Spondylodiscitis after percutaneous discectomy,

A case diagnosed by MRI

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Case report

A 50-year-old woman presented with a 4-year history of recurrent attacks of low back pain and right side sciatica. A clinical diagnosis of disc prolapse at the L4-5 level was made, and this was confirmed by CT, which showed a right disc herniation at this level. The ESR and routine blood count were normal.

Under local anesthesia, the patient underwent a percutaneous discectomy (PLD) in a conventional orthopedic operating theatre in clean-air enclosure. The skin was prepared in the usual way with a betadine scrub. No prophylactic antibiotics were administered. Under image intensifier, PLD was carried out. A satisfactory amount of disc fragment was removed. The procedure lasted 30 minutes and was uneventful. Four days later, the patient reported substantial improvement of her symptoms, and she was discharged from the hospital.

On the tenth postoperative day, the patient complained of severe back pain. All the spinal movements were restricted and painful, with radiation to the abdomen. She was apyrexial, the surgical wound had healed, and there was no obvious infection. Blood tests showed a white blood count of 7.3 and an ESR of 30 mm/h, and 3 days later 33 mm/h. Blood cultures were negative.

Because of the pain, tenderness, and elevated ESR, a provisional diagnosis of discitis was made, and antibiotic treatment was started intravenously using cloxacillin and ampicillin.

A week later, the patient had improved; the ESR was 27 mm/h. Repeat radiographs of the lumbar spine did not reveal any pathologic findings. An MRI showed a typical infection, with changes in the disc space and the adjacent bodies of the L-4 and L-5 vertebrae. This infection was visualized on the T1-weighted images as areas of low signal intensity. On T2-weighted images, high signal intensity was observed crossing the involved bone disc space with irregularity of the cortical margins (Figure 1).

Antibiotics were continued for another 6 weeks. Six months after the operation, the patient was mobile and almost completely free of pain.

Discussion

It was 12 years after Hijikata's (1975) description of PLD that the first case of postoperative spondylodiscitis was described (Blankstein et al. 1987). The delay in recognition of this major complication of PLD reflects the considerable difficulties in making the diagnosis.

Since the first report of Blankstein et al. (1987), there have been a few sporadic reports of discitis following PLD (Scaffer and Kambin 1991). Hijikata (1989) himself reported 1 case; and by reviewing the literature, Helms et al. (1989) estimated that the rate of this complication was less than 0.2 percent of the cases. However, the real rate is still unknown because of insufficient documentation. The documentation is difficult because in these cases there is usually no evidence of a wound infection, and there may be absence of fever and an elevated leukocyte count. In addition, severe pain is often attributed to failure of the surgery rather than to an infective process. Besides, with an organism of low virulence or good host response, it seems possible that discitis would resolve spontaneously without the condition being recognized (Bircher et al. 1988).

Blankstein et al. (1987) described in detail how the intervertebral space is initially infected with bacteria that are introduced directly into the disc space at the time of surgery. The infection later invades the adjacent vertebrae, a process that is opposite to the one that occurs in hematogenous spondylodiscitis, and which has also been described as a complication of conventional laminectomies. The same authors emphasized that an infection originating in the disc space following PLD, as in our case, may be particularly dangerous due to inadequate drainage and deficient vascular perfusion of the disc space.

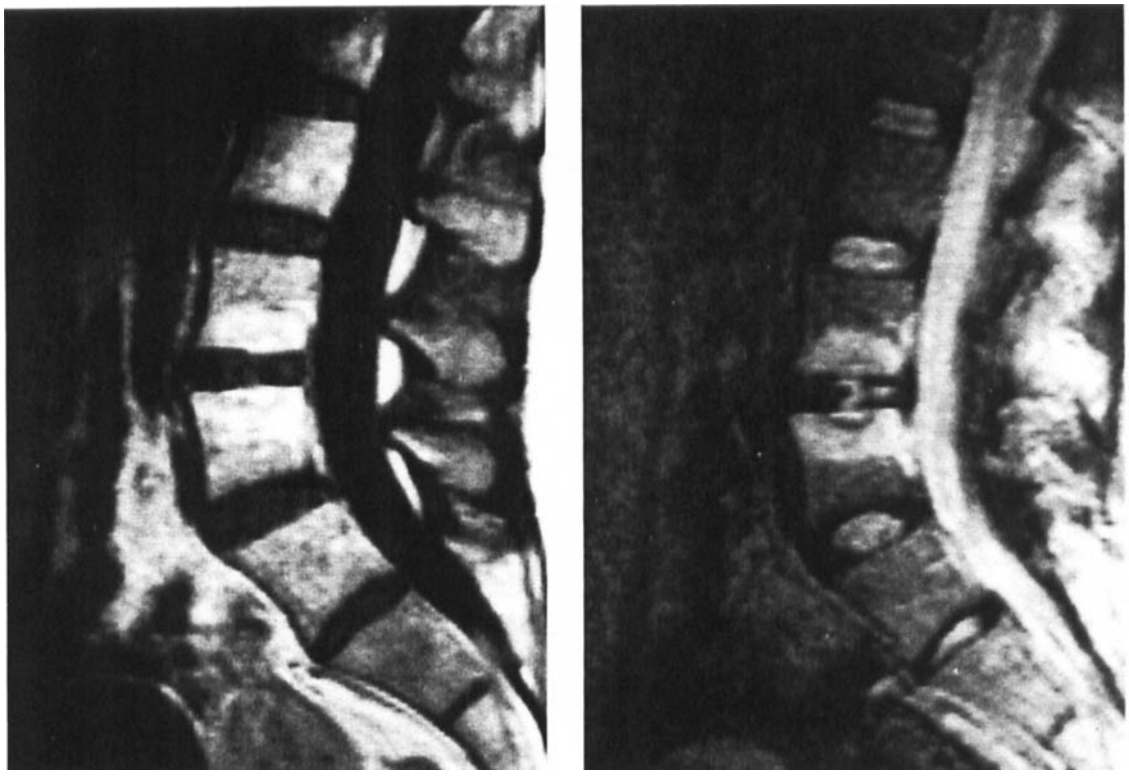

Figure 1. Discitis L4-5 and osteomyelitis of the adjacent vertebrae. Low signal intensity marrow involvement of L-4 and L-5 crossing the discovertebral junction images with low signal intensity on the T1-weighted image (left) and with high signal intensity on the T2-weighted image (right).

Our patient had the pattern of pain that is usually, but not necessarily, associated with discitis. The pain was severe and presented after the second week. However, this was the only symptom compatible with this complication, and was present without signs of an infective process. Frequently, there is an absence of an elevated leukocyte count and fever in postoperative (postlaminectomy) disc infections. The ESR of 33 mm/h, however elevated, in this patient, was well below the level of 50 mm/h, which is the level consistent with discitis (Bircher et al. 1988). Thus, to confirm the diagnosis, further investigations were required.

Plain radiographs and CT scanning are relatively insensitive to postoperative spondylodiscitis, and show changes, such as end-plate erosion, only several weeks after infection (Enzmann et al. 1990). The most effective method of early demonstration of suspected spondylodiscitis seems to be magnetic resonance imaging, because, as in our case, it reveals end-plate changes and expansion of the infection to the vertebral bodies long before any changes are apparent radiographically (Enzmann et al. 1990).

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