Discogenic Low Back Pain

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KEYWORDS

• Low back pain • Discogenic low back pain • Lumbar disk disease • Annular tear
• Lumbar disk herniation • Internal disk disruption

KEY POINTS

- The intervertebral disk can be a common source of acute or chronic low back pain.
- A thorough history and physical examination are needed to assess patients with discogenic low back pain.
- Lifestyle modifications including smoking cessation, proper lifting mechanics, ergonomics, and lower body mass index are helpful.
- Discogenic pain results from changes in the nucleus pulposus and tears in the annulus fibrosis.
- Psychological support, including treatment of underlying or coexistent depression, is highly recommended.
- Conservative treatment, including home exercise for chronic lower back pain, is recommended.
- Injection of corticosteroids for discogenic lower back pain shows mixed efficacy and should be performed judiciously.
- Surgery for this condition should be reserved for cases of significant refractory cases with profound disability and should be confirmed with diskography.

INTRODUCTION

Nature of the Problem

Chronic low back pain is a common and challenging problem presenting to a variety of practitioners. It is estimated that up to 90% of people experience significant lower back pain in their lifetime.[1] Although low back pain was traditionally believed to be self-limited in most cases, data have emerged showing that many low back pain sufferers have recurrences or go on to a more chronic course.[2]

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The economic burden of lower back pain is staggering. It is estimated that the annual cost in the United States for the treatment of lower back pain is between 20 and 50 billion dollars.[3] The pain can be debilitating in many cases and is a major reason for absence from work.[4]

In most cases of acute lower back pain, a definite pain generator cannot be defined.[5–7] For chronic low back pain sufferers, an estimated 39% of cases can be attributed to the intervertebral disk.[8,9] This situation is complicated because not all degenerated or herniated disks are painful.[10] Disk degeneration is a natural part of the aging process and normally begins about the third decade of life. In the right clinical setting, the disk can become a primary source of pain. The following sections address the challenging problem of pain emanating from the damaged and sensitized lumbar intervertebral disk.

Anatomy

The human intervertebral disk consists of a firm, collagenous exterior annulus fibrosis and a gelatinous interior nucleus pulposus. It is often compared with a jelly donut. This structure offers shock absorption and allows for the dispersion of axial and torsional forces at each level through the spine. The annulus is thicker anteriorly than superiorly, therefore herniations and tears are more common posteriorly.[11] The nerve supply to the human intervertebral disk consists of contributions from the sinuvertebral nerves and gray rami communicantes.[12–14] These nerves are segmental and from the dorsal ramus, but recent studies have shown some contributions from the sympathetic chain in a nonsegmental distribution.[15]

Pathophysiology

Pain from lumbar disks can occur from 3 main causes, as described extensively by Bogduk.[16] These causes include disk infection, torsion injury, and internal disk disruption (IDD). This review focuses on torsion injury and IDD.

Torsion injury is believed to result from forcible rotation of the intervertebral joint.[16] Rotational forces around an impacted zygapophysial joint produce a lateral shear force on the disk, which can lead to circumferential tears.[16–19] This cause of disk pain is difficult to prove with objective testing. It is a clinical diagnosis related to patient history, often related to a rotational strain injury.[16]

IDD results from lumbar disk degradation, its nuclear components, and development of radial fissures that extend from the nucleus into the annulus. IDD is believed to be the most common type of discogenic pain.[16] Disk disruption is not to be confused with degenerative changes, which are a normal part of aging.[16] Studies have suggested IDD to be independent of degenerative changes that are seen with aging.[20] Furthermore, it is well known that degenerative disk changes related to aging do not correlate with pain.[21]

The development of radial fissures is believed to be related to repetitive shear, axial loads, and compression of the disk. This process leads to vertebral endplate fractures, in which the fissures can develop followed by disk degradation over time as nuclear material leaks out of the disk or desiccates.[22]

Radial fissures may correlate with pain. A process of sensitization and neural ingrowth are proposed mechanisms of the development of IDD.[12,13] Pain-associated proinflammatory mediators including calcitonin gene-related peptide, tumor necrosis factor α, interleukin 1, interleukin 6, and substance P have been isolated from disks with this morphology.[12,13,23,24]

The modified Dallas diskogram scale is a descriptive way to categorize the severity of IDD after diskography (**Fig. 1**).[25] Using this scale, grade 1 and 2 fissures often produce no pain, whereas grade 3 and 4 fissures frequently produce pain.[26]