The Value of Magnetic Resonance Imaging of the Lumbar Spine to Predict Low-Back Pain in Asymptomatic Subjects

A Seven-Year Follow-up Study

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Background: In 1989, a group of sixty-seven asymptomatic individuals with no history of back pain underwent magnetic resonance imaging of the lumbar spine. Twenty-one subjects (31%) had an identifiable abnormality of a disc or of the spinal canal. In the current study, we investigated whether the findings on the scans of the lumbar spine that had been made in 1989 predicted the development of low-back pain in these asymptomatic subjects.

Methods: A questionnaire concerning the development and duration of low-back pain over a seven-year period was sent to the sixty-seven asymptomatic individuals from the 1989 study. A total of fifty subjects completed and returned the questionnaire. A repeat magnetic resonance scan was made for thirty-one of these subjects. Two neuroradiologists and one orthopaedic spine surgeon interpreted the original and repeat scans in a blinded fashion, independent of clinical information. At each disc level, any radiographic abnormality, including bulging or degeneration of the disc, was identified. Radiographic progression was defined as increasing severity of an abnormality at a specific disc level or the involvement of additional levels.

Results: Of the fifty subjects who returned the questionnaire, twenty-nine (58%) had no back pain. Low-back pain developed in twenty-one subjects during the seven-year study period. The 1989 scans of these subjects demonstrated normal findings in twelve, a herniated disc in five, stenosis in three, and moderate disc degeneration in one. Eight individuals had radiating leg pain; four of them had had normal findings on the original scans, two had had spinal stenosis, one had had a disc protrusion, and one had had a disc extrusion. In general, repeat magnetic resonance imaging scans revealed a greater frequency of disc herniation, bulging, degeneration, and spinal stenosis than did the original scans.

Conclusions: The findings on magnetic resonance scans were not predictive of the development or duration of low-back pain. Individuals with the longest duration of low-back pain did not have the greatest degree of anatomical abnormality on the original, 1989 scans. Clinical correlation is essential to determine the importance of abnormalities on magnetic resonance images.
this prospective investigation was to determine the association between abnormal findings on magnetic resonance images of the lumbar spine and the development and duration of low-back pain in the cohort of sixty-seven asymptomatic individuals described by Boden et al. in their study published in 1990. The hypothesis of the present study was that abnormalities on the magnetic resonance images would not be predictive of the development of back or leg pain over a seven-year period.

**Materials and Methods**

In 1989, volunteers were recruited for the study through advertising in newspapers or by word-of-mouth. Review by the Institutional Review Board was not required by the sponsoring institution at that time. Respondents and their spouses were chosen to obtain a correct balance for gender in each age-range. For a volunteer to be included in the study, he or she had to have no history of pain in the back, sciatica, or neurogenic claudication. Any episode of nonradiating low-back pain that had lasted for more than twenty-four hours or had necessitated an absence from work was grounds for exclusion from the study. Volunteers were also excluded if they had sciatica (pain or sensory abnormalities in the buttocks or lower limbs) or if walking caused pain or sensory deficits distal to the knee.

Once the subject had been entered into the study in 1989, multiplanar magnetic resonance imaging was done from the first lumbar to the first sacral vertebra with a 1.5-tesla imaging system (Signa; General Electric, Milwaukee, Wisconsin). Sagittal and axial localizing series were performed. Technical details of the scans are described in our previous report.

In 1996, the entire cohort of sixty-seven asymptomatic individuals with scans from the previous study was contacted by letter. Written informed consent was obtained from all patients. The study was approved by the chairman of our Institutional Review Board. The letter requested completion of an eighteen-item questionnaire. The questionnaire asked for information concerning age, gender, episodes of low-back pain

<table>
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<th>TABLE I Definitions and Associated Scores of Spinal Findings</th>
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<tr>
<td><strong>Finding</strong></td>
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<tr>
<td>Herniated nucleus pulposus</td>
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<td>Normal</td>
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<td>Protrusion (nucleus pulposus contained in the annulus fibrosus but with contour abnormality)</td>
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<td>Extrusion (nucleus pulposus extending through the annulus fibrosus but still contiguous with the host nucleus)</td>
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<td>Free fragment (migration of herniated fragment away from the disc space)</td>
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<tr>
<td>Stenosis of the canal (nondiscogenic loss of normal epidural fat with compression of neural tissues within the canal)</td>
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<td>Normal</td>
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<tr>
<td>Mild (flattening of the ventral thecal sac)</td>
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<td>Moderate (triangularization of the spinal canal with loss of the posterior epidural fat pad)</td>
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<td>Severe (compression of the canal with loss of epidural fat in all planes)</td>
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<td>Disc bulge</td>
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<td>Normal</td>
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<td>Asymmetric</td>
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<td>Diffuse (nondiscogenic, nonosseous material extending beyond the normal disc space in a circumferential manner)</td>
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<td>Disc degeneration</td>
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<tr>
<td>Normal</td>
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<tr>
<td>Mild (slight dehydration of the disc on T2-weighted images)</td>
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<td>Moderate (disc dehydration and mild loss of disc height)</td>
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<td>Severe (total disc dehydration with nearly complete loss of disc height)</td>
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since 1989, duration of pain, absence from work, sensory abnormalities in the buttocks or lower extremities, abnormalities associated with walking, a validated pain diagram, visits to a health-care provider, specific diagnoses, tests performed, treatment received, treatment outcome, coexistent illnesses, surgical procedures, and concurrent medications. Eight individuals who did not wish to complete the long questionnaire were asked for information concerning the presence and duration of back pain and the location of pain in the lumbar area and/or either leg during the past seven years. Also included with the questionnaire was a request for a repeat magnetic resonance scan of the lumbar spine. Individuals who did not respond were contacted by telephone. A location service was used to find the volunteers who had moved in the seven-year period.

In 1996, the thirty-one individuals who agreed to participate had repeat multiplanar magnetic resonance imaging with use of a 1.5-tesla unit (Magnetom Vision; Siemens Medical Systems, Erlangen, Germany). Three sagittal image series (T1, proton density, and T2) were obtained with a 4-mm slice thickness from the superior end plate of the third lumbar vertebra to the superior end plate of the first sacral vertebra. Angled axial cuts of the third lumbar to the first sacral disc spaces were also performed. The image series were done without contrast enhancement. A spine surface coil (quadrature form) was used for signal reception.

The fifty 1989 magnetic resonance studies of the individuals who completed the questionnaire (or answered questions regarding back pain) were randomly mixed with the thirty-one magnetic resonance studies from 1996. The eighty-one scans were reviewed, in random sequence, by two board-certified neuroradiologists (C.P. and D.S.) and a board-certified orthopaedic spine surgeon (W.C.L.), who were blinded with regard to the clinical status of the subjects. A determination concerning the presence of a herniated nucleus pulposus, stenosis of the canal, bulging, and degeneration was made at each disc level. Each category was rated independently at each disc level. In addition to rating the severity of the abnormality, the evaluators rated their certainty about the diagnosis (definite, probable, or possible) (Table I).

Each disc level was graded on an ordinal scale, ranging from 0 (normal) to 3 points (free disc fragment, severe degeneration). At least two evaluators needed to agree on the presence of an abnormality (a score of ≥1 point) at a disc level in order for the level to receive a score above 0 points. For example, a grading of severe disc degeneration (a score of 3 points) by one evaluator was not adequate to categorize a level as abnormal unless at least one other evaluator gave the level a score of ≥1 point.

A change in the average score of ≥1 point was considered indicative of progressive disease, whereas a decrease of ≥1 point was indicative of improvement. Only the findings that the interpreters had labeled as definitely or probably abnormal were tabulated. Therefore, an abnormal ranking required two interpreters to give a score of ≥1 point with definite or probable certainty at the same disc level. Once a level was scored as abnormal in this manner, the ratings of all three evaluators were averaged.

Some individuals had more than one abnormality in the lumbar spine on the magnetic resonance scan. The rank order of disc and canal abnormalities used to place a subject in a radiographic category, from the most to the least severe, was disc herniation, stenosis of the canal, disc bulging, and disc degeneration. The degree of severity of the abnormality did not preempt this rank order. For example, an individual with moderate spinal stenosis (a score of 2 points), a diffuse disc bulge (a score of 2 points), and severe disc degeneration (a score of 3 points) was listed once in the spinal stenosis category for statistical analysis and was not included in the disc-bulge or disc-degeneration categories.

The clinical condition of the subjects (the presence of back or leg pain or sensory abnormalities, for example) in the seven-year study period was matched with the abnormalities on the magnetic resonance images made in 1989. For analysis of the correlation of the magnetic resonance abnormalities and back pain and for analysis of agreement among the evaluators of the magnetic resonance studies, only the more severe alterations (an extruded or free fragment for the herniated disc category, moderate or severe stenosis, diffuse bulge, and moderate or severe degeneration) were considered anatomically important abnormalities. Correlation was not measured for milder anatomical abnormalities, such as disc protrusion, asymmetric disc bulge, or mild disc degeneration.

Statistical Methods

Analysis of the correlation between the duration of pain (measured on an ordinal scale from 0 points for no pain to 5 points for pain of more than six weeks’ duration) and the magnetic resonance abnormality consisted of the Spearman rank-order correlation. The predictive correlative value of magnetic resonance abnormalities and back pain was tested with chi-square analysis with significance set at p < 0.05. The degree of progression of magnetic resonance abnormalities from 1989 to 1996 was evaluated with use of the Wilcoxon signed-rank test. The statistical method utilized for rating agreement between pairs of evaluators was a 2 × 2 table with a kappa coefficient. The three kappa coefficients were averaged. A kappa value of ≥0.5 indicated good agreement. The maximum rating of severity of any of the four anatomical changes at any level of the spine was the value utilized for analysis. This test was used for the fifty magnetic resonance scans made in 1989 and the thirty-one magnetic resonance scans made in 1996. Data analysis was performed with SAS software (version 6.12; SAS Institute, Cary, North Carolina).

Results

During the study period, two male subjects died and two others moved from the United States. Thirteen additional subjects did not reply to repeated telephone calls and written requests for clinical information. The average age of the fifty
subjects from the 1989 study who answered the survey (or questions regarding back pain) in 1996 was 43.6 years compared with 42.0 years for the total group of sixty-seven subjects from the original study. The average age of the cohort of thirty-one subjects who underwent a repeat magnetic resonance scan was fifty-two years (range, twenty-six to sixty-eight years).

Analysis of the fifty magnetic resonance scans made for the 1989 study revealed that thirty-two had normal findings and eighteen had abnormal findings (see Appendix). Over the seven-year study period, pain in the low back or leg developed in twenty-one individuals, including twelve (38%) of those with a normal scan, five of the six subjects with a herniated intervertebral disc at any level, three of the four subjects with spinal stenosis, none of the six subjects with disc bulging, and one of the two subjects with moderate disc degeneration. Of the twenty-one individuals who had pain, eight described discomfort radiating into the thigh and leg. The 1989 magnetic resonance scans of these eight subjects revealed an extruded disc between the fifth lumbar and the first sacral vertebrae in one, a protruded disc between the first and second lumbar vertebrae in one, and moderate stenosis of the canal between the fourth and fifth lumbar vertebrae in two. The remaining four subjects had a normal magnetic resonance scan or mild disc degeneration.

We analyzed the results for the thirty-one subjects who had a repeat magnetic resonance imaging study in 1996 (see Appendix). In the seven-year period, a herniated or protruded disc developed in ten of them, mild-to-severe stenosis developed in ten, a bulging disc developed in twenty, and moderate or severe disc degeneration developed in fourteen. Eight of the thirty-one subjects continued to have no magnetic resonance abnormality.

Five of the six subjects with a herniated intervertebral disc on the magnetic resonance scan made in 1989 had a repeat study. Disc protrusion or extrusion persisted at one level or more in all five subjects. Two disc extrusions resolved to a protruded state, and one totally resolved. Only one of the five subjects had radiating pain lasting longer than two weeks. During the seven-year study period, five new disc herniations developed in four subjects who had had normal findings on the magnetic resonance images made in 1989 and in one subject who had had evidence of spinal stenosis. Two of these subjects, including one with an extruded disc, had no back pain. Two individuals had pain for one week, and another had pain for six weeks.

In one of the four subjects in whom stenosis of the lumbar canal had been the sole abnormality in 1989, repeated scans showed that a protruded disc had developed at the level of mild stenosis. During the seven-year period, stenosis of the canal developed in nine additional individuals, four of whom had a concomitant herniated disc. Stenosis was mild to moderate in nine individuals, and one of the nine also had severe stenosis at another level but no pain. A single level was involved in six subjects, and three levels were involved in four. None of the subjects with only spinal stenosis on the scans made in 1996 had pain.

A disc bulge was seen on the 1996 scans of twenty (65%) of the thirty-one subjects and was the sole finding in eight subjects (26%). New abnormalities had developed in eight individuals during the study period. Three of these individuals with no other abnormality had back pain lasting six weeks or longer. A single disc level was involved in one, and two levels or more were involved in the remaining subjects. The twelve subjects with disc bulges noted in 1989 had progression of bulging, and there was involvement at additional interspaces in seven individuals.

Disc degeneration was noted in twenty-three of the thirty-one subjects who had scans made in 1996. Eighteen individuals had involvement at two or more levels.

The average severity of involvement increased significantly in all four magnetic resonance categories. Fifteen individuals with disc degeneration and fifteen with a bulging disc had the greatest increases (p < 0.0001). An increase of one grade or more was noted in five subjects with a herniated disc (p < 0.003). These anatomical alterations were identified by all three evaluators of the magnetic resonance images. The duration of pain in these subjects ranged from no pain to one week of pain. Eight subjects had an increase in spinal stenosis of one grade or more (p < 0.0002). The duration of pain in these individuals ranged from no pain to six weeks of pain. During the seven-year period, four subjects, ranging in age from thirty-nine to forty-seven years, had no progressive change in any category of abnormality of the intervertebral discs. Three additional individuals, who were forty-seven, fifty-two, and fifty-seven years old, had only mild degenerative changes at a single disc level. None of these seven individuals had low-back pain during the study period.

There was radiographic improvement of some of the intervertebral disc abnormalities; however, there was only one instance in which all three evaluators reached a consensus with regard to a one-grade improvement in the rating for disc bulging.

**Predictive Value of Magnetic Resonance Scans**

With regard to the ability of magnetic resonance scans to predict low-back pain, a positive trend was noted in each category but none were significant.

The relationship between low-back pain and the presence of magnetic resonance abnormalities on the 1996 scans was investigated. A significant correlation between the duration of low-back pain and the presence of a herniated nucleus pulposus (p = 0.01) or moderate degenerative disc changes (p = 0.04) was found. Although the presence of a herniated disc or degenerative disc disease on the original (1989) scan did not predict the development of back pain, there was a clinical correlation between back pain and the simultaneous existence of disc herniation and degeneration. The relative risk that low-back pain would develop in individuals with worsening abnormalities on magnetic resonance scans was 3.5.
Agreement Among Evaluations of Magnetic Resonance Scans

The three evaluators of the 1989 and 1996 magnetic resonance scans agreed most frequently on the anatomical changes associated with disc degeneration ($\kappa = 0.801$) and disc herniation ($\kappa = 0.633$) in 1989. The greatest degrees of disagreement, with kappa averages of <0.5, included stenosis of the canal in 1989 ($\kappa = 0.4341$) and 1996 ($\kappa = 0.448$), disc herniation in 1996 ($\kappa = 0.244$), and disc bulges in 1989 ($\kappa = 0.362$).

Clinical data gathered from the questionnaires revealed that four of the fifty subjects missed an average of twelve days (range, one to twenty-one days) of work. No patient had persistent pain at the time that the questionnaire was completed. A total of nine new specific diagnoses were made that four of the fifty subjects missed an average of twelve days (range, one to twenty-one days) of work. No patient had persistent pain at the time that the questionnaire was completed. A total of nine new specific diagnoses were made.

Discussion

Correlation between the symptoms and signs and the magnetic resonance findings is necessary to determine the clinical importance of anatomical abnormalities identified by this radiographic technique. Questions remain concerning whether radiographic findings can be used to predict the development of future low-back pain in individuals with anatomical abnormalities of the lumbar spine. A study by Wilberger and Pang suggested that asymptomatic individuals with disc herniations identified on spinal myelography may be at risk for low-back pain. They reported that symptoms of lumbosacral radiculopathy developed in 64% of 108 patients with asymptomatic lumbar disc herniations who were followed for three years.

The present study demonstrated the inability of magnetic resonance scans of the lumbar spine to predict the development of low-back pain over a seven-year period. Magnetic resonance scans confirm the presence of anatomical abnormalities that are suspected on the basis of the history and physical examination of a patient. Our study suggests that the anatomical findings most closely associated with clinical symptoms and duration of back pain are a herniated nucleus pulposus and moderate degenerative disc disease.

Low-back pain developed in a total of twenty-one of the fifty subjects over a seven-year period. The largest group of individuals in whom low-back pain developed during this period consisted of those who had had no specific magnetic resonance abnormalities in 1989. The predictive value of magnetic resonance imaging was diminished by this large group of subjects who had pain but no anatomical abnormalities on magnetic resonance imaging.

In five of the six individuals with an extruded or protruded disc on the 1989 magnetic resonance scan, pain developed in the back (three subjects) or in the leg (two subjects). A positive trend was noted between the presence of a herniated disc and the development of pain ($p = 0.2$), but it was not significant with the numbers evaluated. Individuals with a herniated or protruded disc appear to be at risk for the development of pain.

A clinical association was found between the development of back or leg pain and the presence of a herniated nucleus pulposus or disc degeneration. Four of the eight patients with leg pain had an extruded disc or moderate spinal stenosis on the 1989 scans. The leg pain corresponded with these magnetic resonance findings. However, one subject in whom an extruded disc developed and two subjects in whom moderate or severe lumbar stenosis developed during the study period remained pain-free.

With regard to the progression or regression of magnetic resonance abnormalities, repeat magnetic resonance scans demonstrated a general progression in all categories of spinal abnormalities. Of the five extruded discs, one resolved entirely, two regressed to a protruded status, and two remained extruded. Stenosis and degeneration did not resolve during the seven-year study period. Of the five individuals with a repeat scan who had had a diffuse bulge on the 1989 scan, one had severe stenosis when they were reexamined in 1996.

The present study has a number of limitations. Seventeen of the sixty-seven subjects did not complete a questionnaire (or answer questions regarding back pain). The loss of younger individuals in that group may have biased the data by decreasing the number of subjects with a herniated disc who had pain compared with those with a herniated disc who may have been asymptomatic. Herniated discs were identified in younger subjects in this study. The inclusion of these younger subjects who were lost from the 1989 study may have increased the number of herniated discs identified by magnetic resonance imaging in 1996. The predictive power of magnetic resonance imaging in the present study might have been increased if these younger individuals had remained in the study. Another possibility is that younger individuals did well and chose not to participate because of an absence of symptoms. Another limitation of the study is the fact that patients were asked questions concerning back pain after a seven-year hiatus. The recall of the patients with regard to the duration or location of pain may have been faulty. Also, two different magnetic resonance machines were used for this study. Magnetic resonance scans from the same machine would have been preferable; however, both machines contained high-field-strength magnets. Finally, the individuals who evaluated the original (1989) magnetic resonance scans were not the same ones who interpreted the 1989 and 1996 scans in the present study.

In conclusion, magnetic resonance scans of the lumbar spine did not predict the development or duration of low-back pain. Individuals with leg pain frequently had a spinal lesion in a location on a magnetic resonance scan that corresponded to the clinical symptoms and signs. The findings discovered by magnetic resonance technology can only confirm the clinical suspicions of the clinician. Therapeutic or prophylactic interventions should not be based solely upon magnetic resonance abnormalities in the absence of clinical indicators.
Appendix

A table showing the age and gender of the patients, the findings on the 1989 and 1996 magnetic resonance images, the location and duration of pain, and the location of the abnormalities is available with the electronic versions of this article, on our web site at www.jbjs.org (go to the article citation and click on “Supplementary Material”) and on our CD-ROM (call 781-449-9780, ext. 140, to order).

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