Lumbar Degenerative Disc Disease and Lumbar Disc Herniation

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45.1 Definition

Low back pain (LBP; Chap. 41) can have a variety of generators including degenerative disc disease (DDD) and lumbar disc herniation (LDH; Chap. 46). DDD can be defined as an age accelerated structural failure of the intervertebral disc (IVD), and thus disc degeneration does not necessarily equal DDD. To make a diagnosis of DDD, the clinical picture must be evaluated in addition to the radiographic presence of disc degeneration. LDH can occur in both degenerated and non-degenerated disc segments. LDH occurs when the nucleus pulposus is able to herniate through the annulus fibrosis. LDH can cause back pain, radicular pain, and sensory/motor disturbances.

45.2 Natural History

LBP is the most common cause of disability in young adults with an indirect loss of >100 billion dollars per year in decreased productivity. Over 80% of young adults will experience an episode of back pain in their lifetime, and an additional 2% to 3% of these patients will get radiculopathy as well. Typically, patients with DDD will

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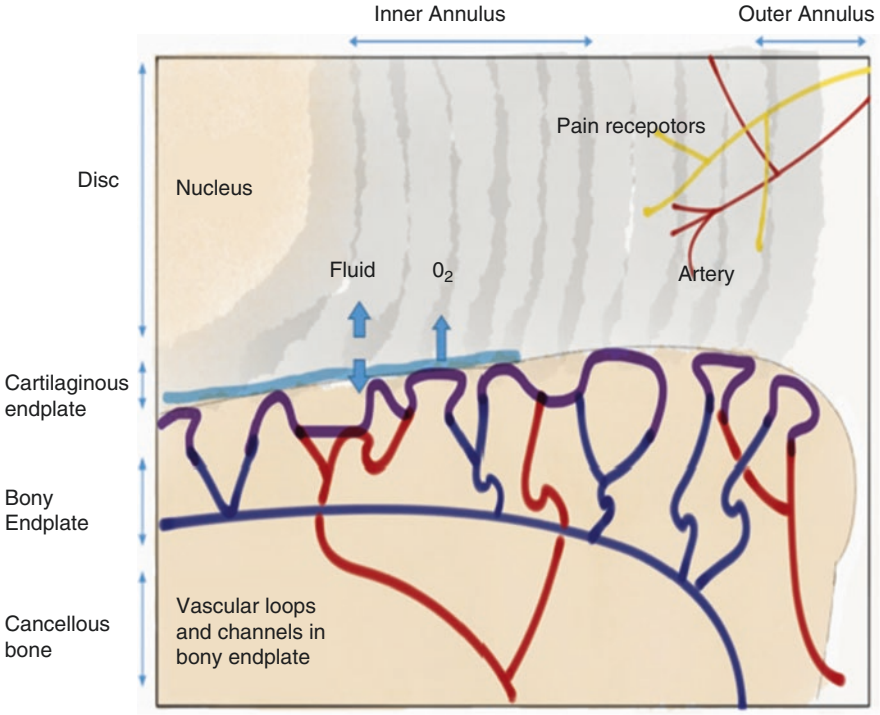

Fig. 45.1 Nutrition of the IVD

have a self-limited course of back pain, with 95% experiencing a recovery in 3 months with appropriate nonoperative treatment modalities. However, chronic back pain is a possibility for these patients. The pathway that leads to DDD is a complex interplay between nutritional, environmental, and genetic factors. Decreased nutritional supply limits the IVD from responding to load (Fig. 45.1), while genetic polymorphisms can affect genes that contribute to IVD structure and upregulate pro-inflammatory pathways (Fig. 45.2). Environmental factors that contribute to DDD include obesity and cigarette smoking. Understanding the degenerative cascade is important for the development of future treatment modalities. For LDH, the vast majority of patients (~75%) will experience relief at 1 year. However, in patients who remain symptomatic beyond 6 weeks, there is a small but statistically significant benefit to undergoing a direct decompression of the affected nerve root.

45.3 Physical Examination

Observation of behavior, gait, and muscle atrophy is important when evaluating patients with DDD and LDH. Observation of Waddell's signs is important for this patient population as well. Patients with a large LDH may be very uncomfortable and

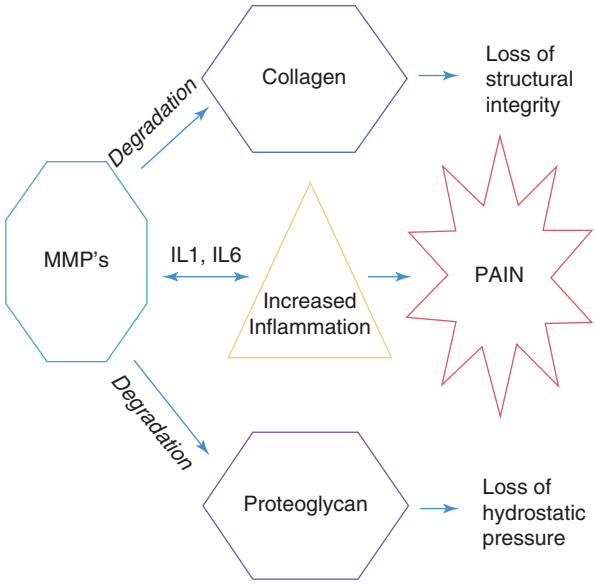

Fig. 45.2 Inflammatory pathways in DDD

be unable to sit down. Patients with DDD will present most commonly with back pain. Midline spinous process tenderness is very common. The range of motion will be limited secondary to pain (Appendix N). Pain that worsens with extension is likely facet-oriented, while worsening of pain with lumbar flexion suggests a discogenic mediator of pain. Radicular pain is very common with disc herniation. There are commonly motor, sensory, and reflex deficits along the affected nerve root as well (Video 45.4). The straight leg raise test can reproduce radicular limb pain. A positive contralateral leg raise test usually indicates a massive or extruded disc fragment.

45.4 Imaging

Radiographs of the lumbar spine should include anteroposterior (AP) and lateral radiographs of the affected area (Fig. 45.3). The AP view may show arthritic changes in the facets or osseous bridging between the disc spaces. The lateral view can show a variety of pathology including loss of disc height, spondylolisthesis, and vacuum disc (Fig. 45.4). Computed tomography (CT) scan will clearly show arthritic changes throughout the spine including in the facet joints, end plate sclerosis, and calcified disc herniation. However, a CT scan is not always required for these patients unless it is to address a specific question or if the patient is unable to obtain magnetic resonance imaging (MRI).

MRI is indicated for patients who fail to recover after 6–12 weeks of proper nonoperative management and for those with any neurological deficit (sensory/

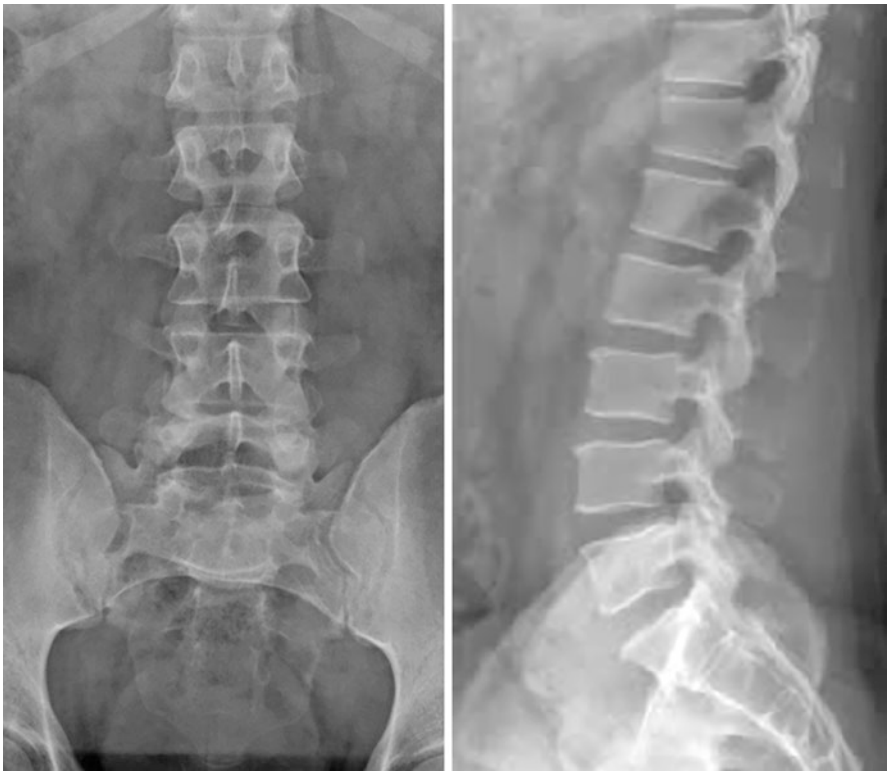

Fig. 45.3 Normal lumbar radiographs (normal disc height, no vacuum phenomenon in the disc, no arthritic changes)

motor) in a radicular pattern. Patients with DDD will typically have a loss of signal within the nucleus pulposus and disc space collapse on MRI (Fig. 45.5). Annular tears can be appreciated as high-intensity zones in the posterior annulus on T2-weighted imaging. Some MRI classifications include the Modic system and the Pfirrmann classification (Fig. 45.6; Tables 45.1 and 45.2). The clinical utility of these classification systems is uncertain, but there is evidence to suggest a higher failure of nonoperative treatment in those with Modic changes on MRI.

45.5 Differential Diagnosis

There is a broad differential diagnosis for DDD and LDH (Table 45.3). Potential pain sources could be viscerogenic (renal/abdominal causes), neoplastic (primary or metastatic bone tumors; Chap. 63), neurogenic (spinal cord tumors/cysts), inflammatory disease (ankylosing spondylitis; Chap. 54), infectious (discitis, osteomyelitis; Chap. 65), psychogenic, and spondylogenic (muscle strains, spinal stenosis, disc disease, facet arthropathy; Chap. 41; Chap. 46). Radiculopathy is most commonly

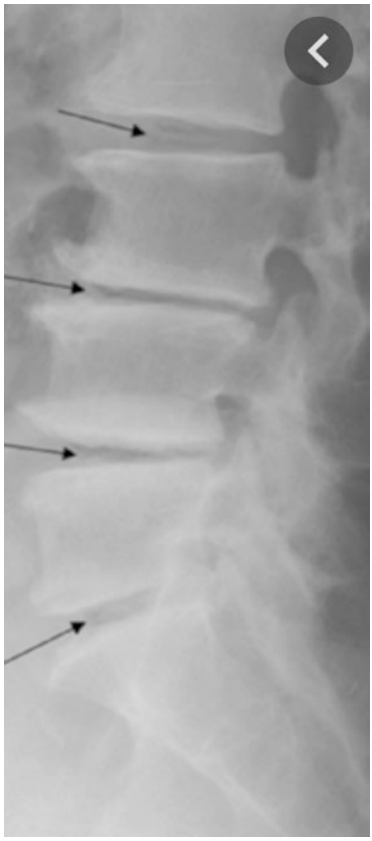

Fig. 45.4 Lumbar lateral radiograph showing vacuum disc phenomenon with accumulation of air in the disc space (black arrows)

caused by a disc herniation but may be mimicked from peripheral nerve compression (e.g., piriformis syndrome) or intraneural pathology (e.g., peripheral nerve sheath tumor).

45.6 Treatment Options

The vast majority of patients undergo nonoperative care for DDD. Physical therapy is a mainstay of treatment, along with lifestyle modification (smoking cessations/weight loss/core strengthening/back education). Anti-inflammatory medications are very effective for treating pain related to DDD and LDH. Steroid medications are useful for radiculopathy. Cognitive behavioral therapy is effective at treating back pain in patients with chronic pain. Alternative therapies are frequently attempted as well, including yoga, CBT, chiropractor, CBD oil, and oral CBD with varying success. In patients with radiculopathy, transforaminal epidural injections can be effective in reducing the chemical/inflammatory component of the radiculopathy but do not change the natural history of the disc herniation.

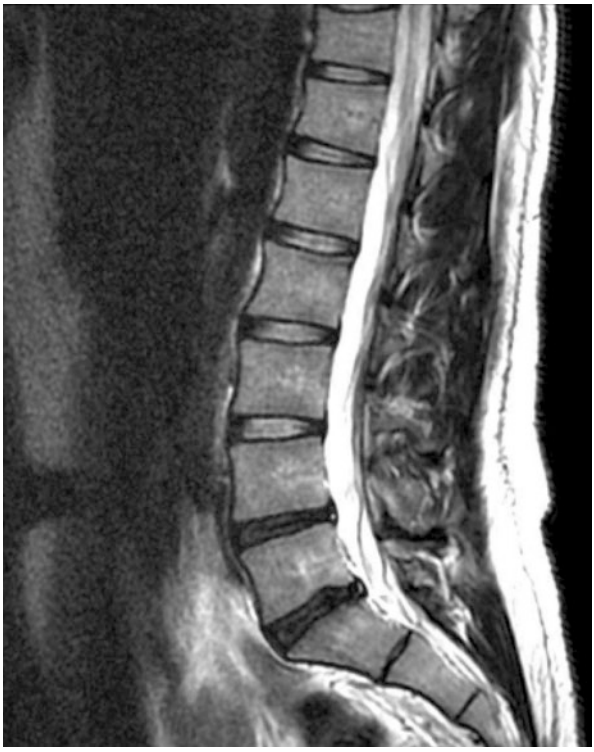

Fig. 45.5 T2 lumbar MRI showing degenerative disc at L4-5, L5-S1 (loss of hydration, disc collapse), no Modic changes

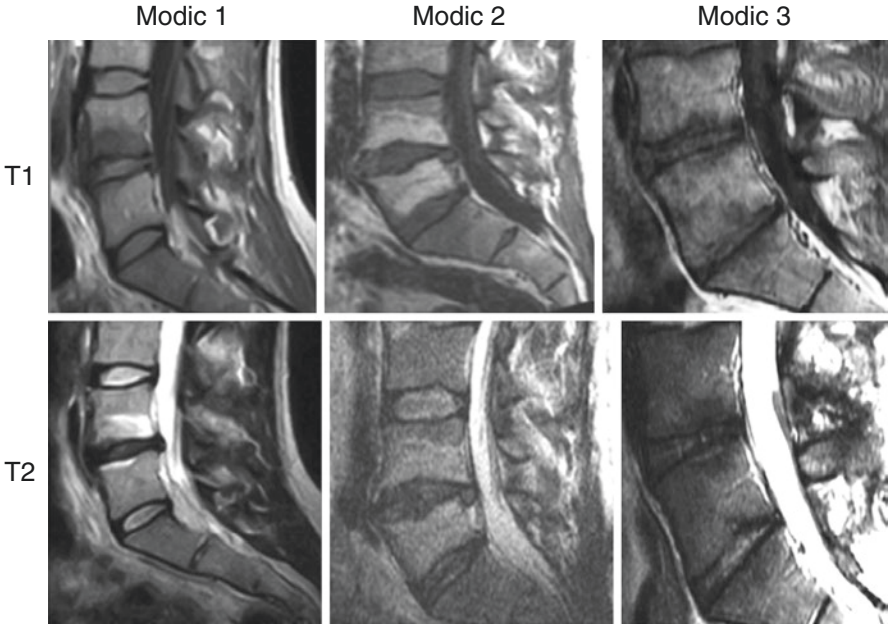

Fig. 45.6 Modic changes (Reproduced from Jones et al.)

Table 45.1 Modic changes as illustrated

Type	T1 MRI signal intensity	T2 MRI signal intensity
I	Hypointense	Hyperintense
II	Hyperintense	Iso or hyperintense
III	Hypointense	Hypointense

Reproduced

Table 45.2 Pfirrmann grades as illustrated

Grade	Structure	Distinction (nucleus and annuals)	T2 MRI signal intensity	Disc space height
I	White, homogenous	Clear	Isointense to cerebrospinal fluid (hyperintense)	Normal
II	Inhomogeneous, with banding	Clear	Isointense to cerebrospinal fluid (hyperintense)	Normal
III	Gray, inhomogeneous	Unclear	Intermediate	Normal to decreased
IV	Gray to black inhomogenous	No distinction	Intermediate to hypointense	Normal to decreased
V	Black inhomogenous	No distinction	Hypointense	Collapsed

Reproduced

Table 45.3 Differential diagnosis of low back pain

Type	Differential
Viscerogenic	Abdominal and renal
Neoplastic	Primary and metastatic bone tumors
Neurogenic	Spinal cord tumors or cysts
Inflammatory diseases	Ankylosing spondylitis, Reiter's syndrome, inflammatory bowel diseases, and psoriatic arthritis (sacroiliitis)
Infectious	Diskitis, osteomyelitis, psoas abscess
Spondylogenic	Myofascial syndromes Iliolumbar syndrome, piriformis syndrome, quadratus Lumborum syndrome, and fibrositis (trigger point syndrome) Motion segment disorders Disk disease, facet syndrome, spinal stenosis Bony problems Fractures, including osteoporotic compression fractures Spondylolisthesis Sacral lesions, coccyx pain Psychogenic

For LDH, in patients who fail conservative treatment for at least 6 weeks, surgical intervention can lead to improved outcomes. For any patient with a progressive neurological deficit or neurological findings (radicular pain/straight leg raise positive), operative management is appropriate (Video 45.4). Any method of lumbar decompression and stabilization is appropriate (open or minimally invasive) for these patients if a direct nerve decompression can be achieved (Video 45.3).

45.7 Expected Outcomes

The majority of patients recover very well from disc herniation. Patients with DDD can have a more variable clinical course due to the complexity of treating LBP. Nonoperative modalities of treatment can provide significant relief for these patients, but for those with chronic unrelenting pain, surgical treatment without any accompanying neurological symptom is very controversial with no guarantee it will relieve pain.

45.8 Potential Complications

The major complication risk profile for lumbar decompression surgery is continued symptoms, nerve root injury, dural tear, re-herniation, and infection. However, the complication of nonoperative care for an LDH in a patient with neurological compromise is chronic pain and permanent loss of muscle function.

45.9 What Should Patient and Family Know?

DDD is a lifelong condition that will require significant efforts in lifestyle modifications and physical therapy for the best chance of a good outcome. Patients with LDH with symptoms lasting longer than 6 weeks or with neurological compromise have better outcomes with surgery. These patients have a great chance for a full functional recovery.

Further Readings

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