Chronic dura erosion and intradural lumbar disc herniation: CT and MR imaging and intraoperative photographs of a transdural sequestrectomy

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Received: 26 July 2011 / Revised: 19 October 2011 / Accepted: 6 November 2011 / Published online: 23 November 2011
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Abstract

Introduction A 47-year-old male with a history of recurrent low-back pain presented with acute left radiculopathy.

Material and methods The CT and MR scans showed a severe osteochondrosis of the L4/5 segment, a broad protrusion of the disc annulus and extrusion of nucleus material into the spinal canal on the left side.

Results The caudally dislocated sequester pieces were visualised intradurally and the intraoperative finding confirmed this rare pathology. After dorsal durotomy-free sequester material was found between the nerve rootlets within the subarachnoid space and altogether ten fragments were removed. Further transdural exploration visualised ventrally a round dura defect surrounded by a thickened arachnoid layer with enlarged veins as a sign of a chronic erosive process.

Discussion Patients history, imaging and the intraoperative findings support the thesis, that chronic degenerative disc disease and adhesions between the posterior longitudinal ligament and the dura are the predisposing pathogenetic factors for an intradural disc herniation.

Conclusion Intradural disc herniation is a rare condition and requires durotomy to remove the pathology. Therefore an actual high resolution MRI is mandatory in all cases of intraspinal space occupying lesions.

Keywords Lumbar disc herniation · Intradural sequester · Transdural sequestrectomy · Dura erosion

Introduction

Intradural disc herniations are very rare and comprise less than 0.3% of all herniated discs with a preponderance of more than 90% at the lumbar spine and the L4/5 segment is the most affected location [1, 5–9, 11, 12, 14]. Since the first report of an intradural sequester was presented by Dandy [4] in 1942, more than 140 cases have been reported in the literature [7].

The clinical symptoms are indistinguishable to those of a “classic” lumbar disc herniation and the differentiation between a “normal” epidural versus a rare intradural sequester can usually be achieved with a high-resolution MR imaging only. Confusion may occur especially with other space-occupying intraspinal pathologies such as micrometastasis, meningioma, neurinoma or cysts [1, 10, 11]. If the preoperative diagnosis of an intradural pathology is missed because of reduced quality of the imaging or simply because there was no suspicion of such a rare location of herniated disc material, the surgeon will be surprised; he will not find the expected situs after routine microsurgical exploration of the epidural space.

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Case report

History

A 47-year-old male patient (neurologist, height 192 cm, weight 84 kg) was admitted because of acute excruciating

left leg pain, tingling and numbness starting suddenly after a normal city walk 1 week ago. He had a history of recurrent short episodes of mild-to-moderate low-back pain for more than 15 years, but pain radiating to the leg was unknown to him before. There was no trauma and no previous surgeries, or percutaneous infiltrations at the lumbar spine. According to the visual-analogue scale (VAS) the patient judged the intensity of his leg pain with 8, the leg-to-back pain ratio was 95–5%. The pain radiated to the lateral aspect of the left leg and foot according to the L5 segment and neurologic examination revealed an L5 hypaesthesia and a moderate weakness of the elevation of the foot and the big toe on the left (grade III–IV MRC). The symptoms did not change in strength, character or radiation during the 7 days before admission. Lab values inclusive CRP and leucocytes were within the normal range.

Preoperative imaging

A CT scan of the lumbar spine (Fig. 1a) was done 2 days after onset of the leg pain and it showed a severe osteochondrosis with considerable loss of the disc height at L4/5 and L5/S1. Additionally, a vacuum phenomenon was detected between the endplates of L4 and L5 (a1) and

extrusion of disc material from L4/5 into the spinal canal dorso-lateral on the left side (a2). Seven days after onset of the complaints the patient was admitted to our hospital. The setting with a typical left L5 radiculopathy and an according left-sided disc herniation at L4/5 in the CT scan seemed to be clear and ready for treatment.

For obtaining more details of the pathology—but without having the suspicion of an intradural herniation after the CT scan—an MRI without contrast agent was performed additionally (Fig. 1b) The sagittal MRI (b1–b3) demonstrates the severe osteochondrotic changes with Modic-signs at the endplates of L4 and L5 and a broad, most likely old and hard fingertip-like protrusion of the disc annulus with elevation/impression of the posterior longitudinal ligament. In addition soft nucleus material prolapsed dorsally and was dislocated caudally through the annulus into the spinal canal. In the midline cut (b3)—free fragments are visible in the complete spinal diameter. At last the axial MRI (b4–b6) revealed the intradural dislocation of sequester material: at the level of the L4/5 disc (b4) the annulus showed a left-sided protrusion and within the dural sac the nerve roots of the cauda equina gathered at the right anterior side. They are pushed to the opposite side because of a large left-sided disc herniation visible,

Fig. 1 Preoperative imaging: Sagittal CT reconstruction (a1) and axial CT scan (a2), sagittal T1w MRI (b1), sagittal T2w MRI (b2, b3) and axial T2w MRI (b4, b5, b6). The CT (a1, a2) shows a loss of disc height with a vacuum phenomenon between L4 and L5 (yellow arrow) and extrusion of disc material dorso-lateral on the left side (red arrow). The sagittal MRI (b1–b3) demonstrates the severe osteochondrotic changes with Modic-signs at the endplates of L4 and L5 (yellow arrows) and a broad (old) protrusion of the disc annulus (orange arrow). Additionally, soft nucleus material prolapsed dorsally through the annulus into the spinal canal (red arrows). The

axial MRI (b4–b6) reveals the intradural dislocation of sequester material; at the level of the L4/5 disc, there is left-sided protrusion of the annulus (orange arrow). All nerve rootlets of the cauda equina (green arrow) are pushed to the right anterior side (b4) because of a large left-sided prolapse (red arrow) visible below at the level of the upper L5 endplate (b5). At this level, it is not clear whether the sequester is epidural or intradural but more below behind the L5 vertebral body (b6)—free intradural pieces of disc material (red arrow) can be identified between the nerve roots

one scan below at the level of the upper L5 endplate (b5). At this level it is not clear whether the prolapse is located in the epidural space lateral of the dura or intradural. One scan below at the height of the L5 vertebral body (b6)-free intradural fragments of disc material can be identified clearly between the nerve rootlets.

Procedure

Because of the persistent severe pain syndrome in conjunction with the sensory and motor deficit, the patient decided to undergo microsurgical decompression of the nerve root. Routine interlaminar fenestration was performed at the left L4/5 level and intraoperative photographs of the findings were obtained at all stages of the procedure (Fig. 2c1–c5).

As expected, no free disc material was found in the lateral epidural space (c1). During the normal manoeuvre of medialisation or elevation of the dural sac spontaneous flow of small amounts of CSF was observed. After

relaxation of the dura, the CSF flow from ventral persisted immediately. The hard protrusion of the annulus could be touched with the small hook below the ventral surface of the dural sac but no free disc fragments were detected.

A dorsal 15 mm durotomy was performed in the midline and sequester material was visualised between the nerve rootlets within the subarachnoid space (c2). With support of the small hook and the sucker, several free fragments were mobilized towards the dura incision (c3) and removed with the forceps (c4). The highly magnified view through the dorsal incision reveals a round defect of 10-mm size within the ventral dura and even further a free transdural view into the open disc annulus (c5) was possible. This spontaneous round defect of the ventral dura was surrounded by a thickened arachnoid layer with enlarged veins (c5).

A collection of two large- (diameter of 10 mm both), four medium- and four small-sized sequester pieces (d) were sent for neuropathologic examination. While the ventral dural defect was sealed with two layers of a

Fig. 2 Intraoperative imaging at different stages of the transdural sequesterotomy. Left interlaminar fenestration L4/5 with dorsolateral exploration of the dural sac and the lateral epidural space (c1). Opening of the dura and visualisation of free disc material within the subarachnoid space (c2). Intradural mobilisation and removal of large sequester pieces with the forceps (c3, c4). Transdural view through the dorsal durotomy and the spontaneous ventral dura defect into the open annulus of the disc. The round defect of the ventral dura is surrounded by a thickened arachnoid layer with enlarged veins as a sign of a chronic erosive and inflammatory process (c5). Collection of the ten removed sequester pieces (d)

fibrinogen–thrombin patch (TachoSil®), the dorsal dura incision was sutured.

Postoperative course

The patient experienced immediate pain relief with reduction of his left L5 radicular pain from VAS 8 to VAS 1 and after 24 h of bedrest (because of the dura opening) he was mobilised. The wound healing was normal and no CSF-leakage occurred. At the time of discharge, 5 days postoperatively, the motor L5 deficit had already regressed nicely while the L5 hypaesthesia still persisted. Full recovery of the residual deficits was achieved within 12 weeks postoperatively.

Histologic diagnosis revealed pieces of severely degenerated disc material without signs of inflammation.

Discussion

With the current high-resolution and contrast-enhanced MRI techniques, the differentiation of an intraspinal/intradural sequester and an intraspinal/intradural tumourous or cystic space-occupying lesion is usually possible. Due to the vascular granulation, tissue around the disc fragment a sequester will show a thin rim enhancement around a non-enhancing solid centre, while a neoplastic tumour will usually show a solid mass with an homogeneous enhancement and a cyst typically shows a fluid signal in the middle of the lesion [1, 3, 12, 16]. In the presented case, the preoperative CT showed a herniated disc and the subsequent MRI clearly revealed an intradural disc herniation with free fragments within the dural sac.

The exact pathogenesis of an intradural or even intradiscal [13] disc herniation is not certain, but there is a general agreement that chronic adhesions between the ventral aspect of the dural sac, the posterior longitudinal ligament (PLL) and the disc annulus are the main preconditioning factors [1, 3, 5, 8]. These dense adhesions may be congenital due to prenatal adhesion formation [17] or they are acquired and occur (1) spontaneously subsequent to degenerative disc disease with or without association to an ossification of the PLL [7] or they (2) develop after the previous spine surgery or trauma due to scar tissue formation within the epidural space [2, 7, 15].

In case of progressive disc degeneration with rupture of the disc annulus at the area of adherence, the herniated nucleus material cannot “escape” cranially or caudally under the posterior ligament or perforate through it into the epidural space. The herniated fragments work like a fingertip and the relative movements of the lumbar spine lead to a repetitive impression of the fixed dural sac and

subsequent to a chronic inflammation and erosion process with thinning of the dura. Finally, the ligament and the adherent dura sheets are perforated and free disc material can herniate into the dural sac.

The clinical history, the imaging and the intraoperative findings in the presented case support the thesis, that chronic degenerative disc disease and adhesions between the posterior longitudinal ligament and the dura are the predisposing pathogenetic factors for an intradural disc herniation. The patient had a long history of low-back pain and the scans showed a severe osteochondrosis at L4/5 and a fingertip-like old protrusion of the disc annulus with impression of the ligament. The continuous degenerative process of adhesion, impression and erosion led to thinning of the spinal dura and an inflammatory reaction of the adjacent arachnoid membrane. Accordingly, the intraoperative inspection of the ventral dural sac did not show a smooth tear (as a sign of an acute rupture of the dura) but a round defect of 10-mm size surrounded by a thickened arachnoid with enlarged veins. This relatively large dura defect and the changes of the arachnoid membrane are signs of a chronic erosive and inflammatory process. Most likely, the slowly progressive perforation of the dura had already been completed before the acute clinical signs occurred and the sequester material penetrated through a kind of tunnel from the disc through the posterior ligament into the dural sac. Finally not a pressure stress to the disc but only a normal city walk led to the acute symptoms caused by the migration of free sequester material into the subarachnoid space.

Conclusion

Intradural disc herniation is a rare condition, but—compared to the “normal” disc prolapse—it requires a different operative strategy and surgical skills due to the necessity of durotomy to remove the pathology. Therefore, the exact preoperative visualisation of the dislocated disc material is essential and an actual high-resolution MRI would be ideal in all cases of intraspinal space occupying lesions.

But reality is different and sometimes disc surgery has to be performed with a CT scan or a low-quality MRI only where the diagnosis of an intradural disc herniation might be missed. In case of the absence of the expected epidural disc fragments and spontaneous flow of CSF without intraoperative injury to the dura, a fluoroscopic control of the operated disc segment should be performed first and after the verification of the correct segment the possibility of an intradural pathology should be considered.

Conflict of interest None of the authors has any potential conflict of interest.

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