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Spontaneous regression of a lumbar disc herniation: case report

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Spontaneous regression of a lumbar disc herniation: case report

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Abstract: Lumbar disc herniation is a common disease that induces back pain and radicular pain. Some cases require conservative treatment or at times relived spontaneously. Spontaneous regression of disc herniation is an atypical clinical presentation, and it has been recognized with the advancement of recent advances in imaging techniques. We present a 35-year-old woman presented a spontaneous regression of a lumbar disc herniation with good outcome after intensive physical therapy program. Spontaneous regression of disc herniation is thought to occur via an inflammatory reaction with molecular mechanisms of phagocytic processes.

Key words: Herniated disc, Regression, Spontaneous

Introduction

Lumbar disc herniation is a common cause of lower leg radiculopathy. It's widely accepted that conservative treatment must be done first exceptionally in neurological deficit, severe unremitting leg pain despite conservative measures, and repeated time loss from work, and equina syndrome signs. The prognosis is generally good.

Spontaneous regression of herniated discs in patients treated non-operatively has been reported in the literature in the past. It involves virtually all spinal compartments i.e. cervical, thoracic, and lumbar (1-4) and becomes clinically present in a variety of ways, including myelopathy and radiculopathy.

Recent advances in imaging techniques: computed tomography (CT) and magnetic resonance imaging (MRI) have facilitated the

precise documentation of this fact (2). However, the underlying mechanism remains unclear.

Here we present a case of lumbar radiculopathy due to a sequestered herniated disc at the L5-S1 level in which neurological improvement coincided with a significant diminution in the size of the extruding disc fragment documented on serial CT scans. The possible mechanism of sequestered fragment disc resorption is discussed.

Case report

A 35-year-old Moroccan woman was admitted with progressive low back and right leg pain along the L5 dermatome with no appearance cause.

One admission she was in good healthy. Body mass index was normal. Somatic

examination showed no abnormality.

On examination the straight-leg-raising test was positive on the right side (40°). Neurological examination showed no sensory impairment or cauda equina symptoms. The Lumbar CT scan showed a herniated disc with inferior migration and sequestered fragment on L5-S1 level (Figure 1).

The patient was treated conservatively with bed rest, muscle relaxants, and nonsteroidal anti-inflammatory drugs for 20 days. After that period he participated in a physical

therapy program during one month: (3 times one week). 40 days after the onset of pain the patient returned to daily activities. He was symptom-free and the serial CT scan performed 4 and 9 months showed spontaneous and almost complete regression of the sequestered fragment on L5-S1 level (Figure 2).

Our patient presented a spontaneous regression of a lumbar disc herniation with good outcome after intensive physical therapy program.

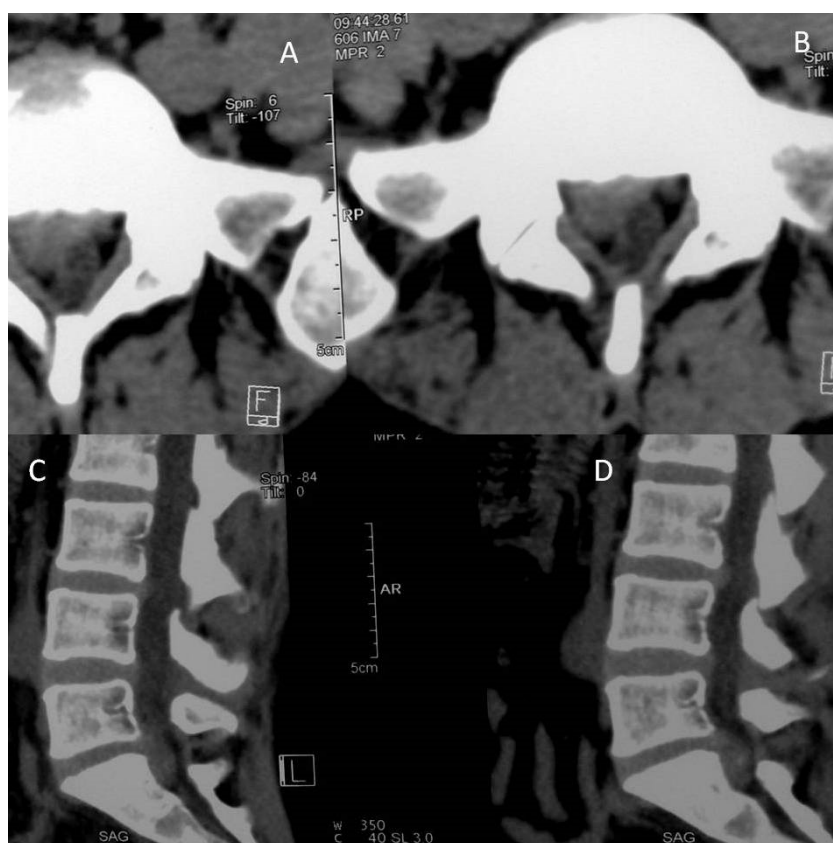


Figure 1 - CT scan of lumbar axial (A-B) and sagittal (C-D) view on the L5-S1 intervertebral level showing image herniated disc with inferior migration and sequestered fragment

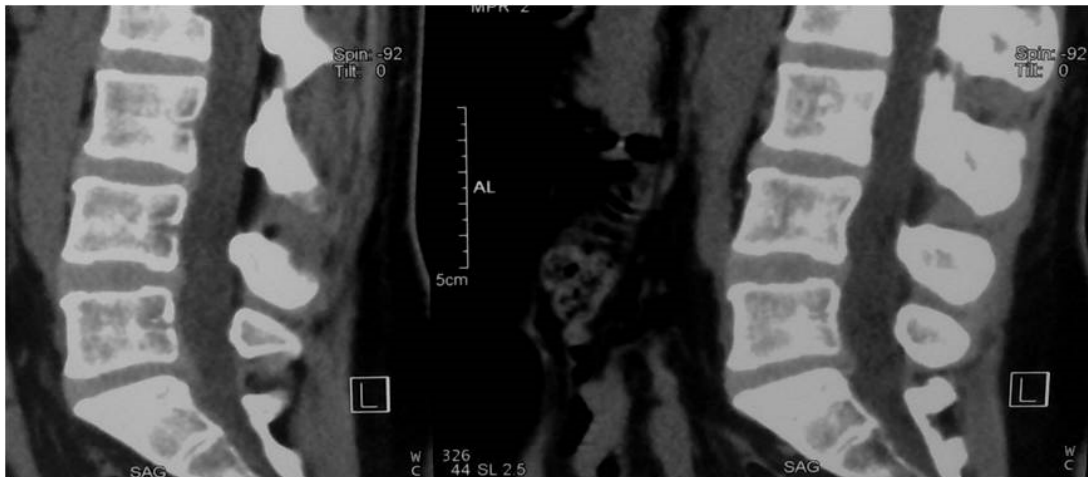


Figure 2 - Follow-up CT image at the same location one year later. Note the almost complete disappearance of the herniated disc fragment seen in Figure 1

Discussion

This event is rare. Recent advances in imaging techniques (CT, MRI) have facilitated the precise documentation of this fact (2-5). However, the underlying mechanism remains unclear.

Although the phenomenon of spontaneous disappearance or decrease in size of herniated disc fragments is well known, the exact mechanism underlying this process remains unclear. Three possible explanations for disc regression are discussed in the literature: retraction into the intervertebral space, dehydration/shrinkage, and resorption due to inflammatory reaction.

Researchers have brought forward three possible explanations which could provide a satisfactory answer (1, 2, and 3). The first, the theory of dehydration, states that the herniated disc fragment disappears due to gradual dehydration and shrinkage. The second hypothesis, the theory of resorption, the most experimentally studied one, supports the idea

of cartilaginous tissue resorption through enzymatic degradation and phagocytosis as a result of an inflammatory reaction and neovascularization. The inflammatory reaction could be the result of an autoimmune response developed when the disc contents extrude into the epidural space and are recognized as “foreign” (3). Studies have revealed neovascularization at the periphery of the sequestered disc and the presence of macrophages and T cells in the vascularized areas. Anti-interleukin-1, intracellular adhesion molecule-1, lymphocyte function-associated antigen, and basic fibroblast growth factor are also expressed on most of the mononuclear cells. Furthermore, the cell infiltration is more prominent in the nucleus pulposus than in the annulus fibrosus.

Finally, blood vessels in the extruded tissue are formed either through metaplasia of undifferentiated mesenchymal cells or they are derived from blood vessels that have invaded the annulus fibrosus as a result of disc degeneration. According to the third

explanation, the theory of retraction, the herniated disc retracts back into the intervertebral space. This is the case if the disc protrudes through the annulus fibrosus but at the same time is not separated from it. It is possible, though, that all three proposed mechanisms play a role in the diminution process.

It is also noted that larger fragments are more frequently observed to diminish than smaller ones, possibly because of their larger water content (dehydration, resorption). (6)

Extruded material exposed to the epidural space may be resorbed more quickly than that beneath the ligament. Vascular supply probably plays a role in the mechanism of resorption. In our previous reported case of posterior migration of lumbar sequestered disc fragment, a MRI showed a peripheral enhancement mimicking a space occupying lesion. The epidural fat is vascular; the presence of fragment in this space will induce an inflammatory response with granulation tissue and neovascularization around the extruded tissue, causing a ring enhancement with gadolinium in the MRI scan. (7). this finding supported the third explanation of spontaneous regression of a lumbar disc herniation (5).

In addition, radiographic changes often follow clinical improvement (relief of pain). Studies have shown maximal clinical improvement within one year, but the reduction in disc size continued thereafter. This phenomenon is explained first by a progressive decrease in the pressure exerted by the fragments on neural structures and second by gradual amendment of the inflammatory response (4).

Conclusion

The regression of sequestered large lumbar disc is very rare but well documented. Spontaneous regression was observed in the sequestration-type lesions. Vascular supply probably plays a role in the mechanism of resorption.

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