Unusual Case of Secondary Scoliosis in a 20-Year-Old Man

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Secondary, or sciatic, scoliosis is a reactive spinal deformity caused by an underlying pain source. Sciatic scoliosis is 1 of 3 scoliosis subtypes first described in 1980 by McPhee and O'Brien.[1] The other 2 subtypes are idiopathic scoliosis and structural defect. In most cases, children and adolescents presenting with idiopathic scoliotic curves have no pain.[2] In contrast, secondary scoliosis usually has a pain component and can occur with disc herniation, spondylolisthesis, and osteoid osteoma. Secondary scoliosis presents as a continuum of physical and radiographic findings. Patients affected by secondary scoliosis are often pediatric patients and have a clearly demonstrable painful lesion. In some cases, however, the pain source is unclear. This de novo spinal deformity begins as a nonstructural type of scoliosis secondary to a painful focus, which is readily reversible with treatment of the painful lesion. Residual structural scoliosis curves may occur, particularly when there is a long delay between diagnosis and treatment.

Signs and symptoms of disc herniation in adolescents and children may develop slowly and insidiously. The clinical presentation differs from the typical picture in the adult population, but the transition point is not precise from adolescent to adult. Adults may bend, list, or tilt in response to pain but seldom have nonstructural scoliosis.

In this report, we present a case that reinforces the importance of serial examination and follow-up even in the absence of neurologic findings or an overt pain source. A review of the literature on epidemiology, pathophysiology, and management is included.

Case Report

A 20-year-old man presented with a 3-year history of back pain thought to be caused when he slid into base (playing baseball) and jammed his left leg. The patient was 6 feet tall, weighed 225 pounds, and was a nonsmoker in overall excellent health except for exertional asthma. Prior surgical history included arthroscopic left knee surgery, inguinal hernia repair, and tonsillectomy. The mechanism of the initial injury was thought to be either a low back sprain or an acute stress fracture of the pars interarticularis that produced symptoms of back and buttock pain, but the pain subsided with physical therapy, before bone scanning was required. After 12 months, the left-side low back pain returned. The pain was aggravated with activities and diminished with rest. The patient initially sought chiropractic care, during which symptoms seemed to worsen.

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The patient eventually returned to our clinic, 3 years after injury. He reported back pain and left buttock pain exacerbated with exercise, standing, walking, and spine extension. The pain was diminished with rest or leg flexion. He was able to heel- and toe-walk without weakness, but flattening of the lumbar lordosis with spasm and listing to the right was observed. He was unable to come to a fully erect position without pain. He had some hamstring spasm on the left, but the neurologic examination was otherwise normal. Hip examination was unremarkable, except for a pelvic obliquity, where the right hemipelvis was slightly higher. The Lasègue test was negative bilaterally. Bowel and bladder functions were normal.

On review of a 2-year-old magnetic resonance imaging (MRI) scan ordered by another physician, a right L5–S1 disc protrusion without nerve root deviation was evident but lacked clinical correlation at the time. No other findings were appreciated on prior imaging, and thus a stress fracture of the pars interarticularis was thought to be less likely. New standing anteroposterior and lateral plain radiographs showed loss of lumbar lordosis. Dynamic radiographs showed no abnormal motion in flexion or extension. No spondylolisthesis or spondylolysis was appreciated. The patient's back showed an atypical lordoscoliosis, with the Cobb angle measuring 15° from T10 to T12. There was more tilting than rotary deformity, as evidenced by minimal pedicle asymmetries. A new MRI scan was positive

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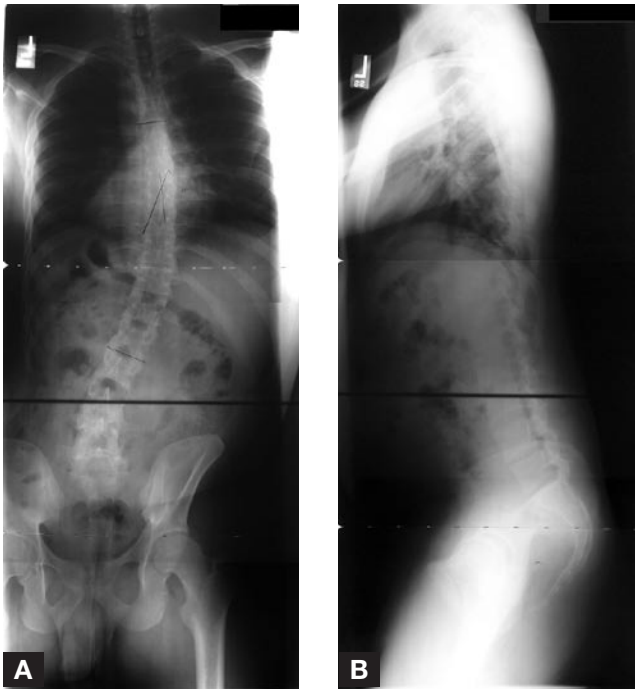
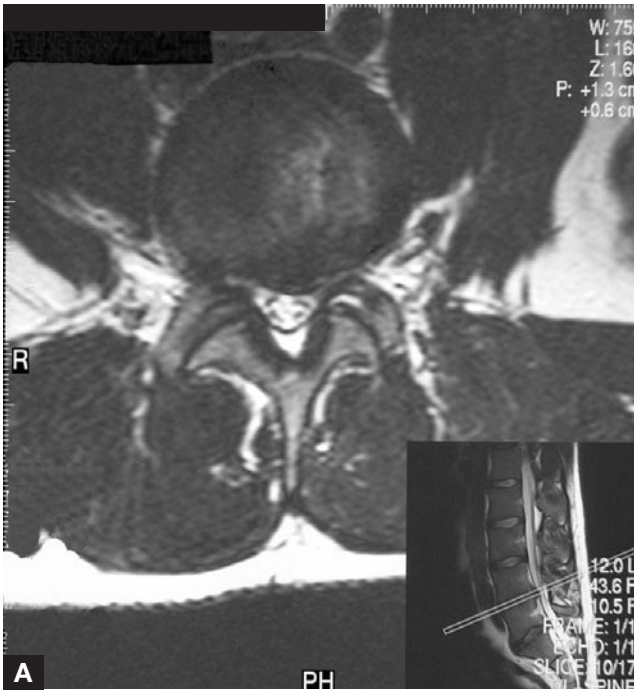


Figure 1. Preoperative radiographs: (A) Anteroposterior view shows thoracolumbar scoliosis with convexity to right measuring 30°; (B) lateral view shows decreased lumbar spinal lordosis with a forward tilt of torso.



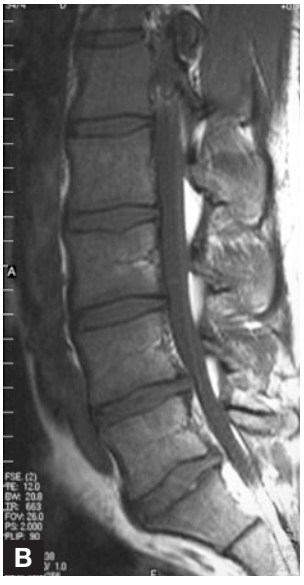


Figure 2. Preoperative magnetic resonance imaging: (A) Axial scan shows left-side paracentral L4–L5 disc herniation where patient developed left L5 radiculopathy; (B) sagittal scan shows disc herniation at L4–L5 level consistent with contained disc displacement.

for disc displacement at L4–L5 and L5–S1, left more than right. Without prior clinical correlation with the initial MRI scan, we thought the left-side paracentral disc protrusion at L4–L5 in the preforaminal region was likely causing the new symptomatic progression, with deformity secondary. Bone scan results were normal. A series of 3 epidural steroids was prescribed along with physical therapy in our initial, conservative approach.

Three weeks after the epidural steroid injection, the patient noted some relief of symptoms. Examination of the neurologic system of the lower extremities showed no evidence of hamstring tightness or straight leg raising sign. No atrophy differentials were noted, and motor strength at the hip, knee, foot, and ankle was all 5+/5+. Deep tendon reflexes at the knee and ankle were 1-2+/4+ with no focal lesions, and the sensory examination was unremarkable.

By radiographic analysis 7 months later, the patient's somewhat atypical thoracolumbar lordoscoliosis curve, from T8 to L2, had increased from 15° to 30° (Figure 1A). Posture was worsening, with increasing forward and left lateral tilt. Lateral radiographs showed decreased lumbar lordosis with a forward tilt of the torso (Figure 1B).

Approximately 3 months later, MRI and computed tomography (CT) myelogram results, from an institution where the patient sought a second opinion, supported the clinical suspicion of reactive scoliosis, but the second opinion was unable to identify the pain source. The only new finding to be compared with older MRI findings was incomplete filling of the left L5 nerve root sleeve. There was no extremity pain, sciatica, or neurologic deficits at this point to precisely correlate with this new neurodiagnostic finding. Our conservative management continued.

Twenty months after our initial evaluation and almost 5 years after injury, the patient returned to our clinic with a classic L5 radiculopathy, which was confirmed with electromyography. At this point, examination revealed 3+/5+ weakness in the left extensor hallucis longus and 4+/5+ weakness in the tibialis anterior. In addition, the patient had a left positive Lasègue sign. MRI showed a left-side L4–L5 paracentral disc herniation (Figures 2A, 2B). The diagnosis of left L5 radiculopathy caused by left L4–L5 disc with reactive scoliosis was confirmed. Unfortunately, the deformity now appeared to have structural patterns with vertebral rotation. Surgical intervention was recommended, as epidural steroids and chiropractic, pharmacologic, and physical therapies had all been unsuccessful.

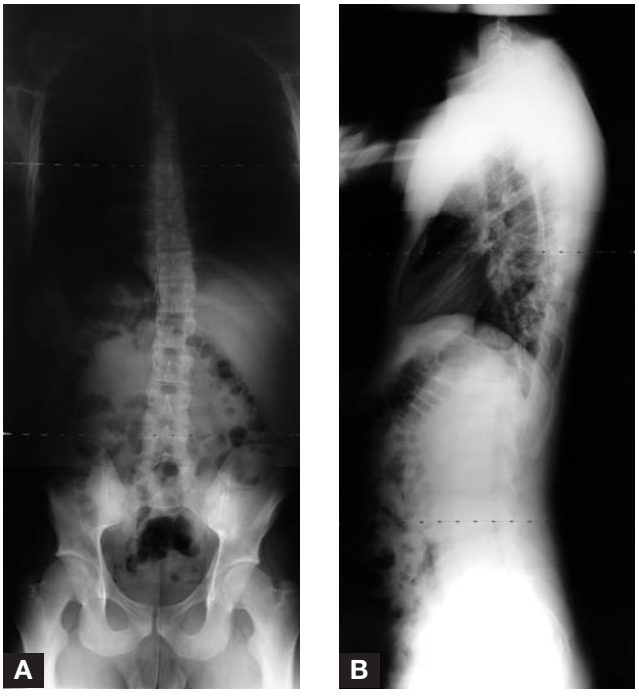


Figure 3. One-year postoperative radiographs: (A) Antero-posterior view shows improvement in thoracolumbar scoliosis (Cobb angle, 17°) with notable improvement in balance; (B) lateral view shows improved lumbar lordosis, absence of forward tilt, and more normal sagittal balance.

Five years after initial injury, the patient underwent a left L4–L5 microlumbar hemilaminotomy with L5 nerve root decompression and discectomy. At this level, complete laminectomy decompression was not necessary. The L4–L5 disc herniation extruding laterally to midline, identified with the new preoperative neurodiagnostic imaging, was confirmed during surgery. The procedure was completed without complications, and the patient noted immediate relief of the leg pain. Accordingly, his posture began improving within 1 to 2 weeks.

Two years after surgery, the patient remained pain-free, and his left L5 radiculopathy was completely resolved. He had a negative left tension sign and 5+/5+ strength of the left extensor hallucis longus and tibialis anterior. He still had mild residual structural scoliosis resulting in a short leg on the right. He had undergone further physical therapy, including a program of stretching and strengthening, but wore a 3/8-in heel lift for comfort. Scoliosis radiographs showed a Cobb angle of only 17° convex to the right between T4 and L2—an improvement from 30° before surgery (Figure 3A). Lateral plain radiographs showed improved lumbar lordosis and an absence of forward tilt (Figure 3B). Overall, our young patient was markedly improved functionally and had returned to normal activities. He continued to come in for monitoring of scoliosis changes; we anticipated stabilization but not improvement in the residual structural scoliosis.

Discussion

Sciatic scoliotic list is a nonstructural scoliosis secondary to nerve root irritation.[3] Disc protrusion after herniation often leads to neural entrapment. Hirayama and colleagues[4] proposed that the spinal reflex caused by painful inputs from a compressed nerve root induces the sciatic scoliosis. Unilateral spasm of the back muscles results in the postural changes.[4] Thus, removing the offending painful stimulus should improve the sciatic scoliosis.

In our patient's case, lumbar disc herniation resulted in a relatively acute onset of secondary scoliosis with progression and subsequent structural features. The diagnosis was not readily apparent but developed slowly, over a period of years. Lumbar disc herniation is uncommon in the pediatric and adolescent population, and the true incidence is not known.[5,6] An estimated 1% of patients operated on for disc herniation are between the ages of 10 and 20.[6,7] In our patient, we observed antecedent trauma, which is reported to be a common factor in 30% to 60% of cases.[5-9] The incidence of structural changes resulting from disc herniation in this population is reported to be 20% to 24%.[6,9]

The direction of sciatic scoliotic list has been suggested to provide information about the original site of disc protrusion. Studies performed by Matsui and colleagues[10] supported an earlier hypothesis by Finneson[11] that, when disc herniation is lateral to the nerve root, convexity is usually toward the side of disc herniation to decrease nerve root compression. However, the location of the curve did not predict the correct level of pain or herniation in our patient. As was not the case in the study by Matsui and colleagues, our patient listed to the side of the sciatica, producing concavity toward the side of disc protrusion. Suk and colleagues[12] indicated that the direction of sciatic scoliotic list was related to the side of disc herniation but not to the location of nerve root compression (lateral, medial, etc). We agree with Matsui and colleagues' conclusion that the list of sciatic scoliosis is only suggestive and is not a predictive factor of the anatomical location of disc herniation.

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Treatment of the deformity depends on correcting the underlying pain source—something that was not clear in our patient's case for an extended period, emphasizing the need to follow such cases closely. Surgical intervention in managing reactive scoliosis in the adolescent and pediatric population must be directed at the pain generator, not at

the deformity per se. In the absence of neurologic deficit, a conservative approach may initially be undertaken safely. Conservative measures, including physical therapy, pharmacologic prescription, chiropractic manipulation, and bed rest, are often unsuccessful. Secondary scoliosis resulting from lumbar disc herniation can be managed by lumbar discectomy if nonoperative measures or the natural history does not relieve the pain. With a short interval between diagnosis and definitive management, the patient is at less risk for developing fixed rotatory deformities, which may lead to persistence of scoliotic curves.

Disc herniation is part of the differential when an adolescent or child presents with a history of back pain without leg pain. Hamstring tightness or pain may be present and is thought to be a type of radiculitis. Performing the Lasègue test in this population produces an increase in back pain with or without hamstring tightness or radiating lower extremity pain. However, the diagnosis of disc herniation in young patients may be difficult to make in the absence of plain radiographic or neurologic changes. As was the case with our patient, who at age 17 initially presented to another physician, the diagnosis of disc herniation was evasive. Presence of extremity pain and sciatica coupled with radicular dermatomal patterns commonly assists in localizing the level of herniation. Without this, the back pain is a symptom almost impossible to pinpoint in most cases. Our patient had disc displacement at 2 levels, neither of which was clearly the pain source initially. Absence of sciatica, as originally seen in our case, may delay the diagnosis, which allows the secondary scoliosis to assume structural changes. A disc injury may result in an annulus tear, which over time herniates and produces extremity pain. This transition may also be complicated by chronologic factors, such as the change from "adolescence" to "young adulthood." One would have been hard-pressed to operate on the L4–L5 disc even with an increase in the scoliosis (15°-30°) with just left-side low back pain.

The reactive scoliosis in this case and others may be atypical (ie, nondiagnostic). The lordoscoliotic deformity pattern was atypical and relatively acute in onset. Back pain was variable in intensity over a 3-year period, and was accompanied by a negative Lasègue test, before any structural components manifested. The initial workup, including plain radiographs, MRI, and bone scan, was inconclusive for an underlying etiology for the back pain. Initial MRI showed disc displacement on the right side at L5–S1 but did not correlate with symptoms. Epidural steroids provided transient pain control, but there was no correction in structural deformity. Serial MRI and CT myelograms revealed a left disc protrusion at L4–L5 with radiculopathy, emphasizing the importance of serial examination, serial neurodiagnostic imaging, and routine follow-up in cases involving a progressive reactive scoliosis, even with an initial negative workup.

Initial workup with painful scoliosis includes plain radiographs and bone scan. Depending on those results, CT, MRI, myelography, and electromyography may be added. Disc pathology should be included in the differential, especially if there is a positive Lasègue sign. Other to-be-ruled-out conditions are congenital malformations, spondylolisthesis, osteoblastoma, osteoid osteoma, calcified discs, and stress fracture.

Conclusions

Back pain is an uncommon presenting complaint in adolescents and children and is usually benign in origin. However, back pain is not normal and mandates further workup to identify the underlying pathologic cause(s), such as infection, stress fracture, or spinal tumor. Lumbar disc herniation as the cause of back pain or scoliosis must also be considered, even though it is uncommon in youth. Lumbar disc herniation may not be clear initially and may require close, sequential clinical and/or neurodiagnostic study follow-ups. Secondary scoliosis may progress slowly and insidiously. Unfortunately, a fixed deformity can develop during a delay in identification of the pain source. Therefore, it is imperative to perform close clinical and radiologic examinations and follow-ups even in the absence of neurologic changes.

Authors' Disclosure Statement and Acknowledgment

The authors report no actual or potential conflict of interest in relation to this article.

Our patient provided written, informed consent for our report of his case to be published.

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This paper will be judged for the Resident Writer's Award.