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Spontaneous regression of a herniated cervical disc in a patient with myelopathy

Case report

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The authors present a case of spontaneous regression of a herniated cervical nucleus pulposus in a patient with myelopathy. This 37-year-old woman developed sudden quadriparesis; she had reported no history of trauma. Magnetic resonance (MR) imaging revealed a large disc herniation and increased signal intensity of the cord at the C5–6 level. The extruded disc fragment was found to have resolved on follow-up MR imaging after 28 months, despite the fact that the patient had undergone no specific treatment. The patient's symptoms had subsided almost totally. This is the first case of MR-documented regression of a cervical disc herniation in a patient with myelopathy.

KEY WORDS • cervical disc • myelopathy • regression

SPONTANEOUS regression of herniated lumbar disc material has been well documented.[3,4,8] This phenomenon has also been reported to occur in the cervical[5,7,9] and thoracic[1] regions. Most such cases are confined to disc herniations that are associated with radiculopathy. We present a case of spontaneous disappearance of a herniated cervical disc in a patient with myelopathy, which has not been reported previously.

Case Report

Presentation. This 37-year-old woman presented at our hospital because of a sudden onset of quadriparesis. She awoke with pain in her neck, paresthesia in her whole body, and quadriparesis. She could not walk without support. There was no history of trauma.

Examination. On physical examination, C-7 sensory level quadriparesis was demonstrated (motor Grade 4+), and mild urinary disturbance. Hoffmann's sign and ankle clonus were present bilaterally, with hyperactive deep tendon reflexes. Plain x-ray films of the cervical spine showed unremarkable findings. Magnetic resonance (MR) imaging of the cervical spine revealed a large disc herniation at the C5–6 level. On T_2-weighted MR imaging the herniated disc material was shown to have compressed the anterior aspect of the spinal cord and to have changed its signal intensity (Fig. 1). We recommended that the patient undergo an anterior discectomy and fusion procedure. However, the patient refused the operation and discharged herself, against medical advice, because her symptoms had begun to show some improvement.

Second Presentation. She returned 28 months after her initial visit and was able to walk on her own. She complained of paresthesia in her whole body. According to her statement, she had improved without receiving any specific therapeutic care. Initially, she regained her motor strength rapidly. She was ambulatory without need of supportive assistance within 2 weeks. By the 2nd month, she no longer experienced urinary difficulty and could perform most housekeeping tasks. Thereafter, she made slow but steady improvement until approximately 6 months after her initial presentation, when she had regained her previous dexterity with chopsticks. However, the fluctuating whole-body paresthesia remained.

Second Examination. Physical examination showed dysesthesia was present below the C-7 level. Examination of motor skills demonstrated normal strength throughout and normal gait. Hoffmann's sign was positive in her left hand. However, there was no ankle clonus, in spite of hyperactive tendon reflexes. Follow-up MR imaging showed complete regression of the disc and an abnormal cord signal (Fig. 2). Therapy with carbamazepine and tricyclic antidepressant alleviated her remaining symptoms.

Discussion

Since Teplick and Haskin[8] first reported a case of spon-

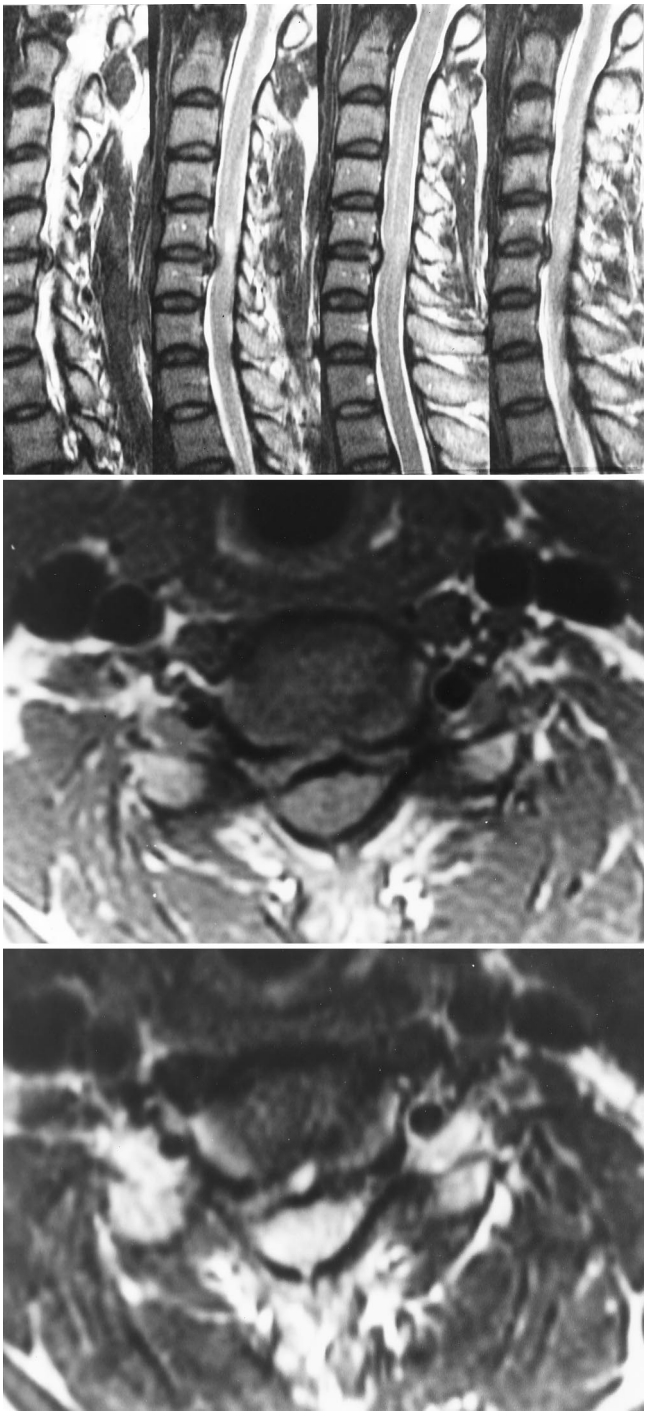

FIG. 1. Magnetic resonance imaging studies. *Upper:* Cervical T_2-weighted image demonstrating large disc extrusion, compressed spinal cord, and increased signal intensities in adjacent cord at the C5–6 level. *Center* and *Lower:* Axial T_1- and T_2-weighted images at the level of C5–6 revealing significant disc extrusion.

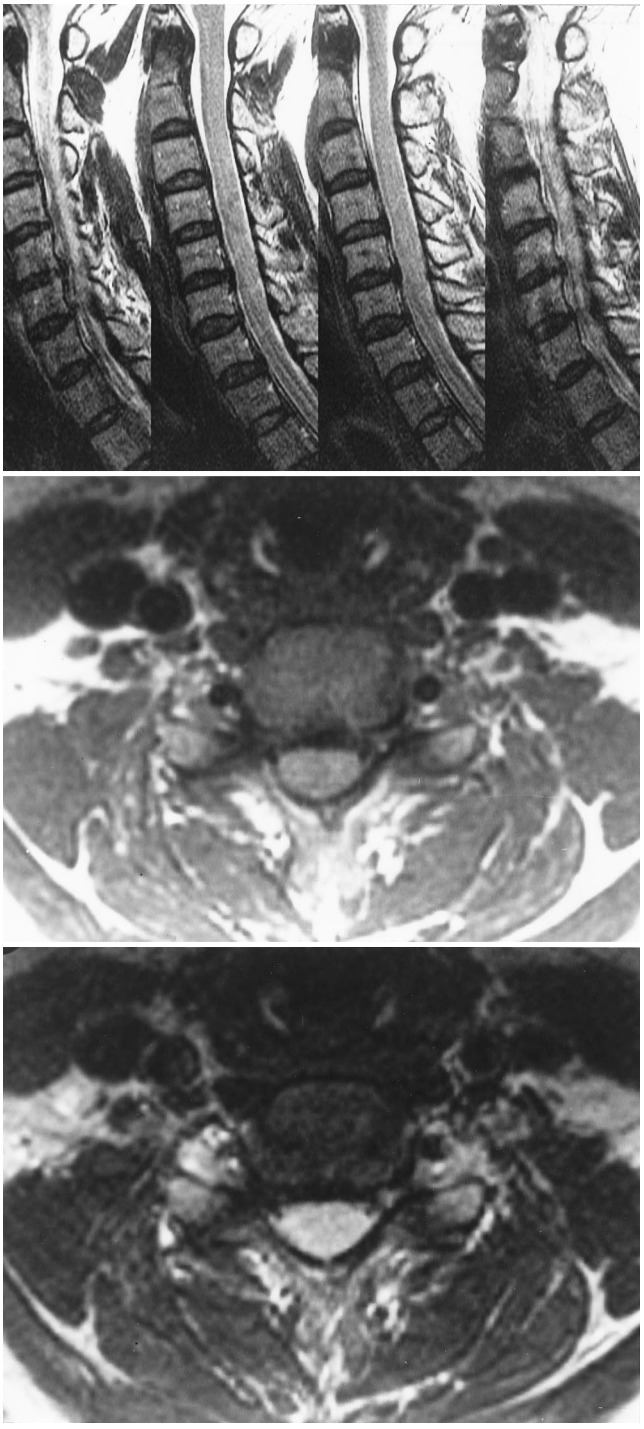

FIG. 2. Follow-up MR images 28 months after initial radiographic examination. *Upper:* Cervical T_2-weighted image revealing complete resolution of abnormal cord signal and almost completely regressed disc fragment. *Center* and *Lower:* Axial T_1- and T_2-weighted images demonstrating the regression of the large disc extrusion at the level of C5–6.

taneous regression a of herniated nucleus pulposus, other authors have described the phenomenon in patients with lumbar disc disease.[2–4,6] Several cases of spontaneous disappearance of cervical[5,7,9] and thoracic disc herniations[1] have also been documented. The authors of these articles have described disc extrusion primarily in patients with radiculopathy; its occurrence in patients with myelopathy has not been described in the literature. This is because herniated discs in patients with myelopathy are usually

treated surgically. Generally, patients with myelopathy, a condition which is more severe than radiculopathy, are impatient for their neurological deficits to resolve. Therefore, doctors are apt to choose more aggressive treatment in the absence of knowledge about the natural history of such patients. To our knowledge, this is the first MR-documented case of spontaneous regression of a cervical disc herniation in a patient with myelopathy.

Some authors have studied the mechanism and factors affecting resorption. It has been suggested that once the herniated disc material is exposed to the vascular environment of the epidural space, cellular mechanisms contribute to regression.[2,6] Because large and extruded discs have wider exposure to cellular resorption mechanisms, they tend to regress more rapidly than contained herniated discs. One can assume that a disc herniation large enough to compress the spinal cord, as in our case, is also reabsorbed well. However, cervical disc extrusion in a patient with radiculopathy is usually managed surgically because fragment migration and compression of the spinal cord might result in quadriplegia. In contrast, Saal, et al.,[7] recently conducted a longitudinal study of the nonoperative management of cervical disc disease in a cohort of 26 patients who had cervical spine herniations and radiculopathy. Progressive neurological deterioration did not occur in any of their patients. The authors concluded that many cervical disc herniations could be successfully managed with nonsurgical care.

The operation for cervical disc disease is a relatively safe procedure, and we recommend surgery routinely to patients who have cervical disc disease with myelopathy. Surgical management might have resulted in more rapid and complete alleviation of symptoms in our patient. However, our case illustrates the potential of spontaneous recovery from cervical myelopathy without undergoing any specific treatment of the cervical disc. This can be interpreted as a natural course. Although generalizations from the results in a single patient would be inappropriate, nonsurgical care may be considered as an option for the treatment of patients with cervical disc herniation and myelopathy, especially patients with complicated medical problems.

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