, the benefit of epidural steroid injections for chronic non-radiating back pain is less clear.

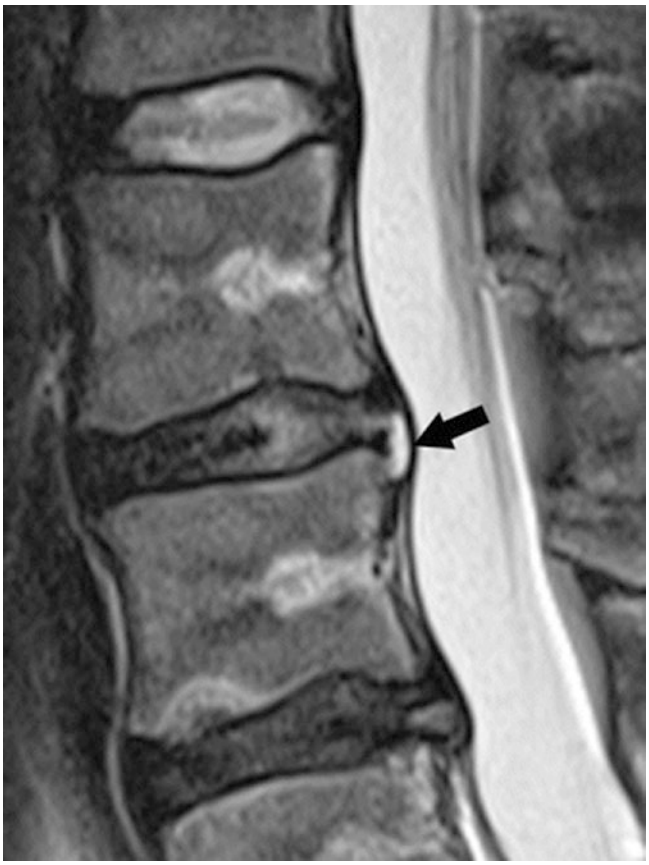

Fig. 14.6 Sagittal MRI images of lumbar spine shows disc degeneration with decrease in signal intensity at L2–L3 and L3–L4 when compared to L1–L2. Also pictured at L2–L3 is a posterior hyperintense zone (HIZ) (arrow). (Reprinted by permission from Springer Nature: Imaging of Degenerative Disk Disease by Guillaume Bierry, Jean Louis Dietemann. Copyright 2016)

Epidural steroid injections, when mixed with local anesthetic, begin to work immediately due to the local anesthetic component in the injectate, but this phase of pain relief usually wears off after a few hours. The onset of pain relief for steroid medication may take up to 2–5 days after the injection, but when effective, treatment with steroid offers sustained relief ranging from a few days to a few months [14, 15]. The goal of an epidural steroid injection is to reduce the patient's pain long enough so that the patient can fully participate in physical therapy and strengthening exercises to ultimately reduce load-bearing stress and improve pain for the long term.

Surgical Intervention

Despite an adequate trial of conservative therapy, some patients will continue to experience an unacceptable level of pain. Patients can choose to avoid activities that elicit pain or opt for surgical intervention. Patients with degenerative disc disease who may benefit from surgical intervention are those specifically with disc herniation or degenerative spinal stenosis [6, 7]. Surgery should likely be reserved for patients with severe pain limiting daily activity, neurologic deficits including lower extremity weakness, or spondylolisthesis. The Spine Patient Outcomes Research Trial (SPORT) compared conservative versus surgical management of lumbar disc herniation and spinal canal stenosis. For patients with lumbar disc herniation who underwent discectomy versus medical management, improvements after 3 months in measures of bodily pain, physical function, and Oswestry Disability Index (ODI) were greater with discectomy [16]. However, after 2 years, these differences narrowed and were not statistically significant.

For patients with lumbar spinal stenosis who underwent decompressive laminectomy, greater improvements were seen in bodily pain, physical function, and the ODI after surgery compared to medical management when assessed at 6 weeks, 3 months, 6 months, and yearly for up to 4 years [17]. However, these benefits diminished in years 4 through 8, with the surgical patients faring no better than those in the medical management group [18]. Thus, patients must be informed that surgical intervention for disc herniation or lumbar stenosis may have favorable outcomes for a number of years, but their pain will likely return. Results for surgery for non-radiating lower back pain are even less predictable.

Summary

Degenerative disc disease represents a broad category of back pain resulting from the degeneration of intervertebral discs. When discs degenerate, whether from aging or excessive or repeated stress, the surrounding axial structures can also be affected, causing pain. These may include the vertebral bodies, facet joints, spinal ligaments, and exiting nerve roots. Thus, symptoms from degenerative disk disease can mirror primary pathology from any of these other structures. It is important for the clinician to elicit a detailed history and physical exam and use appropriate imaging modalities in order to determine the cause of a patient's low back pain. Treatment for degenerative disc disease begins with conservative therapy, including physical therapy and anti-inflammatory oral medications. Epidural steroid injections can also relieve pain, with the purpose of facilitating improved participation in physical therapy and core-strengthening exercises so that stress on the degenerated discs may be reduced. If these conservative therapies fail, surgical options are available for patients with radiculopathy or lower extremity weakness, including discectomy or decompressive laminectomy. Pain relief from these surgeries may be superior compared to medical management in the short term, but the benefits are likely not sustained after

a few years. Thus, degenerative disc disease can pose significant challenges to the physician and patient when multiple strategies for treatment have been attempted.

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