

Lumbar Discitis Complicating Percutaneous Laser Disc Decompression: Case Report and Review of Literature

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Introduction

Percutaneous methods for treating herniated lumbar discs are relatively new procedures and include chemonucleolysis¹, automated discectomy² and laser disc decompression^{3,4}. The advantages of these procedures are minimal invasion and complications²⁻⁴. However, discitis, bowel nerve root and great vessel injuries have been reported^{1,5}.

Although chymopapain injected into the intervertebral discs, employed in more than 16,000 patients, was withdrawn following a double-blind study that indicated that the enzyme had no significant benefit over its diluent; enthusiasm for percutaneous lumbar discectomy utilizing an automated nucleotome or Nd: YAG laser still exists in certain centres^{2,5}. The rationale for these procedures is that by removing a small portion of the nucleus, one reduces the intradiscal pressure^{5,6}. The validity of these procedures as safer and less invasive alternative to open discectomy/laminectomy has not been proved and in the absence of a well designed, prospective randomized study, it would be prudent to regard this entire area of surgery with appropriate skepticism⁵. In this report, a disabling complication of laser discectomy is presented.

Case Report

A 53 year old man presented with a 4 month history of low back pain and bilateral sciatica (right. more than left), not responding to medical treatment. Pain was associated with an unpleasant tingling sensation in right leg and foot, which felt clumsy and weak when walking. Clinical examination revealed minimal weakness of dorsiflexors of right foot and hypoalgesia of L5 dermatome. Lumbar myelography followed by CT scan showed disc protrusion at L4-L5. During August, 1993, he was treated by percutaneous laser disc decompression (PLDD) elsewhere. Three weeks later, the patient was admitted with very severe back and paraspinal muscle pain that was aggravated by slightest movement. He noted that his legs were weak and numb and he could no longer walk. Low grade fever (100°F) was present. On examination, there was marked tenderness at L3 to S1 region. Straight leg raising (SLR) was restricted bilaterally. Neurological examination showed decreased strength in both lower extremities and hypoaesthesia of right perianal skin and dorsum of foot. Ankle jerks were depressed bilaterally and dorsiflexors of right foot were weak. Erythrocyte sedimentation rate (ESR) was elevated (60) and C-reactive protein (CRP) level was markedly raised (120 mg/L), MRI scan (Figure 1)



Figure 1. MRI showing changes of post-PLDD discitis involving the L4 and L5 vertebral bodies as well as the intervening disc space (arrow heads).

on 12-9-93 showed erosion of adjoining parts of the bodies of L4 and L5. A diagnosis of discitis was made. Lumbar myelogram followed by CT scan (Figure 2)

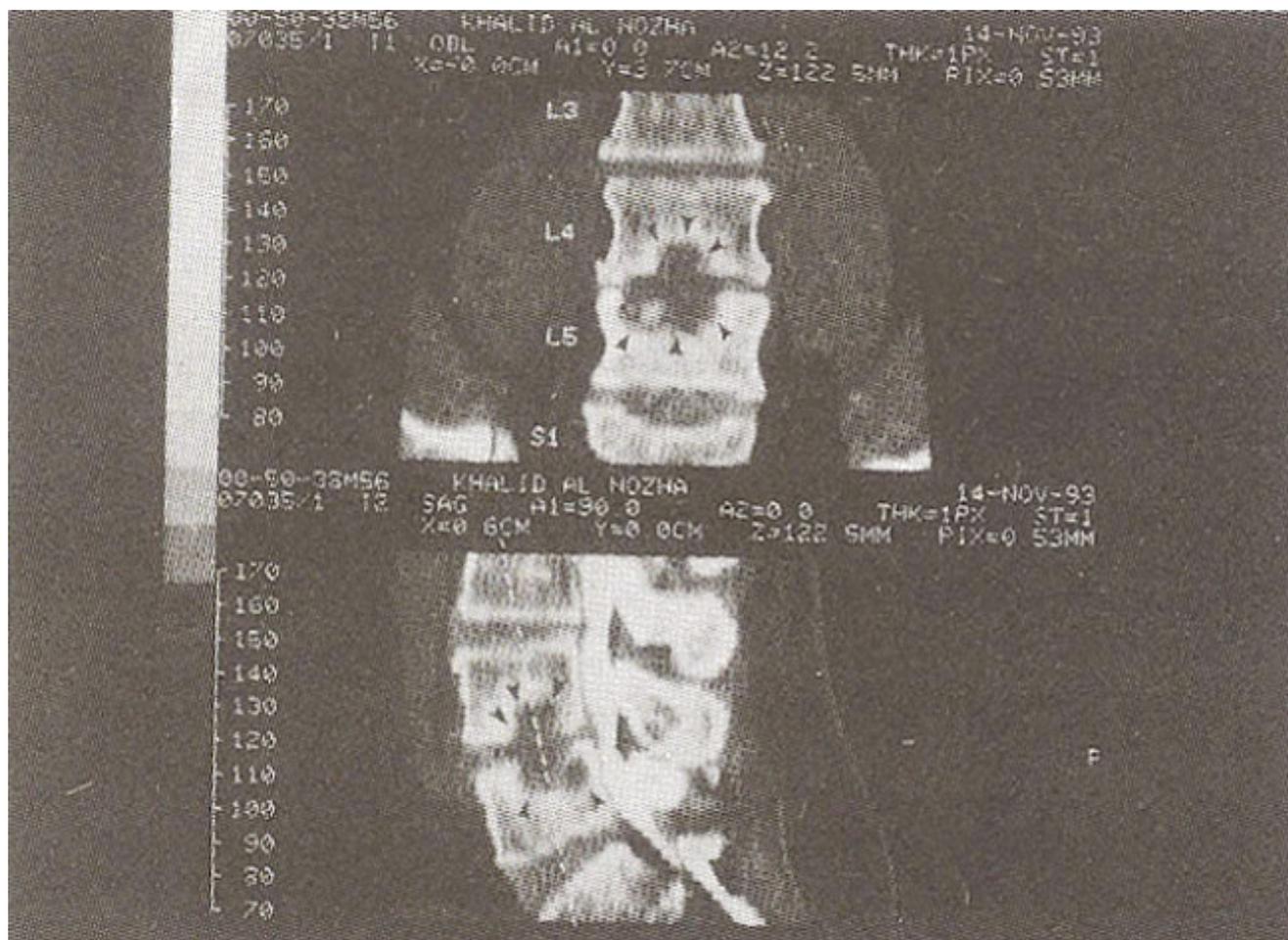


Figure 2. CT-myelography coronal and sagittal reconstruction showing typical picture of discitis. Note the destruction beginning in the disc and spreading to the lower half of the body of L4 and upper half of the body of L5 (arrow heads).

showed a disc prolapse between L4 and L5 as well as extensive bone destruction on either side of the L4-L5 intervertebral disc space - the site of PLDD. Six weeks of conservative treatment consisting of bed rest and anti-staphylococcal medication (Flucloxacillin) failed to relieve the severe back pain and spasm. In view of the unrelieved symptoms and evidence of epidural compression at L4-L5, laminectomy was carried out. At operation, a large protruded disc at L4-L5 was excised and L4-L5 disc space explored. No pus was found but a large cavity of bone destruction containing granulation tissue and cartilaginous fragments was cleared. Biopsy revealed inflammatory granulation tissue with polymorphonuclear infiltration, but was negative for bacterial and acid fast bacilli culture. Anti-staphylococcal therapy (Vancomycin) was started on the day of operation and continued for two weeks. Post-operative period was marked by rapid relief of pain and resolution of neurological symptoms. Serum CRP and ESR monitored post-operatively, rapidly came back to normal. Gradually increasing mobilization was pain free. Follow-up CT scan at 3 months showed the cavity to be healing. Return to full activity thereafter was smooth.

Discussion

Discitis as a complication of conventional operative treatment of lumbar disc herniation is rare, with a

reported incidence of 0.8%⁷. Incidence of this complication following percutaneous procedures especially with the use of laser and automated neurotome, although claimed to be zero^{2,3}, is not known. Diagnostic clinical features of discitis, as typified by our patient, are extreme pain and lumbar spasm related to motion. An elevated ESR is often the only laboratory indication of discitis⁷; although the C-reactive protein (CRP) correlated more closely with the clinical and radiological progress of the condition in our case. Based on this case and previous observations⁸, it is suggested that CRP levels may be useful for monitoring the effectiveness of treatment in patients with discitis, especially when used in combination with the clinical response and CT/MRI scans.

The X-ray changes are subtle and delayed⁷ and therefore, less helpful in the diagnosis of discitis than the CT or MRI. Typical bone destruction and sclerosis as seen in CT scans (Figure 2) appears earlier and correlates well with the progress of disease. MRI (Figure 1) was found to be even more sensitive in detecting early changes caused by discitis.

Pathogenesis and management of discitis following laser disectomy is no different than that following other procedures. Surgery is indicated in the face of increasing pain especially if the laboratory, CT and MRI monitoring shows expansion of the lesion. Under these circumstances, surgical intervention appears to shorten overall disability. Two approaches, posterior and retroperitoneal have been described for surgical exploration and debridement in the management of lumbar discitis⁹. Posterior approach was chosen in our case because of the evidence of cauda equina compression. At operation there is usually no pus, only granulation tissue and loosened cartilaginous fragments⁷, as was seen in our case. Once the offending granulomatous material is removed, drainage established and appropriate antibiotic coverage based on bacteriological studies instituted, recovery is usually rapid.

To conclude, sterile or bacterial discitis can complicate PLDD, and that its incidence may be underestimated due to its being a new procedure.

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