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Chiropractic Rehabilitation of a Patient with S1 Radiculopathy Associated with a Large Lumbar Disk Herniation

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ABSTRACT

Objective: To describe the nonsurgical treatment of acute S1 radiculopathy from a large ($12 \times 12 \times 13$ mm) L5-S1 disk herniation.

Clinical Features: A 31-year-old man presented with severe lower back pain and pain, paresthesia, and plantar flexion weakness of the left leg. His symptoms began 5 days before the initial visit and progressed despite nonsteroidal anti-inflammatory drugs and analgesic medication. An absent left Achilles reflex, left S1 dermatome hypesthesia, and left gastrocnemius/soleus weakness was noted. Magnetic resonance imaging demonstrated a large L5-S1 disk herniation.

Intervention and Outcome: Initial treatment of this patient included McKenzie protocol press-ups to reduce and centralize symptoms, nonloading exercise for cardiovascular fitness, and lower leg isotonic exercises to prevent atrophy. Counseling was provided to reduce abnormal illness behavior risk. Later, flexion distraction and side-posture manipulation were provided to improve joint function. Sensory motor training, trunk stabilization exercises, and trigger point therapy were also used. He returned to modified work 27 days after symptom onset. A follow-up, comparative magnetic resonance imaging (MRI) study was unchanged. He was discharged as asymptomatic (zero rating on both the Oswestry and numerical pain scales) after 50 days and 20 visits, although the left S1 reflex remained absent. Reassessment 169 days later revealed neither significant symptoms nor lifestyle restrictions.

Conclusion: This case demonstrates the potential benefit of a chiropractic rehabilitation strategy by use of multimodal therapy for lumbar radiculopathy associated with disk herniation. (J Manipulative Physiol Ther 1999;22:38-44)

Key Indexing Terms: Chiropractic; Intervertebral Disk; Rehabilitation; Lumbar Vertebrae

INTRODUCTION

The therapeutic value of various nonsurgical treatments for radiculopathy caused by lumbar disk herniation has a great deal of support.[1-3] Treatments have included a combination of medications and active, or exercise, protocols. However, little has been published regarding the benefit of a multimodal protocol of various manual and active care techniques[4] compared with individual manual techniques, such as the McKenzie protocol,[5,6] manipulation,[7,8] or flexion/distraction mobilization therapies.[9,10] Unfortunately, the fact remains that the diagnosis of radiculopathy associated with disk herniation generally indicates the patient will likely have a significant episode of pain; lifestyle restriction; disability; financial hardship from work loss, medical costs, or both; and the psychosocial effects of these stressors. The risk of decompressive surgical intervention further complicates this difficult picture. Therefore new strategies and techniques are necessary to more effectively reduce the patient's pain and associated costs of this condition.

Chiropractic rehabilitation seeks to restore locomotor system function by use of various manual procedures, active procedures, and psychosocial counseling while documenting outcomes with accepted measures. The emphasis on this model is on normalizing the pathophysiologic condition rather than the pathoanatomic condition. The purpose of this article is to provide a case study that uses such a chiropractic rehabilitation method, integrating multimodal techniques used in the management of a patient with acute radiculopathy associated with a very large lumbar disk herniation.

CASE REPORT

A local physician referred a 31-year-old man, a manager for a cargo-shipping carrier, for chiropractic care. The referring diagnosis was lumbar left sciatica. Five days earlier the patient had attempted to quickly rise from a prolonged seated position at his desk at home and rotated his torso to the right to reach for a book. He recalled feeling immediate severe left-sided lower back pain. Over the ensuing several hours, the pain with paresthesia extended down the outside of his leg to the left lateral side of the foot to the fourth and fifth digits. Analgesic and nonsteroidal anti-inflammatory medication provided only minimal temporary relief. His condition rapidly worsened and prevented him from working.

The patient, who was 5 feet, 11 inches tall and weighed 200 pounds, complained of severe and unrelenting low back pain, which was greater on the left side than the right side, and left leg pain and paresthesia, which extended to the left

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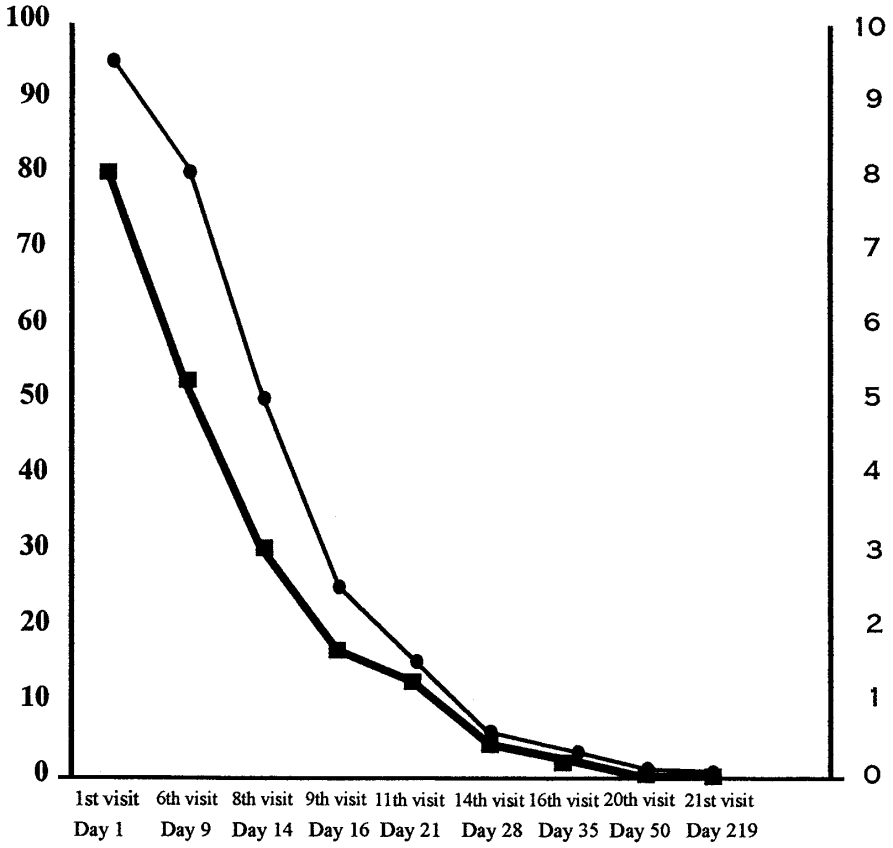

Fig 1. *Graph of measured outcomes, using the revised Oswestry disability questionnaire and the numerical pain scale, during the course of treatment. Thick line with small squares represents the Oswestry rating and correlates with the 101-point scale on the left x-axis. Thin line with small triangles represents the numerical pain rating and correlates with the 11-point scale on the right x-axis. This graph demonstrates a consistent, measured improvement of a significant left S1 radiculopathy over a relatively short clinical period.*

lateral 2 toes. He was unable to stand on his left toes. He had no bowel or bladder dysfunction. His symptoms were aggravated by coughing, sneezing, straining, bending, sitting, standing up, and sitting down and were not relieved by rest. He appeared to be exhausted and anxious, admitting to sleep deprivation as a result of his symptoms.

Physical risk factors for chronicity were assessed. Aside from his current condition, the patient believed that he was in generally good health.[11] He admitted to being deconditioned. His job did not require heavy physical demands and was sedentary in nature. He smoked 1 pack of cigarettes per day. He had no history of leg pain below the knee or prior prolonged disability.[11] He had had more than 4 prior episodes of low back pain, which is considered a risk factor for chronicity in the Mercy Guidelines.[12]

Psychosocial risk factors for chronicity such as abnormal illness behavior were assessed. The patient was happily married, and his wife was in the last trimester of pregnancy with their second child. The patient was gainfully employed in a job that he found rewarding and was financially stable. He denied a history of chemical abuse. He was not involved in, nor did he intend to initiate, a litigious action associated with his back injury.[11]

The patient rated the intensity of his pain as 9 to 10 on a 10-point numeric pain scale. Disability, measured with the 101-point Revised Oswestry disability questionnaire, was rated at 80 of 100 or 80% (Fig 1).[13-17]

The patient's posture was antalgic to the right, and he limped on his left leg. He appeared to be in significant distress as a result of low back and left leg pain. Dejerine's triad was present, provoking left-sided low back and leg pain extending to the left foot. Lumbar spine mobility was reduced 100% in flexion and right lateral flexion and 75% in left lateral flexion and extension. All upright movements increased his lower back pain and left leg pain, extending into the left lateral 2 toes. The straight leg–raise test produced left leg pain to the lateral foot at 30 degrees. The well leg–raise test produced lower back pain and left leg pain to the lateral foot at 40 degrees. Seated straight–leg raise provoked left-sided low back pain that extended to the lateral toes at 30 degrees of knee extension. While in the prone position, the patient was asked to perform press-ups with his trunk in 3 different positions (pelvic neutral, pelvic shift right, pelvic shift left). The left and neutral pelvic positions aggravated his leg symptoms. The right pelvic shift position brought a slight reduction in the pain and paresthesia in the

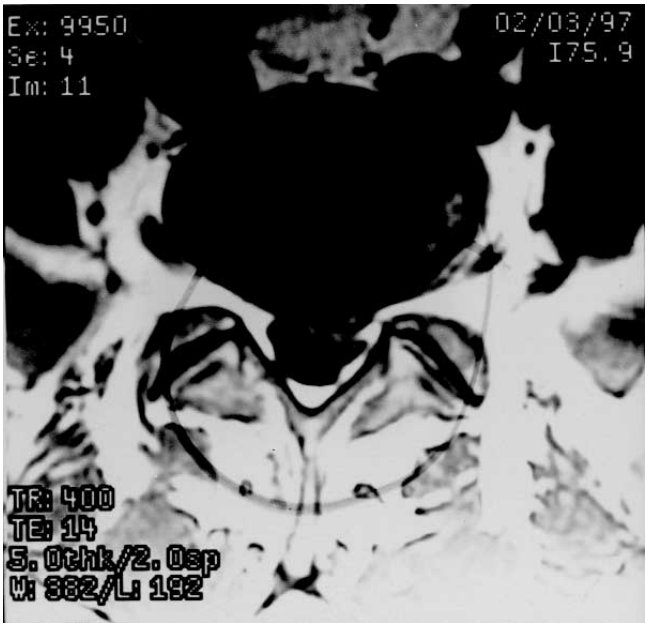

Fig 2. *Initial MRI study. T1-weighted axial image at L5-S1 level reveals large central disk herniation (measured at 13 × 12 × 12 mm) impinging the dural sac centrally and somewhat to the left. The herniation in this view appears to be larger in diameter than the dural sac. The neural canal is relatively large and accommodates both the herniation and sac. The initial radiologist circled the herniation of this key frame in the scan as an aid to the referring physician.*

Fig 3. *Initial MRI study. T1-weighted sagittal image reveals large L5-S1 disk herniation extending far into the neural canal. The initial radiologist also circled this key image.*

left lateral foot while slightly increasing his low back pain. Prone low back flexion-distraction aggravated his left leg symptoms. He was unable to tolerate lying on either side because of increased left leg pain.

The left Achilles reflex was absent, and the right reflex was 2+. The patient was hypoesthetic to pinprick testing over the left S1 dermatome. The patient was unable to toe walk on the left foot. Plantar flexion strength was rated at 3 of 5 on the left and 5 of 5 on the right.

Radiographs of the lumbar spine revealed mild narrowing at the L5-S1 disk space. Mild end plate eburnation was present at that level. Except for the patient's right laterally flexed position, this standing study was otherwise normal.

The initial diagnosis was suspected L5-S1 disk herniation with attendant left S1 radiculopathy complicated by a posterior derangement syndrome, category 6 (McKenzie classification).[6] The derangement syndrome is one of the 3 McKenzie syndrome patterns characterized by a derangement of intradiscal material or other substance. Posterior derangement suggests that this material has migrated posteriorly. The derangement, category 6 in this case, was defined by an antalgic posture, and asymmetric (left greater than right) low back symptoms extending to the left buttock and leg below the knee.

The initial clinical plan was to use manual procedures to reduce and centralize the patient's symptoms from the left leg toward the low back, order an MRI of the lumbar spine to confirm probable L5-S1 disk herniation, recommend a neurologic consultation, and perform in-office and home exercises to avoid deconditioning. His diagnosed condition was explained to him in detail. He was counseled regarding the prognosis with conservative care, the success of which would depend greatly on his level of compliance. McKenzie press-up exercises were prescribed at 10 repetitions every 1 to 2 hours, with the appropriate position (pelvis-neutral or shifted position), whichever best centralized his left leg symptoms, being re-established at the beginning of each set of exercises.[6,18] He was instructed to maintain a lordotic lumbar posture and use a lumbar roll while seated or supine, although lying prone while propped up on his elbows ("sphinx position") was preferable. He was also instructed to apply cold packs to his low back for 10 to 15 minutes every 1 to 2 hours.

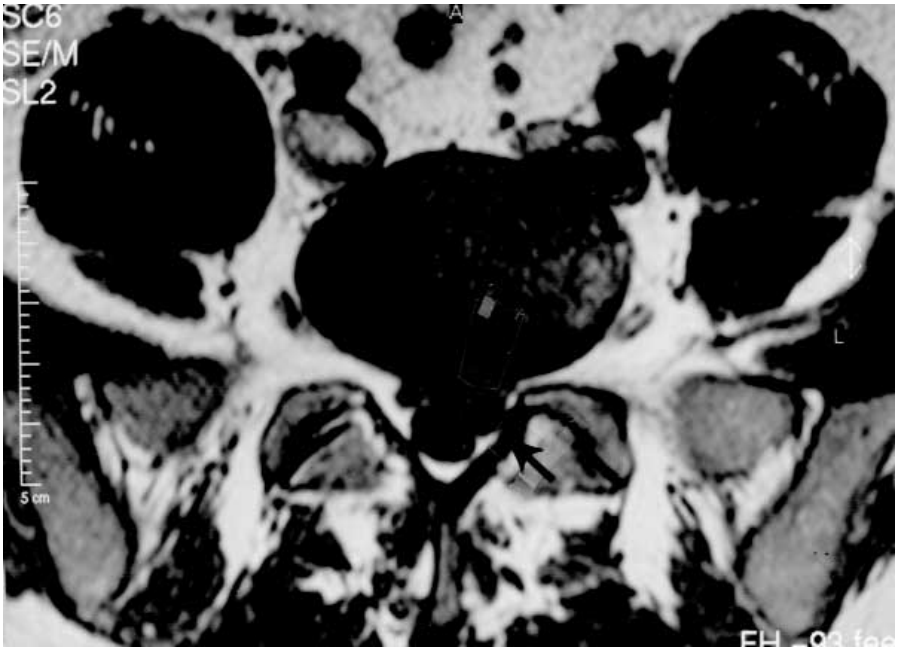

Fig 4. *Follow-up MRI comparison study. This study was performed on a scanner superior to the first and read by a different radiologist. The T1-weighted axial image reveals large central disk herniation unchanged in size or location from the initial study (see Fig 1). The resolution of this film better delineates the herniation (arrows) from the dural sac than the initial study. White measurement scale on the left side, graded in 2-mm segments, confirms the initial radiologist's estimated size of the herniation.*

The patient returned the following day noting a slight decrease in his left foot pain and paresthesia, unchanged weakness in his left foot, and unchanged severity of low back pain. His anxious wife, quite concerned for her husband, accompanied him. His condition was again explained in great detail and all questions were answered. They were then counseled on the prognosis and importance of a positive attitude, proper rest, maintaining conditioning, and working as a team. Treatment consisted of press-up exercises in a right pelvic–shifted position, which again reduced his leg left symptoms. After treatment, the couple was instructed to have him perform 10 isotonic gastrocnemius/soleus-strengthening exercises ("heel raises") concentrically and eccentrically in a supine position with lumbar support while his wife provided resistance. They were instructed to perform these exercises every 1 to 2 hours. This strategy proved to be effective in reducing both of their anxiety levels by recruiting them as active participants in the treatment. He was also instructed to continue with his home press-up exercises and cold packs.

The patient returned on the third visit with decreased antalgia, noting a 50% decrease in left leg and foot paresthesia. He appeared to be well rested and far less anxious than on previous visits. His low back pain was essentially unchanged. Increased mobility was noted in low back extension and straight-leg raising. Therefore his home exercise routine of press-up repetitions and heel raises was increased to 15 repetitions each. He was then asked to pedal a stationary bicycle from behind while lying in a supine position with the lumbar support placed under his low back. This non-weight-bearing activity was quite tolerable, so he was allowed to continue for 5 minutes at resistance level 4 for a total distance of 1.6 miles.

A neurologist subsequently assessed the patient. He concurred with the initial diagnosis and recommended continued chiropractic care in light of the patient's improvement to date. He also supported the initial recommendation of a lumbar MRI scan. Finally, he recommended a neurosurgical consultation because of the significant left S1 nerve root compromise and the likelihood of eventual surgical intervention.

On the fourth visit, the patient noted a further decrease in left leg paresthesia. In addition to the press-up exercises, flexion-distraction was briefly attempted by use of the protocol described by Cox.[19] This procedure neither increased his low back pain nor increased, or peripheralized, left leg symptoms. Therefore flexion-distraction was added to the in-office treatment protocol in an effort to further restore low back flexion as soon as tolerably permitted.

The patient underwent a lumbar MRI the following week. The scan confirmed a large central disk herniation at L5-S1, measuring $13 \times 12 \times 12$ mm, impinging on the dural sac centrally and somewhat to the left (Figs 2 and 3). Fortunately, the scan also revealed a sufficiently large neural canal at that level, which appeared to accommodate both the herniation and dural sac.

On the sixth visit, the patient noted further improvement in his condition, particularly his left leg. An updated Oswestry questionnaire was scored as 52%. His pain was rated as an 8 of 10 because of his low back. His gait was less painful, and his lumbar range of motion was slightly improved. His attitude was positive.

Over the next week, he noted a gradual decrease in distal left leg paresthesia and pain, along with decreasing low back pain. Hypomobile dysfunction of the left proximal tibiofibular junction was present on palpatory joint springing. Multiple active trigger points were found in the left gastrocnemius/soleus muscle group.[20] Postisometric relaxation procedures to these muscles and mobilization of the left fibular head significantly reduced the remaining calf and foot pain and paresthesia.[21] In-office supine bicycling was increased in both time (12 minutes) and resistance level (level 5). Cold pack applications were tapered off.

On his ninth visit the revised Oswestry score was 16%, indicating significant improvement. He stated that approximately 75% of the time he felt no pain in his back or leg. The rating of pain severity was now 2½. Left S1 motor function had increased to 4 of 5, and he noted increased sensitivity to pinprick. Because of the patient's decreased symptoms, his exercise program was increased to include walking.

The patient was compliant with the prescribed exercise regimen and continued to improve. He returned to work, with restrictions, after his 11th visit, 3 weeks after the initiation of treatment. In light of his improvement, the consulting neurosurgeon continued conservative care.

On the 13th visit, the patient was finally able to rise up onto his left toes again without the use of his right foot. He noted occasional tingling in the left lateral foot and some recurrent low back pain. Straight-leg raising on the left had increased to 80 degrees. For the first time, the patient was able to perform push-ups and flexion/distraction mobilization, and gentle manipulation was used on the left sacroiliac and L5-S1 joints with the patient lying on his right side. He was placed on a uniplanar rocker board for sensory motor training and increased activation of postural stabilizing musculature.[22,23] Standing heel lift exercises were added to his home regimen.

On his 15th visit, the patient was instructed to perform tolerable pelvic tilt exercises after a negative Valsalva maneuver. Abdominal hollowing and bracing exercises were also added because of weakness and incoordination of the abdominal musculature.[24] Sensory motor training was increased with the inclusion of balance sandal walking.[22,23] He was returned to unrestricted work status.

At his final treatment (the 20th visit and 50th day of chiropractic care) the patient noted only occasional minor low back stiffness. The revised Oswestry score was now 0% and the pain was also 0 of 10. On examination, S1 motor strength was now 5 of 5 bilaterally. The left Achilles reflex remained absent. However his gait was normal and lumbar range of motion was full and painless. He was instructed to continue with his home exercise program indefinitely to maintain conditioning. This regimen consisted of stretching and lumbopelvic stabilization exercises in addition to walking. A follow-up MRI was performed 1 month later for academic purposes and revealed that the L5-S1 disk herniation was unchanged in size or position (see Fig 4).

On follow-up, almost 6 months after the final treatment, the patient related that he occasionally experienced minor localized low back pain on waking, which resolved after taking a hot shower. He continued to perform his home exercises, admittedly with less consistency as time passed. He noted that he had a rather significant fall 2 to 3 months earlier, which caused moderate local left-sided low back pain for 1 or 2 days before resolving. He denied any left leg pain or paresthesia since his last visit. The revised Oswestry score remained at 0% and his pain also remained at 0 of 10. His clinical findings remained unchanged from his prior visit, with the notable exception of an improvement in his left Achilles reflex to 1+.

DISCUSSION

This case demonstrates the successful use of the chiropractic rehabilitation model for acute lumbar herniation with associated S1 radiculopathy. The key strategy was based on McKenzie's centralization principle; that is, procedures, movements, or positions that reduce distal radicular symptoms from their periphery and shift the remaining symptoms toward the midline torso would be of benefit and therefore should be continued. As such, centralization of symptoms while performing press-ups with a right pelvic shift on this man's initial visit provided a clinical "glimmer of hope."

Other factors may have improved this patient's chance of recovery. The risk factors for chronicity were relatively few. I suspect that the large, accommodating neural canal in the lumbosacral spine may have been helpful. Finally, the opportunity for early intervention, 5 days after trauma, allowed me to address such issues such as abnormal illness behavior and deconditioning syndrome from a preventive standpoint.

As the patient improved, other manual techniques, reported to be of benefit in similar case studies, were used. Flexion distraction and side-posture manipulation previously aggravated his leg symptoms on the first visit and were therefore not initially used. These procedures were later incorporated into the treatment plan when found to be "nonperipheralizing" and quite tolerable. Because the rehabilitation approach focuses on function, the strategy for inclusion of these techniques was that each might uniquely promote improved joint function. For example, press-ups may assist in increasing extension function, whereas flexion distraction may assist in improving joint flexion function while tractioning the joint. Similarly, thrust manipulation may assist in increasing coupled spinal rotation and lateral flexion.

As the patient's symptoms receded, a change in the emphasis from passive care to active care occurred. As outlined by Murphy,[25] this strategic continuum allows for a smooth transition back to the normalized function of the locomotor system and to unrestricted activities of daily living. The suc-

cess of this strategy is suggested by the consistent improvement in the outcome measures (see Fig 1).

One question raised by this case is what exactly was the generator of the radicular symptoms? Was it chemical radiculitis, mechanical compression, a combination of both, or neither? If the radicular symptoms were chemical in nature, it would seem logical that chemical anti-inflammatory countermeasures such as the trial course of nonsteroidal anti-inflammatory drugs would have been effective instead of the manual, or mechanical, means that seemed to be of benefit. Conversely, if the cause of the radicular symptoms was mechanical in nature, why was the follow-up MRI unchanged? Unfortunately, the pictures visualized in the MRI tell little about the truly dynamic interrelationship between the herniated disk, thecal contents, and surrounding tissues. Perhaps a momentary reduction of the disk on the theca occurred, allowing for a reduction in the dysesthesia from nerve decompression, nociceptive irritation of the dura, or both. Perhaps the treatments allowed a normalization of the vasa nervorum, which had been compromised when the herniation occurred and led to a localized ischemic acidosis of the theca and contents. Perhaps key changes occurred in the mobility of the theca and nerve root, such as the release of an early adhesion tethering the bundle to the herniation.[26-28] Finally, a reduction of compression to the dorsal root ganglia may have occurred, resulting in the reduced levels of such potential nociceptive neuropeptides as P substance or calcitonin gene–related peptide.[29]

Clearly, the puzzle regarding the cause(s) and resolution of radicular symptoms associated with disk herniation is not yet complete. As such, we can only speculate as to what led to this patient's centralization of symptoms. We must patiently accept the likelihood that more questions than answers will be brought forward in the short term. More research on this complex subject is indeed necessary for long-term answers.

CONCLUSION

This case demonstrates a patient with an acute severe lumbar radiculopathy associated with a large disk herniation, who had a successful and timely outcome after receiving multimodal treatment of mobilization, manipulation, counseling/education, muscle relaxation techniques, proprioceptive training, trunk stabilization, and conditioning exercises. This case further demonstrates that significant locomotor system pathophysiologic conditions can be reversed while an associated significant pathoanatomic condition remains unchanged. The absence of a recurrence of radicular symptoms or significant low back pain in this case, combined with an increase in the left S1 reflex over the ensuing months without treatment, suggests a gradual stabilization of locomotor system function. In light of the relatively minimal risk of harm and significant potential benefit, further research to better understand and use chiropractic rehabilitation strategies and interventions for radiculopathies and associated disk herniations appears to be warranted.

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