Lumbar DDD is manifest clinically by a spectrum of disorders, including disc extrusion with or without migrated fragment, disc protrusion, (central, paracentral, intraforaminal, or far-lateral) disc bulge, and internal disc disruption. Of these, frank disc rupture causing mono-radiculopathy or cauda equina syndrome is a well-established entity. Very little, if any, controversy exists with regard to its clinical diagnosis or management, although there may be minor differences of opinion about the choice of options for treatment. In the past two decades, the syndrome of disc resorption without disc herniation has been recognized as a definable entity amenable to surgical treatment.11,12 Historically, disc rupture with monoradiculopathy or cauda equina syndrome is a well-established entity. Very little, if any, controversy exists with regard to its clinical diagnosis or management, although there may be minor differences of opinion about the choice of options for treatment. In the past two decades, the syndrome of disc resorption without disc herniation has been recognized as a definable entity amenable to surgical treatment.11,12 Historically, disc rupture with monoradiculopathy was thought to be a clinical syndrome amenable to surgery, originating with the initial description of the syndrome by Mixter and Barr.30 Patients presenting with axial back-dominant pain but with minimal or absent radicular pain were not thought to be good candidates for surgical intervention. There have been advances in several related fields, including a better understanding of the anatomical, physiological, and biochemical features of pain generators in the intervertebral disc, refinements in the technique of lumbar discography, improved resolution in MR imaging, development of newer anterior approaches to the lumbar disc (open or laparoscopic), evolving concepts about the usefulness of bone morphogenetic proteins, and critical evaluations of surgery-related results following lumbosacral fusion. These advances are contributing to the rapid contemporary evolution in the understanding of discogenic pain syndrome.

Several terms have been applied to this discogenic pain syndrome and the differences are minor; these include trauma-induced internal disc disruptions, black disc disease, isolated disc resorption, and segmented instability.

**CLINICAL SYNDROME**

Discogenic back pain syndrome appears to be a disease of adulthood. Although disc disease is well recognized in teenagers and even younger children, the childhood syndrome is one of disc herniation at a single or multiple levels. The incidence of lumbosacral DDD is higher in young athletes such as gymnasts or ballet dancers, but in our experience, they present with disc herniation rather than black disc disease. This difference may be the result of age-related biochemical changes in the intervertebral disc.

The cardinal manifestation of internal disc disruption is back pain. Although a patient’s description may seemingly suggest diffuse low-back pain, we have found that when specifically questioned and asked to run a finger horizontally across the back at the site of maximum pain, the accuracy of this pain localization matches that defined by MR imaging in approximately 80% of the cases. This observation may be related to the segmental nature of innervation of the anulus, which is the most pain-sensitive structure.23 O’Brien35,36 has observed focal tenderness at the anterior lumbosacral region with transabdominal palpation. He attributed this to the rich innervation of the anulus, which is the most pain-sensitive structure.23 O’Brien35,36 has observed focal tenderness at the anterior lumbosacral region with transabdominal palpation. He attributed this to the rich innervation of the anulus, which is the most pain-sensitive structure.

**KEY WORDS** • disc degeneration • internal disc disruption • magnetic resonance imaging • discography • discogenic pain • lumbar fusion

**Abbreviations used in this paper:** DDD = degenerative disc disease; MR = magnetic resonance; PLIF = posterior lumbar interbody fusion.
Defects in the DNA for collagen have been identified in most instances to consider back surgery. If a patient fails to participate in a weight-reduction program, it will be futile to consider surgical intervention until serious measures are undertaken for weight reduction. These may include sustained, systematic reduction in caloric intake under medical supervision, increased physical activity with a tailored exercise program, pharmacological therapy for appetite suppression and enhanced fat excretion, and, as a final resort, gastric bypass surgery. If the patient fails to participate in a weight-reduction program, it will be futile to consider surgical intervention.

CAUSATIVE FACTORS

Unquestionably, repetitive or continuous axial overloading is the key determinant in the pathogenesis of lumbar degenerative disease. Morbid obesity continues to be a major public health issue in the United States and, to a lesser extent, in other Western nations. The clinical triad in the obese individual is intractable low-back pain, bilateral knee pain, and flat feet with bilateral ankle pain. The primary pathological process is cartilaginous degeneration in the intervertebral discs and the cartilage in the knee joint. Although axial loading is the obvious factor implicated, accelerated degeneration due to fatty infiltration of cartilage may not be ruled out. It is unwise in this setting to consider surgical intervention until serious measures are undertaken for weight reduction. These may include sustained, systematic reduction in caloric intake under medical supervision, increased physical activity with a tailored exercise program, pharmacological therapy for appetite suppression and enhanced fat excretion, and, as a final resort, gastric bypass surgery. If the patient fails to participate in a weight-reduction program, it will be futile to consider surgical intervention.

Genetic factors have an influence in the incidence of the DDD. Defects in the DNA for collagen have been identified in family clusters predisposed to degenerative disc disease. Other genetic defects resulting in impaired proteoglycan synthesis are being explored. Videman, et al., noted that polymorphism associated with the vitamin D receptor gene correlated with intervertebral disc degeneration. Richardson, et al., confirmed, through an epidemiological survey, the presence of a familial disposition for back pain. Degenerative disorders involving family clusters tend to manifest as multilevel disc herniations at a younger age. Elfering, et al., noted a high incidence of disc degeneration in individuals working night shifts because of the relative dessication of the disc at night.

Occupation is a very important determinant. Workers performing typical repetitive work in an assembly line setting are prone to back problems, especially if the work involves repetitive bending, turning, and lifting. Jobs necessitating lifting and carrying heavy loads are associated with a high incidence of lumbar degenerative disease; examples include furniture movers, landscapers, and medical assistants working in nursing homes. Authors of epidemiological studies point to whole-body vibratory forces such as driving trucks, earthmovers, or tractors as contributing to low-back pain.

Vigorous and compulsive athletic activities in a competitive setting predispose to accelerated degeneration of discs. Examples include weightlifting and gymnastics. Cigarette smoking is implicated in DDD, but a direct link has not been proven.

PAIN GENERATION

It is crucial to understand and localize the DDD-related pain generator to tailor the surgical treatment and eliminate the source of pain. As stated previously, O'Brien has noted pain in the anterior anulus on direct palpation of this disorder is diagnosed has had a preexisting problem that is only aggravated by the accident. Loading injuries do not occur even in high-velocity accidents with the individual in the seated position.

MAGNETIC RESONANCE IMAGING FINDINGS

There are certain consistent MR imaging changes indicative of DDD, but the findings should always be interpreted in light of clinical presentation because it is impossible to differentiate symptomatic from incidental syndromes based on MR imaging studies alone. A defining characteristic is the decrease in signal intensity on T2-weighted sequences obtained in the nucleus pulposus compared with the adjacent disc (Fig. 1). The outline of the nucleus pulposus becomes irregular and the disc height decreases. An intense dotlike high-intensity signal in the posterior anulus signifies an anular tear. The cortical endplate and the adjacent marrow show changes in three steps, well described by Modic.
Black disc disease

Fig. 1. Sagittal T2-weighted MR image demonstrating black disc disease at L5–S1.

ROLE OF DISCOGRAPHY

Although the role of provocative discography in the diagnosis of discogenic pain syndrome remains controversial, in the past 2 to 3 years its role has become better defined.1,4,9,28,33,47 The results are still somewhat controversial, in the past 2 to 3 years its role has become better defined.

The diagnosis of discogenic pain syndrome remains controversial. However, the role of provocative discography has become clearer in the past 2 to 3 years. The results are still somewhat controversial, in the past 2 to 3 years its role has become better defined.

In our practice, we limit discography to two specific clinical settings: 1) a patient with credible pain, well-defined low-back tenderness, some objective signs of nerve root irritation, and yet no evidence of abnormality on MR imaging (in this setting, the suspected pathological disc is injected first and then the adjacent disc); and 2) a patient with a well-defined degenerative disc at one level with marginal changes in the immediately adjacent disc (one needs to determine if the second disc needs to be included in the fusion).

PATIENT MANAGEMENT

As a sound surgical principle, general conservative measures should be instituted first. These may include a long-term exercise program for conditioning, physical therapy with various modalities, a trial of epidural steroid injections, and a corset worn only when the patient is active. All of these physical measures are supplemented by pharmacological therapy involving nonsteroidal antiinflammatory drugs, muscle relaxers, and low-potency narcotic agents. A patient’s lifestyle or vocation may have to be modified to avoid repetitive injury. Restrictions at the worksite and establishment of an ergonomic environment in the workplace, with the assistance of an occupational therapist, might help reduce the likelihood of repetitive injury.

Failure of conservative treatment over a period of 3 to 6 months heralds the need for surgical treatment. Table 1 provides a summary of the surgical choices available today. The number of choices is increasing with the introduction of percutaneous placement of pedicle screws and the impending approval of bone morphogenetic protein for clinical use. The choice of surgical procedure is also governed by published results pertaining to long-term follow-up in patients who have undergone surgery via various techniques.

Cloward13 pioneered the technique of PLIF in which structural allograft was used. Although he reported 80 to 90% fusion rates without the use of pedicle screws, others4 have not been able to reproduce his success rate. Lin25 and Ma27 have refined the PLIF technique. Poor success rates have led to other techniques. Kuslich, et al.,1622 introduced the use of threaded cages, but analysis of recent results indicates that there is high failure rate with stand-alone cages introduced anteriorly or posteriorly, unless they are supplemented by pedicle screw stabilization. Anterior interbody fusion with femoral ring in single-level disease yields a success rate of 80 to 90%, but the rate drops precipitously in two-level procedures unless supplemented by posterior stabilization. The Harrms transforminal fixation technique41 involves the unilateral removal of facet joint, radical discectomy, anterior column support in which cages are supplemented by bone, and pedicle screw stabilization. This is a viable alternative to combined 360° decompression and fusion and is currently our preferred option for black disc disease. The choice of surgical technique46,16,20,21,24,26,32,34,48,50,51 is left to the surgeon as long as the following principles are adhered to: near-total excision of the intervertebral disc; placement of spacer to maintain anterior column support and lordosis; use of adequate bone graft, bone extender, and bone enhancers; and surgical stabilization.

References


TABLE 1

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<th>Surgical options in black disc disease*</th>
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<tr>
<td>ALIF alone</td>
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<td>ALIF w/ transfacetal screw</td>
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<td>ALIF w/ pedicle screw stabilization (open or percutaneous)</td>
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<tr>
<td>PLIF w/ pedicle screw stabilization</td>
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<td>transforminal interbody fusion with pedicle screw stabilization</td>
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* ALIF = anterior lumbar interbody fusion.


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