Lumbar disc disease: the natural history

NEVAN G. BALDWIN, M.D.

Texas Tech University Health Sciences Center, Lubbock, Texas

Symptomatic lumbar disc disease represents a major cost to societies providing modern care for these conditions. The impact of any treatment cannot be assessed without an understanding of the natural history of the disease process. The majority of individuals with degenerative disc disease are asymptomatic. Although the natural history of sciatica is associated with a good overall prognosis, that of discogenic low-back pain is less promising. For patients with symptomatic lumbar disc herniations, the results of discectomy are better than those predicted by the natural history of the disease process.

KEY WORDS • lumbar spine • intervertebral disc • natural history • outcome

“Timing has an awful lot to do with the outcome of a rain dance.” Conventional Cowboy Wisdom

Symptomatic lumbar disc disease is responsible for a tremendous cost to society. It is believed to be a major contributor to the estimated 60 to 80% lifetime incidence of low-back pain in the general population.^{8,24} The cost of treatment for low-back pain in the United States alone is measured in 10s of billions of dollars per year.⁶ Patients with radiculopathy represent another large segment of the population who consume care costs related to lumbar disc disease. Several relatively recent innovative technologies have been developed for the treatment of lumbar disc disease. As with most new technologies, there are associated costs. Although high-tech innovations may ultimately reduce the cost of treating lumbar disc disease, the cost-related advantage or disadvantage associated with these technologies cannot be determined without a clear knowledge of the natural history of the disease process. Applying an expensive treatment to a disease that has a high likelihood of spontaneous improvement may well yield results similar to those of a well-timed rain dance.

LUMBAR DISC DISEASE AND LOW-BACK PAIN

Whereas lower-extremity radicular pain is well accepted as a symptom of lumbar disc disease, the role of disc disease as a cause of nonradicular back pain remains more

controversial. In numerous studies investigators have demonstrated that, during lumbar surgeries performed after administration of a local anesthesia, pressure to the annulus fibrosis results in low-back pain. Falconer, et al.,⁵ noted that the application of pressure to lumbar discs generated low-back pain during such operations. Kuslich, et al.,¹⁴ were able to generate back pain similar to the preoperative pain in 70% of the patients in a consecutive series of 193 cases when stimulation was applied to the annulus fibrosis or the posterior longitudinal ligament.¹⁴ This pain response was blocked with the application of a local anesthetic.

The existence of discogenic back pain is perhaps the subject of the most controversy relating to the anatomical arguments for its presence. Opponents of the discogenic pain hypothesis have argued that because there are no pain receptors within the intervertebral discs, discogenic pain cannot occur. The presence of the sinuvertebral nerve to the spinal canal was described in the 19th century.⁶ More recent staining techniques, however, have allowed precise anatomical description of this neurological structure. Groen, et al.,¹¹ identified the sinuvertebral nerve as a derivative of the rami communicantes arising as a postganglionic structure branching into segments ending in the posterior longitudinal ligament and the outer lamina of the annulus fibrosis. Arguments for the existence of discogenic low-back pain are therefore supported by both empirical clinical data and neuroanatomical research.

Abbreviation used in this paper: MR = magnetic resonance.

Natural History

Although lumbar disc disease may well be a cause of low-back pain, it is important to note that disc degeneration can occur in the absence of back pain or any other symptoms. Paajanen, et al.,²⁰ found that in 35% of healthy male volunteers significant disc degeneration was demonstrated on MR imaging. In a series of 600 autopsy specimens, Miller, et al.,¹⁷ showed that 90% of all lumbar discs had evidence of degeneration in individuals by the age of 50 years. The presence of disc degeneration and the presence or absence of low-back pain may be considered analogous to the occurrence of gallstones and abdominal pain. Although most individuals with gallstones are asymptomatic, if a patient has gallstones and the appropriate symptoms, those symptoms may be attributed to the gallstones.

Although low-back pain results in significant disability, 95% of these patients return to their previous employment within 3 months of symptom onset.⁶ Failure to return to work within 3 months may be considered a poor prognostic sign. In patients who experience total disability after 1 year, the likelihood of returning to work is less than 20%. After 2 years of disability, the probability of returning to work is less than 2%.¹

Numerous imaging studies have demonstrated that with nonsurgical treatment lumbar disc herniations will subside over time in the majority of cases.^{2,13,23} In a series of 32 patients studied with serial MR imaging, Matsubara, et al.,¹⁵ found regression of disc herniations in 62%. They also demonstrated that the more degeneration seen initially in the discs, and the larger the initial herniation, the more the size of the herniated disc fragment was observed to decrease.

Although degenerative disc disease is common in asymptomatic populations, large compressive lesions are uncommon. Patients with large compressive lesions are also generally believed to be more ideally suited to surgical intervention. These same patients, however, are those most likely to experience spontaneous regression of their lesions and they have a high rate of clinical improvement with noninvasive treatments.²³

Clinical improvement following a symptomatic lumbar disc herniation does not necessarily correlate with the radiographically documented resolution of the herniation. Fraser, et al.,⁷ performed a study in which they examined the MR imaging findings in patients who had undergone treatment for lumbar disc herniation 10 years earlier. Approximately one third of these patients had undergone a saline injection as a control treatment for the study of chymopapain as a treatment for lumbar disc herniation. Overall, a persistent herniated disc was found in 37%, and the incidence of persistent herniation was similar in patients treated with chymopapain, surgery, or saline injection. Interestingly, the presence or absence of persistent disc herniation had no significant bearing on outcome. Analysis of their findings indicated that long-term improvement of symptoms after treatment of a disc herniation may occur with or without resolution of the herniated disc.

The term "disc herniation" in this article refers to a process in which there has been rupture of the annulus fibers and subsequent displacement of the central mass of the disc in the intervertebral space, common to the posterior

or posterolateral aspect of the disc. Posterior protrusion may be visualized as a bulging disc or, if the outer fibers of the annulus are penetrated, as an extruded disc. The phenomenon, when it occurs in conjunction with clinical symptoms, most commonly occurs in individuals between the ages of 30 and 50 years.²⁶ Although it is seen in both younger and older individuals, acute disc herniation is far less commonly observed in these populations. There is no definitive information regarding the incidence or prevalence of disc herniations in a sizable population that would define the norm. Risk factors for disc herniation include driving of motor vehicles, sedentary occupation, vibration, smoking, previous full-term pregnancy, physical inactivity, increased body mass, and a tall stature.^{19,26} With regard to occupations that require strength, it has been shown that an increasing degree of disparity between the strength required and the strength of the individual results in higher rates of symptomatic low-back injury.^{4,12}

Preventative efforts in lumbar disc disease, other than by modification of the aforementioned risk factors, have not been successful. Intuitively, one might assume that physical fitness would be preventative with regard to disc rupture. Although the literature on this topic is not entirely uniform in opinion, the strongest evidence probably suggests that physical fitness does little to protect or provoke attacks of sciatica. Physical fitness, however, likely is advantageous as a factor affecting rehabilitation after disc surgery.²⁵

It is well supported by clinical evidence that sciatica can be managed by lumbar surgery.^{10,16} The early recovery rate and the rate of return to work are improved following surgical intervention. Postoperatively, recovery of the efferent conduction system is considerably better than that seen in the afferent portion.

The mechanism for radiculopathy-related pain production has been a subject of considerable debate. Compression of the nerve has been shown to create edema formation and eventually lead to intraneural inflammation and hypersensitivity.²¹ This then results in increased mechanosensitivity of the nerve root with regard to compression and the induction of pain. It is now generally accepted that a combination of mechanical and abnormal biochemical events is involved in the generation of radicular pain.²²

It has been shown that most patients hospitalized for sciatica will have suffered their first episode of acute low-back pain while in their third decade of life.²⁵ The initial episode is usually provoked by an acute traumatic event. An approximate mean of 10 years will pass before the onset of the first radicular symptoms. Weber²⁵ attributed this long interval between onset of low-back pain and the onset of radicular pain to intradiscal degeneration and regeneration forces. Weber and colleagues²⁷ were also able to demonstrate improvement over the course of 4 weeks in 70% of their 208 nonhospitalized patients with sciatica in whom nonsurgical intervention was performed. Sixty percent of those patients returned to work by the 4-week interval.

Saal, et al.,²³ reported on a series of 64 patients with verified herniated lumbar discs who received nonsurgical treatment during a 1-year period. Treatments included epidural steroid injection as well as oral steroid therapy, transcutaneous electrical nerve stimulation, acupuncture,

Natural history of lumbar disc disease

exercises, and physical therapy modalities, in addition to nonsteroidal antiinflammatory drugs. In that 1-year trial, satisfactory recovery was demonstrated in 90% of the patients.

Bush, et al.,³ reported on 165 consecutive patients (mean age 41 years) who suffered sciatica due to lumbosacral nerve root compression. The patients were treated primarily with epidural steroid injections and local anesthetics. All but 23 of these patients (14%) experienced satisfactory clinical recovery at 1 year. Those 23 patients in whom no improvement occurred underwent decompressive surgery. Although the use of epidural steroid injections to treat acute radiculopathy and/or acute low-back pain is somewhat controversial, there is evidence to suggest that a favorable effect is more likely when its used to treat patients with acute onset of symptoms.

In the aforementioned studies, the authors obtained excellent results with nonsurgical interventions. In fact, those results are so good that one would be hard pressed to suggest that surgery could improve upon such impressive rates of satisfactory outcome. In controlled trials in which patients were randomized to surgical and nonsurgical treatment groups, however, surgery has been shown to be advantageous.²⁵⁻²⁷

It has long been accepted that there are definite indications for surgical intervention. These include the cauda equina syndrome, progressive loss of motor strength, and severe intractable pain. The empirically observed rates of halting disease progression in cases in which these indications exist is so good that, given such symptoms, randomized clinical trials would be unethical. Such patients, however, represent a minority of the lumbar disc herniation population and study of the far larger group who are less symptomatic is therefore warranted. Weber²⁵ reported on herniated lumbar discs in 208 patients among whom 126 were incapacitated by sciatica but in whom indications for surgery were believed to be uncertain. This subgroup was therefore randomized to surgical and nonsurgical treatment for purposes of comparison. The patients were then followed for a 10-year period postoperatively.²⁵ In this controlled prospective randomized trial Weber found a statistically significant improvement at 1 year in cases in which surgery was performed. Ninety percent of those patients who had undergone surgery reported a good outcome compared with 60% in the control group. Seventeen patients in the control group had also undergone surgery for persistent or worsening pain. After 4 years, the surgery-treated patients continued to experience better results than those in the control group. At the 4-year interval, the differences were not statistically significant. At 10 years posttreatment, results in the groups were essentially identical. Motor deficits in both groups resolved nearly completely. More than one third of the patients suffered persistent sensory deficits at the conclusion of the study. A key finding was also that in patients in whom symptoms persisted for more than 3 months preoperatively, a far worse prognosis was demonstrated.

CONCLUSIONS

A few tenets of surgical treatment of lumbar disc disease are well supported by multiple studies. It has been

shown that for the best results, surgery should be performed preferably within the first 3 months of the onset of sciatica. The value of repeated surgery for persistent radiculopathy after an initial operation is usually questionable. Nachemson¹⁸ reported that only 25% of patients undergoing revision surgery experienced any improvement within a 3-year follow-up period. Nachemson recommended repeated surgery only if a new disc herniation was demonstrated. In patients suffering low-back pain and/or sciatica for more than 6 months, the likelihood of successful rehabilitation is only approximately 40%.

An evidence-based review of the existing data on the surgical treatment of lumbar disc disease supports the use of discectomy in carefully selected patients with sciatica due to lumbar disc herniation.^{9,10} Discectomy has been shown to provide faster relief from the acute attack, although the effect of surgery, positive or negative, on the lifetime natural history of disc disease remains unclear. There is evidence of moderate scientific quality that the clinical outcomes of microdiscectomy are comparable with those of standard discectomy.^{9,10,16} There is moderate evidence that automated percutaneous discectomy produces poorer clinical results than standard discectomy or chemonucleolysis.^{9,10} There is suitable evidence to underscore that discectomy (or microdiscectomy) affects the disease outcome in a manner that exceeds the expected outcomes associated with the natural history alone.^{9,10}

References

1. Andersson GB, Svensson HO, Oden A: The intensity of work recovery in low back pain. **Spine** 8:880-884, 1983
2. Boos N, Semmer N, Elfering A, et al: Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: predictors of low back pain-related medical consultation and work incapacity. **Spine** 25:1484-1492, 2000
3. Bush K, Cowan N, Katz DE, et al: The natural history of sciatica associated with disc pathology. A prospective study with clinical and independent radiologic follow-up. **Spine** 17:1205-1212, 1992
4. Chaffin DB: Manual materials handling: the cause of over-exertion injury and illness in industry. **J Environ Pathol Toxicol** 2:31-66, 1979
5. Falconer MA, McGeorge M, Begg AC: Cause and mechanism of symptom-production in sciatica and low-back pain. **J Neurol Neurosurg Psychiatry** 11:13-26, 1948
6. Fischgrund JS, Montgomery DM: Diagnosis and treatment of discogenic low back pain. **Orthop Rev** 22:311-318, 1993
7. Fraser RD, Sandhu A, Gogan WJ: Magnetic resonance imaging findings 10 years after treatment for lumbar disc herniation. **Spine** 20:710-714, 1995
8. Frymoyer JW, Cats-Baril WL: An overview of the incidences and costs of low back pain. **Orthop Clin North Am** 22:263-271, 1991
9. Gibson JN, Grant IC, Waddell G: The Cochrane review of surgery for lumbar disc prolapse and degenerative lumbar spondylosis. **Spine** 24:1820-1832, 1999
10. Gibson JNA, Grant IC, Waddell G: Surgery for lumbar disc prolapse (Cochrane Review), in **The Cochrane Library**, issue 2. Oxford: Update Software, 2002
11. Groen GJ, Baljet B, Drukker J: Nerves and nerve plexuses of the human vertebral column. **Am J Anat** 188:282-296, 1990
12. Keyserling WM: Strength testing as a method of evaluating ability to perform strenuous work, in Stanton-Hicks M, Boas RA (eds): **Chronic Low Back Pain**. New York: Raven Press, 1982, pp 149-156

13. Komori H, Shinomiya K, Nakai O, et al: The natural history of herniated nucleus pulposus with radiculopathy. **Spine 21**: 225–229, 1996
14. Kuslich SD, Ulstrom C, Michael CJ: The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. **Othop Clin North Am 22**:181–187, 1991
15. Matsubara Y, Kato F, Mimatsu K, et al: Serial changes on MRI in lumbar disc herniations treated conservatively. **Neuroradiology 37**:378–383, 1995
16. McCulloch JA: Focus issue on lumbar disc herniation: macro- and microdiscectomy. **Spine 21 (Suppl 24)**:45S–56S, 1996
17. Miller JA, Schmatz C, Schultz AB: Lumbar disc degeneration: correlation with age, sex, and spine level in 600 autopsy specimens. **Spine 13**:173–178, 1988
18. Nachemson AL: Advances in low-back pain. **Clin Orthop 200**: 266–278, 1985
19. Nachemson AL: Prevention of chronic back pain. The orthopaedic challenge for the 80's. **Bull Hosp Jt Dis Orthop Inst 44**:1–15, 1984
20. Paajanen H, Erkintalo M, Kuusela T, et al: Magnetic resonance study of disc degeneration in young low-back pain patients. **Spine 14**:982–985, 1989
21. Rydevik BL, Pedowitz RA, Hargens AR, et al: Effects of acute, graded compression on spinal nerve root function and structure. An experimental study of the pig cauda equina. **Spine 16**: 487–493, 1991
22. Saal JA: Natural history and nonoperative treatment of lumbar disc herniation. **Spine 21 (Suppl 24)**:2S–9S, 1996
23. Saal JA, Saal JS, Herzog RJ: The natural history of lumbar intervertebral disc extrusions treated nonoperatively. **Spine 15**: 683–686, 1990
24. van Dieen JH, Weinans H, Toussaint HM: Fractures of the lumbar vertebral endplate in the etiology of low back pain: a hypothesis on the causative role of spinal compression in aspecific low back pain. **Med Hypotheses 53**:246–252, 1999
25. Weber H: Lumbar disc herniation. A controlled, prospective study with ten years of observation. **Spine 8**:131–140, 1983
26. Weber H: The natural history of disc herniation and the influence of intervention. **Spine 19**:2233–2238, 1994
27. Weber H, Holme I, Amlie E: The natural course of acute sciatica with nerve root symptoms in a double-blind placebo-controlled trial evaluating piroxicam. **Spine 18**:1433–1438, 1993

Manuscript received July 8, 2002.

Accepted in final form July 23, 2002.

Address reprint requests to: Nevan G. Baldwin, M.D., Texas Tech University Medical Office Plaza, 3502 9th Street, Suite 240, Lubbock, Texas 79407. email: nevan3@cox.net.