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Posterior epidural migration of a lumbar disc fragment causing cauda equina syndrome: Case report and review of the relevant literature

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Abstract Posterior epidural migration (PEM) of free disc fragments is rare, and reported PEM patients usually presented with radicular signs. An uncommon case involving a patient with cauda equina syndrome due to PEM of a lumbar disc fragment is reported with a review of the literature. The patient described in this report presented with an acute cauda equina syndrome resulting from disc fragment migration at the L3–L4 level that occurred after traction therapy for his lower back pain. The radiological characteristics of the disc fragment were the posterior epidural location and the ring enhancement. A fenestration was performed and histologically confirmed

sequestered disc material was removed. An early postoperative examination revealed that motor, sensory, urological, and sexual functions had been recovered. At late follow-up, the patient was doing well after 18 months. Sequestered disc fragments may occasionally migrate to the posterior epidural space of the dural sac. Definite diagnosis of posteriorly located disc fragments is difficult because the radiological images of disc fragments may mimic those of other more common posterior epidural lesions.

Keywords Cauda equina syndrome · Disc migration · Lumbar disc · Sequestered disc

Introduction

Disc sequestration (noncontained herniations) is defined as a perforation of the fibrous ring and posterior longitudinal ligament (PLL), and migration of the fragment to the epidural space. The sequestered disc is completely separated from the parent disc and disc space. Free fragments generally migrate within the spinal canal in superior, inferior, and lateral directions [1, 5, 9]. In contrast, disc extrusion (contained herniations) is characterized by rupture of the fibrous ring, but a variable degree of connection with the parent disc or disc space. Extruded disc fragments may migrate subligamentously behind the PLL [1, 9].

The most common path of disc migration is in a posterior and posterolateral direction to the anterior epidural space, and the most commonly recognized clinical syndrome is a radiculopathy [1, 4, 7, 8]. Posterior epidural disc

migration is an uncommon event, and posterior migration causing cauda equina syndrome is exceptionally rare [1, 2, 4, 6, 7]. In this report, a patient with cauda equina syndrome resulting from a posteriorly migrated disc material is presented and difficulties in diagnosis which might delay appropriate therapy are discussed.

Case report

A 47-year-old man who had been suffering mild lower back pain for 6 weeks was given medical treatment for 1 month. His symptoms did not subside, and he underwent subsequent traction therapy by a chiropractor. His condition suddenly deteriorated and was followed at home for 15 days prior to admission. When he was admitted to our hospital, a neurological examination revealed bilateral leg weakness and bowel and bladder retention. The Laseque and provocative tests were negative bilaterally. Motor strength was 80% of normal proximally and distally in both lower extremities. Diminished pinprick and light touch sensation was detected below

Fig. 1 A Axial T₁-weighted magnetic resonance imaging (MRI) demonstrates an iso- and slightly hyperintense lesion in the posterior epidural space at the L3–L4 level. B The same lesion was hyperintense on T₂-weighted images

Fig. 2 On T₁-weighted sagittal images, the lesion at the L3–L4 level was extended cranially. The height and intensity of L3–L4 and adjacent intervertebral disc spaces below and above the lesion were similar to each other

the L3 dermatome bilaterally, and this was worse on the left. Cremaster reflex and bilateral deep tendon reflexes at the knee and at the ankle were absent. The patient had erectile dysfunction.

Fig. 3 Cyst-like appearance of the lesion after contrast administration on T₁-weighted axial image. The absence of communication of the lesion with the disc space and the facet joint is shown after contrast injection

Plain radiograms were unremarkable. Magnetic resonance images (MRI) showed a left L3–L4 posterior epidural lesion. MRI characteristics of the lesion are shown in Figs. 1 and 2. The lesion showed ring-like enhancement after contrast injection (Fig. 3).

An emergency left-sided L3–L4 fenestration was performed. A large sequestered disc fragment was identified posterior and lateral to the dural sac. There was no communication between the disc material and the facet joint posteriorly or the intervertebral disc space anteriorly. The lesion was not attached to the dura and was

easily dissected and removed as a single mass. Examination of the L3–L4 interspace showed a rent in the lateral PLL, and discectomy was performed. The pathological examination confirmed that the specimen was a disc. At the follow-up examination 18 months post-operatively, the patient had fully recovered motor, sensory, urinary, and sexual functions.

Discussion

Free disc fragments typically tend to migrate laterally due to the anatomical properties of the anterior epidural space formed by the PLL posteriorly and the vertebral body anteriorly. A “sagittal midline septum” (septum posticum) separates this space into two compartments and limits sideward migration [1, 9]. A “lateral membrane” (peridural membrane) is another band that is attached to the PLL medially and to the wall of the spinal canal laterally. This anatomical structure limits posterolateral migration [1, 6, 9]. Other important ligaments are the midline and lateral dural ligaments of Hoffmann, which connect the anterior dural surface to the PLL and posterior vertebral periosteum [11]. These structural properties of the PLL and ligaments are the reasons for the lateral or posterolateral path of migration [7, 9]. A disc fragment may migrate within the spinal canal in a caudal, cranial, or lateral direction and may be located above or below the axilla of the root, intra- or extraforaminally and intradurally. The PLL, ligaments, dura, epidural vessels, fat, and root itself act as anatomical barriers against migration and limit further posterior migration. In some rare cases, the fragment may migrate to the posterior epidural space [1, 5, 7, 8].

Clinical symptoms of a free fragment are usually radicular pain due to root compression; however, posteriorly migrated fragments may also cause the cauda syndrome in addition to radiculopathy [7]. In the patient presented, possible extruded disc herniation which could not be evaluated was thought to be the reason of initial low back pain lasting 6 weeks. However, unreliable and faulty traction by a traditional chiropractor (who may intervene in orthopedic emergencies) was thought to be the reason why the previously extruded fragment migrated and the symptoms changed from lower back pain to the cauda syndrome. One of the known contraindications of traction therapy is extruded disc herniation, as in our patient [3]. Presumably, a free fragment just in and in front of the PLL rent moved secondary to closure of the PLL rent edges due to stretching. Moreover, stretching of other ligaments after traction pushed or displaced the fragment further into the spinal canal. In normal conditions, traction of the spine causes negative intradiscal pressure, fluid uptake into the disc, and widening of the intervertebral spaces [3]. The other possible explanation of posterior migration of a free fragment is this pressure gradient.

MRI is the first diagnostic tool in evaluation of cauda compression. The differential diagnosis of a disc fragment should be made using MRI characteristics before surgical

Table 1 Differential diagnosis of posterior epidural lesions [1, 5, 7, 8]

Type	Disease
Metabolic disorder	Gout
Infection	Epidural abscess Cysticercosis Echinococcosis
Tumor	Chordoma Lipoma Lymphoma Cystic neurinoma Elastofibroma Metastasis
Degenerative disease	Sacral cysts Ligamentum cysts Synovial cysts of the facet joint Osteophyte of the facet Pigmented villonodular synovitis Unilateral flaval ligament hypertrophy Migrated disc fragment
Trauma	Hematoma
Iatrogenic disorder	Postoperative fibrosis

Table 2 Literature review of patients with cauda equina syndromes caused by posterior epidural migration of a sequestered free fragment

Reference	Year	Level
Lichter [4]	1989	L2–L3
Lutz et al. [5]	1990	L2–L3
Sekerci et al. [10]	1992	L3–L4
Bonaroti and Welch [1]	1998	L2–L3
Robe et al [7]	1999	L3–L4
Presented case	2000	L3–L4

intervention (Table 1). MRI intensities of a posteriorly migrated fragment are similar to disc herniation in other regions. However, no direct communication with the disc space is observed and ring-like contrast enhancement can be seen after injection. A peripheral enhancement and cyst-like appearance is the result of the wrapping of the lesion by richly vascularized epidural fatty tissue and by the inflammatory and vasogenic properties of disc content [7].

Posterior epidural migration is an infrequent event, and posterior migration causing cauda syndrome is extremely rare. Cauda syndrome caused by posterior disc migration has only been reported five times to our knowledge (Table 2). In the patient presented, cauda equina syndrome was thought to be primarily due to a mass lesion rather than to disc disease because of the negative Laseque test, second motor neuron-type paraparesis, and the posterior location of the lesion. A definite diagnosis is difficult to make because

the images of a fragment in a posterior location may show characteristics similar to other more common epidural lesions. A free disc fragment should therefore be considered in

the differential diagnosis of posteriorly located epidural lesions. Surgical results after early diagnosis are encouraging.

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