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Spine

Simultaneous occurrence of spinal epidural abscess and disk herniation causing irreversible neurologic deficits: A case report and review of the literature

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A 69-year-old male patient was admitted to our clinic with a left leg radiating pain and a gradually developing back pain over a 4-week period. Three weeks before the admission, he also noticed left leg weakness. He was diagnosed with epileptic seizures 2 years before the admission and was medically treated with 500 mg of levetiracetam twice daily otherwise, he was healthy. He had difficulties to urinate several days before admission. The neurologic examination revealed paresis for hip flexion graded 3 of 5, knee extension graded 4 of 5, paralysis for ankle dorsiflexion and toe extension (clinical drop foot), paresis for plantar flexion graded 4 of 5, and loss of sensation in the first digit on the same side. The patellar and Achilles reflexes were absent on the left side.

Magnetic resonance imaging (MRI) of the lumbar region performed 1 week earlier by the general practitioner revealed disk herniation on the left side at the L5-S1 level (Fig. 1). The herniated disk migrated cranially and could therefore affect both L5 and S1 nerve roots.

The left L4 nerve root appeared swollen, emitting higher signal intensity compared with the right nerve root (Fig. 2). Unfortunately, the first MRI examination was performed without intravenous contrast and standard blood samples were not taken at admission.

A microdiscectomy was performed the same afternoon at the level of L5-S1 on the left side to treat the radiating pain and to see if the paresis could improve even though the

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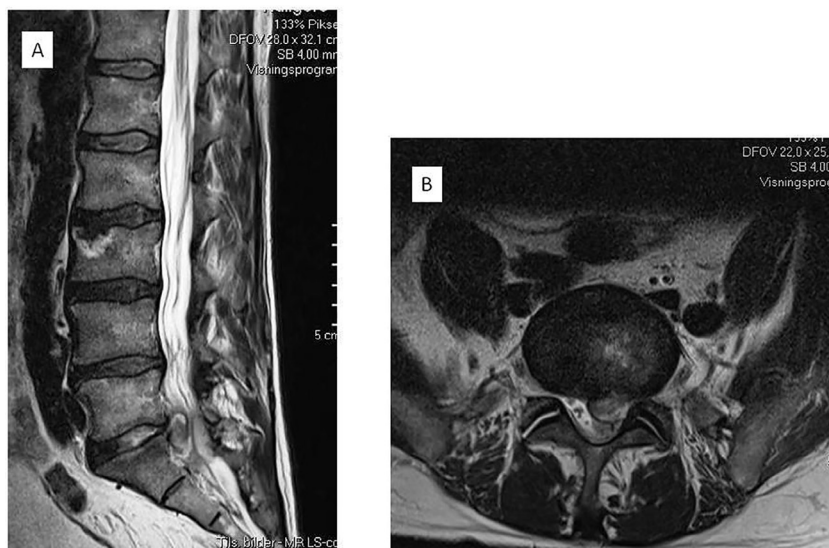


Fig. 1 – Magnetic resonance imaging of the lumbosacral column. T2-weighted sagittal (A) and axial (B) images showing disk herniation at level L5-S1 migrating cranially that could affect both the left L5 and S1 nerve roots with compression of the dural sac.

symptoms had lasted 3 weeks. Intraoperatively, a collection of pus was found in the epidural room. Tissue samples were collected for microbiological analysis and revealed beta-hemolytic group B *Streptococcus*. The day following surgery, the left leg radiating pain completely subsided; however, the paralysis for ankle dorsiflexion and toe extension persisted as the extension deficit of the knee worsened. The patient was not able to empty the urinary bladder adequately, which required the installation of a urinary catheter. There was no obvious explanation for the worsening of knee extension paresis graded 2 of 5 after the surgery, compared with 4 of 5 before the surgery. During a re-evaluation of the first magnetic resonance (MR) images, the epidural abscess was detected dorsally at level of L5-S1, which was not evident preoperatively (Fig. 3).

Two days after the surgery, an MRI follow-up examination of the lumbar spine with intravenous gadolinium showed a lesser residual of the L5-S1 herniated disk. The left L4 nerve root was swollen, as seen on the preoperative MR images, with contrast enhancement, confirming the persistent nerve root inflammation (Fig. 4). There were no signs of spondylitis or spondylodiscitis at any level of the lumbar spine. Because of the complete cessation of the radiating pain postoperatively, it was decided not to proceed with a secondary surgery.

The following day after the surgery, the laboratory tests showed an elevation of the erythrocyte sedimentation rate of 106 mm/h, the C-reactive protein of 260 mg/L, and the leukocytosis of 13.9×10^9 per liter. Because of the elevated infection parameters and intraoperatively cultured beta-hemolytic group

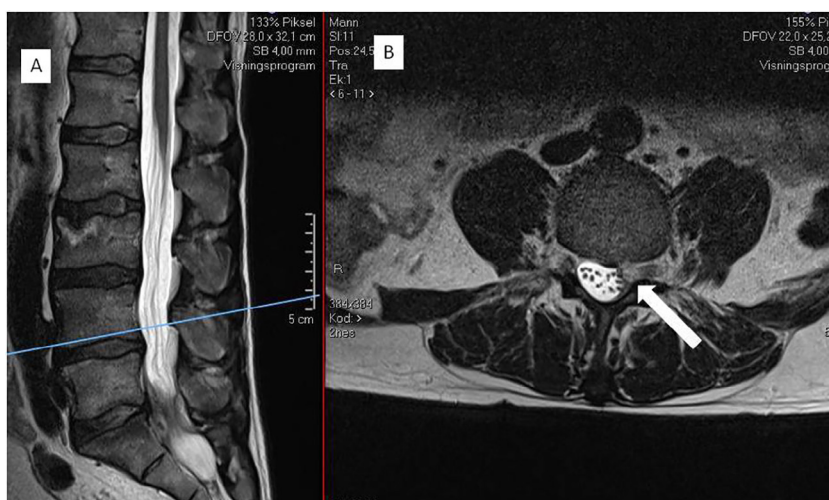


Fig. 2 – Magnetic resonance imaging of the lumbar spine. T2-weighted sagittal (A) image of level L4-L5 with the left L4 nerve root appearing to be swollen with higher signal intensity (arrow) compared with the right nerve root on the axial image (B).

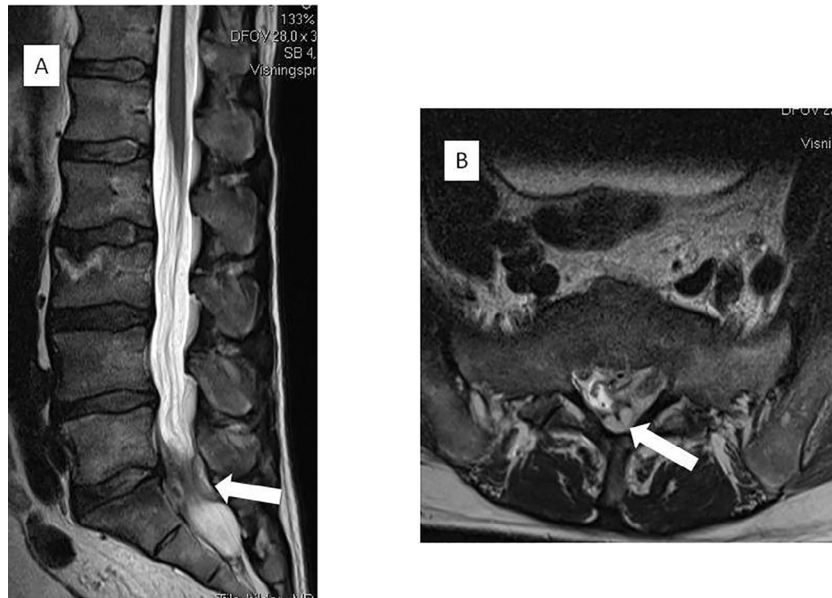


Fig. 3 – T2-weighted sagittal (A) and axial (B) images. Reevaluating the first magnetic resonance image, we could detect an epidural abscess dorsally at level L5-S1 (arrows) that was not evident preoperatively.

B Streptococcus, an intravenous penicillin treatment was initiated and continued for 4 weeks, followed by an oral administration for 4 additional weeks. Three weeks later, an electromyogram showed nerve root affection from L2 to S1 level, in the left lower extremity.

The patient was followed for 1.5 years after the primary surgery. A new examination revealed a persistent paralysis for ankle dorsiflexion and toe extension, as well as the knee extension deficit. The urinary retention was permanent, necessitating daily intermittent sterile catheterization. An MRI examination with intravenous gadolinium 1.5 years postsurgery

did not show any disk herniation at the L5-S1 level, and the dura sac expanded to its normal dimension (Fig. 5).

The left L4 nerve root was no longer swollen or hyperintense (Fig. 6).

Discussion

To our knowledge, there are no previous reports on the simultaneous occurrence of disk herniation and spinal epidural

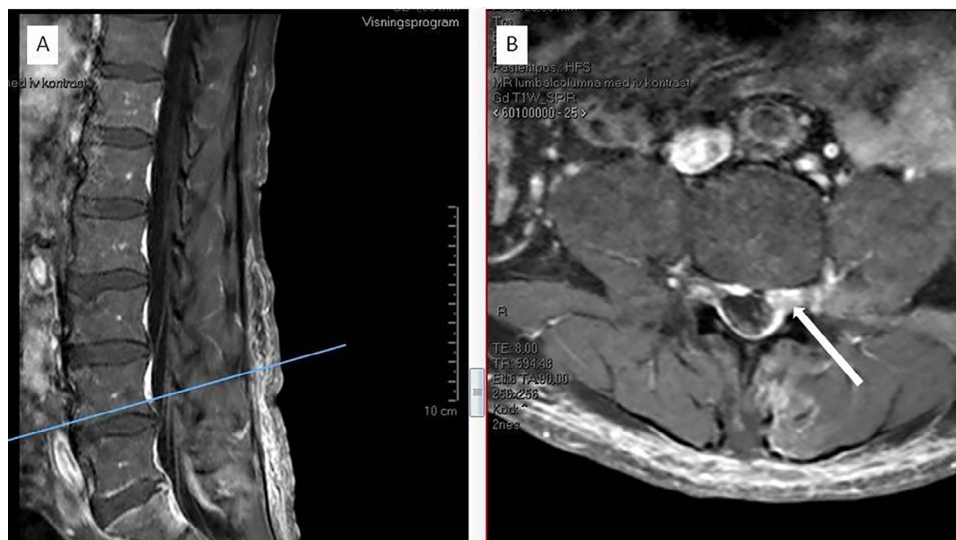


Fig. 4 – Postsurgery T1-weighted magnetic resonance imaging with gadolinium showing level L4-L5 on sagittal image (A) and on axial image (B), the L4 nerve root (arrow) appeared with high signal intensity with contrast enhancement, confirming the persistent nerve root inflammation.

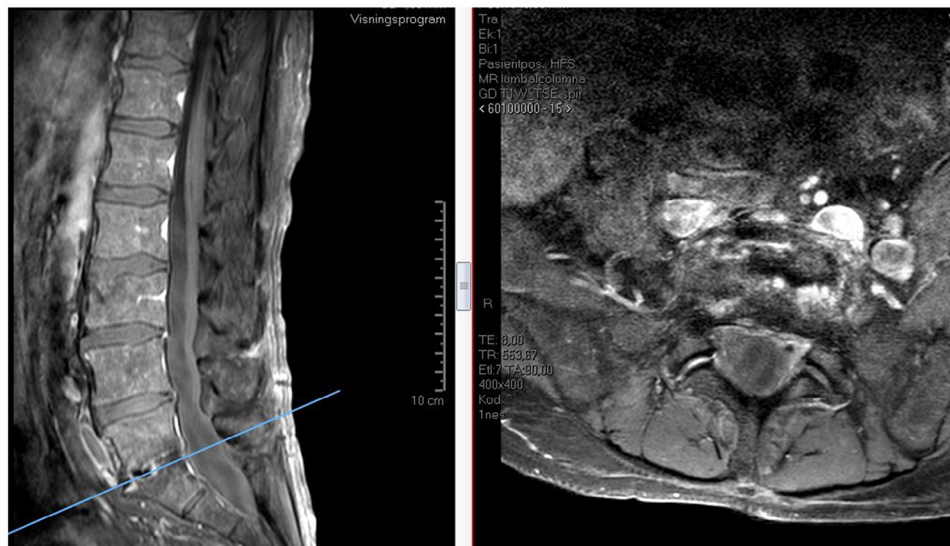


Fig. 5 – T1-weighted magnetic resonance imaging with gadolinium at 1.5 years follow-up did not show any disk herniation at the L5-S1 level, and the dural sac had expanded to normal dimension.

abscess (SEA) causing neurologic deficits and irreversible nerve damages. Yilmaz et al. described a case with *Brucella* discitis as a cause of lumbar disk herniation [1].

SEA was first described by Giovanni Morgagni in 1761 and the condition is relatively rare [2]. SEA has an estimated incidence of 1 per 10,000 population per year [2,3]. It is described as a potentially devastating pyogenic infection encased between the spinal dura mater and the vertebral periosteum. The incidence of SEA is increasing, and it is expected to further increase among patients at risk, such as those patients with weakened immune system and chronic diseases [4]. Intravenous drug use and alcohol abuse are the most frequently reported risk factors; whereas, diabetes and hepatic disease remain the most commonly reported medical comorbidities [2,3].

SEA occurs most commonly in adults in their fifth and sixth decades of life. Men are slightly overrepresented compared with women. Because SEA can be challenging to diagnose, a significant delay may occur between the first symptoms and the diagnosis. Delayed diagnosis accounts for increased morbidity and mortality [4-6].

Back pain is the most common presenting symptom of SEA. Fever, radiculopathy, neurologic deficits, and bladder and bowel dysfunction, have been reported in up to half of the cases [4]. In general, motor deficits at presentation are associated with a poor prognosis for recovery [3]. Increased awareness of SEA and increased use of MRI with intravenous gadolinium contrast may explain the increased incidence of SEA [4,5]. MRI with gadolinium has a specificity and sensitivity over 90% for detection of SEA, and is the diagnostic method of choice [5,7].



Fig. 6 – T1-weighted magnetic resonance imaging with gadolinium showing level L4-L5 with the left L4 nerve root that was no longer swollen and with normal signal intensity at 1.5 years follow-up.

There are 3 routes of pathogen spread: hematogenous, which is the most common, direct external inoculation, and spread from contiguous foci of infection such as the psoas muscle or the vertebra [8,9]. The most common pathogen found in either blood or tissue cultures is *Staphylococcus aureus* (60%-90%), whereas *Streptococcus* species, as in our case, is found in 6.8%-18% of the cases [2-4,9]. SEA can be situated at any level in the spine; but it is most frequently located in the lumbar section (48% of cases). It is detected in the thoracic and cervical spine in 33% and 22% of cases, respectively [3].

A recent review showed that most patients with SEA are treated surgically (60%). Patients with back pain only, without neurologic deficits, receive medical management more often [2]. Antibiotic treatment for SEA is preferred in patients with panspinal involvement, patients with a paralysis lasting for 72 hours or longer, or when the risk of complications to surgical treatment is not acceptable [4]. The objectives of surgical management are neurologic decompression, microbiological source control, and occasionally spinal instrumented fixation.

Our patient had both epidural abscess and disk herniation contributing to neurologic deficits. The findings on the first MRI examination were misinterpreted as disk herniation only at L5-S1 level; and the dorsal epidural abscess was not detected before surgery. The lack of gadolinium-enhanced MR imaging before surgery would limit the ability for radiological diagnosis of the epidural abscess in the shadow of the disk herniation. It is quite difficult to affirm whether the epidural abscess preceded the disk herniation, or vice versa. Theoretically, the epidural abscess could evolve firstly because back pain and radiculopathy were present before the paresis in our case. The SEA probably developed by the hematogenous route and would thereafter spread to the L5-S1 disk space with subsequent disk herniation. Yilmaz et al. hypothesized that an inflammatory process in the nucleus pulposus of the infected disk could cause expansion, thereby increasing the intradiscal pressure, which could lead to a disk herniation again [1]. The MRI images with or without intravenous contrast in our case revealed no evidence of infection in the L5-S1 disk space.

The disk herniation and the SEA were confined to the L5-S1 level making it difficult to explain why our patient developed multiple myotomal paresis. The affection of several nerve roots (from L2-S1) was confirmed by electromyography, in line with the clinically observed neurologic deficits. Bond and Manian [4] postulated that neurologic deficits caused by SEA are often attributed to its direct compression of the spinal cord resulting in ischemia and injury, disturbing the local blood flow. Local circulatory disruption due to venous stasis or thrombosis of spinal arteries has also been implicated. This hypothesis could explain the difficulty in predicting the tempo of neurologic complications following the onset of symptoms in patients with SEA. However, that view is not universally accepted. Vasculitis infarction has also been implicated as a possible cause of neurologic deficit without specific nerve root compression found on MRI [6].

Both the preoperative and postoperative MRI images of our patient revealed that the L4 nerve root was thickened. It is unclear whether this thickening was caused by local circulatory

disruption or a vasculitis infarction due to the SEA. Another explanation could be that the infection had spread to the L4-L5 level without initial formation of abscess. Aycan et al. [10] reported a case of SEA at the L4-L5 level on the first MR examination; but later on, the infection had expanded from Th12 to S1 only in a couple of days.

Our patient had paralysis in his ankle dorsiflexion and toe extension. The former could be caused by the L4 nerve root affection although the L5 nerve root affection can also cause an ankle dorsiflexion paresis. An affection of the L4 nerve root can clinically manifest with weakness of both ankle dorsiflexion and knee extension. However, in the present case, it is uncertain if compression alone, caused by the disk herniation or epidural abscess at the L5-S1, could explain a paresis for ankle dorsiflexion and knee extension. Theoretically, a local circulatory disruption or vasculitis infarction of the left L4 nerve root caused by the SEA could have contributed to the clinical status.

Finally, the focus of infection leading to the SEA in our patient was unknown. The patient was diagnosed with epileptic seizures 2 years earlier but was healthy otherwise. We can only speculate that the infection could be due to the patient's weakened immune system exacerbated by increased alcohol consumption of 3-4 units daily. Alcoholism has previously been suggested as a risk factor for SEA [2,3,6].

In conclusion, based on our patient's clinical presentation and data from the literature, the advent of disk herniation and clinical signs of infection should alert the clinician about the possibility of a concurrent intraspinal abscess.

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