

Spontaneous regression of lumbar disc herniation

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ABSTRACT

We present 3 cases of spontaneous regression of lumbar herniated disc. The disc regression correlated with clinical improvement documented by MRI studies. Although the phenomenon of spontaneous disappearance of decrease in size of herniated disc fragments is well known, the exact mechanism underlying this process remains unclear. We discuss 3 possible explanations for disc regression: retraction into the vertebral space, dehydration/shrinkage, and resorption due to inflammatory reaction.

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The fact that neurological symptoms caused by a disc herniation may frequently improve without surgical interventions is well known.^{1,2} The herniated part of the disc may decrease in size over a period of months or years. Recently, CT and MRI have been used to document this regression in different spinal compartments.^{1-5,6,7} We present 3 cases of lumbar radiculopathy caused by a herniated disc, one of them at the level L3-L4 and the others at the L4-L5 level, in which clinical improvement was associated with a significant decrease in size of the extruded disc fragment, documented on MRI scans. Our objective is to show that disc herniations can improve without surgical interventions.

Case Report. Patient 1. A 31-year-old man admitted in May 1997 with a 2-month history of

low back and left leg pain with no obvious cause. An MRI of the lumbar spine obtained 2 months after his symptoms began revealed a large extruded disc fragment (Figure 1a). There was compression of the dural sac with displacement by this fragment. We managed him conservatively, followed with serial neurological examinations and medical treatment. We obtained a second MRI study in January 2001, approximately 4 years after the first MRI. This showed regression of the extruded fragment that had been located posterior of L3 vertebral body, and a little compression of the dural sac. Straight leg raising (SLR) test was positive at 70° and there were no neurological deficits. We started an exercise program and conservative treatment, and followed him up for another 3 years. In January 2004, we obtained a third MRI, which showed significant regression of the extruded fragment of the lumbar disc herniation (Figure 1b). The height of L3-L4 disc space remained decreased compared to other levels and was unchanged from the previous MRI examinations.

Patient 2. A 31-year-old woman admitted in September 1997 with a 6-month history of low back and right leg pain. We obtained an MRI of the lumbar spine 3 months after her symptoms began, and this revealed a large disc fragment at the L4-L5 level, with compression and displacement of the dural sac. A trial of conservative management failed to relieve the patient's pain and so we offered surgery, however, she refused. We followed her with serial neurological examinations and medical treatment. Over the next 8 months, her pain gradually improved to the point that she did not require any medication and was essentially pain-free. We obtained a second MRI one year later, which showed the protruded part of the disc was smaller than the first MRI. After 5 years, control examination and a third MRI showed no evidence of compression or displacement of the dural sac by the protruded fragment presently located at the L4/L5 level.

Patient 3. A 47-year-old man admitted in September 2001 with a one-month history of low back and left leg pain. An MRI of the lumbar spine obtained one month after his symptoms began revealed a large disc fragment at L4-L5 (Figure 2a). A trial of conservative management failed to relieve the patient's pain and so we offered surgery, which he refused. We followed him with serial neurological examinations and medical treatment. Over the next 12 months, his pain gradually improved to the point that he did not require any medication and was essentially pain-free. Control examination and a second MRI after 4

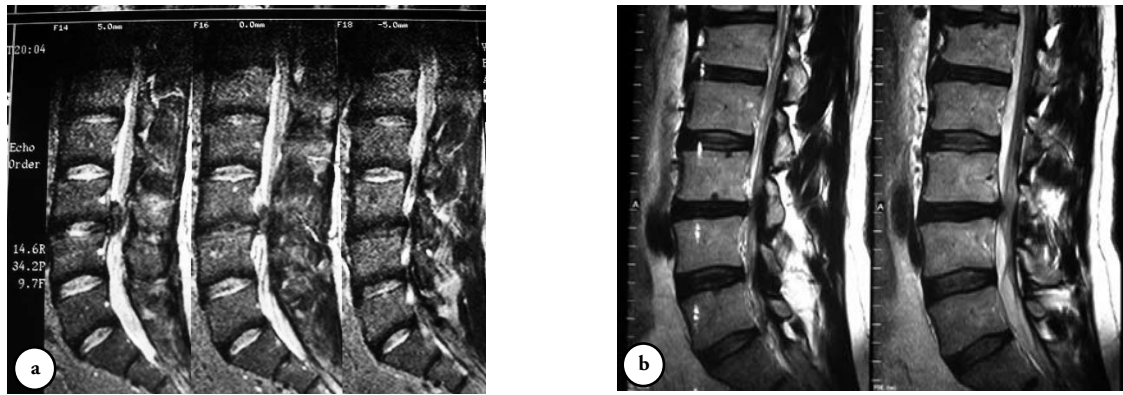


Figure 1 - An MRI of patient 1 a) obtained in 1997 showing a large extruded disc fragment. Sagittal images obtained through the extruded disc, b) obtained in 2004 showing significant regression of the extruded lumbar fragment.

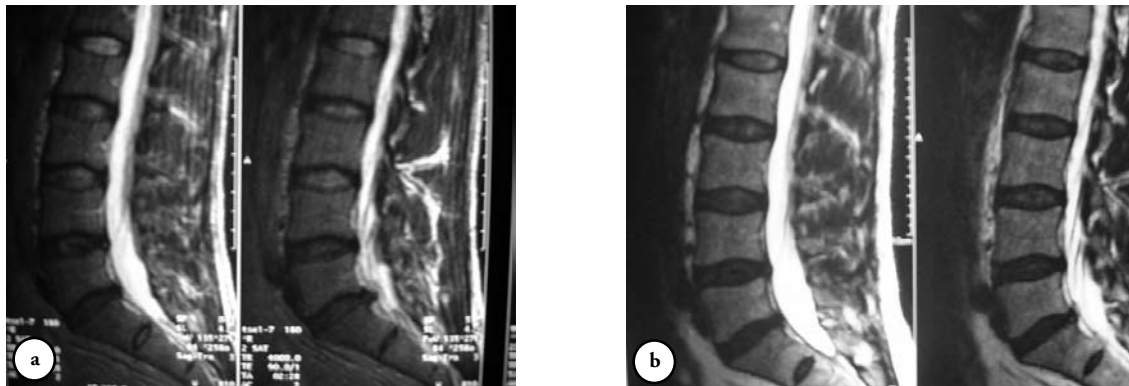


Figure 2 - An MRI of patient 3 a) showing L5-S1 disc hernia, b) after 4 years showed a bulging disc of the L5-S1.

years (Figure 2b) showed no evidence of compression or displacement of the dural sac by the protruded fragment located at the L4-L5 level.

Discussion. The literature contains several reports of regression or disappearance of herniated intervertebral discs without surgery, and CT scanning allows direct demonstration of the spontaneous regression of a herniated disc.^{3,4,6} Before CT, myelography documented reduction in the size of extradural defects. Following the introduction of MRI, we can obtain more detail of disc herniations and their natural development.^{1,2,5} Studies describe regression of herniated discs at different levels and with various clinical presentations, including lumbar-thoracic-cervical discogenic radiculopathy and myelopathy,^{1-3,7-9} however, the exact mechanism of this process remains unknown. Three possible explanations exist.¹ According to the first hypothesis, the herniated disc retracts back into the intervertebral space.⁶ This may occur theoretically if there is a disc bulge or if the disc material protrudes through the annulus fibrosis but is not separated from it,⁴ however, it would be unlikely in cases of completely extruded or migrated fragments.

The second hypothesis states that the herniated part disappears because of gradual dehydration and shrinkage. According to the third hypothesis, disc herniation into the epidural space causes an inflammatory reaction and neovascularization, resulting in gradual resorption of the cartilaginous tissue through enzymatic degradation and phagocytosis.^{2,9} The tissue extruded from the intervertebral disc showed obvious signs of degeneration such as changes in osmotic pressure. Macrophages were observed to be the mechanism of spontaneous regression.¹⁰

Clinical improvement frequently correlates with radiographic disc regression.² It appears that symptomatic improvement may occur without significant morphological changes, or that such clinical improvement precedes the radiographic changes. We can explain this discrepancy by the progressive decrease of pressure exerted by herniated fragments on neighboring neural structures and the gradual improvement of the inflammatory response that accompanies the herniation.

We confirm the validity of non-surgical management of herniated lumbar discs in the absence of neurological

deficits. Medical treatment alone or physiotherapy together may result in improvement of clinical symptoms.

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